EARTHJUSTICE

ALASKA CALIFORNIA FLORIDA MID-PACIFIC NORTHEAST NORTHERN ROCKIES
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COMMENTS BY EARTHJUSTICE ON BEHALF OF SIERRA CLUB
ON THE
DRAFT/PROPOSED TITLE V AIR OPERATION PERMIT RENEWAL FOR THE OKEELANTA CORPORATION'S OKEELANTA SUGAR MILL AND REFINERY (Facility ID No. 0990005) AND THE NEW HOPE POWER COMPANY'S OKEELANTA COGENERATION PLANT (Facility ID No. 0990332)

Public Notice of Intent to Issue Air Permits Published March 15, 2015

Comments Submitted April 14, 2015

I. INTRODUCTION

Under Florida's implementation of the Clean Air Act's Title V program, major sources of air pollution in Florida must obtain operating permits.[1] The Department of Environmental Protection ("DEP") has issued a draft/proposed renewal of the Title V permit for the Okeelanta Corporation's Sugar Mill and Refinery, together with the New Hope Power Company's Okeelanta Cogeneration Plant,[2] notice of which was published on March 15th, 2015.[3] Pursuant to section 403.0872(4) of the Florida Statutes, DEP must "accept public comment with respect to" the permit for 30 days following publication of notice of the permit's renewal. **This letter, submitted on behalf of the Sierra Club, constitutes such a public comment. Because of the substantial public interest in this permit, the Sierra Club also requests that a public hearing be held on this proposed permit renewal.**

[1] § 403.0872, Fla. Stat. (2014).

[2] FLA. DEP'T OF ENVTL. PROT., DIV. OF AIR RESOURCE MGMT., OFFICE OF PERMITTING & COMPLIANCE, TITLE V AIR OPERATION PERMIT RENEWAL, PERMIT NO. 0990005–040–AV (2015) [hereinafter DRAFT PERMIT].

[3] *Public Notice of Intent to Issue Air Permits*, PALM BEACH POST, Mar. 15, 2015.

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II. OVERVIEW

The Okeelanta Corporation's Sugar Mill and Refinery, together with the New Hope Power Company's Okeelanta Cogeneration Plant (collectively, the "Okeelanta Facility"), processes sugarcane grown on nearby land owned and/or operated largely by the Okeelanta Corporation ("Okeelanta").[4] Prior to being harvested, cane is burned in the fields to remove dead leaves and other undesirable biomass.[5] This practice, which is unnecessary from both a technical and economic standpoint, has serious health consequences for the public: sugarcane burning has been linked to an increased incidence of asthma in nearby populations,[6] genotoxicity in field workers,[7] and even cancer.[8] Prompted in part by these public health concerns, sugarcane growers around the world have been switching to "green harvesting" techniques—techniques for harvesting sugarcane that don't involve pre-harvest burning.[9] In addition to being more protective of public health, green harvesting techniques have the significant advantage of being sustainable—something that can't be said for the practice of pre-harvest cane burning.[10]

Despite the advantages of green cane harvesting, Okeelanta continues to burn its sugarcane prior to harvest.[11] While this practice is conducted pursuant to open burning permits

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[4] *Our Story*, FLORIDA CRYSTALS, https://www.floridacrystals.com/content/131/our-story.aspx (last visited Apr. 14, 2015) (attached as Appendix C).

[5] L.E. Baucum & R.W. Rice, UNIV. OF FLA., INST. OF FOOD & AGRIC. SCIS., COOP. EXT. SERV., AN OVERVIEW OF FLORIDA SUGARCANE 6 (2009), *available at* http://ufdc.ufl.edu/IR00003414/00001 (attached as Appendix D).

[6] Marcos Abdo Arbex et al., *Air Pollution from Biomass Burning and Asthma Hospital Admissions in a Sugar Cane Plantation Area in Brazil*, 61 J. EPIDEM. & COMMUNITY HEALTH 395 (2007) (attached as Appendix E).

[7] Henrique César Santejo Silveira et al., *Emissions Generated By Sugarcane Burning Promote Genotoxicity in Rural Workers: A Case Study in Barretos, Brazil*, 12:87 ENVTL. HEALTH (2013) (attached as Appendix F).

[8] *See, e.g.*, Helena Ribeiro, *Sugar Cane Burning in Brazil: Respiratory Health Effects*, 42 REVISTA DE SAÚDE PÚBLICA 370 (2008) (attached as Appendix G).

[9] *See, e.g.*, *Brazil Sugarcane Mills Agree to End Burning By '17*, REUTERS.COM (Oct. 22, 2007), http://www.reuters.com/article/2007/10/22/environment-brazil-cane-harvest-dc-idUSN2245768620071022 (attached as Appendix H).

[10] ANDREW WOOD, TIME FOR A TRANSITION FROM PRE-HARVEST BURNING OF SUGARCANE TO GREEN CANE HARVESTING IN THE EVERGLADES AGRICULTURAL AREA (2015) (attached as Appendix A).

[11] *See, e.g.*, Baucum, *supra* note 5.

issued by the Florida Forest Service,[12] the massive amounts of hazardous air pollutants (HAPs) released during the burning process are inexplicably not included in Okeelanta's Title V operating permit.[13] Not listing Okeelanta's burning cane fields as HAP emissions units in its Title V permit runs afoul of federal and state air pollution laws. It also makes little sense, as the HAP emissions from pre-harvest sugarcane burning are likely many times greater than the HAP emissions from Okeelanta's refining and milling operations; DEP is essentially keeping its eye fixed on a candle while a bushfire rages next door. In order to meets its statutory obligations and ensure that Florida's implementation of the Title V program as applied to Okeelanta serves its intended purposes, DEP must include Okeelanta's cane fields as HAP emissions units in its Title V permit.

III. OKEELANTA'S OPERATIONS

Okeelanta operates a sugar mill and refinery located in western Palm Beach County.[14] This facility is located directly next to a cogeneration plant owned and operated by the New Hope Power Company, itself an affiliate of Okeelanta;[15] the cogeneration plant uses waste biomass from the sugar mill (along with other fuels) to generate electricity and process steam,[16] some of which is used to power the refinery and mill.[17] The refinery/mill and the cogeneration plant are considered to be one facility for Title V purposes.[18]

In addition to owning and operating the mill, refinery, and cogeneration plant, Okeelanta owns and/or operates thousands of acres of sugarcane fields surrounding the mill.[19] These various operations—cane fields, mill, refinery, cogeneration plant, and even a packaging and

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[12] *See FAQ for Open Burning in Florida*, FLORIDA DEPARTMENT OF AGRICULTURAL AND CONSUMER SERVICES, http://www.freshfromflorida.com/Divisions-Offices/Florida-Forest-Service/Wildland-Fire/Resources/FAQ-for-Open-Burning-in-Florida (last visited Apr. 13, 2015).

[13] DRAFT PERMIT, *supra* note 2.

[14] *Id.*

[15] *See, e.g.*, *Company Rules and Regulations*, FLORIDA CRYSTALS (Dec. 12, 2012), https://www.floridacrystals.com/content/181/company-rules-regulations.aspx (attached as Appendix I).

[16] OKEELANTA CORPORATION & NEW HOPE POWER COMPANY, TITLE V PERMIT RENEWAL APPLICATION (2014).

[17] *Id.*

[18] DRAFT PERMIT, *supra* note 2, at 2.

[19] *Our Story*, *supra* note 4.

distribution center—comprise one integrated agro-industrial process devoted to producingsugar and turning it into a variety of marketable sugar products.[20] The cane fields are where that process begins: each year, Okeelanta and its operatives harvest cane from over 100,000 acres of sugarcane fields, burning each field just prior to harvesting.[21] This burning is done for a number of reasons: it gets rid of the dry outer leaves of the cane stalk; rids cane fields of snakes and insects; ; and quickly and cheaply removes excess biomass, which is referred to in the industry as "trash."[22,23]

Once the cane fields have been burned, the cane is harvested by machine and transported to the mill via trailer.[24] At the mill, the cane is pressed to extract the juice; the fibrous material left over is then used as fuel for the cogeneration facility.[25] The juice then goes through a purification and filtration process to further remove solids; the washed juice is then heated to produce a syrupy substance.[26]

IV. ADVERSE PUBLIC HEALTH EFFECTS OF SUGARCANE BURNING

The modest economic benefits of pre-harvest sugarcane burning come at a great cost to public health and welfare: when burned, sugarcane releases a number of pollutants and toxins, including particulate matter (PM), various polycyclic aromatic hydrocarbons (PAHs), dioxins, carbon monoxide (CO), carbonyls such as formaldehyde, and volatile organic compounds (VOCs) such as benzene.[27] While sugarcane field workers and residents in communities neighboring sugarcane fields are the populations at the greatest risk of being exposed to these

[20] *See id.*

[21] Baucum, *supra* note 5.

[22] *Id.*

[23] *Burning Agricultural Waste: A Source of Dioxins*, COMM'N FOR ENVTL. COOPERATION (Jan. 2014), http://www3.cec.org/islandora/en/item/11405-la-quema-de-residuos-agr-colas-es-una-fuente-de-dioxinas-en.pdf (attached as Appendix J).

[24] *Growing Sugar Cane*, FLORIDA CRYSTALS, https://www.floridacrystals.com/content/123/growing-sugar-cane.aspx (last visited Apr. 14, 2015) (attached as Appendix K).

[25] *Id.*

[26] *Id.*

[27] Danielle Hall et al., *PAHs, Carbonyls, VOCs and $PM_{2.5}$ Emission Factors for Pre-Harvest Burning of Florida Sugarcane*, 55 ATMOSPHERIC ENV'T 164 (2012) (attached as Appendix L).

dangerous releases, plumes from sugarcane burning can travel great distances and affect populations far from the actual burning.[28] Populations that are especially susceptible to the pollutants and toxins released by sugarcane burning include the very young, the elderly, and individuals with compromised immune systems.[29,30,31]

Far from having a negligible effect on air quality, sugarcane burning is likely one of the largest sources of many dangerous air pollutants in the Everglades Agricultural Area (EAA) and even, in the case of some pollutants, statewide. A recent study conducted by researchers at the University of Florida estimated that cane burning may be responsible for as much as 86% of the formaldehyde emitted in Palm Beach County each year and as much as 16% of the formaldehyde emitted in Florida.[32] The same study estimated that cane burning is a significant source of PAHs both in Palm Beach County and statewide, accounting, for instance, for up to 69% of the acenaphthylene emitted in Palm Beach County and 11% of acenaphthylene emitted in Florida.[33] PAHs can cause mutagenic and carcinogenic effects,[34] and PAHs from sugarcane burning in particular have been linked to adverse public health effects: in a 2014 study, researchers in Brazil concluded that the risk of cancer from PAH inhalation was greater in persons who were longtime residents of regions where sugarcane was burned.[35]

Perhaps the most serious health threat from pre-harvest sugarcane burning comes in the form of the particulate matter released during the burning process. A 2005 study of one area in

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[28] LA. DEP'T OF AGRIC. & FORESTRY, LOUISIANA SMOKE MANAGEMENT GUIDELINES FOR SUGARCANE HARVESTING 3 (2000), *available at* https://www.lsuagcenter.com/NR/rdonlyres/8AAEF1B2-EFA6-40A0-AC59-654C15894EE9/12567/smoke_management3.pdf (attached as Appendix M).

[29] *Smoke from Biomass Burning*, DEP'T OF THE ENV'T & HERITAGE, AUSTRALIAN GOV'T (2005), http://www.environment.gov.au/resource/smoke-biomass-burning (last visited Apr. 9, 2015) (attached as Appendix N).

[30] J.E. Cançado et al., *The Impact of Sugar Cane-Burning Emissions on the Respiratory System of Children and the Elderly*, 114 ENVTL. HEALTH PERSPECTIVES 725 (2006) (attached as Appendix O).

[31] Ribeiro, *supra* note 8.

[32] Hall, *supra* note 27.

[33] *Id.*

[34] AGENCY FOR TOXIC SUBSTANCES & DISEASE REGISTRY, CASE STUDIES IN ENVIRONMENTAL MEDICINE: TOXICITY OF POLYCYCLIC AROMATIC HYDROCARBONS (2009) (attached as Appendix P).

[35] Joao V. de Assunção et al., *Airborne Polycyclic Aromatic Hydrocarbons in a Medium-Sized City Affected by Preharvest Sugarcane Burning and Inhalation Risk for Human Health*, 64 J. AIR & WASTE MGMT. ASS'N 1130 (2014) (attached as Appendix Q).

southeastern Brazil reported that sugarcane fires were the largest source of particulate matter in the area, contributing 60% of the area's fine particulate matter ($PM_{2.5}$) and 25% of its coarse particulate matter ($PM_{10\text{-}2.5}$).[36] Particulate matter less than 10 micrometers in size, including fine particles less than 2.5 micrometers, can penetrate deep into the lungs.[37] In recent studies, exposure to particulate pollution—either alone or together with other air pollutants—has been linked with premature death,[38] difficulty breathing,[39] aggravated asthma,[40] cancer,[41] increased hospital admissions and emergency room visits,[42] and increased respiratory symptoms in children.[43] In a 2002 Louisiana study, researchers observed an increase in the number of asthma attacks during the sugarcane burning season, and a 50% increase in hospital admissions for respiratory problems during that time of year.[44]

Other toxins and pollutants released by sugarcane burning include dioxins, carbon monoxide, and ozone.[45,46,47] Dioxins are released during agricultural combustion processes where chlorinated pesticides, such as pentachlorophenol fungicide and 2,4-dichlorophenoxyacetic acid (2,4-D), are used. Dioxin emissions increase by 150 times when

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[36] L.L. Lara et al., *Properties of Aerosols from Sugar-Cane Burning Emissions in Southeastern Brazil*, 39 ATMOSPHERIC ENV'T 4627 (2005) (attached as Appendix R).

[37] *Id.*

[38] Robert D. Brook et al., *Particulate Matter Air Pollution and Cardiovascular Disease*, 121 CIRCULATION 2331 (2010) (attached as Appendix S).

[39] *Particulate Matter Research,* EPA.GOV, http://www.epa.gov/airscience/air-particulatematter.htm (last visited Apr. 9, 2015) (attached as Appendix T).

[40] *Id.*

[41] C. Arden Pope III et al., *Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution*, 287 J. AM. MED. ASS'N 1182 (2002) (attached as Appendix U).

[42] Cançado, *supra* note 30.

[43] *Id.*

[44] R. Boopathy et al., *Sugar Cane (Saccharum officinarum* L*) Burning and Asthma in Southeast Louisiana, USA*, 68 BULL. ENVTL. CONTAMINATION & TOXICOLOGY 173 (2001) (cited in Ribeiro, *supra* note 8).

[45] S.I. Shih et al., *Significance of Biomass Open Burning on the Levels of the Polychlorinated Dibenzo-p-dioxins and Dibenzofurans in the Ambient Air,* 153 J. HAZARDOUS MATERIALS 276 (2007) (attached as Appendix V).

[46] Q. Zhang et al., *Polychlorinated Dibenzo-p-dioxins and Dibenzofurans Emissions from Open Burning of Crop Residues in China Between 1997 and 2004*, 151 ENVTL. POLLUTION 39 (2008) (attached as Appendix W).

[47] *Burning Agricultural Waste*, supra note 23.

biomass treated with 2,4-D is burned.[48] Dioxins can cause a number of health effects, including reproductive effects in women, diabetes, harmful effects to the immune system, chloracne, and various types of cancer.[49] Carbon monoxide may prevent blood from carrying enough oxygen, leading to a condition called hypoxia.[50] And even at low concentrations, ozone can affect the lungs and can cause coughing, choking, breathlessness, mucus production, burning of the throat, irritability, nausea, and a decrease in lung function during exercise.[51]

Finally, sugarcane burning can have a dramatic effect on visibility and atmospheric conditions. Fine particles emitted during cane burning can scatter and absorb light, creating a haze that can reduce visibility.[52] In some areas, sugarcane burning has caused visibility problems so severe that roads have had to be shut down to prevent accidents.[53]

V. HAP EMISSIONS FROM OKEELANTA'S BURNING OF SUGARCANE FIELDS MUST BE INCLUDED IN ITS TITLE V PERMIT

Under DEP regulations implementing Florida's Title V program, a "Title V Source" includes "[a] facility containing an emissions unit, or any group of emissions units, … that emits or has the potential to emit, in the aggregate, 10 tons per year or more of any one [HAP] … or 25 tons per year or more of any combination of HAPs."[54] An "emissions unit," in turn, is defined broadly as "[a]ny part or activity of a facility that emits or has the potential to emit any air pollutant,"[55] while a "facility" is defined as "[a]ll of the emissions units which are located on one or more contiguous or adjacent properties, and which are under the control of the same person

[48] *Id.*

[49] *Id.*

[50] J. Malilay, *A Review of Factors Affecting the Human Health Impacts of Air Pollutants from Forest Fires, in* HEALTH GUIDELINES FOR VEGETATION FIRE EVENTS—BACKGROUND PAPERS 255 (Kee-Tai Goh et al. eds., 1999) (attached as Appendix X).

[51] *Ribeiro, supra* note 8.

[52] *Agricultural Burning*, EPA.GOV, http://www.epa.gov/agriculture/tburn.html (last visited Apr. 14, 2015).

[53] Wendy Osher, *Smoke From Cane Fire Forces Intermittent Closures on Kuihelani*, MAUINOW.COM (June 12, 2013), http://mauinow.com/2013/06/11/smoke-from-cane-fire-forces-intermittent-closures-on-kuihelani/ (attached as Appendix Z).

[54] Fla. Admin Code R 62-210.200(173).

[55] *Id.* R. 62-210.200(113).

(or persons under common control)."[56] The fields in the EAA from which Okeelanta obtains its sugarcane, together with the Okeelanta Facility, clearly meet the regulatory definition of a "Title V Source" of HAPs. Because, generally speaking, each "Title V Source" must operate in compliance with the provisions of Chapter 62-213 of the Florida Administrative Code,[57] Okeelanta's Title V permit must include its sugarcane fields as HAP emissions units.[58]

A. Emissions Unit

DEP's broad definition of an "emissions unit" clearly encompasses a sugarcane field, which emits air pollutants when burned.[59] A cane field also falls under the Clean Air Act's definition of a "stationary source" as "any building, structure, facility, or installation which emits or may emit any air pollutant."[60] The Environmental Protection Agency ("EPA") has rejected the position that this broad definition excludes agricultural operations.[61] Moreover, a stationary source does not require a smokestack, either literally or figuratively: EPA regulates municipal landfills as stationary sources,[62] and concentrated animal feeding operations—whose emissions come in large part from animal waste found in open lagoons and ponds[63]—"plainly fit the definition of stationary source[s]," according to EPA.[64] Each field of burning sugarcane is thus clearly a "stationary source" of hazardous air pollutants.

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[56] *Id.* R. 62-210.200(121).

[57] *Id.* R. 62-213.400.

[58] The fields in the EAA from which Okeelanta obtains its sugarcane together with the Okeelanta Facility also meets the federal statutory definition of a "major source" found in 42 U.S.C. § 7412. Because every § 7412 "major source" must obtain a Title V permit, federal law also requires Okeelanta's permit to include HAP emissions from its sugarcane fields.

[59] *See, e.g.*, Hall, *supra* note 27.

[60] 42 U.S.C. § 7411(a)(3).

[61] *Ass'n of Irritated Residents v. Fred Schakel Dairy*, 61 Env't Rep. Cas. (BNA) 1801 (E.D. Cal. Dec. 2, 2005) ("[I]t is the EPA's position that the CAA does not exempt major stationary agriculture sources.")

[62] *See* 40 C.F.R. § 60.30c *et seq.* (2014)

[63] EPA, EMISSIONS FROM ANIMAL FEEDING OPERATIONS (DRAFT) (2001), *available at* http://www.epa.gov/ttnchie1/ap42/ch09/draft/draftanimalfeed.pdf.

[64] 67 Fed. Reg. 63,551, 63,556-57 (Oct. 15, 2002)

B. Common Control

Emissions units (or stationary sources under federal law) must be under "common control" in order to be aggregated for HAP purposes. "[P]roperties that are owned, leased, or operated by the same entity, parent entity, subsidiary, or any combination thereof" are considered to be under common control.[65] Even when properties do not fit this description, however, common control obtains when one entity has the "power to direct or cause the direction of the management and policies" of all relevant entities having ostensible property or source ownership, "whether through the ownership of voting shares, contract, or otherwise."[66]

All of the sugarcane fields shown colored-in in Figure 1 are either owned outright by Okeelanta or one of its affiliates, leased by Okeelanta, or operated by Okeelanta—in any case, Okeelanta is responsible for directing the agricultural operations on all of these properties. It is clear that Okeelanta exercises control over these properties and over the Okeelanta Facility itself, and that the "common control" prong of the aggregation test is therefore met.

C. Contiguous Or Adjacent Properties

In order for HAP emissions units to be aggregated together into a single facility, they must be "located on one or more contiguous or adjacent properties."[67] This does not mean that the property upon which each emissions unit sits must be touching some other property containing an emissions unit; rather, two properties or sites may be aggregated if they are near one another.[68] This interpretation of "contiguous or adjacent" takes into account the fact that railroads, highways, and similar features routinely cut across major sources, and that to claim that such features should split major sources into multiple smaller sources "would be an artificial distinction, and … is contrary to the intent of the statutory definition of major source."[69]

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[65] *See, e.g.*, 60 Fed. Reg. 43,244, 43,265 (Aug. 18, 1995); 77 Fed. Reg. 22,848, 22,939 (April 17, 2012).

[66] *See* 45 Fed. Reg. 59,874, 59,878 (Sept. 11, 1980) (quoting 17 C.F.R. 210.1-02(g) (1980)).

[67] Fla. Admin. Code R. 62-210.200(121). Under federal law, stationary sources of HAPs should be aggregated together into a major source if they are "located within a contiguous area." 42 U.S.C. § 7412(a)(1).

[68] *See* 59 Fed. Reg. 12,408, 12,412 (Mar. 16, 1994).

[69] *Id.*

In determining whether two sources are sufficiently close to one another for aggregation purposes, EPA until recently considered the functional interrelatedness of the sources.[70] That is, EPA looked to the "nature of the relationship between the facilities" as well as their physical distance from each other in determining whether they could be aggregated together.[71] Pursuant to the Sixth Circuit Court of Appeals's decision in *Summit Petroleum Corporation v. EPA*,[72] EPA is currently obligated to consider only physical proximity in determining whether stationary sources are "adjacent" for purposes of Title V aggregation.[73]

Summit, however, has limited applicability to the question of whether Okeelanta's cane fields are "located on one or more contiguous or adjacent properties" or "located within a contiguous area" with its mill and refinery. First, *Summit* dealt with the term "adjacent," *not* the term "contiguous."[74] There is little doubt that emissions from sources located on *contiguous* properties—that is, properties that border one another[75]—can be aggregated. Second, *Summit* does not foreclose the grouping of emissions from sources located on neighboring—but not contiguous—properties, but merely disallows the consideration of functional interrelatedness when conducting the adjacency analysis.[76] Under *Summit*, two sources may still be aggregated even if the properties on which they sit are not abutting so long as those properties are physically close.

The vast majority of Okeelanta's cane fields in the EAA—including those located on land leased and operated by Okeelanta—comprise a single area connected to the Okeelanta Facility. The properties on which these fields are located can be grouped into two categories: Category A and Category B. Category A includes those properties from which it would be possible to reach the Okeelanta Facility without crossing any non-Okeelanta-operated land, save

[70] *See Summit Petrol. Corp. v. EPA*, 690 F.3d 733, 739 (6th Cir. 2012).

[71] *Id.* at 740.

[72] *Id.*

[73] *See Nat'l Envtl. Dev. Ass'n's Clean Air Project v. EPA*, 752 F.3d 999, 1010 (D.C. Cir. 2014). Note, however, that EPA has the option to revise its regulations to incorporate a functional interrelatedness component into its definition of "adjacent."

[74] *Summit*, 690 F.3d at 741.

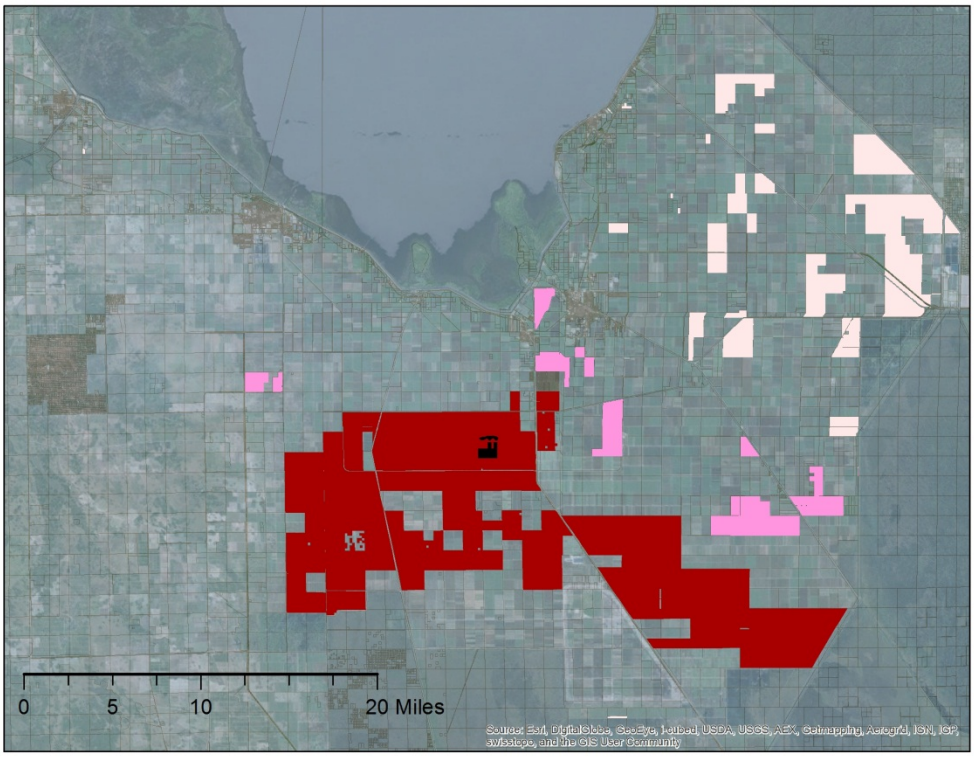
[75] *Id.*

[76] *Id.* at 741-43.

for roads, canals, and similar features. That is, Category A comprises a contiguous area surrounding the Okeelanta Facility. Category A properties are shown in red in Figure 1.

Category B consists of properties and contiguous blocks of properties adjacent to properties in Category A. The emissions from these properties can be aggregated together with the emissions from the Okeelanta Facility and from the fields/properties in Category A because the properties on the edge of Category B are physically close to properties on the edge of Category A. Moreover, all of the fields in Category B are clearly part of the same agro-industrial process as the fields in Category A and the Okeelanta Facility itself, and this functional relationship counsels in favor of finding the fields in Category B to be adjacent to the rest of the operation. However, even ignoring the obvious functional interrelatedness between the fields in Category B and the remainder of Okeelanta's operation, properties from Category B can be aggregated with properties from Category A and with the Okeelanta Facility on the basis of physical proximity alone. Properties in Category B are shown in pink in Figure 1.

Figure 1: Okeelanta Properties in the EAA



The remainder of the Okeelanta lands (shown in Figure 1 in off-white) should also be included in Okeelanta's Title V permit as part of a single HAP-emitting facility. Although these properties are not as close to the core of Okeelanta's operation as the properties in Categories A and B, they are nonetheless sufficiently physically proximate to—not to mention intimately functionally related with—that core for aggregation purposes under Title V.

D. HAP Emissions Potential

Because the Okeelanta Facility is already a Title V Source with respect to HAPs,[77] the addition of the HAP emissions from Okeelanta's cane fields would obviously render the entire aggregation a Title V Source. However, it is important to note that the fields may be, and likely are, a much larger source of HAPs than the refinery/mill facility by itself; Okeelanta's current Title V permit thus only includes a sliver of the entire operation's HAP emissions.

It is difficult to calculate precisely how much additional hazardous air pollution is released each year by Okeelanta's pre-harvest burning practices, but even a very conservative estimate places the emissions from the Category A fields alone at approximately 100 tons per year. A more realistic estimate, using average values of emissions and fuel loading factors, is approximately 600 tons per year for Category A fields, almost 700 tons per year for Category A and Category B fields combined, and over 800 tons per year for all Okeelanta-controlled fields—likely much more than the HAP emissions from the Okeelanta Facility alone.[78] Table 1 shows high, low, and medium estimates for the total HAPs released each year by the burning of sugarcane fields on Okeelanta-operated land; the methodology used to compute the data in this table is explained in an appendix to this comment letter.[79]

[77] *See* DRAFT PERMIT, *supra* note 2.

[78] We were not able to locate total HAP emissions data for the Okeelanta Facility. However, a decent estimate can be obtained by taking the HAP emissions data from U.S. Sugar's facility in Clewiston, which processes a similar amount of sugarcane. *See* SUGAR LOAN PROGRAM, *infra* note 84. In 2011, the Clewiston facility emitted 267.8528 tons of HAPs according to DEP data.

[79] *See* Appendix B.

Table 1: Estimated Yearly HAP Emissions from Cane Field Burning

	Low	Medium	High
Category A Properties	102 tons/year	593 tons/year	2302 tons/year
Category A+B Properties	118 tons/year	685 tons/year	2659 tons/year
All Okeelanta Properties	139 tons/year	809 tons/year	3140 tons/year

DEP regulations require a Title V permit renewal application to "include information sufficient to determine all applicable requirements for the Title V source and each emissions unit."[80] As discussed above, the proposed Title V permit renewal—and Okeelanta's application for that permit renewal—fails to include emissions units that, together with the Okeelanta Facility, comprise a major source of HAPs and thus a "Title V Source." DEP must ask Okeelanta to revise its application to include those emissions units (i.e., Okeelanta's sugarcane fields) in order for the application to be complete.[81]

VI. OKEELANTA CAN AFFORD TO USE GREEN HARVESTING TECHNIQUES

Okeelanta's practice of burning sugarcane before harvesting poses a serious risk to public health. This risk is wholly unnecessary, as an economically feasible—and more sustainable—alternative to pre-harvest burning exists: green cane harvesting. Indeed, green cane harvesting has proven profitable for the sugar industry even in countries without the artificial price increase system that subsidises Okeelanta and the rest of the sugar industry here in the United States. Given the existence of that system in this country, Okeelanta's failure to use more environment-friendly harvesting techniques is inexcusable.

A. Corporate Welfare At The Expense Of U.S. Consumers

The sugar industry has created what amounts to a corporate welfare system in the United States, with a multi-pronged approach aimed at guaranteeing lucrative profits at the expense of U.S. consumers. The practice of inflating U.S. sugar prices is an anachronism from early in our

[80] Fla. Admin. Code R. 62-213.420(3).

[81] *Id.* R. 62-213.420(1)(b)3.

country's history: the very first Congress imposed tariffs on foreign sugar in 1789.[82] The purpose of those tariffs was to encourage domestic sugar production in the new nation. That purpose was accomplished two hundred years ago.

Today, sugar industry profits are dramatically increased by three mechanisms that artificially control the market and inflate prices for consumers: 1) price supports, 2) market allotments, and 3) import quotas.[83]

Price supports are provided through a nonrecourse loan program authorized by the 2014 Farm Bill and administered through the Farm Service Agency.[84] The U.S. sets a loan rate at 18.75 cents per pound for raw cane sugar for each of the 2011 through 2018 crop years adjusted by region, and sugar processors pledge sugar as collateral.[85] In 2013, the rate for Florida was guaranteed at a bottom of 18.19 cents per pound.[86] If the processor cannot obtain a higher price on the market, it can forfeit the sugar to the U.S. government who must accept it as full repayment of the loan. In 2013, the world raw sugar price was 17.46 cents per pound, and now it is less than 14 cents per pound.[87]

The U.S. sugar market is additionally carved into delegated allotments and sugar produced beyond these allotments is blocked from sale in the United States, further forcing prices higher and eliminating the need for producers to compete with one another.[88] Allotments are made between cane sugar and beet sugar and then divided to each company or cooperative

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[82] *See* JOSE ALVAREZ & LEO C. POLOPOLUS, UNIV. OF FLA., INST. OF FOOD & AGRIC. SCIS., DOC. NO. SC 019, THE HISTORY OF U.S. SUGAR PROTECTION (2012) (attached as Appendix BB).

[83] *See Sugars & Sweeteners Policy*, USDA ECONOMIC RESEARCH SERVICE (Nov. 14, 2014), www.ers.usda.gov /topics/crops/sugar-sweeteners/policy.aspx (attached as Appendix CC).

[84] USDA, FARM SERV. AGENCY, SUGAR LOAN PROGRAM, SUGAR MARKETING ALLOTMENTS, AND FEEDSTOCK FLEXIBILITY PROGRAM (2014) (attached as Appendix DD).

[85] *Id.*

[86] *Id.*

[87] *Table 3b—World Raw Sugar price, ICE Contract 11 Nearby Futures Price, Monthly, Quarterly, and by Calendar and Fiscal Year*, USDA ECONOMIC RESEARCH SERVICE (Apr. 2, 2015), http://www.ers.usda.gov/datafiles/ Sugar_and_Sweeteners_Yearbook_Tables/World_and_US_Sugar_and_Corn_Sweetener_Prices/table03b.xls (attached as Appendix EE).

[88] *See* SUGAR LOAN PROGRAM, *supra* note 84.

that markets raw cane sugar or refined beet sugar.[89] Initially for 2015, Florida Crystals (the brand name for the Okeelanta operations) was allotted over 950,000 tons to produce, raw value.[90]

Finally, import quotas restrict the quantity of sugar entering the U.S. from other countries.[91] USDA establishes the annual quota volumes and the U.S. Trade Representative allocates the volumes among countries.[92] Only sugar paying a higher, over-quota tariff may enter the country in unlimited quantities, again restricting supply and inflating prices.[93] The tariff applies to imports of raw cane sugar, refined sugar, sugar syrups, specialty sugars and sugar-containing products.[94]

The end result of these measures is a hyper-inflated U.S. sugar price ultimately paid by consumers. In 2014, the U.S. raw sugar price was 24.15 cents per pound versus 16.34 cents per pound in the world market.[95] With 1.5 million tons of sugar produced by Florida sugar cane each year, the sugar industry in the EAA annually pockets an additional $234 million dollars from U.S. consumers, and Okeelanta takes home $92 million of it.[96] In other words, instead of receiving $326 per ton of raw sugar like the rest of the world, they receive $483 per ton, with U.S. consumers paying roughly 50% more for their sugar.

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[89] *Id.*

[90] *USDA Announces 2014-Crop Sugar Loan Rates and FY 2015 Sugar Program Revisions*, USDA FARM SERVICE AGENCY, http://www.fsa.usda.gov/FSA/newsReleases?area=newsroom&subject=landing&topic=ner&newstype=newsrel&type=detail&item=nr_20140926_rel_0152.html (last visited Apr. 13, 2015) (attached as Appendix FF).

[91] *Sugar Import Program*, USDA FOREIGN AGRICULTURAL SERVICE, http://www.fas.usda.gov/programs/sugar-import-program (last visited Apr. 13, 2015) (attached as Appendix GG).

[92] *Id.*

[93] *Id.*

[94] *Id.*

[95] *Compare Table 3b*, *supra* note 87, *with Table 4—U.S. Raw Sugar Price, Duty-Fee Paid, New York, Monthly, Quarterly, and By Calendar and Fiscal Year*, USDA ECONOMIC RESEARCH SERVICE (Apr. 2, 2015), http://www.ers.usda.gov/datafiles/Sugar_and_Sweeteners_Yearbook_Tables/World_and_US_Sugar_and_Corn_Sweetener_Prices/Table04.xls (attached as Appendix HH).

[96] *See* MARK MOSSLER, UNIV. OF FLA., INST. OF FOOD & AGRIC. SCIS., DOC. NO. PI 171, FLORIDA CROP/PEST PROFILE: SUGARCANE (2014) (attached as Appendix II); *see also USDA Announces 2014-Crop Sugar Loan Rates*, *supra* note 90.

B. Sugar Production Is Profitable With Green Harvesting

Sugar producers in other parts of the world run profitable and successful businesses without these corporate subsidies, even when they use green harvesting practices. Other countries, such as Australia, have moved away from burning cane.[97] In Australia, only a fraction of the sugarcane producers continue to burn cane.[98] The remaining areas all employ green harvesting techniques to harvest sugarcane, leaving the extra trash (leaves and stalks) on the ground to act as a blanket and delivering unburned cane to the mill.[99]

While it takes longer to harvest green cane, the practice avoids spoilage that occurs from burning because the cane is still alive when it is harvested and thus lasts longer.[100] Also, the trash blanket left on the ground significantly reduces weeds and cuts the cost of herbicide use or other mechanical removal, reducing growing costs.[101] Trash blankets also conserve soil moisture and reduce evaporation losses, thus allowing for less frequent irrigation.[102] Additionally, they prevent erosion and improve soil fertility.[103] In most cases, trash blanketing results in higher yield crops.[104] Green harvesting also allows for harvesting during wet conditions and fewer mill stoppages.[105] The increased trash on green cane brought to the mill also provides an additional fuel source.[106] These advantages of green cane harvesting—and these are only some of the advantages, as further discussed in the attached report from Dr. Andrew Wood, an agronomist specializing in sugarcane who has extensive experience in advising sugarcane producers[107]—outweigh the minor disadvantages of the practice. Consequently, it is economical to employ green harvesting even at world market prices.

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[97] WOOD, *supra* note 10 (attached as Appendix A).

[98] *Id.*

[99] *Id.*

[100] *Id.*

[101] *Id.*

[102] *Id.*

[103] *Id.*

[104] *Id.*

[105] *Id.*

[106] *Id.*

[107] *Id.*

C. Okeelanta Already Uses The Equipment Needed To Harvest Green

Okeelanta and its affiliates need make no equipment changes to harvest green cane. All harvesters are capable of harvesting either burnt or unburnt cane.[108] With green cane, harvesters simply have to slow down the forward speed of the machine and adjust the speed in the extractor fans.[109] Though Florida long employed harvesting by hand using migrant workers,[110] Florida's sugarcane industry is now fully mechanized.[111] Mechanical harvesters were first brought to Florida from Australia.[112] Later, American companies copied the design and produced similar models.[113] In fact, about 15 percent of the sugar cane in the EAA is harvested green.

Okeelanta could be harvesting green today. Instead, the industry uses its inflated income to hire fleets of lobbyists to push for less stringent environmental regulation. From 1990 to 2013, the sugar cane and beet industry spent more than $41.7 million lobbying the government.[114] Okeelanta is content to make windfall profits, ignore established harvesting technology, and keep the fires burning.

VII. CONCLUSION

Okeelanta's Title V permit needs to include the HAP emissions from its pre-harvest burning operations, because Okeelanta's sugarcane fields, together with its mill and refinery operations, constitute a single "facility" under Title V for HAP purposes. Furthermore, any renewal of Okeelanta's permit should be conditioned upon the phasing out of its pre-harvest cane burning practices over the next five years.

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[108] *Id.*

[109] *Id.*

[110] *Id.*

[111] MOSSLER, *supra* note 96.

[112] *Cane Syrup Mill–Detail*, PIONEER FLORIDA MUSEUM AND VILLAGE, http://www.pioneerfloridamuseum.org/index.php?option=com_content&view=article&id=34:cane-syrup-mill-detail&catid=35:grounds-section&Itemid=39 (last visited Apr. 13, 2015) (attached as Appendix JJ).

[113] *Id.*

[114] *Sugar Cane & Sugar Beets*, OPENSECRETS.ORG, http://www.opensecrets.org/industries/indus.php?ind=A1200 (last visited Apr. 13, 2015) (attached as Appendix KK).

Dated April 14, 2015

David Guest on Behalf of The Sierra Club

APPENDIX A

Time for a transition from pre-harvest burning of sugarcane to green cane harvesting in the Everglades Agricultural Area

Andrew Wood, PhD

1. Qualifications and work experience in the sugar industry

My university training was in physical geography and soil science (BSc. Hons. Geography, University of Durham; MSc. Soil Science, University of Aberdeen; PhD. Geography, specializing in farming systems, University of Papua New Guinea, 1984). My whole working life has been spent in tropical areas with a wide range of agricultural crops and cropping environments. This has included 4 years of soil mapping and land resource assessment with a land resource survey team in Nigeria, 6 years of university teaching and agricultural research in Papua New Guinea, and 34 years as a sugarcane agronomist and agricultural consultant in Queensland, Australia. I learnt a great deal about tropical soils and different food crops whilst working in Nigeria. In Papua New Guinea, my research focus was on understanding the relationship between different cropping systems and changes in soil fertility over time.

I was recruited to the Australian sugar industry to investigate soil degradation under long-term sugarcane cropping and to develop strategies for restoring soil fertility. By comparing the soil fertility of sugarcane fields and adjacent areas that had not been farmed I was able to conclude that soil physical properties (bulk density and porosity) and chemical properties (organic matter, cation exchange capacity, acidity, available Ca and Mg) had degraded under the system of sugarcane cropping in the Herbert district. I attributed the degradation to long-term monoculture, burning of both sugarcane and harvest residues and the application of large amounts of nitrogen fertiliser (Wood, 1985). I considered that the first and most important step was to phase out cane burning so that the large amounts of crop residues from sugarcane crops (leaves and tops) could be returned to the soil and thus rebuild soil organic matter levels.

I started working with a small number of cane farmers who were interested in harvesting their cane green (unburnt) but were unsure of the longer-term consequences. Together we developed a series of farm trials with 3 major treatments: cane harvested green and trash burnt on the ground, cane harvested green and trash incorporated into the soil, and cane harvested green and trash left as a surface blanket with no cultivation. The trials showed improved cane yields, higher sugar content and a progressive increase in soil organic matter over time when the trash was left on the soil surface as a trash blanket (Wood 1991) .

Cane growers were made aware of the research results through visits to on-farm demonstration trials and through shed meetings and field days. We held a symposium on green cane harvesting in Ingham in 1983 and expected around 50 people to attend. We were amazed when over 500 people came from all over northern Queensland. Clearly farmers were eager to seek alternative ways of harvesting and managing their sugarcane crops because the price of sugar on the world market at that time was quite low and they were seeking farming practices that would reduce their costs. The research and extension activity was instrumental in accelerating the adoption of green cane harvesting in the Ingham area, which increased from less than 1% of the area in 1981 to over 70% in 1987. The Ingham area led the way in

the transition from burnt cane to green cane. Now virtually the entire Australian sugarcane crop, apart from the Burdekin and northern New South Wales districts, is harvested green.

The promotion of green cane harvesting and trash retention was followed by research into ways of achieving better use efficiency of nitrogen fertiliser. Collaborative research with Griffith University and CSIRO showed that high losses of applied nitrogen often occurred when urea was applied to the surface of sugarcane trash. Cane growers are now much better informed of ways in which nitrogen losses by volatilisation can be minimised and have changed to applying their fertiliser beneath the trash blanket.

2. Previous visits to Florida's sugar industry

I first visited the Florida sugar industry in 1989 and visited it a second time in 2001. My first visit was part of a study tour of sugar research around the world when I visited sugar research facilities in Mauritius, South Africa, Brazil, Florida, Louisiana and Hawaii. My second visit occurred when I was travelling to Dominican Republic. I spent a few days in Florida and was hosted by Dr David Anderson, an agronomist based at the Everglades Research and Education Centre in Belle Glade. I had met David at a symposium on sugar research held in Brisbane a few years earlier. I agreed to give a seminar at Belle Glade on my work in Australia with green cane harvesting. However to my surprise I learnt that the seminar had been cancelled because of pressure exerted by the major sugar companies in Florida. Clearly they were opposed to a visitor from Australia talking about the benefits of green cane harvesting.

My observations in the Everglades Agricultural Area include:

- *Muck soils.* Most of the sugarcane was grown on fertile, black, highly organic peat soils. The muck soils, once they have been drained, gradually oxidise, causing the depth of soil to shrink and the level of the land to become progressively lower. I learnt that no new areas of muck soils were being released and that sugarcane production was expanding onto much more infertile sandy soils which were low in organic matter and which required frequent irrigation.
- *Harvesting cane by hand cutting.* Australia had been harvesting cane mechanically since the 1960s so I was very surprised to discover that the richest country in the world was still cutting cane by hand. The cane was burnt prior to harvest and groups of migrant labourers from the Caribbean cut the cane stalks, removed the tops and left the whole stalks in the field. Groups of mechanical loaders then loaded the stalks into trucks for transport to the sugar mill. Now the Florida industry uses the same high capacity mechanical harvesters as used in Australia and manufactured by the John Deere Company. They are based on the harvester technology developed in Australia.
- *Extinguishing fires in the muck soils.* It is not uncommon for fires to occur in the muck soils following a cane fire. Groups of labourers would walk through the cane fields after a fire and extinguish any soil fires. I was told that fires could smoulder beneath the soil surface for days. Later I learnt that a sugarcane harvester had once disappeared into one of the holes caused by a smouldering fire.
- *Planting cane by hand.* Cane was planted by making furrows in the field, laying whole stalks of cane in the furrows and cutting them into billets. Tractors were then used to cover the cane with soil.

- *Environmental issues*. The Florida sugar industry was increasingly subject to regulation in order to reduce the impact of sugar production on the adjacent wetlands of the Everglades.

3. The benefits of green cane harvesting for farming and the environment in Australia

Trash left as a "blanket" in cane fields after green cane harvest results in a significant reduction in weed germination and growth. Fewer herbicides need to be used, thus reducing growing costs. Conversion to green cane harvesting in Australia occurred in the 1980s before herbicides were widely used. At that time farmers used mechanical cultivation (discs, weeder rakes etc.) to destroy weeds. Trash blanketing and no cultivation of ratoon crops resulted in a significant reduction in growing costs. Fewer tractors and implements were required resulting in a big reduction in fuel use. Farmers spent much less time in the field allowing them to farm larger areas. Trash blankets conserve soil moisture and reduce evaporation losses. Consequently less frequent irrigation is required thus lowering the cost of water and pumping. The benefits are particularly evident on dry, sandy soils where there is much better cane growth and higher yields. Trash blankets also protect soils from erosion on sloping land. In the high rainfall areas of north Queensland where slopes are quite steep, significant reductions in soil loss by erosion have been recorded. One disadvantage is that a trash blanket poses a fire risk in very dry conditions and trash fires are difficult to extinguish.

Trash blankets improve soil fertility particularly in the top few centimetres of soil. They increase soil organic matter and cation exchange capacity (ability of the soil to store nutrients). Trash contains nutrients (nitrogen, potassium, calcium and magnesium) and slowly releases these as it decomposes. If the cane had been burnt, most of the nitrogen and some of the other nutrients in the trash would have been lost. Trash blankets improve soil biological activity and significant increases in earthworm populations and soil microbial biomass have been recorded. Trash blankets also reduce the range of soil temperatures (lower maximum temperatures, higher minimum temperatures) which is generally better for crop growth although growth following cold nights can be slowed. Burning is preferred in areas consistently experiencing temperatures close to freezing, such as northern New South Wales.

Whilst trash retention in most cases results in higher crop yields, particularly in drier areas and on well drained soils, reduced yields may occur where drainage is poor and where water ponds due to irregularities in topography because the trash keeps the soil wetter for longer. Most sugarcane farmers in Queensland now grade their fields using laser-guided land levelling equipment to ensure that this does not occur. Another strategy is to grow cane on raised beds.

Significant benefits for the environment accrue from not burning cane. There is no air pollution from pre-harvest cane burning or from burning harvest residues (tops and trash) on the ground. Green harvesting and trash blanketing increase the sequestration of carbon into the soil and reduce carbon dioxide emissions. However trash blankets have been implicated in increased emissions of nitrous oxide, a potent greenhouse gas. The micro-organisms responsible for denitrification in wet soils require a carbon source and this is available in the trash blanket. Good surface drainage is thus essential to avoid denitrification losses particularly in high rainfall environments. Nitrification inhibitors can also be used, which prevent the conversion of ammonium into nitrate, but they are expensive.

One of the factors that appears to affect the build-up of carbon in the soil with continued trash retention from green cane harvesting is that at the end of each crop cycle (usually consisting of a plant crop and 3-4 ratoons) the crop is disced and the trash incorporated into the soil. This usually results in an acceleration in oxidation of organic matter and a reduction in soil carbon. It appears likely that in order for carbon sequestration in the soil to be enhanced, the trash blanket should not be disturbed. To achieve this a system of zero tillage is required where any new plantings of sugarcane are direct drilled into the soil beneath the trash blanket using a double disc opener planter. Some cane farmers in Australia are now using a farming system following these principles and are adjusting their practices to suit different soils and climatic conditions.

4. Harvesting green cane

In the 1980s when cane farmers in Australia were showing a lot of interest in harvesting green cane the biggest impediment was the lack of a suitable harvester. Growers used their existing machines but had to harvest very slowly otherwise the harvester choked with the mass of cane and trash. Australia through the Toft company and numerous grower led innovations led the way in the development of harvesters with the capability of cutting green cane. Now there is no longer a manufacturer of cane harvesters based in Australia. New cane harvesters have to be sourced either from John Deere based in Louisiana or from Case based in Brazil. All current harvesters are capable of harvesting cane either burnt or unburnt cane.

Green cane harvesting allows harvesting to proceed in wet or showery weather. Prior to the change to green harvesting, mills in Australia often had to stop in wet weather because farmers were unable or unwilling to burn their cane and this extended the milling season and increased mill costs. The increased ability to operate in wet conditions resulted in far fewer mill stoppages and harvester operators upgrading their equipment so that they could operate efficiently in wet field conditions. Many more harvesters now have caterpillar tracks rather than wheels and infield cane transporters are equipped with high flotation tyres or tracks to improve trafficability in wet field conditions and reduce soil compaction.

Harvesting large crops of >60 tons per acre green is possible but harvesters need to travel very slowly so that they can separate the cane from the trash. In the two areas in Australia with high yielding crops (Burdekin and northern New South Wales), less than 50% of the cane is harvested green. Higher harvesting rates (more tons cane per hour) can be achieved in burnt cane. Harvesting costs are thus lower in burnt cane.

Cane losses occur with mechanical harvesting with both green and burnt cane, but are higher with green cane. Cane loss during harvest can be reduced by slowing down the forward speed of harvesters and reducing the speed of extractor fans. The reduction in extractor fan speed results in higher levels of trash in the harvested cane being sent to the mill. Since there is much less trash in burnt cane, losses of cane during harvest and trash levels in harvested cane are lower.

In Australia whilst there is no difference in the price charged by harvester contractors for cutting burnt or green cane, time spent harvesting, fuel use and harvester wear are higher when cutting green. The true cost of harvesting green would be 5-10% higher than burnt cane.

5. Transport of cane to the mill

The volume of material to transport to the mill with green cane is greater than with burnt cane due to higher amounts of trash mixed with the cane. Hence cane transport costs are likely to be at least 10% higher. Experimentation is in progress to test methods of post-harvest cane cleaning for reducing the amount of trash in cane delivered to sugar mills. The cane cleaning can occur either at small cleaning plants located at cane delivery points where the harvested cane is collected for transport to the mill, or at larger plants at the mill itself. However cane cleaning represents an additional cost which is not particularly attractive at today's relatively low world prices for sugar (12-13 cents per pound). In Florida, where the price is approximately 10 cents more per pound, this would be more attractive.

6. Processing cane at the mill

When sugarcane is processed into raw sugar at the mill there are at least 9 stages in the process. The first stage is shredding where the billets of cane are smashed with large hammers so that the sugar can be more easily removed. The next stage is crushing or milling where the shredded cane passes through 4-6 combinations of mill rollers to extract the juice. Water is added to the crushed cane to wash out as much sugar as possible. The third stage is clarification where lime is added to the dirty juice and the soil, fibre and some other impurities flocculate. This flocculated material is extracted through a filtration process and the solid material is known as filter mud or filter cake. The mud is transported to the cane fields and is spread on the soil. It is an effective soil conditioner which is high in calcium (from the lime) and phosphate. The next stage is evaporation where the juice is boiled under pressure and the excess water evaporates. The juice is now known as syrup. The syrup is boiled again under vacuum at the pan stage to further concentrate the sugar content. The next stage is crystallisation where sugar crystals begin to form but are coated in dark molasses. The molasses is removed at the next stage which is known as centrifugation. Once most of the molasses is removed the sugar crystals are a golden brown colour and proceed to the sugar drying process. This material is known as raw sugar. Australian sugar mills mainly produce raw sugar. White sugar is produced by refining the raw sugar, and removing more of the molasses and impurities.

When green cane is processed at the mill it is much fresher than burnt cane and usually has a higher sugar content of about 1 percentage point greater than its normal percentage concentration. The sugar stored in cane starts to deteriorate as soon as the cane is burnt, and the rate of deterioration is faster in hot humid conditions. If there are delays in getting the harvested cane to the mill or if the mill breaks down, considerable processing difficulties are encountered at the mill and additional costs are incurred. One of the main processing difficulties with stale cane is the production of Dextran. High dextran content in raw sugar juice creates operational problems in the mill. The large dextran molecules increase the viscosity of syrup, massecuite and molasses, thereby hampering the sucrose crystallization process. In addition, sucrose crystal elongation may occur which further reduces molasses exhaustion. As a result, factory capacity and yield are lowered and in many cases it is necessary to shut down the mill for equipment cleaning.

Increased trash levels in green cane sent to the mill provide additional fuel which can be used for mill operation, electricity generation or for production of ethanol and other chemicals.

However the increased fibre leads to higher milling costs and can result in increased sugar losses to bagasse. Other issues include sugar colour, which is likely to be higher with green cane resulting in slightly higher refining costs.

7. Summary of advantages and disadvantages of green cane

Advantages:

- Reduced weed germination and growth.
- Lower herbicide costs.
- Reduced cultivation (discing).
- Fewer implements and tractors.
- Big reduction in fuel use for cultivation.
- Lower labour costs because of substantially reduced time necessary to work in fields.
- Reduced soil moisture losses.
- Less frequent irrigation.
- Reduced costs of water and pumping.
- Reduced soil loss by erosion.
- Increased soil fertility.
- Reduced oxidation of muck soil.
- Increased return of organic matter to the soil.
- Recycling of nutrients.
- Increased soil nutrient storage capacity.
- Improved soil biological activity.
- Increased earthworm populations.
- Improved soil structure.
- Reduced range of soil temperature (lower maximums and higher minimums).
- Reduced air pollution.
- Increased sequestration of carbon.
- Reduced carbon dioxide emissions.
- Fewer mill stoppages for wet weather.
- Fresher cane with a higher sugar content.
- Reduced rate of cane deterioration.
- Reduced processing difficulties with stale cane.
- Increased fuel for electricity generation.

Disadvantages:

- Increased fire risk.
- Reduced harvesting rate because harvesters have to move more slowly.
- Higher harvesting costs.
- Increased cane losses during harvesting.
- Increased cane trash going to the mill.
- Higher cane transport costs.
- Higher milling costs.
- Increased sugar loss to bagasse.

- Increased sugar colour.

On balance, the advantages outweigh the disadvantages, so that green cane harvesting produces a larger net profit.

8. A vision for a more sustainable sugarcane farming system in the Everglades Agricultural Area

Agriculture as currently practised in the Everglades Agricultural Area has a limited future due to the continued oxidation of the peaty soils and their destruction by burning. Extensive land subsidence has already occurred on the muck soils and this is likely to continue rapidly whilst crops like sugarcane are burnt and little organic matter is returned to the soil. A system of sugarcane production where the soil is always covered with a thick trash blanket would be much more sustainable, and easy to achieve because the harvesters already in use can be used in green cane harvesting without any modifications. This system would be equally applicable to sugar production on the very sandy soils outside the muck area. A trash blanket would greatly reduce irrigation needs and would return much needed organic matter to the soil. A farming system with the following characteristics is suggested:

- No further burning of sugarcane crops or crop residues.
- Trash from green cane harvesting be left as a trash blanket covering the soil surface and protecting the peaty soils from further oxidation.
- Sugarcane grown on preformed beds with a row spacing of around 1.8m to avoid compaction of the soil close to the cane row by harvesting equipment. Growing cane on raised beds should avoid problems associated with excessive wetness when the water table is high. Ideally both cane planting and harvesting would be under GPS guidance.
- Fertiliser is applied beneath the trash using a fertiliser applicator with coulters to avoid ammonia volatilisation losses.
- The cane crop at the end of a crop cycle is removed by killing it with glyphosate herbicide.
- A break crop is recommended for planting between sugarcane crop cycles in order to break the sugarcane monoculture and control soil pathogens.
- The next sugarcane crop is then be planted through the trash onto the preformed beds using a double disc opener planter with coulters to cut through the trash.

This type of sugarcane farming system with green cane harvesting, trash blanket, little or no tillage and break crops is being successfully used in parts of Australia.

The amount of innovation and experimentation in the Australian sugar industry has been much greater than that in the Everglades Agricultural Area. Pressure to lower costs in farming, harvesting, and milling, is far greater in Australia as the industry has to exist on world sugar price. The sugar industry in the United States receives very substantial subsidies and has fallen behind other parts of the world in adopting more modern growing methods.

9. References

Wood, A.W. (1985) Soil degradation and management under intensive sugarcane cultivation in north Queensland. *Soil Use and Management* 1: 120-124.
Wood, A.W. (1991) Management of crop residues following green harvesting of sugarcane in north Queensland. *Soil and Tillage Research* 20: 69-85.

APPENDIX B

Estimate of Total Hazardous Air Pollutant Emissions from Okeelanta-Operated Fields

In order to estimate the HAP emissions from the pre-harvest burning of Okeelanta-operated sugarcane fields in the EAA and surrounding areas, we used the following formula:

$$E_{TOT} = A \times \sum_i EF_i,$$[1]

where E_{TOT} is the total emissions of HAPs, A is the activity factor, and EF_i is the emissions factor for HAP type *i*. The sum is taken over all HAPs for which emissions factor data is available. The activity factor A is the amount of cane biomass burned per year on Okeelanta-operated fields:

$$A = (burn\ area\ of\ fields) \times (fuel\ loading\ factor),$$

where the fuel loading factor is the average number of tons per acre burned during pre-harvest burning each year.[2] The table below shows the low, medium, and high values used for all relevant variables.

Variable	Units	Low	Medium	High	Sources
fuel loading factor	tons/acre	3	7	17	AP 42[3]; Hall[4]
burned field area total	acres	-	124,698	-	Florida Forest Service[5]
burned field area Cat. A	acres	-	91,428	-	Florida Forest Service
burned field area Cat. B	acres	-	14,167	-	Florida Forest Service
Benzene EF	mg/kg	14.61	16.5	18.39	Hall

[1] *See, e.g.*, EPA, COMPILATION OF AIR POLLUTANT EMISSION FACTORS—VOLUME I: STATIONARY POINT AND AREA SOURCES (1995) (attached as Appendix LL).

[2] *Id.* § 2.5.

[3] EPA, COMPILATION OF AIR POLLUTANT EMISSION FACTORS—VOLUME I: STATIONARY POINT AND AREA SOURCES § 2.5 tbl.2.5-5 (1995).

[4] Danielle Hall et al., *PAHs, Carbonyls, VOCs and $PM_{2.5}$ Emission Factors for Pre-Harvest Burning of Florida Sugarcane*, 55 ATMOSPHERIC ENV'T 164 (2012) (attached as Appendix L).

[5] Burned acreage estimated from approved burn requests on Okeelanta-operated land in the EAA during the 2013-14 season. Information on burn approvals obtained via public records request from the Florida Forest Service.

Toluene EF	mg/kg	4.26	5.2	6.14	Hall
Ethyl Benzene EF	mg/kg	0.66	0.81	0.96	Hall
Styrene EF	mg/kg	0.1	0.35	0.6	Hall
m, p-Xylenes EF	mg/kg	0.49	0.94	1.39	Hall
o-Xylenes EF	mg/kg	0.1	0.29	0.48	Hall
Naphthalene EF	mg/kg	2.79	5.24	7.69	Hall
Formaldehyde EF	mg/kg	208	524	840	Hall
Acetaldehyde EF	mg/kg	125	323	521	Hall
Propionaldehyde EF	mg/kg	17.4	51	84.6	Hall

To calculate the high, medium, and low estimates reported in the body of the comment letter, we simply took the high, medium, and low values for fuel loading and emissions factors and plugged them into the above equation.

APPENDIX C

Our Story

Florida Crystals Corporation is a privately owned and diversified agriculture, consumer products, real estate and energy company.

Florida Crystals is a leading sugar producer and the United States' first fully integrated cane sugar company, guiding our sugar from the field to the table. In Palm Beach County, Florida Crystals owns 155,000 acres of land, two sugar mills, a sugar refinery, a rice mill, a packaging and distribution center, and a renewable energy facility.

We are America's first and only domestic producer of certified organic sugar, grown and harvested in Florida. We also pioneered certified organic and natural rice production in the state.

Florida Crystals proudly owns and operates the largest biomass power plant in North America, which produces eco-friendly energy that powers our operations and tens of thousands of homes.

Florida Crystals is headquartered in Palm Beach County, Florida.

Origins

The origins of Florida Crystals trace back five generations, when the Fanjul family began sugar farming and production in Cuba in the 1850s. After the communist takeover of Cuba, Alfonso Fanjul led the effort to reestablish the business in the rich and fertile soils of South Florida in 1960. Throughout the following decades, under the leadership of Alfonso "Alfy" Fanjul and Jose "Pepe" Fanjul, the company has grown to be one of the largest, most innovative and successful agriculture companies in the world.

Today, along with our subsidiaries, as the world's largest sugar refiner, our production capacity exceeds 7 million tons of sugar per year with operations in Florida, California, Louisiana, New York, Maryland, Canada, Mexico, England and Portugal. Our products are sold under the Domino®, C&H®, Florida Crystals®, Redpath®, Jack Frost® and Tate & Lyle® brands.

Chocolate Filled Valentine Sugar Cookies

APPENDIX D

SS-AGR-232

UF UNIVERSITY of FLORIDA
IFAS Extension

An Overview of Florida Sugarcane[1]

L. E. Baucum and R. W. Rice[2]

Sugarcane is a crop that can be grown throughout Florida. In many areas of the state, sugarcane is grown only as a hobby crop for syrup production or as a source of “chewing cane." However, in South Florida, near Lake Okeechobee, sugarcane is grown commercially for the production of crystal or "white" sugar.

This overview was prepared to answer the most frequently asked questions about the commercial Florida sugarcane industry and to describe the production of sugarcane and sugar.

What is sugarcane?

Sugarcane is a tropical grass native to Asia, where sugarcane has been grown in gardens for more than 4,000 years. The product of interbreeding four species of the *Saccharum* genus, sugarcane is a giant, robust, sugary plant.

Methods for manufacturing sugar from sugarcane were developed in India about 400 BC. Christopher Columbus brought the plant to the West Indies in the sixteenth century, and today sugarcane is cultivated in tropical and sub-tropical regions throughout the world. Roughly 75 percent of the world's sugar comes from sugarcane.

Where is sugarcane grown in Florida?

Palm Beach County accounts for approximately 75 percent of the commercial sugarcane acreage in Florida and 75 percent of the total harvested sugarcane tonnage in Florida. The remainder of Florida's commercially produced sugarcane is grown in Hendry, Glades, and Martin counties. These counties–along with Palm Beach County–are all adjacent to Lake Okeechobee, which is the northernmost portion of the historic Florida Everglades system. This area of commercial sugarcane production is so compact, most visitors to the Sunshine State never see a commercial sugarcane field.

1. This document is SS-AGR-232, one of a series of the Agronomy Department, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida. First printed July 1992. Revised April 2006 for publication in EDIS. Original authors included T. J. Schueneman, extension agent IV, Palm Beach County, Belle Glade, FL. This publication, most recenlty revised in August of 2009, is a part of the Florida Sugarcane Handbook, an electronic publication of the Agronomy Department. For more information, contact the editor of the Sugarcane Handbook, Ronald W. Rice (rwr@ufl.edu). Visit the EDIS Web site at http://edis.ifas.ufl.edu.

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Figure 1. An aerial view of commercial sugarcane fields near Belle Glade, FL, June 2008. In the foreground are variety test plots managed by sugarcane growers for the purpose of evaluating promising germplasm lines, a process required for the release of new cultivars. Credits: James M. Shine, Jr., Sugar Cane Growers Cooperative of Florida

With renewed interest in the possibility of growing sugarcane as a source of biofuel for ethanol, small amounts of sugarcane are also being grown on a demonstration basis in some counties that neighbor this region of commercial sugarcane production in Florida.

Why is sugarcane grown near Lake Okeechobee?

Fertile organic soil and the warming influence of Lake Okeechobee are the primary reasons Florida's sugar industry originally located in this region. Killing cold temperatures occur very infrequently near the lake. Adequate water and abundant sunshine were also important factors that led to the location of Florida's commercial sugarcane industry in this region. The expensive proposition of establishing any new infrastructure has ensured that commercial sugarcane production in Florida remains fairly close to the existing sugar mills.

Is South Florida a good place to grow sugarcane?

Fair. The muck soils of the Everglades are rich in nitrogen and support vigorous cane growth. Nitrogen-rich soils, in combination with the favorable South Florida climate, can sometimes lead to excessive sugarcane growth, which results in large stalks, but with reduced sugar content.

Sporadic sub-freezing temperature conditions damage some cane almost every winter, particularly young growth from newly planted cane or young re-growth from ratoon “stubble” crops. South Florida also has its share of environmental concerns, thus growers design and implement Best Management Practices (BMP's), which typically increases production costs.

While Florida sugar mills are modern and efficient, labor costs are extremely high by global standards. Underlying high labor costs and other economic factors led to the industry's rapid conversion to mechanical harvesting in the early 1990s and ultimately to a reduction in the number of sugar mills.

How much sugarcane is grown in Florida?

In recent years commercial sugarcane production in Florida has declined slightly. Sugarcane acreage has decreased from a high of 454,400 acres (183,727 hectares) in the 2000-2001crop year to approximately 400,000 acres (161,874 hectares) for the 2008-2009 crop year. Florida's 2000-2001 sugarcane crop year yielded over 17.3 million U.S. tons (15.7 million metric tons) of stalks and 2.02 million U.S. tons (1.83 million metric tons) of raw sugar. In comparison, Florida's 2007-2008 sugarcane crop year yielded 13.3 million U.S. tons (12.1 million metric tons) of stalks and 1.55 million U.S. tons (1.41 million metric tons) of raw sugar.

Placing the current size of Florida's sugarcane crop into perspective historically, prior to the Cuban embargo in 1961, Florida had only 50,000 acres (20,234 hectares) of sugarcane.

What is the value of Florida's sugar crop?

In Florida's sugarcane industry, recoverable sugar, by weight, has increased from below 10 percent in 1984 to 11.6 percent in 2000-01. In 2008 Florida's raw sugar crop was valued at approximately

$450 million. (http:www.ers.usda.gov/StateFacts/FL.htm).

Most of the fibrous portion of the cane stalks (bagasse) is burned as fuel for the mills, saving sugar producers an estimated 113 million gallons of fuel oil or 2.1 billion kilowatt hours of electricity.

Sugarcane is Florida's most economically valuable field crop, worth more than the combined value of Florida-grown corn, soybean, tobacco, and peanut crops. In 2004 sugarcane ranked third in Florida's agricultural economy; by 2008 sugarcane had fallen to sixth in the state's overall agricultural economy.

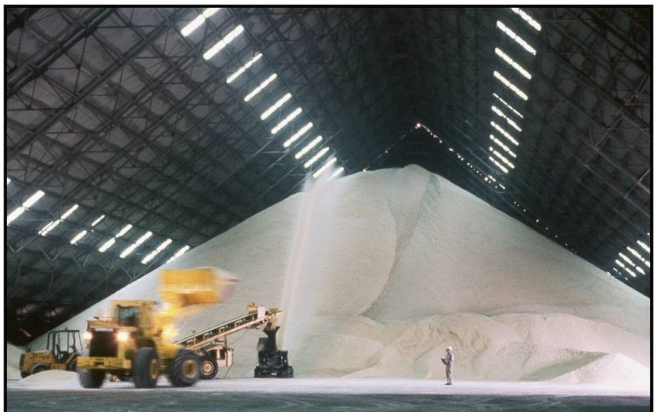

Figure 2. Raw sugar, produced from Florida-grown sugarcane, in temporary storage in a warehouse in South Florida, March 1998. In the sugar-production process, raw sugar is moved from warehouse to refinery, where the sugar is re-dissolved and refined to obtain the purity required for white sugar crystals. Credits: Sugar Cane Growers Cooperative of Florida

What is Florida's contribution to the U.S. production of sugar?

In 2007-08, Florida contributed an estimated 48 percent of the cane sugar and 24.3 percent of the total (from sugarcane and beets, combined) sugar produced in the United States.

Where does the rest of the sugar produced in the U.S. come from?

Florida is the largest producer of sugarcane in the United States followed by Louisiana, Hawaii, and Texas in order of production. Sugar beets are grown in Minnesota, North Dakota, Idaho, Michigan, California, and in six other states. The sugar beet industry provides approximately 50 percent of the sugar produced in the United States, and sugarcane production accounts for the remaining 50 percent of domestic sugar production.

All U.S. sugar production combined still falls short of U.S. consumption. As a result, almost one-fifth of the sugar consumed in the United States is imported.

What research facilities support the Florida sugarcane industry?

Production of improved varieties is the primary mission of the U.S. Department of Agriculture-Agricultural Research Service (USDA-ARS) Sugarcane Field Station at Canal Point, Fla. Originally established to produce seed for the Louisiana sugarcane industry, this station now has an extensive program for developing and testing sugarcane varieties adapted to Florida conditions. High sugar content, disease resistance, rapid growth, and tolerance to high water tables are among the traits sought in new sugarcane varieties. Additionally, a new focus of the variety development program is aimed at improving yields of sugarcane grown on sandy mineral soils.

In Palm Beach County, the University of Florida Everglades Research and Education Center at Belle Glade, Fla. (EREC) cooperates with the USDA-ARS at Canal Point in a sugarcane variety development program and conducts research on sugarcane nutrition and physiology, pest control, water use, and associated agronomic problems. In Immokalee, Fla., in Collier County, on Florida's west coast, the University of Florida Southwest Florida Research and Education Center (SWFREC) conducts research to meet the needs of sugarcane producers who plant on the sandy mineral soils of that region. Research results and recommendations (fertilization, weed control, etc.) are presented in reports and meetings sponsored by the University of Florida Cooperative Extension Service.

Major sugarcane companies – including the United States Sugar Corporation, Florida Crystals Corporation, and the Sugar Cane Growers

Cooperative of Florida – conduct their own research and also support research programs at University of Florida (UF) and USDA facilities.

Figure 3. Sugarcane cultivar 'CP 00-1101' with lower leaves stripped away to reveal the segmented physiology of sugarcane stalks, August 2007. Credits: Wayne Davidson, Florida Sugar Cane League.

How is sugarcane planted?

Sugarcane planting takes place from late August through January. Because sugarcane is a multi-species hybrid, sugarcane seeds will produce plants that differ genetically from the parents. For this reason, commercial planting of sugarcane using seeds is impractical. Instead, a favorable sugarcane variety is planted by using sections of stalks from the mother plant, which then sprout daughter plants (clones), which are genetically identical to the mother plant variety. This process is called vegetative propagation.

Concerns surrounding high labor costs have supported a growing interest in developing machinery and agronomic practices for the mechanical planting of sugarcane. However, as of 2008, hand planting is still a common practice for planting sugarcane.

In this scenario, portions of mature sugarcane fields are reserved for “seed cane” (the cane used to vegetatively propagate the next planted crop). Instead of this sugarcane being mechanically harvested for the sugar mill, stalks of seed cane are mechanically harvested by “whole stalk harvesters”, loaded onto wagons, transported to the target field, and dropped horizontally into shallow furrows roughly 3-8 inches deep (8-20 cm deep). Typically, these sugarcane stalks are dropped as pairs, for a double line of sugarcane stalks throughout the furrow. To increase sprouting potential, stalks are then cut into shorter segments before being covered with soil.

Conventional row spacing for commercial sugarcane production in Florida is 5 feet (1.5 m). Cane stalks have buds (“eyes”) every 2-6 inches (5-15 cm), and each of these buds have the capability to sprout rapidly when buried in moist soil. Within two to three weeks, shoots emerge and, under favorable conditions, produce secondary shoots to give a dense stand of cane.

How often is a cane field replanted?

Typically, a sugarcane field is replanted every two to four years. After a field has been harvested the first time, it is maintained free of weeds, and a second crop of stalks, called a ratoon, grows from the old plant stubble. The second crop is harvested about one year after the first harvest. On average, 3 annual crops are harvested from one field before the field is replanted. When annual production declines to an unacceptable level due to insect, disease, or mechanical damage, the old sugarcane crop is plowed under after harvest, and the land is prepared for replanting with new seed cane.

If the last harvest of the ratoon sugarcane crop occurs early enough during the harvest season (possibly before January), the field will likely be replanted to sugarcane (termed “successive planting”). If, however, the last harvest of the ratoon crop occurs later, a decision may be made to delay replanting in sugarcane until the following season. Instead, the fallow sugarcane field may be planted to another crop, such as rice or sweet corn (termed, “fallow planting”).

Doesn't sugarcane produce seed?

A sugarcane plant is capable of producing seed, but seeds are not used for commercial planting. Sugarcane seeds are so small (1,000 seeds per gram or almost 454,000 seeds per pound), they cannot be planted directly in the field. Furthermore, sugarcane does not breed true. As a result, seeds from a single plant will produce seedlings that are genetically different from each other. Thus, seeds are not used for planting since varietal uniformity is important for commercial sugarcane production.Finally, only under strictly controlled temperature and day-length conditions will sugarcane seed be viable. Under normal conditions in Florida, sugarcane seed produced in the field will not germinate. For these reasons, only stalks are used as planting material for the vegetative propagation of sugarcane.

What are the white or reddish plumes seen in some sugarcane fields during winter and spring?

Plumes are the flowers and seed heads of the sugarcane plant. Each plume consists of several thousand tiny flowers. Each flower is capable of producing one seed. The cool winter weather in Florida ordinarily prevents development of viable seeds. During breeding for variety development, special precautions are taken to produce viable seed.

What attention does a cane field require after planting?

After sugarcane planting, weeds are controlled through cultivation and herbicides. Water must be pumped out of the field when rainfall is excessive. When the soil gets too dry, the crop is irrigated by allowing the water to flow back into the ditches that are normally used for drainage. Water readily seeps from the lateral field ditches throughout the entire sugarcane field, flowing on top of the underlying limestone bedrock and irrigating the porous organic muck soils. Sugarcane grown on nutrient-poor sand soils will require supplemental fertilizer applications to achieve optimal growth.

Figure 4. Sugarcane flowers, which produce seeds, are pictured in this November 2001 photo. Because seeds from a single sugarcane flower differ genetically, pieces of sugarcane stalk are planted, rather than seeds, to produce new plants that are genetically identical to the parent plant. Credits: James M. Shine, Jr., Sugar Cane Growers Cooperative of Florida

When and how is the cane harvested?

Sugarcane is harvested from late-October through mid-April. Given no damaging effects from freezes, sugar yields are typically highest after December. However, to complete the entire South Florida sugarcane harvest within the optimal time period, some fields must be harvested before sugarcane plants have reached maximum yield

potential. For this reason, agronomic research that documents variety-specific sugar yields over time is a useful tool for identifying varieties that can best be harvested in early-, mid-, and late-season. (For more on this topic, see EDIS Publication SSAGR221, *Maturity Curves and Harvest Schedule Recommendations for CP Sugarcane Varieties*, http://edis.ifas.ufl.edu/SC069.)

Figure 5. Cattle Egrets (Bubulcus ibis) forage for insects and soil grubs during a pause in a sugarcane harvest operation in fields west of Clewiston, FL, December 2005. Credits: Ronald W. Rice, IFAS/UF

In years past, sugarcane was hand-harvested, using cane knives. Conversion to mechanical harvesting began in the mid-1980s, and by 1993 the entire South Florida sugarcane crop was harvested mechanically.

Why are sugarcane fields burned before harvest?

Controlled fires in sugarcane fields burn off dead leaves and other biomass "trash," which would otherwise impede the harvest operation, increase transportation costs to the mill, interfere with milling machinery, and absorb sugar during the extraction process. (Absorbed sugar cannot be recovered.)

Fields are burned immediately before harvest. The fires are rather spectacular, but of short duration; a 40-acre field burns in 15-20 minutes. Burning is done only in the daytime (through a permitting process with the Florida Division of Forestry), and only when dispersal of the smoke by air currents causes minimum nuisance.

Once the cane is cut, how does it get to the mill?

After the sugarcane fields are burned, mechanical harvesters deposit the cut cane directly into field wagons. Four-wheel drive tractors haul 16 tons of cane out of the field with each four-wagon load. At special ramps near the field, the cane is dumped from the wagon into highway trailers or rail cars for transport to the mills. Rail cars carry 25-30 tons each. Highway trailers carry 20 tons per load.

How is the Florida sugar industry organized?

All commercially produced Florida sugarcane is destined for one of four sugarcane mills in South Florida. Corporations that grow their own sugarcane and extract the sugar in their own mills produce more than 65 percent of the cane. Independent growers for grower-owned cooperative mills produce another large portion, 25-30 percent. The remaining cane is grown by independent producers and sold to one of the four mills in South Florida.

What happens to the cane at the mills?

At the mill, the sugarcane is crushed between heavy rollers to squeeze out the juice. Small amounts of hot water are then added, and the fiber is again squeezed to remove as much juice as possible. This process is repeated several more times to maximize sugar extraction from the crushed cane stalks.

Lime is then added to the juice to help filter out cane fibers and soil particles and to prevent conversion of the desirable sugar (sucrose) into other sugar forms that won't crystallize. This sucrose solution is concentrated by evaporating off the water (which is also recovered and reused by the mill) until raw sugar crystals form.

For each pound of sugar produced, 6.5 pounds or 3 quarts of water must be boiled off. Because sugarcane is more than 50 percent water when harvested, enough water is recovered during sugarcane processing that sugar mills actually become producers of water, rather than consumers. Most of what appears as “smoke” emitted from sugar mills is actually water vapor, steam generated from boilers in the mill.

Raw sugar is a coarse, brownish material containing impurities that must be removed in a separate refining process.

What is bagasse? (“baa-gas”)

Bagasse is fibrous plant material that remains once all of the juice has been squeezed from the sugarcane stalk. Bagasse consists mostly of stalk fibers, but also contains leaves and other biomass components inadvertently brought to the mill by harvest trucks or railcars. Bagasse is about half water and half dry matter (plant material).

In Florida, most bagasse is burned as fuel for the mills. Some of the mills also burn excess bagasse to generate electricity for the public grid.

What is blackstrap molasses?

Blackstrap molasses is the dark, viscous liquid that remains when as much sucrose as practical has been removed as sugar crystals from boiled cane juice. Blackstrap molasses is used primarily as animal feed.

What is done with the raw sugar?

Raw sugar is stored in high piles in large warehouses to await shipment to a refinery. Having the consistency of very coarse sand, raw sugar is moved by belt conveyors, front-end loaders, or dump trucks to be loaded into large dump trucks, railcars, barges, or ships for transportation to refineries.

Are there sugar refineries in Florida?

Yes. Two refineries in South Florida produce both granulated sugar and liquid sugar. About 65 percent of the raw sugar produced in Florida is processed in these refineries. One refinery is in South Bay, Fla. and the other refinery is in Clewiston, Fla.

The granulated sugar is packaged in consumer-size bags. Liquid sugar is shipped in tanker trucks for use in beverages, processed foods, and bakery goods.

Can members of the public visit commercial sugar mills and refineries in Florida?

Sugarcane milling is an industrial process and, as such, is dusty, noisy, and potentially dangerous to non-trained personnel. However, special interest group tours are available during the milling season at selected mills. Tours must be arranged in advance.

How much sugar is there in one stalk of Florida sugarcane?

An average sugarcane stalk weighs about 3 pounds (1.4 kilograms) and is roughly 85 percent liquid. An average stalk, therefore, has about 2.6 pounds (1.2 kilograms) of juice, which is roughly 11 percent sugar by weight. Thus, an average stalk contains about 0.3 pounds (0.14 kilograms) of sugar.

Which is better, cane sugar or beet sugar?

Cane sugar and beet sugar are identical in chemistry and quality. Sugar beets, which provide roughly 25 percent of the world's sugar, are grown in temperate areas. Sugarcane is grown primarily in the tropics and sub-tropics.

How much sugarcane is required to supply one American with sugar for one year?

In 2008 the average refined sucrose sugar consumption in the United States was approximately 66.3 lbs. per person per year. This total could be produced from roughly 221 stalks of sugarcane. At 30,000 stalks per acre, one acre would supply sugar for roughly 137 Americans for a year.

In 2004, per capita consumption in the United States was 61.5 pounds of refined sugar, 78.1 pounds of corn-derived sweeteners, and 1.3 pounds of honey and edible syrups, for an annual total caloric-sweetener consumption of roughly 141.0 pounds. By 2008 per capita sugar consumption had increased slightly to 66.3 pounds while per capita use of corn-derived sweeteners had dropped slightly to 69.3 pounds.
http://www.ers.usda.gov/Briefing/Sugar/data/table50.xls

Per capita sugar consumption has decreased from about 100 pounds in the early 1970s to 66.3 pounds in 2008. Consumption of high fructose corn syrup (HFCS) has increased from two pounds per capita in the early 1970s to 69.3 pounds in 2008.

What determines the price of sugar?

Neither Florida nor the United States exports sugar, so changes in the Florida crop have little effect on world sugar prices. Worldwide, individual government tariffs and trade policies play a paramount role in determining sugar prices.

World demand for sugar is rather constant and, under normal growing conditions, production of sugar matches the demand, resulting in stable prices for raw sugar. However, good growing conditions sometimes result in higher-than-normal sugar yields in the major sugar beet and sugarcane producing areas of the world – Brazil, India, and the European Union.

When surplus quantities of sugar enter the world market, surplus sugar can be purchased well below the normal market price. However, to prevent unpredictable disruptions to the U.S. sugar industry, the federal government annually adjusts import quotas on foreign sugar. These quotas help insulate U.S. consumers, as well as sugar producers, from the sometimes rapidly fluctuating sugar prices on the world market.

APPENDIX E

EVIDENCE BASED PUBLIC HEALTH POLICY AND PRACTICE

Air pollution from biomass burning and asthma hospital admissions in a sugar cane plantation area in Brazil

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Objective: To evaluate the association between the total suspended particles (TSPs) generated from preharvest sugar cane burning and hospital admission due to asthma (asthma hospital admissions) in the city of Araraquara.

Design: An ecological time-series study. Total daily records of asthma hospital admissions (ICD 10th J15) were obtained from one of the main hospitals in Araraquara, São Paulo State, Brazil, from 23 March 2003 to 27 July 2004. The daily concentration of TSP ($\mu g/m^3$) was obtained using Handi-vol equipment (Energética, Brazil) placed in downtown Araraquara. The local airport provided the daily mean figures of temperature and humidity. The daily number of asthma hospital admissions was considered as the dependent variable in Poisson's regression models and the daily concentration of TSP was considered the independent variable. The generalised linear model with natural cubic spline was adopted to control for long-time trend. Linear terms were used for weather variables.

Results: TSP had an acute effect on asthma admissions, starting 1 day after TSP concentrations increased and remaining almost unchanged for the next four days. A 10 $\mu g/m^3$ increase in the 5-day moving average (lag1–5) of TSP concentrations was associated with an increase of 11.6% (95% CI 5.4 to 17.7) in asthma hospital admissions.

Conclusion: Increases in TSP concentrations were definitely associated with asthma hospital admissions in Araraquara and, despite using sugar cane alcohol to reduce air pollution from automotive sources in large Brazilian urban centres, the cities where sugar cane is harvested pay a high toll in terms of public health.

Epidemiological studies on asthma suggest that its prevalence and severity increase constantly in many countries.[1 2] In the US, from 1980 to 1994, the prevalence of asthma increased by 75%.[3] The tendency to develop asthma can be inherited, but genetic factors alone are unlikely to explain the increases seen over the past 20 years.[4] Environmental exposure is one of the many proposed reasons for these increases.[5]

In this respect, the most extensively studied environmental factor so far is air pollution, as it can increase the risk of asthma attacks through different mechanisms such as (1) a direct effect on sensitive airways, (2) a toxic effect on the respiratory epithelium, (3) the triggering of bronchial hyper-reactivity, both allergen-specific and non-specific or (4) a change in immune response by increasing susceptibility to an immunological trigger.[6 7]

An association between increased pulmonary morbidity, including asthma exacerbations, and particulate matter (PM) pollution has been noticed through observational and experimental studies in various countries.[8 9] A report from the Committee of the Environmental and Occupational Health Assembly of the American Thoracic Society[10] suggests that each 10 $\mu g/m^3$ increase in the ambient concentration of inhalable particulates is associated with a 3% increase in asthma exacerbations.

Biomass burning is a major contributor to toxic and greenhouse gases, as well as the build-up of suspended particles throughout the world, thus resulting, in most cases, in exposure to high levels of air pollution.[11]

The world oil crisis of the 1970s led Brazil to look for an alternative, locally produced, renewable fuel source as a replacement for gasoline. Being the world's biggest producer of sugar cane, ethanol derived from that crop was the obvious choice. Today, 70% of the country's sugar cane is burnt before being harvested manually for ethanol production.

Biomass fuels are seen as cleaner alternatives to oil. The air quality in major Brazilian urban centres has improved since the decision to use ethanol as the automotive fuel was taken.[12] Notwithstanding this, the burning of the sugar cane crop produces large amounts of particles and toxic gases that affect the entire population of the nearby settlements for at least 6 months of every year. A study has shown that burning is the major source of fine and ultrafine particles in sugar cane producing areas during the crop-burning periods.[13] The World Health Organization[11] suggests that vegetation fires produce adverse respiratory health effects, including an increase in the number of emergency room visits and hospitalisation events. Sadly, in the city of Araraquara, with 192 000 inhabitants, sugar cane burning is already associated with an increase in emergency room visits for inhalation therapy.[14]

This being so, this study was designed to evaluate the association between total suspended particles (TSPs) generated from the burning of the sugar cane crop and the increase in hospital admissions due to asthma (asthma hospital admissions) in the city of Araraquara.

METHODS

This is an ecological time-series study using secondary health data. Total daily records of asthma hospital admissions (ICD 10th J45) were obtained from one of the main hospitals in the

Abbreviation: TSP, total suspended particles

Table 1 Descriptive analysis of the main variables used in the study

Variables	Mean (SD)	Minimum	Maximum
Asthma (HA)*			
Total	1.3 (1.3)	0	7
Burning*	1.5 (1.4)	0	7
Non-burning	0.96 (1.2)	0	4
TSP (μg/m^3)			
Total	46.8 (26.4)	6.7	137.8
Burning*	56.86 (25.07)	8.1	137.8
Non-burning	28.45 (17.53)	6.7	99.9
Temperature (°C)†	16 (3.5)	2	26
Humidity (%)‡	62 (14.5)	20.5	93.3

*Hospital admission (HA); different from non-burning period ($p<0.001$, Mann–Whitney U test).
†Minimum temperature.
‡Mean relative humidity.

city of Araraquara, São Paulo State, Brazil, between 23 March 2003 and 27 July 2004. Asthma hospitalisation is herein defined as an admission in which asthma was the primary diagnosis.

Of the 493 days considered for the study, sugar cane was burnt on 318 days. Daily TSP concentration (μg/m^3) data were obtained through the use of a Handi-vol sampler (Energética, Brazil), with fibreglass filtering elements, and a certified flow rate of 3 litres/min. The equipment was placed in downtown Araraquara under a canopy to protect it from the rain. Filters were weighed before and after each 24 h period of particle collection. The daily concentration of TSP in micrograms per cubic metre of air was estimated using the following equation:

$$PC = \frac{\Delta M}{TV_{24h}}$$

where PC is the TSP concentration (μg/m^3) under standard conditions of temperature and barometric pressure, ΔM is the mass difference in filter weight (g) before and after use and TV_{24h} is the total volume (m^3) of sampled air during each 24 h interval. The local airport authority provided the daily mean figures for temperature and humidity.

The Mann–Whitney U test was used to compare the mean TSP concentrations and the daily asthma hospital admissions during the burning and non-burning periods. The daily number of asthma hospital admissions was considered as the dependent variable in generalised linear Poisson regression models,[15] and the TSP concentration was considered the independent variable. Natural cubic spline[16] was used to control for long-time trend. We used 5 degrees of freedom to smooth the time trend. The number of degrees of freedom for the natural spline of the time trend was selected to minimise the autocorrelation between the residuals and the Akaike Information Criterion.[17] After adjusting for the time trend, no remaining serial correlation was found in the residuals and therefore the use of autoregressive terms was not necessary.

Araraquara is a warm city, and previous analysis has shown that local temperatures present a linear effect on respiratory diseases.[14] Hence, we decided to use linear terms (lags 0 and 1) to control for the effect of temperature and humidity on asthma hospital admissions. Moreover, an indicator for days of the week was included as an additional means of control for short-term seasonality.

The elapse of time between an increase in air pollution and its effect on health has been analysed using different approaches. This study used the lag structure from 0 to 9 days before hospital admission using a fourth degree polynomially distributed lag model,[18] which imposes some restraint but allows enough flexibility in the estimation of a biologically plausible lag structure, thereby better controlling multicollinearity than an unconstrained lag model would do. The standard errors of the estimates for each day were adjusted for overdispersion.

Having defined the lag structure for TSP, our models included lag 0 or moving averages to estimate the cumulative effects of the pollutant in the adopted outcome. Also, to assess potential differences in TSP effects between burning and non-burning periods, a model was constructed using an interaction term for period.

To explore the presence of thresholds in TSP effect, additional analyses were conducted using categories (quintiles) for daily concentrations, instead of using a continuous variable. The effect of air pollution was presented as a percentage increase or relative risk and 95% CI in asthma hospital admission owing to a 10 μg/m^3 increase in TSP. The statistical analyses were performed in SPSS V.11 and Splus 4.5.

RESULTS

Table 1 shows the descriptive analysis of the variables used in the study for the entire period, and for the burning and non-burning periods. Mean TSP concentration remained below the air quality standard during the burning period and despite this, the mean TSP value was twice that observed during non-burning periods ($p<0.001$).

Asthma admissions varied substantially between the two periods. There were 640 asthma hospital admissions during the 493 days of the study. As observed for TSP concentrations, during the burning period (318 days) asthma hospital admissions were 50% higher than those observed during the 175 days of the non-burning period (477 and 163 asthma hospital admissions, respectively; $p<0.001$).

Weather variables showed that Araraquara is located in a region with moderate temperature and very dry days during winter.

Figure 1 shows daily TSP concentrations and asthma hospital admissions within the study period. Both variables increased during the burning period and in 2004, they seem to be more closely correlated than in 2003.

Asthma hospital admissions showed a low but positive and statistically significant correlation with TSP concentrations (Pearson's correlation coefficient: 0.13; $p<0.001$) but the same was not observed for weather variables.

Figure 2 shows the estimated lag structure for the effect of TSP on total asthma hospital admissions using a polynomial distributed lag model. There was a lagged effect starting one day after TSP concentrations increased, which remained almost unchanged until the fifth day after exposure. Other estimates were done for longer periods (up to 14 days after exposure). However, there was no statistically significant effect after day 5.

Once the lag structure was identified, we used moving averages ranging from 2 to 7 days to estimate the cumulative effects of TSP on asthma hospital admissions (table 2). Because effects were positive from lag 0 to lag 7 (in the polynomial distributed lag models), an increase in the cumulative effect until the 6-day moving average is totally plausible.

The 7day cumulative effect of a 10 μg/m^3 increase in TSP concentrations on asthma hospital admissions represented twice the effect observed on the day of the increase and the day after (table 2). When lags 0 and 6 were excluded from initial effect estimates, the cumulative effect from lag 1 to lag 5 reached almost 12%. When the analyses were stratified to non-burning and burning periods, it was observed that for the same variation of 10 μg/m^3 in TSP concentration, asthma hospital admissions increased by 9.7% (95% CI 2.6 to 17.2) and 12.7% (95% CI 2.2 to 24.3), respectively.

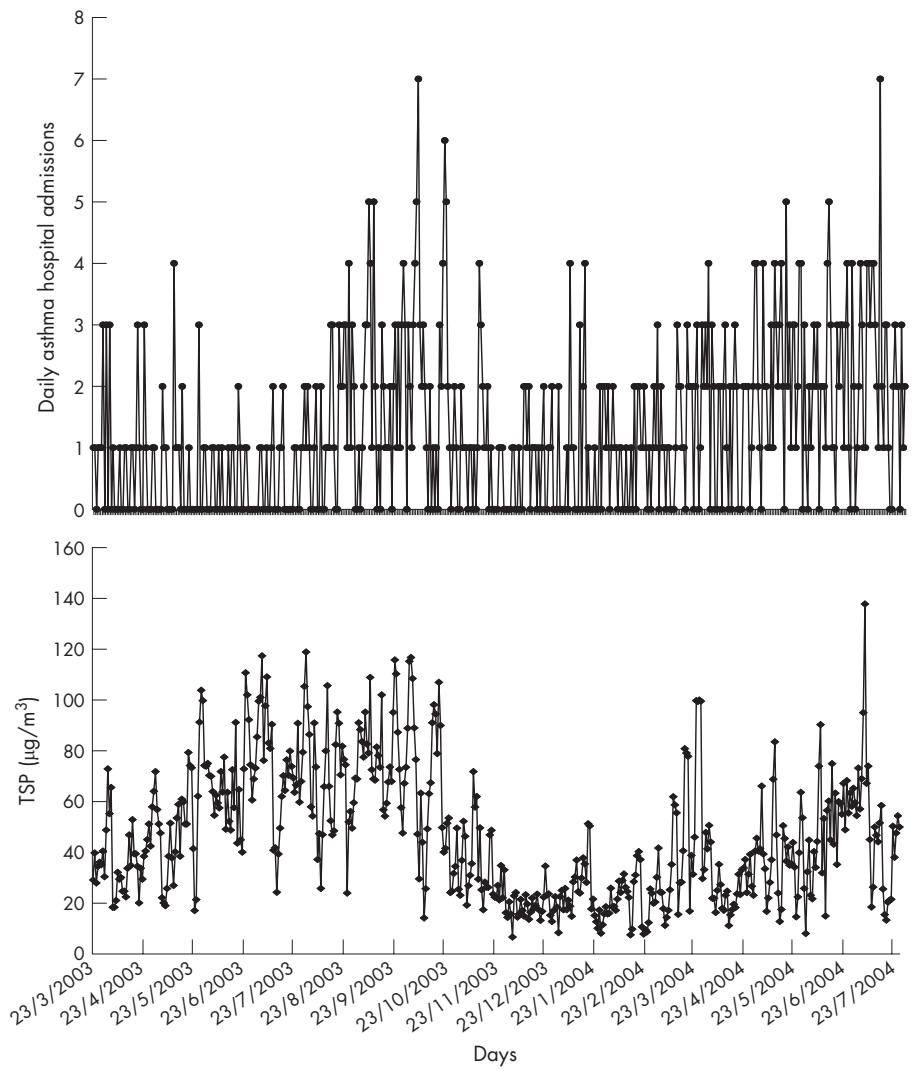

Figure 1 Daily number of asthma hospital admissions and daily concentrations of total suspended particles (TSP).

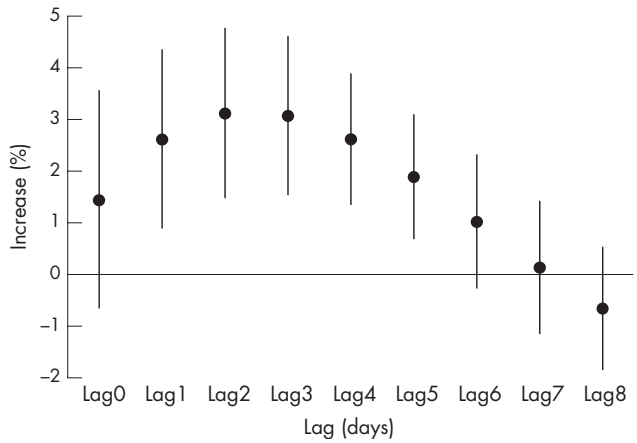

Figure 2 Percentage increases and 95% CI in asthma hospital admissions in the concurrent and six subsequent days following a 10 μg/m^3 increase in total suspended particle concentrations.

Models including categories (quintiles) of TSP moving averages with different ranges instead of continuous variables were also considered. Table 3 shows the results for the quintiles of the 5-day moving average (lag 1–lag 5).

As observed for the moving averages actually adopted in the study (tables 1 and 2), as well as for the one used in table 3 (5-day moving average one day lagged), the relative risk of asthma admissions had a dose–response relationship with the pollutant, without any trace of threshold.

Table 2 Percentage increases and 95% CIs for asthma hospital admissions using moving averages of total suspended particle concentrations

TSP moving average	Percentage increase (95% CI)
2-day (lag0–lag1)	6.96 (1.4 to 12.86)
3-day (lag0–lag2)	9.09 (3.12 to 15.40)
4-day (lag0–lag3)	10.28 (4.05 to 16.90)
5-day (lag0–lag4)	11.63 (5.46 to 19.18)
5-day (lag1–lag5)	11.63 (5.42 to 17.73)
6-day (lag0–lag5)	12.61 (5.68 to 20.00)
7-day (lag0–lag6)	12.56 (5.47 to 20.13)

TSP, total suspended particles.

Table 3 Relative risk (RR) and 95% CIs of the total suspended particles 5-day moving average (lag 1–lag 5) quintiles

TSP moving average	RR (95% CI)
5-day (lag1–lag5)(μg/m^3)	
9.25–28.45	1.00
28.46–48.85	1.55 (0.45 to 5.77)
48.86–69.06	2.46 (1.08 to 5.60)
69.07–88.44	2.77 (1.32 to 5.84)
88.45–108.91	2.94 (1.48 to 5.85)

TSP, total suspended particles.

DISCUSSION

This study shows that in a region with characteristics similar to those of Araraquara, where periodic preharvest biomass burning is common practice, TSP concentrations and asthma hospital admissions are very likely to be higher during the burning periods. Moreover, there is a statistically significant association between asthma hospital admissions and TSP concentrations. Also, the effect was acute, occurred 1 day after the increase in TSP concentrations, and remained for almost a week. These results show almost the same lag structure seen for respiratory diseases in our other studies.[14 19 20]

The all-ages approach used in this study, as opposed to stratified age group analysis of asthma hospital admissions, was taken because, as shown in the descriptive analysis above, these are not common events and stratifying by age could possibly decrease, distort or even hide the true picture. Instead, working with the whole spectrum of patients with asthma may provide a much closer estimate of the effect of TSP generated from burning sugar cane in a specific community.

Health data were collected from the city's main hospital and the precision of asthma diagnosis was assured by its permanent, well-trained staff. Moreover, the diagnosis of all patients admitted to hospital is reviewed by a senior physician. In Brazil, respiratory diseases, including asthma, are more prevalent throughout the country during autumn and winter, both in large urban centres where air pollution is generated mainly by fossil fuel combustion, and in areas where the main source of pollutants is biomass burning. Weather and seasonality have been recognised as confounders of the association between air pollution and respiratory disease, and effective control for these confounding factors has been matter of concern among environmental epidemiologists. This study uses different sets of controls for weather and season, which are the standard approaches and have been extensively used by other authors.[14 19 21] In a recent study[20] we used the method of stratification by burning/non-burning periods. In it, we found a remarkable difference in the total number of hospital admission for respiratory ailments in each of these periods. In the present study, we used an interaction term for period and the results showed at least a 30% greater effect during the burning period.

Our statistical analyses comprised a widely adopted time-series method that is robust and less sensitive to effect estimate bias.[19 20] Some authors have recently chosen to use case-crossover analysis to estimate the acute effects of air pollution. Results, however, have shown that the appropriate use of either case-crossover or time-series techniques produces similar results for estimates.[22] Hence, the adoption of generalised linear models in our study cannot be considered a weakness.

Some recent studies on the adverse health effects derived from particulate matter have looked at fine instead of coarse particle or TSP data. This was not possible in this study as the region has no monitoring stations and PM_{10} measurements are rarely conducted by the São Paulo State Environmental Agency (CETESB) and, when conducted, are of short duration. To be adequately performed, a time-series analysis requires a long period of regular data collection for both exposure and outcome. This being so, we considered that using daily TSP concentrations instead would be a better choice. Moreover, previous studies have shown that most particles emitted during biomass burning fall into the ultrafine category (<0.1 μm).[11] The relationship between particulate matter with diameter <10 μm and total particulate emission in agricultural burning emissions is approximately 90%.[23] Ward *et al*[24] found that total particulate matter emitted by flaming-combustion forest fires is composed of 80–95% fine particles ($PM_{2.5}$). Therefore it is reasonable to assume that daily TSP variations can be used as a proxy of daily variation of fine and ultrafine particles, which are widely recognised as inducing damage in the airways of patients with asthma.[5]

Several toxicological studies have shown that particles can cause inflammatory reactions both in vivo and in vitro. Particles collected directly from the natural environment cause inflammatory reactions in rat lungs, rat cell lines and human cell lines.[25–27] Free radical activity or the oxidative capacity of particulate matter plays an essential role in provoking these inflammatory responses. It has also been suggested that the inflammatory properties of ultrafine particles are mediated by their large number, small size and high penetration rate into the interstitium, independently of their chemical composition.[28]

Patients with pre-existing respiratory or cardiac illnesses seem to be at particular risk of the most severe adverse health effects on exposure to inhalable particles, including those with asthma. Previous studies from several countries have shown a relationship between hospital admissions for asthma and fossil-fuel-generated particulate matter, independently of the size of the particles and the age of the population.[29-36] Although there are very few epidemiological studies on this subject, it is possible to subdivide them into three groups: (1) residential wood burning, (2) agricultural residuals and (3) forest fires. Lipsett *et al*[37] working in Santa Clara County showed a 6% increase in asthma-related emergency room visits after increases of 10 μg/m^3 in ambient wintertime PM_{10}. In this case the main source of PM_{10} was residential wood smoke. In 1993, wood burning was found to be the dominant source of PM_{10} pollution in Seattle, ranging from 60% in summer to 90% in winter. Schwartz *et al*[38] found a 3.7% increase in emergency room visits for every 10 μg/m^3 rise in PM_{10}, and the positive association remained for an average lag of 0–4 days, suggesting a delayed response. Norris *et al*,[39] again in Seattle, found that for every 11 μg/m^3 increase in PM_{10}, there was a 1.15% increase in the relative risk of asthma-related emergency room visits. Also in Seattle, Sheppard *et al*[40] showed an association among PM, PM_{10} and $PM_{2.5}$, and asthma hospital admissions in patients aged <65 years, with a 1 day lag.

Long *et al*[41] evaluated 428 subjects surveyed for their respiratory symptoms following exposure to combustion emissions from agricultural burning (straw and stubble). During the 24 h period of exposure, average PM_{10} concentrations increased from 15–40 μg/m^3 to 80–110 μg/m^3; 63% of those surveyed experienced worsening symptoms owing to the air pollution. Subjects with asthma and chronic bronchitis were most affected.

Forest fires are usually brief events in which particulate matter reaches extremely high concentrations and a concomitant increase in asthma-related emergency room visits has been observed.[42 43] Occasionally, forest fires are long lasting such as the one in Southeast Asia, which lasted from July to October 1997. During this period asthma-related emergency room visits and hospital admissions in many countries in the region were higher than usual.[11] A study performed at that time on young

What is already known

- Indoor biomass burning is strongly associated with high exposure to particulate matter and this situation leads to increases in adverse health effects.
- Episodic vegetation fire is also associated with adverse health effects.
- Some regions face regular episodes of biomass burning due to agricultural activities and with lower concentration of particulate matter. Sugar cane preharvest burning was already associated with increases in emergency room visits and hospital admissions, mainly in elderly and children.

What this paper adds

- This is one of the few available manuscripts analysing the adverse health effects of regular biomass burning in communities surrounded by sugar cane plantations.
- Most of the existing studies on biomass burning deal just with episodic events in which there was a very high concentration of particulate matter.
- This study shows that, in urban centres, the effects of particles produced by burning biomass rival those produced by the burning of fossil fuel.

Policy implications

- The population and authorities have devoted much more of their attention to analysing the effects of air pollution generated from fossil fuel combustion than evaluating air pollution caused by biomass burning, which they have associated mainly with wood burning or episodic events such as forest fires.
- If alcohol is to be considered an alternative to fossil fuel, it is important to show that cities surrounded by sugar cane plantations where preharvest burning is routine practice have been paying an extremely high price in terms of adverse public health.
- We also recommend that mechanised harvesting is introduced as soon as possible and that biomass burning is banned to balance better the advantages and disadvantages of producing fuel alcohol.

and healthy military recruits in Singapore showed an association between air pollution and the increase in peripheral blood cell counts,[44] linking this specific kind of air pollution with systemic inflammatory responses.

Johnston *et al*[45] conducted a time-series ecological study in Darwin, Australia, during a bushfire (7 months) in 2000, and showed a significant increase in asthma hospital admissions owing to increases in PM_{10} concentrations. However, the effect was not linear, only occurring on days with PM_{10} concentrations above 40 $\mu g/m^3$.

All of these studies reported episodic events showing adverse respiratory effects associated with relatively brief haze episodes. In the cities surrounded by the sugar cane plantations, exposure to air pollution generated by biomass burning is continuous for at least 6 months each year but particle concentrations are lower, and the effects were very similar to those observed in other regions with episodic events. Analysis of specific toxicity or of the elemental composition of the particles is required to shed more light on the size of the effect.

The main concern about alcohol production is the need to burn the sugar cane before manual harvesting, a process that reduces the cost of production. To minimise adverse effects from this pollution, researchers and doctors have recommended that the population avoid overexposure during burning periods and restrict physical activity. The weather forecast also provides information on weather quality and pollutant dispersion, allowing people to plan their activities. São Paulo state is gradually implementing mechanised harvesting, with the aim to be fully mechanised by 2031.[46] However, we have concerns about the length of time required to mechanise given the adverse health effects of the burning process.

Biomass burning is not just a Brazilian problem and has been a common practice in other countries. In Brazil, however, the problem is more acute as the country is the biggest producer of sugar and alcohol in the world. The problems faced in Brazil from this specific kind of air pollution may help to consolidate the relevance of biomass burning as a public health issue, leading to more restrictive policies in Brazil and other countries planning to implement alcohol-based fuel programmes.

In summary, increased TSP concentrations were associated with asthma hospital admissions in Araraquara and, despite the benefits of alcohol produced from sugar cane in reducing air pollution from automotive sources in large Brazilian urban centres, the cities where sugar cane is harvested have paid a high price in terms of public health. We recommend the banning of preharvest sugar cane burning to balance better the advantages and disadvantages of producing fuel alcohol.

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APPENDIX F

Emissions generated by sugarcane burning promote genotoxicity in rural workers: a case study in Barretos, Brazil

ENVIRONMENTAL HEALTH

RESEARCH Open Access

Emissions generated by sugarcane burning promote genotoxicity in rural workers: a case study in Barretos, Brazil

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Abstract

Background: To determine the possible genotoxic effect of exposure to the smoke generated by biomass burning on workers involved in manual sugar cane harvesting.

Methods: The frequency of micronuclei in exfoliated buccal cells and peripheral blood lymphocytes was determined in sugarcane workers in the Barretos region of Brazil, during the harvest season and compared to a control population, comprised of administrative employees of Barretos Cancer Hospital.

Results: The frequency of micronuclei was higher in the sugar cane workers. The mean frequency in blood lymphocytes (micronuclei/1000 cells) in the test group was 8.22 versus 1.27 in the control group. The same effect was observed when exfoliated buccal cells were considered (22.75 and 9.70 micronuclei/1000 cells for sugar cane workers and controls, respectively).

Conclusion: Exposure to emissions produced by the burning of sugar cane during harvesting induces genomic instability in workers, indicating the necessity of adopting more advanced techniques of harvesting sugar cane to preserve human health.

Keywords: Sugar cane workers, Micronuclei, Genomic instability, Human lymphocytes, Exfoliated buccal cells

Background

Biofuels have been considered a cleaner and more sustainable alternative compared to fossil fuels. To reduce the emissions of greenhouse gases as well as local pollutants, several countries are including biofuels in their energy policies. Sugar cane ethanol is one of the most widely used sources of ethanol in Brazil, which has run a program for using ethanol as an automotive fuel since the mid-1970s. Thus, Brazil contains 25% of the total land worldwide that is planted with sugarcane. The state of São Paulo is the largest producer of this crop; between 2001 and 2011, the production of sugarcane grew 121% due to the use of biofuels in cars.

When projecting a future scenario with an increased production, it is important to focus on the health effects associated with the different steps of sugar cane production. Currently, the sugarcane harvest is associated with straw burning for reasons of productivity as well as to avoid contact of workers with the sharp leaves and poisonous animals in the sugar cane plantation. However, the burning process results in a high exposure to smoke, which is still present during harvest.

The environmental impact due to the burning of sugar cane has diminished with the implementation of legislation to suspend the use of burning to use mechanized harvesting as an alternative in São Paulo [1]. These laws have not been extended to the rest of Brazil, where sugarcane plantations are expanding, and in the Central America and Africa, where the burning of sugar cane is still practiced [2].

Biomass burning is a major source of toxic gases [3]. Several products are generated during this process that result in adverse effects on the health of the exposed population [4], such as particulate matter, polycyclic

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aromatic hydrocarbons (PAHs), carbon monoxide, aldehydes, organic acids, volatile and semi-volatile compounds of nitrogen and sulfur, ozone and inorganic chemical species. Taking into account the health effects associated with sugar cane burning, it is important to focus on fine and ultrafine particulate matter (PM10 and PM2.5), which consists of a mixture of liquids, gases and solids deposited on particles such as PAHs, which are derived from an incomplete organic combustion process [5]. PAHs are pollutants that cause mutagenic and carcinogenic effects [6,7]. In the Brazilian city of Araraquara (an area with high production of sugar cane in São Paulo State), a significant increase in PAHs, especially the benzopyrene fractions of particulate matter (PM10 and PM2.5), occurs during the sugar cane harvest [7]. Populations in areas surrounded by sugarcane plantations are exposed to the particles produced by biomass burning continuously for at least six months to a year, and increased hospital admissions due to asthma occur during these periods [8,9].

Sugar cane workers are exposed to high levels of particulate matter, thermal overload, and intense physical exertion during the harvest period, and these conditions induce muscle lesions, changes in blood coagulation and heart rate, systemic oxidative stress, and high blood pressure [10]. Sugarcane products have been shown to cause respiratory problems in workers [8]. Furthermore, sugar cane workers are exposed to various genotoxic compounds, including PAHs [11]. Considering the aforementioned evidence, it is important to evaluate genomic instability in sugarcane workers. Micronucleus (MN) assessment is a biomarker test of genotoxic events and manifestations of chromosomal instability that are frequently observed in diseases such as cancer and can thus evaluate the potential risk of disease in exposed populations [12]. The MN test in blood lymphocytes and buccal mucosal cells is widely used to assess the extension of chromosomal genetic alterations promoted by the exposure to environmental toxicants [13-15]. The aim of the present study was to evaluate the MN frequencies in blood lymphocytes and exfoliated buccal cells of sugar cane workers during the harvest season.

Methods

Study groups

The present study was approved by the Barretos Cancer Hospital ethical committee (n° 361/2010), and all participants signed an informed consent prior to any intervention.

The study population consisted of a group of male sugarcane workers (n = 23) in the Barretos region, and the control group (n = 30) was taken from the administrative staff of Barretos Cancer Hospital. The age of the subjects under study ranged from 22 to 45 years. Every participant answered a questionnaire including general information and occupational data, such as previous occupational background, working time in the current job and past and previous smoking habits.

Determination of micronucleus frequency in blood lymphocytes

Blood was collected in vacuum tubes (BD TriPath Imaging, Burlington, N.C., USA). The samples were transported carefully to avoid heat and agitation. Blood lymphocytes were isolated with FicollPaque (Invitrogen). The cultures were grown in RPMI medium at 37°C for 72 hours, and phytohemagglutinin was added (Lifetechnologies). After 44 hours, citochalasin B (Sigma) was added to a final concentration of 6 μg/mL following [16]. Then, to resuspend the cells after cultivation for 72 hours, SurePath preservative liquid (BD TriPath Imaging, Burlington, N.C., USA) was used with Liquid-based cytology medium (BD TriPath Imaging, Burlington, N.C., USA), to ensure a single layer of cells with excellent smear quality. This fixative solution is a mixture of ethanol, methanol and isopropyl alcohol. One ml of this solution was transferred to an incubation chamber mounted on a support (BD TriPath Imaging, Burlington, N.C., USA) consisting of a PrepStain Settling Chamber (BD TriPath Imaging, Burlington, N.C., USA) placed on the top of a glass slide. A cell suspension from each sample was dropped onto a clean glass slide. After one hour, the slides were stained automatically, as specified by the manufacturer. The slides were previously encoded and analyzed under an optical microscope at a magnification of 1000X. One thousand cells were counted per sample. The MN frequencies were expressed as MN per thousand cells. All experiments were carried out in duplicate.

Determination of micronucleus frequency in exfoliated buccal cells

Exfoliated buccal cells were sampled with the assistance of a cytobrush (BD TriPath Imaging, Burlington, N.C., USA), which was stirred in Surepath preservative liquid (BD TriPath Imaging, Burlington, N.C., USA). The material was centrifuged at 1500 rpm for 10 minutes, and the supernatant was discarded, thus obtaining a precipitate with a high concentration of cells. Smears were produced and stained with Papanicolaou staining. Then, the cells were dehydrated in ethanol and clarified in xylene. The slides were mounted with coverslips. Then, 1000 cells were counted, and the micronucleus frequencies in both groups were determined. The slides were stained by Papanicolaou staining, 1000 cells per sample were counted, and the MN frequencies were determined. The cells were counted under an optical microscope using a magnification of 1000X.

Table 1 Demographic characteristics of the subjects

	Controls	Sugarcane cutters
Individuals (N)	30	23
Mean age (± SD*)	30.50 (5.5)	31.00 (6.7)
Work hours, mean (± SD)	46.33 (8.3)	51.39 (3.9)
N** of Smokers (± SD)	7	5
N** Ex-smokers	1	0
Mean age for beginning cigarette smoking (± DP)	17.00 (2.0)	16.40 (2.3)
N Risk of inhalation of harmful substances*** (in earlier jobs)	9	-
N Medication (Current)	30	21
No (%)	26 (86.7)	18 (85.8)
Yes (%)****	4 (13.3)	3 (14.2)
N Diseases related to work	30	23
Yes (%)*****	1 (3.3)	1 (4.3)
No (%)	29 (96.7)	22 (95.7)

SD* = Standard Deviation; N** = Number of subjects; ***Harmful substances: dust, smoke, gases and chemicals; ****Anti-inflammatory, anti-allergic, anti-muscarinic, antihypertensive and analgesic; *****1 control with wrist synovial tendinitis and 1 sugar cane cutter with muscle aches and cramps.
(–) Not applied to the cutter group.

Statistical analysis

Statistical analysis was conducted with the aid of SPSS software for Windows version 19.0. Initially, the data were tabulated based on descriptive statistics (mean, standard deviation, minimum, maximum and quartiles) for quantitative data and frequency tables for qualitative data. The mean MN frequencies in lymphocytes and exfoliated buccal cells in the control group and the sugarcane workers' group were compared with Student's t-test, for which the samples were considered independent groups. A p-value < 0.05 was considered statistically significant in all analyses.

Results

The characteristics of the groups are presented in Table 1. The age and smoking habits were similar in the exposed and control groups. Only one individual from each group had a history of occupation-related disease. Four sugar cane workers and three controls were using medications that are not associated with genotoxic effects (Table 1).

A total of 51,000 binucleated lymphocytes were analyzed for the verification of micronuclei. The MN frequencies in peripheral lymphocytes (micronuclei/1000 cells and Additional file 1) were higher ($p < 0.001$) in the sugar cane workers (mean = 8.22, SD = 4.18) compared with the control group (mean = 1.27, SD = 1.34), as shown in Table 2.

Adequate smears of exfoliated buccal cells were possible in 43 individuals (16 sugar cane workers and 27 controls) Additional file 1. Again, a higher micronucleus frequency in exfoliated cells was obtained in the group of sugar cane cutters (mean = 22.75, SD = 5.78) compared with the controls (mean = 9.70, SD = 4.76), with a statistically significant p-value <0.001 (Table 2). The distribution of the groups according to their MN frequencies is shown in Figure 1.

The MN frequency in exfoliated buccal cells was higher in comparison to that determined in pair-wise peripheral blood lymphocytes (the mean difference in the control and cutter groups was −8.59 and −13.87, respectively, p-value <0.001), as shown in Table 3.

Additionally, the Curve Receiver Operating Characteristics (ROC) curves were generated to evaluate the sensitivity and specificity of the two methods (peripheral lymphocytes and exfoliated mucosal cells) in the characterization of the controls and sugar cane workers. Thus, the respective areas of the ROC curves were 0.969 for the MN frequency in lymphocytes and 0.973 for the MN frequencies in exfoliated buccal cells (Figure 2).

Discussion

The results obtained in the present study revealed that workers exposed to emissions generated by sugar cane burning exhibit a higher micronucleus frequency in peripheral blood lymphocytes and buccal mucosa exfoliated cells. Indeed, because the harvesting process begins when smoke emissions are still present, these workers are exposed to a high level of air pollution during intense physical activity (due to the intense effort required for manual cutting and harvesting), which demands

Table 2 Comparison of the MN frequencies by Student's t-test, considering independent groups

					Confidence interval		
		Mean	SD	DM	Lower	Upper	*p-value*
Lymphocytes	Control (N = 30)	1.27	1.34	−6.95	−8.58	−5.32	<0.001
	Cutters (N = 23)	8.22	4.18				
Buccal cells	Control (N = 27)	9.70	4.76	−13.05	−16.33	−9.76	<0.001
	Cutters (N = 16)	22.75	5.78				

N number of subjects per group considered in the analysis, *SD* Standard deviation, *DM* differences of the mean, considered statistically significant when p <0.05.

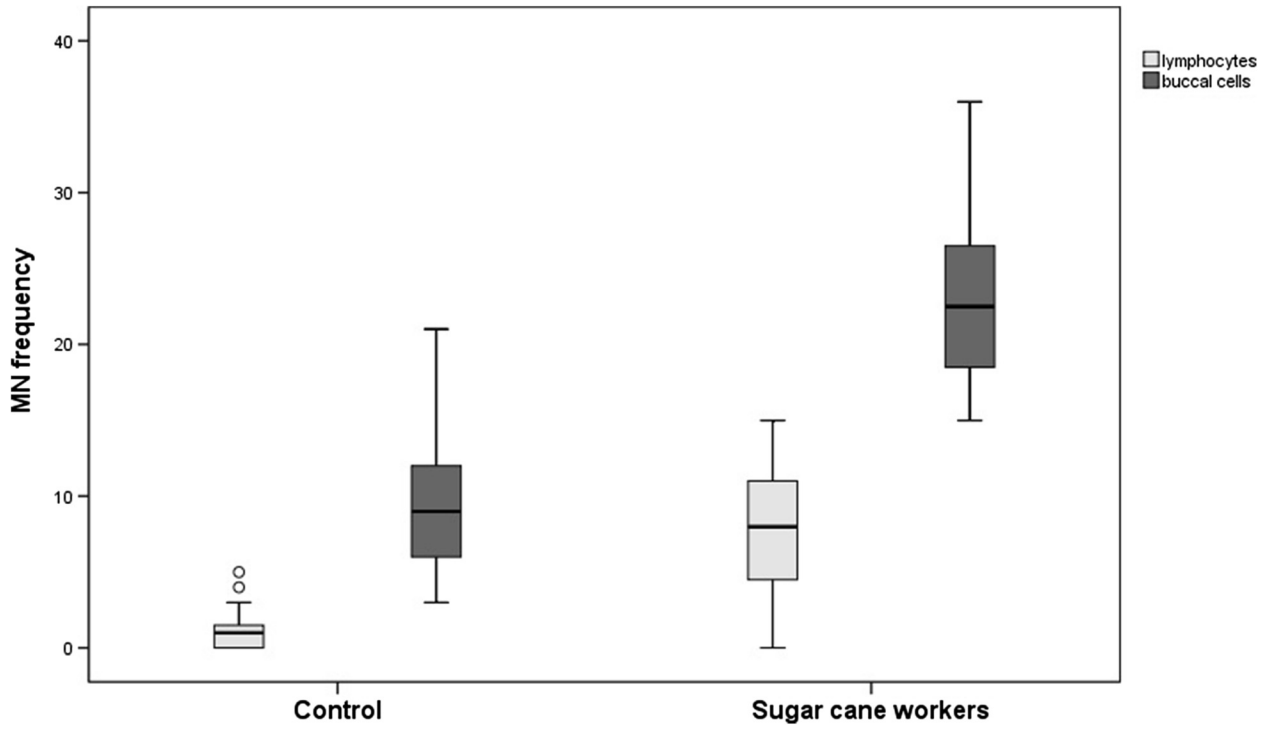

Figure 1 Graphic representation of distribution of both groups according to their MN frequency. The MN frequency in lymphocytes (light gray) and the MN frequencies of exfoliated buccal cells (dark gray) were considered for this analysis. (°) indicates discrepancy points, bars indicate percentile values, and lines indicate minimum and maximum standard deviations.

higher pulmonary ventilation [10]. Considering the two biomarkers used in our study, our results indicate that this group of workers developed a significant degree of genotoxicity as a result of compounds emitted by biomass burning. The results are in agreement with a previous study by our group in the Brazilian Amazon region, in which MN frequencies in exfoliated buccal cells of children were utilized to assess the genotoxicity potential of biomass burning pollutants [17].

In recent decades, biomarkers have been used to assess exposure to genotoxic agents, and the increase in these biomarkers, such as MN, in early cellular events is associated with changes related to diseases such as cancer. Assuming that the cellular mechanisms for the induction of chromosomal damage are similar in different tissues, the extension of DNA damage in lymphocytes and other tissues most likely reflects the level of cancer risk [12,18]. Furthermore, studies have shown that in Western countries, lung cancer is a major cause of mortality related to malignant tumors, and its pathogenesis involves the accumulation of various molecular abnormalities during long periods of time and has been associated with exposure to PAHs [19-21].

Recently, a comparison of different studies that assessed the MN frequency in blood lymphocytes and exfoliated buccal cells of the same individuals

Table 3 Analysis of mean MN frequencies versus MN frequencies of lymphocytes and MN frequencies of buccal cells

						Confidence interval		
		Mean	SD	DM	SD of difference	Lower	Upper	*p-value*
Control	Lymphocytes (N = 27)	1.11	1.28	−8.59	0.89	−10.43	−6.75	<0.001
	Buccal cells (N = 27)	9.70	4.76					
Cutters	Lymphocytes (N = 16)	8.87	4.05	−13.87	1.78	−17.68	−10.07	<0.001
	Buccal cells (N = 16)	22.75	5.78					

N number of subjects per group considered in the analysis, *SD* Standard Deviation, *DM* Differences of the mean, considered statistically significant when $p < 0.05$. We only considered both individuals for which the obtained information was collected in these analyses.

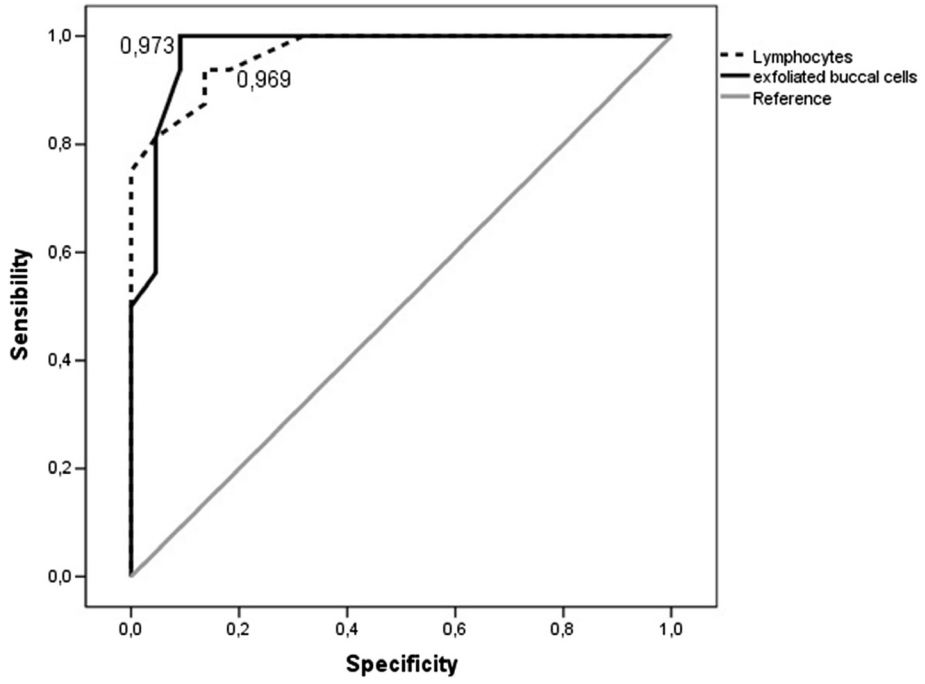

Figure 2 Graph representing of the ROC curve. Receiver operating characteristics curves of the MN frequency in lymphocytes (trace) and the MN frequency in cells exfoliated from the mouth (black). Reference line (gray). The proximity of curves to the upper left corner of the most accurate methods was analyzed.

simultaneously revealed a strong correlation between the MN frequencies in both tissues [22]. Thus, it is possible that measurements of MN frequency in oral epithelial cells can be potentially used as a screening alternative for larger studies focusing on cancer risk [23,24]. Thus, MN scoring can be used as a biomarker to identify several pre-neoplastic conditions much earlier than the clinical manifestations appear [25]. MN scoring in occupational groups reporting exposure to solvents, polycyclic aromatic hydrocarbons, gasoline, arsenic and antineoplastic drugs showed an increase in the MN frequency compared to the corresponding control group [12]. An increase in MN frequency is suggestive of but not diagnostic for cancer risk. However, MN frequency should be considered with respect to the detailed clinical history and examination [25] because MN frequency is the result of the balance between exposure to genotoxic agents and the genetic susceptibility of each individual [25]. Interestingly, previous studies have reported associations of MN frequency in peripheral blood lymphocytes not only with cancer risk [12,24] but also with cardiovascular diseases [26] and neurodegenerative disorders [27]. If these observations turn out to be valid, it is possible that MN frequency can be employed in the screening of high-risk populations [28].

Conclusions

Our results indicate that sugar cane cutters exhibited increased MN frequencies compared to a control group, possibly due to exposure to emissions derived from sugar cane burning. Future studies are necessary to characterize the mechanism responsible for DNA damage in this group. Additionally, the demonstration of a significant degree of genotoxicity in these individuals clearly indicates the necessity of adopting modern, safe harvesting practices in the sugar cane industry.

Additional file

Additional file 1: Micronuclei/1000 cells data.

Abbreviation

MN: Micronuclei; PAHs: Polycyclic aromatic hydrocarbons; PM10: PM less than 10 microns in diameter; PM2.5: PM less than 2.5 microns in diameter.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

HCSS conceived, developed and led the overall study, conducted the data reviews and the analysis, and prepared the manuscript. MSC and EHS participated in the data collection and the analysis. CS participated in the analysis. ALF and ALC helped design the study and data analysis. RMVR

assisted with the data analysis and critically reviewed the manuscript. PHNS conceived and provided advice during the study development and helped in the manuscript preparation. All authors read and approved the final manuscript.

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APPENDIX G

Helena Ribeiro

Sugar cane burning in Brazil: respiratory health effects

ABSTRACT

The article aimed to update scientific literature information about respiratory health effects caused by sugarcane burning, considering the expansion of sugarcane plantations in Brazil and in the state of São Paulo. Articles published between 1996 and 2006, which deal with the health effects of sugarcane burning and/or air pollutants originating from this burning, were discussed. These studies suggest that part of the population – especially the elderly, children and asthmatics – suffers health effects of sugarcane burning. As a result, these people require health care, thus affecting health services and their families.

DESCRIPTORS: Combustion, adverse effects. Saccharum. Air Pollutants, adverse effects. Respiratory Tract Diseases. Agricultural Zones. Review [Publication Type].

INTRODUCTION

There has been great expansion of the sugar and alcohol sector in Brazil, with the construction of more than 100 new mills and the expansion of alcohol participation in the energy matrix. This increase has risen from 6.8% in 1978 to 13.5% in 2004, regardless of debates about possible public health impacts. This country is the largest world producer and exporter of alcohol, considered as clean fuel, because it originates from sustainable sources. The Brazilian production of sugarcane reached 436.8 million tons in the 2005/2006 harvest. The state of São Paulo, except for the coast and the Serra do Mar and Vale do Ribeira areas, produces about 60% of the Brazilian sugarcane.

Sugarcane harvesting is mechanized in 25% of the Brazilian production and 40% of São Paulo's production; the rest is manually cut and suffers pre-harvest burning. In the state of São Paulo, sugarcane harvest happens between May and November, coinciding with the low rain precipitation period and worst conditions of air pollutant dispersion. Thus, it is more likely that fires will have a negative impact on the air quality and the health of those who live in the sugarcane regions.

Scientific literature regarding the effects of sugarcane burning on health is still very limited. There are studies on health effects caused by biomass burning in general, which, at times, mention sugarcane. Ribeiro & Assunção (2002)[21] published a review of these studies, focusing on emissions in the lower atmosphere that originate from biomass burning. The present study updates this review, covering the 1996-2006 period. Its objective is to comment on current scientific production about the effects of sugarcane burning on respiratory health and their main conclusions, aiming to contribute to the analysis of this issue and suggest questions for future research.

The bibliographic research was made on the following databases: Cambridge Sociological Abstracts databases – environmental sciences and pollution management; University of California Libraries Database; Science Direct; and

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Web of Science. Articles that dealt with health effects of sugarcane burning and/or the measurement of air pollutants produced by this burning were researched. This research focused on the impacts of sugarcane burning on the atmosphere and the effects on respiratory health, excluding other environmental impacts on soils and water bodies.

Brazilian library archives were also consulted – at the University of São Paulo and at the *Companhia de Tecnologia de Saneamento Ambiental* (CETESB – Environmental Sanitation Technology Company), as well as American archives – at Berkeley University in California and at the Environmental Protection Agency (EPA) in San Francisco, in 2006.

HEALTH EFFECTS CAUSED BY SUGARCANE BURNING

Burning of sugarcane residues is a common practice all over the world, especially in developing countries, though not exclusive to them. This type of burning significantly contributes to pollution in southeast Louisiana, in the United Sates, where this practice has increasingly been objected to by the population (Boopathy et al[6] 2002). Such practice is allowed by the state legislation, arguing that there is no scientific evidence of negative impacts. In order to provide information on possible health effects, a study based on hospital visits of 6,498 patients diagnosed with asthma was carried out, during the years of 1998-1999, at a hospital in the city of Houma, in this same American state. Temporal analysis and control table with three standard-deviation limits were used to analyze the already existing observations. During two years, the monthly average of hospitalizations due to asthma was 270.8. Women comprised 56.9% of patients, and babies showed the highest rates, with 1,639 visits, followed by the 5-to-10-year-old children group. The months with the highest number of hospitalizations were October and December (33.06% of all hospitalizations), thus indicating an increase in the tendency of asthma-related hospitalization during the months of sugarcane straw burning (Boopathyet al[6] 2002).

In India, case-control studies among sugarcane and sugar mill workers, fitted to confounding factors, indicated increased risk of lung cancer among workers who always worked in sugarcane plantations (odds ratio – OR=1.92, 95% confidence interval – 95% CI: 1.08;3.40). Higher risks were found for work involving soil preparation and after-harvest burning (OR=1.82, 95% CI:0.99;3.35). Workers involved in sugarcane burning for more than 210 days of their lives had a 2.5 times higher risk compared to those who had never been involved in the burning. Among smokers who worked with burning, the risk was six times higher. This risk increased according to the time spent in burning practices and the number of cigarette packs smoked (Amre et al[3] 1999).

In Brazil, research focuses on the state of São Paulo, where the population living in sugarcane areas is exposed to air pollutants originating from burning. Several studies have been performed in the city of Araraquara, in the state of São Paulo, as there are vast sugarcane plantations around it. Zancul[a] (1998) carried out air quality assessment of the city, surveyed by one of the mobile laboratories of CETESB's telemetry network, located in a completely urbanized, central area. For a period of 49 days, during the sugarcane harvest time, Zancul noticed that the air quality index remained good for CO, SO_2, inhalable particles and nitrogen oxides on most days. However, due to the presence of ozone, the index in Araraquara was regular during 85% of the sample days and inadequate during 10% of these. The study did not find evidence of the origin of precursor gases, but suggests they have been released by sugarcane burning and vehicles, or have been transported by wind from other regions. A survey on the number of inhalations, conducted in the city's health centers, revealed higher numbers during the burning period, compared to the productivity of some of the region's sugar and alcohol factories, the precipitation index and the annual seasons.

Moreover, an epidemiological survey was conducted in Araraquara, from June 1st to August 31st, concluding that sugarcane burning may have harmful effects on the population exposed (Arbex et al[4] 2000). A total of four containers were strategically placed in this city to collect particles. These data were compared to the number of hospital visits and of patients who needed inhalation in one of the main hospitals of the city. The association between sediment weight and number of visits was assessed by the regression model, which was controlled by season, temperature, day of the week and rain. The authors found significant dose-dependent relationship between the number of visits and the amount of sediment. The relative risk (RR) of hospital visits associated with the increase in 10mg of sediment weight was RR=1.09 (95% CI:1;1.19) and RR=1.20 (95% CI:1.03;1.39) for inhalation on the most polluted days. However, Arbex et al[5] (2004) argued that several factors contributed to worsen the air quality during the sugarcane harvest time in addition to burning, such as the heavier flow of trucks and machines, as well as the dust on the roads.

In another study conducted in the city of Piracicaba, in the state of São Paulo, daily hospitalizations due to respiratory diseases were quantified, in children, adolescents (below 13 years of age) and elderly people

[a] Zancul A. O efeito da queimada de cana-de-açúcar na qualidade do ar da região de Araraquara [dissertação de mestrado]. São Carlos: Escola de Engenharia de São Carlos da USP; 1998.

over 65 years of age, by means of data from the *Departamento de Informática do Sistema Único de Saúde* (DATASUS – Brazilian Health System's Computer Department). Analyses indicated that biomass burning and re-suspension of eroded soil material are responsible for 80% of fine particulate matter (PM 2.5). Relative risk of hospitalizations due to respiratory diseases in children and adolescents was significantly associated with inter-quarter variation of PM10, PM2.5, black carbon of aluminum, silicon, manganese, potassium and sulfur. PM increase in 10 μg/m^3 was associated with an increase in 21% in hospitalizations. Among the elderly, the relative risk of hospitalizations due to respiratory diseases was associated with the inter-quarter variation of PM10, black carbon and potassium. The burning period had 3.5 times more hospitalizations than the non-burning period (Cançado 2003).[a] However, the author warns of confounding factors such as the air temperature and precipitation, as a great part of the burning period coincides with winter and dry weather, not controlled by the study.

Lopes & Ribeiro[15] (2006) analyzed spatial correlations when gathering the following into a geographic information system: fires, sugarcane areas, and hospitalizations due to respiratory diseases, recorded by the DATASUS, from 2000 to 2004, in the state of São Paulo and, on a regional scale, in the city of Bauru. On both scales it was possible to verify higher rate of hospitalizations due to respiratory diseases in areas where there is sugarcane burning.

Other studies measured atmospheric emissions by different pollutants originating from sugarcane burning, regardless of health effects. However, by means of these results, possible human health risks can be inferred.

In the United States, in 1975, the EPA analyzed sugarcane burning emissions in an experiment with whole sugarcane and only with straw in an incinerator. Emissions of particulate matter, carbon monoxide, hydrocarbons, benzopyrene (BaP), and trace metals – beryl, cadmium, chrome, copper, and nickel – were determined. It was verified that 90% of the particles were less than 0.5 μm in diameter. The emission factors found, with a 99% confidence level, were: 4.1-6.5 pounds per ton of particulate matter; 47.7-71.2 pounds per ton of carbon monoxide; 2.3-14 pounds per ton of hydrocarbons (Darley & Lerman, 1975).[b] Particles that were smaller than 10μm (PM10), carbon monoxide and hydrocarbons are harmful to the health.

Godói et al[9] (2004) analyzed aerosol samples with less than 10μm in diameter, in the city of Araraquara, during harvest time, using a Hi-vol air sampler, 5 km away from sugarcane plantations. The authors found daily concentrations of total particulate matter that varied from 76.3 μg/m^3 to 181.8 μg/m^3 and average concentrations of 103 μg/m^3. These concentrations are below the daily standard air quality, but above the annual standard established by the Brazilian legislation. The total concentrations of polycyclic aromatic hydrocarbons (PAHs) measured were between 13 and 94 ng/m³, comparable to the values found in Naples, in Italy, and above those measured in Santiago de Chile, and in Seoul, in South Korea. The PAH analysis indicated high levels of B[a]P (1.9 ng/m³), of special interest due to its carcinogenic properties and the fact that it is at higher levels in major cities in the world such as London, in England. As benzopyrene is usually present in grass burning, the authors suggest that, in Araraquara, it originates from sugarcane burning, not from urban activities.

Oppenheimer et al[19] (2004) used a compact ultraviolet spectrometer to measure nitrogen dioxide (NO_2) emission from sugarcane burning in the state of São Paulo. NO_2 emission from a 10-hectare plot of land reached a peak of 240g (NO_2)/s, and added up to approximately 50kg of N, or about 0.5g (N)/m². Nitrogen emission such as NOx (NO+NO_2) was estimated at 2.5g (N)/m², equivalent to 30% of nitrogen fertilizer applied to crop fields.

During a period of one year, aerosols were measured in the city of Piracicaba. The average concentrations of PM 2.5 and coarser particles (2.5 to 10μm) were statistically higher in the dry season than in the rainy one. Component analysis indicated that sugarcane burning was the main source of PM 2.5 (60%), Soil dust was responsible for 14%, while factories and combustion, 12% each. Re-suspended soil dust was the major source of coarser particles, followed by industrial emissions and sugarcane burning. The authors concluded that sugarcane and farming practices were the main sources of inhalable particles around the city, also affecting the chemical composition of rainwater (Lara et al[13] 2001; Lara et al[14] 2005).

Measurements outside the areas immediately surrounding the fires indicated the background level and the fact that, during the burning period, smaller particles were more acid, contained higher concentrations of sulphates, nitrates and organic species, but insufficient NH_4+ and K+ to reach neutrality. In the winter, the relative contribution of emissions from transportation and factories decreased due to the increase in emissions from biomass combustion and other harvest time-related activities. The concentrations of pollutants were lower than what is usually found in polluted urban zones. Nonetheless, the authors concluded that pre-harvest sugarcane burning influences the aerosol chemistry on a regional scale; also, that the differences between air masses from

[a] Cançado, JED. A poluição atmosférica e sua relação com a saúde humana na região canavieira de Piracicaba – SP [tese de doutorado]. São Paulo: Faculdade de Medicinada USP; 2003.

[b] Darley EF; Lerman SL. Air pollution emissions from burning sugar cane and pineapple residues from Hawaii. North Carolina: Environmental Protection Agency; 1975. (EPA Publication, 450/3-75-071)

distinct origins were small. On the other hand, they assessed that, in urban areas and their surroundings, vehicle emissions had a greater impact (Rocha et al[23] 2005; Allen et al[1] 2004; Rocha et al[22] 2003).

HEALTH RISKS

Despite their restrictions and cautious conclusions, the studies analyzed indicate health risks in adverse atmospheric conditions, caused by sugarcane straw burning. These risks can be higher among children, elderly people and asthmatics, mainly resulting in higher demand for health care. Until recently, studies on sugarcane were mostly concerned about workers in the productive process, such as Phoolchund's investigation[20] (1991), which showed that sugarcane cutters were at higher risk of lung cancer as a consequence of foliage burning. As the global environmental crisis worsened and people became more aware of this issue, especially as regards climate changes resulting from polluting human activities, there has been an increase in biofuel production. Among these fuels, sugarcane is the fastest-growing one. However, its burning has increasingly been opposed by public opinion, allegedly due to its environmental and human health impact, even though Brazilian health organs have had little participation in this discussion. In the state of São Paulo, due to the environmentalists' pressure, the law that foresees gradual elimination of fire utilization to facilitate sugarcane cutting, until 2021 for mechanized areas, and until 2031 for non-mechanized areas, was approved in 2002.

The few studies on the effects of sugarcane burning hint at the health impacts on the general population, though many questions are still left unresolved.

On the other hand, research on the health effects of biomass burning, especially as regards uncontrolled forest fires (Ribeiro & Assunção[21] 2002), may help to define a health policy for this issue and guide future research.

Frankenberg et al[8] (2005) concluded that individuals exposed to biomass smoke experienced more difficulty in their daily activities, even though general and respiratory health effects were more difficult to interpret.

Kunii et al[12] (2002), while assessing the effects of Indonesian forest fires, including interviews and pulmonary function tests in 54 people, verified that more than 90% presented with respiratory symptoms and that elderly people suffered severe deterioration of their health condition. By means of multivariate analysis, the study showed that gender, history of asthma and frequency of mask use were associated with the severity of the respiratory problem.

Negative effects of Indonesian fires were also assessed in the Malaysian population (Sastry[25] 2002). Mott et al[18] (2005) investigated the exposure effects on the cardiorespiratory health of hospitalized people in the Kuching region, in Malaysia. The authors selected admissions from 1995 to 1998 to verify if hospitalizations during or after fires in neighboring countries exceeded the predicted number of hospitalizations, in accordance with historical records. There was statistically significant increase in the number of hospitalizations due to respiratory diseases, especially asthma and chronic obstructive diseases. Survival analysis indicated that people over 65 years of age, who had been previously hospitalized for any reason, with any respiratory, cardio-respiratory, or chronic obstructive pulmonary disease, were more likely to be hospitalized again after the burning period. These cited articles reveal the relationship between non-localized, cross-border pollution caused by biomass burning and the vulnerability of some specific groups of the population, especially elderly people and those who suffer from any of the foregoing diseases.

According to Sapkota et al[24] (2005), in addition to affecting neighboring communities, pollution originated from forest fires can travel thousands of miles to heavily populated urban areas. Fire effects in Canada resulted in a high concentration episode (up to 30 times higher) of particulate matter, especially finer one, in the city of Baltimore, in the United States. In 2003, forest fire smoke in Siberia was tracked by means of airplane and ground observations, thus indicating their transportation to North America. This caused an increase in background pollution in Alaska, Canada and the northeast Pacific Ocean by 23-37 ppbv of carbon monoxide and 5–9 ppbv of ozone. This increase in background ozone contributed to the air quality standard for ozone being exceeded in the northeast Pacific Ocean. According to the authors, regional air quality and health are connected to global atmospheric processes (Jaffe et al[11] 2004). Similarly, research has pointed to the effects of sugarcane burning on a regional scale. Nonetheless, as this burning may have greater spatial influence, the size of the population under the risk of health effects would be larger.

According to Jacobson[10] (2004), the elimination of particles originated from burning may cause an increase in atmospheric temperature in the short run, and cooling of the climate in the long run due to elimination of carbon dioxide. Analytically, biomass burning always leads to carbon dioxide accumulating, even when vegetation recovery and sprouting cycles are equivalent to emission flows. Thus, Jacobson concluded that biomass energy is only partly renewable, because its burning contributes to global warming.

Another concern related to burning is the transport of fungus spores and bacteria for long distances. Mims & Mims III[17] (2004) verified the presence of fungus spores and bacteria, including the *Alternaria, Cladosporum, Fusariella* and *Curvularia* genera, in smoke originating from biomass burning. The authors argue that sugarcane rust caused by the *Puccinia melanocephala* fungus in the Dominican Republic was transported from Africa by convection originated from the fire, which would have taken spores to the upper atmosphere, where they would

have moved on to the Caribbean. Thus, burning could contribute to the spread of pathogenic microbes. Fungus spores (such as *Alternaria*) cause allergic reactions and trigger asthma attacks, as well as smoke inhalation.

Recently, epidemiological studies have showed evidence of the relationship between air pollution and cardiovascular diseases, particularly myocardial infarction. According to Vermylen et al[26] (2005), ozone can have direct, harmful cardiovascular effects, whereas other gases can increase the negative effects of particulate matter. Particles with smaller diameter have a greater impact, but PM10 can quickly penetrate and deposit on the trachea and bronchioles. PM2.5 can reach narrow airways and alveoli, whereas ultra-fine particles, smaller than 100 nm (0.1μm), have high alveolar deposition. The total number of deposited particles may increase four to five times during exercise, due to higher ventilation. Ultra-fine particles represent most part of the particulate matter and have higher area/mass ratio, which would increase biological toxicity, as they can go directly into the bloodstream. The populations at highest risk are the elderly, those with chronic pulmonary or coronary disease, and diabetics. Whereas acute air pollution can trigger myocardial infarction in hours or days among those who are susceptible, chronic exposure to pollutants increase the risk of cardiovascular diseases that may be related to chronic pulmonary inflammation.[26] Sugarcane burning would simultaneously have both effects: acute air pollution in the neighboring areas and diffuse air pollution in the long run, on a regional scale.

During burning, combustion is incomplete, with the formation of compounds that are not completely oxidized, thus irritating to the respiratory system and, in some cases, carcinogenic. Malilay[16] (1999) affirmed that fine particulate matter reach the alveoli and in great concentrations get into the bloodstream or stay in the lungs, resulting in chronic diseases such as emphysema. Toxic organic vapors such as PAHs are possibly carcinogenic. Carbon monoxide may cause hypoxia by preventing blood from carrying enough oxygen. Fetuses are particularly susceptible, as they cannot compensate for the oxyhemoglobin reduction without sustained increase in cardiac frequency. Aldehydes are irritating to the mucosa and some, such as the formaldehyde, can be carcinogenic. Volatile organic compounds may irritate the skin and eyes, and cause drowsiness, cough and wheezing; in addition, some of them are carcinogenic. In high concentrations, ozone can affect the pulmonary function; whereas, in low concentrations, it can cause cough, choking, breathlessness, mucus, throat burning and irritability, nausea, and a decrease in pulmonary function when exercising (Malilay[16] 1999).

FINAL CONSIDERATIONS

Despite the weight of farming residue burning as an old practice, spread around tropical countries to control plagues and eliminate harvest residues, there are public health issues that need to be further studied at this time of expansion of biofuel production.

Yevich & Logan[28] (2003) estimated that, in 1985, 400Tg of farming residues were burned in fields, and that Brazil was the country that most generated these residues in Latin America, especially sugarcane straw. According to the authors, this biomass burning has a significant impact on the global atmospheric chemistry, as it produces great amounts of carbon monoxide, nitrogen oxides and hydrocarbons, thus representing non-neglectable contribution with negative effects, particularly in the regional sphere.

Pre-harvest sugarcane burning aims, above all, to eliminate its residue, straw, in order to facilitate manual harvesting or decrease its volume for incorporation into the soil. Among the difficulties for total elimination of burning, as foreseen by the state of São Paulo's legislation, are the producers' restrictions regarding the price and deficiencies of mechanic harvesters; as well as the manual cutters', who earn per harvest and can perform better and be at a lower risk of harm from poisonous animals found in the burned sugarcane (Braunbeck & Magalhães[7] 2004; Weekes[27] 2004). Sugarcane plantations represent the highest farming workforce demand in the state of São Paulo (35%), especially the ones with low level of education (Braunbeck & Magalhães[7] 2004). Furthermore, there have been unexplained deaths of workers in the plantations. Alves[2] (2006) attributes them to the physical effort to achieve better cutting productivity. However, there could be a relationship between sugarcane burning and these deaths, as heavy physical exercise performed during cutting, in a place where there is a high amount of soot, would contribute to increase respiratory health risks. This relationship needs to be investigated.

Moreover, future studies on this issue need to focus, in addition to respiratory diseases and their symptoms – particularly excessive asthma cases –, on other effects and risks, such as tumors, cardiovascular diseases, impacts on daily activities of the affected people, re-hospitalization of the elderly and biological risks.

Replacement of burning by mechanic harvesters will certainly benefit health conditions of those living in the sugarcane areas. However, to actually guarantee improvement in the cutters' health, programs for their re-qualification and hiring need to be developed and executed immediately.

Finally, even with the enforcement of the law that forbids burning in the state of São Paulo, sugarcane will continue to be burned in the rest of the country and its residues will still be burned in power plants (Rocha et al[23] 2005). Thus, a national policy to prevent health hazards will also depend on the efficacy of fire control across the country, as well as power plant emissions.

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APPENDIX H

Green | Mon Oct 22, 2007 2:21pm EDT Related: ENVIRONMENT

Brazil sugarcane mills agree to end burning by '17

SAO PAULO

Canefarmers "burn off" prior to harvesting at a cane farm at Giru, located in a sugarcane belt stretching over 625 miles in northern Australia August 5, 2004. Almost 100 sugar and ethanol mills in Brazil's main sugar cane state Sao Paulo have agreed to stop the practice of...

(Reuters) - Almost 100 sugar and ethanol mills in Brazil's main sugar cane state Sao Paulo have agreed to stop the practice of burning cane fields by 2017, the Sugar Cane Industry Union (Unica) said on Monday.

These mills crush more than 50 percent of the cane output in Sao Paulo, Brazil's No. 1 cane producing state that accounts for around 63 percent of the national crop.

In June, Unica had signed an agreement with the state government in which mills were to ban cane burning in the state by 2017, well before 2031 target mandated by a state law.

This pact's success still depends on mills' signing on. The protocol is not mandatory but is seen as a move toward protecting the sector from environmental objections.

"The number of mills is surprising and shows how the sector has complied with the demands to end the cane burning," Unica's president, Marcos Jank, said in a news conference.

"It puts an end to the environmental incoherence," Jank added, referring to the environmental impact of burning and the clean benefits of renewable cane-based ethanol fuel.

In September, Unica said it expected 70 to 80 mills to sign the document by year's end. There are 96 mills already subscribed to it, a number expected to rise in coming months.

The cane burning ban already is ahead of the curve in Sao Paulo state, where more than

40 percent of the harvest is mechanized, with only 30 percent mandated by law, Jank said.

Cane burning facilitates manual harvesting, but causes clouds of smoke and endangers public health in nearby communities, especially when humidity is low. Mechanized harvesting does not require burning.

The end of burning fields has economic advantages too. Extra plant matter can be burned in cogeneration electric power plants and one day may be used to produce cellulosic ethanol. Mechanized harvesting frequently is cheaper than the manual cutting because cane cutters enjoy strong labor rights.

Less than 2.2 million hectares of cane are expected to be burned in Sao Paulo this year, down from the 2.5 million hectares last season, despite an increase of more than 10 percent in the planted area to cane in this period.

The protocol determines that flat areas ban cane burning by 2014 and hilly areas, by 2017. But Jank said the practice should be stopped before that.

"I'm convinced that in 10 years there will not be burning of cane in Sao Paulo, and other states will follow," he said.

There are around 200,000 cane cutters in the state nowadays, and the industry is now trying to prepare at least part of this group to take new duties in the mills.

The forecast expansion of the ethanol industry in the coming years should absorb 70,000 of these workers, Unica said.

APPENDIX I

Company Rules & Regulations

PROCUREMENT TERMS & CONDITIONS

SALES TERMS & CONDITIONS

COMPANY RULES & REGULATIONS

If Vendor is performing work or supplying services, the following "COMPANY RULES AND REGULATIONS" shall apply.

FLORIDA CRYSTALS CORPORATION AND SUBSIDIARIES
COMPANY RULES AND REGULATIONS
(Effective December 12, 2012)

Contractor and Contractor's Workforce shall be subject to and abide by these Rules at all times while on Company Property. Each Company Property may supplement these Rules or have additional rules and regulations specific to their location.

1. Defined Terms. As used in these Rules (including the foregoing preamble), the following terms have the meanings specified below:

"*Affiliates*" means any entity which controls, is controlled by or is under common control with Company. The term "*control*" means the ownership, directly or indirectly, or the power to direct the voting or disposition, of fifty percent or more of the voting stock or equity interests of the subject entity.

"*Company*" means the company to whom Contractor is providing services or work – that being, Florida Crystals Corporation, Okeelanta Corporation, Osceola Farms Co., Sem-Chi Rice Products Corp., Sugar Farms Co-Op, Florida Crystals Food Corp., New Hope Power Company and/or any of their respective Affiliates.

"*Company Property*" means a property that is owned or controlled by Company, which may include, but not limited to, refineries, power plants, packaging, storage, and distribution facilities, labs, offices, hospitality centers.

"*Contractor*" means any person or entity providing services to the Company on Company Property.

"*Contractor's Workforce*" means any employee, subcontractor, agent, supplier, independent contractor, or materialman of Contractor.

"*Laws*" means any federal, state, regional or local laws, rules, regulations or ordinances.

"*Rules*" means these Company Rules and Regulations.

"*Services*" means any services or work being or to be performed by Contractor or Contractor's Workforce for the Company Property.

"*Violation*" means any failure of Contractor or Contractor's Workforce to comply with the Rules, including the attached Visitor Release and Confidentiality Agreement, notwithstanding any oral or written contractual provision to the contrary.

2. Contractor's Safety Responsibilities.

- Prior to commencing Services, Contractor shall:
  a. Deliver to Company a Visitor Release and Confidentiality Agreement in the form attached hereto as **Exhibit 1** executed by Contractor and each individual member of Contractor's Workforce that will be entering upon Company Property; and
  b. Provide Company with applicable Certificates of Insurance for Contractor and Contractor's Workforce in a form and with policy limits and conditions satisfactory to Company;
- Contractor and each member of Contractor's Workforce performing Services will attend safety and process meetings if requested by Company to do so.
- Visitors are not permitted on Company Property. If it becomes essential that a Visitor be present, the Visitor must sign a Visitor Release and Confidentiality Agreement, be subject to these Rules, be escorted by the Contractor to the site, and be accompanied by Contractor at all times. The purpose of the visit must be limited to the Services and jobsite.


Chocolate Filled Valentine Sugar Cookies

3. Security and Access.

- Company will advise Contractor of the gate and parking requirements at the applicable Company Property. Contractor may be assigned a separate gate for entry and exit and provided a designated area for parking and if gate passes are required, the Company may require that each vehicle used by Contractor Workforce obtain one. Passes are not to be shared between vehicles unless expressly permitted by the Company.
- All vehicles, packages, and lunch buckets of Contractor are subject to inspection by Company or Company's security officers. Persons attempting to remove Company or third party property from the jobsite or Company Property without authorization may be barred from the Company Property and subject to prosecution.
- Cameras and recording devices of any type are not allowed on Company Property without prior written permission of Company. Cellphone cameras shall not be used while on Company Property.
- Firearms, deadly weapons, explosives, alcohol or drugs are prohibited on Company Property.
- Contractor and Contractor's Workforce shall comply with the Company's security requirements which may include, amongst other requirements, producing government issued photo identification and the Company taking a photo of the Contractor and Contractor's Workforce in order to be granted access to a Company Property.

4. Vehicle and Pedestrian Traffic Safety.

- Contractor will be limited to only those pieces of mobile equipment required to perform the Services and such mobile equipment must not block Company Property roads, fire lanes, hose houses, fire hydrants, or emergency egress routes.
- All vehicles are to be operated at a safe speed and in accordance with all applicable Laws and all traffic signs are to be obeyed.
- Contractor and Contractor's Workforce must watch out for and yield to cranes and fork trucks which shall be given the right of way.
- When performing Services on roadways or near moving equipment, a reflector vest must be worn at all times.
- Crawling under, over, or between connected railcars is prohibited. All persons must walk around the ends of the trains and must maintain a minimum distance of eight feet from the end of the railcar.
- Riding on the back (bed) of vehicles, including pick-up trucks, is prohibited.

5. Contractor's Workforce.

- Contractor's Workforce is physically limited to the jobsite and approved routes to be taken to and from the jobsite. Unless prior approval is granted by Company, access is prohibited to Company cafeterias, lunchrooms, vending machine areas, break rooms, and sanitary facilities. Contractor will supply chemical toilets and drinking water for Contractor's Workforce, unless prior written approval has been provided by Company for other arrangements.
- Contractor is responsible for providing:
    a. A means of communication with Contractor's Workforce and with Company while such personnel are on Company Property and such means of communication shall be acceptable to Company.
    b. Contractor's Workforce with the necessary and appropriate safety equipment.
- Contractor shall ensure that Contractor's Workforce:
    a. Wear long pants and shirts that are tucked into trousers.
    b. No sleeveless shirts, or loose fitting clothes be worn.
    c. No jewelry shall be worn.
    d. Long hair must be restrained.
    e. Footwear must be leather steel-toed shoes.
    f. Only smoke in designated smoking areas.

6. General Procedures and Safety Requirements.

- **APPROVAL MUST BE OBTAINED FROM A COMPANY REPRESENTATIVE BEFORE SHUTTING DOWN ANY COMPANY SYSTEMS OR EQUIPMENT.**
- Contractors are to utilize ground fault circuit interrupters on all 120 volt equipment. GFCIs shall be used in manner where the GFCI is plugged into the electrical outlet box being used with all extension cords and equipment then plugged into the GFCI.
- Compressed air is not to be used for cleaning, including the cleaning of dust from clothing.
- Compressed gas cylinders must be secured at all times and caps on when not on welding carts. Acetylene and Oxygen must be stored separately at least 25 feet from each other.
- Electric extension cords, welding electric cords etc. with kinks or cuts are not to be use on any job, and electrical tape is not allowed to fix same, a certified electrician is the only person that can properly fix electrical cords, otherwise they will have to be replaced.
- Contractor shall not tie into any energy source (electric, gas, air pressure, steam, etc.) without prior approval from Company.
- The use of fiberglass ladders is preferred in the performance of Services and is required for electrical Services. Where scaffolding is required, Contractor is responsible for assuring its construction and use in accordance with applicable safety standards.
- The possession and use of matches, lighters, strikers, or other potential ignition sources on Company Property are strictly prohibited, except to the extent same is integral in Contractor's performance of the Services. Before welding, grinding, or using open flames, torches, and other types of equipment producing sparks, a "Hot Work" permit must be obtained from Company.
- Fire doors must not be propped open or obstructed. Emergency exits must not be obstructed or fastened shut.
- Contractors must properly barricade holes in floors, excavations, and other openings at all times. When persons are working overhead, the area below

must be barricaded and warning signs installed. Contractors must provide ground-men for overhead work as necessary or if requested to do so by Company.

- A "Confined Space Entry" permit must be obtained from Company and completed by Contractor before entering into any confined space, vessel, or equipment. All personnel must be trained in all relevant duties prior to entry and Contractor shall furnish their own monitoring and emergency equipment.
- For control of hazardous energy sources to any equipment and/or work area, a "Lockout/Tagout" procedure shall be reviewed prior and followed at all times. Contractor shall supply all necessary control devices.
- Prior to bringing any hazardous, toxic or radioactive chemicals, materials or substances (collectively, "***Hazardous Materials***") on to Company Property, Contractor must obtain written permission from Company and provide Company with all applicable Material Safety Data Sheets ("***MSDS***"). Contractor will comply with all applicable Company requirements, Laws and EPA rules and regulations regarding the use, handling, labeling and storing of Hazardous Materials at all times while on Company Property. The MSDS must be available for inspection at all times at the jobsite. Contractor will work with the Company's Environmental Manager for the Company Property regarding compliance with the foregoing. The introduction of any radioactive materials onto Company Property must be supervised by the Company's Radioactive Safety Officer for the Company Property.
- Contractor must provide Company with information about any possible Hazardous Materials that may occur from Contractor's performance of the Services. All Hazardous Materials generated as waste will be reported to the Company, including quantity, along with a copy of the manifest. Contractors disposing of Hazardous Materials must supply a copy of DOT training to the Company; and no such Hazardous Materials can be removed without proof of such training. Contractor will work with the Company's Environmental Manager for the Company Property regarding compliance with the foregoing.
- Flammable liquids will be appropriately labeled and stored in U.L. approved safety containers which are properly grounded. Oily rags, waste, waste paper and other flammable or combustible materials must be properly stored in tightly closed metal containers. No glass containers are allowed in any operating facilities of Company Property. All containers must be properly labeled and stored.
- Areas to be used by Contractor for offices, storage trailers, or fabrication will be arranged through Company and all such areas, including the jobsite, shall be maintained by Contractor in an orderly fashion.
- To prevent product contamination and to avoid the creation of an unsafe condition, all debris and garbage produced by Contractor must be cleaned up and properly disposed of on a daily basis by Contractor. Such disposal will follow all applicable Laws, including EPA regulations.
- Many internal drains return to process and yard/storm drains which may discharge into lakes, rivers and streams. Consequently, prior approval must be obtained from Company before the use of any building and sanitary drains. Other than precipitation, no water of any type, including clear, clean potable water, nor any other materials may be discharged into storm water drains.
- The storage of Contractor's or Contractor's Workforce's property, including, but not limited to, equipment, tools, vehicles, materials and personal property (collectively, "***Contractor's Property***") on Company Property must be approved by Company. Such storage is at Contractor's risk and Company is not responsible for any loss or damage to, or to provide security for, Contractor's Property while stored on Company Property.
- In the event Contractor is authorized to utilize any Company equipment, tools, vehicles or materials (collectively, "***Company Equipment***"), then Contractor shall do so at its own risk and is responsible for any loss or damage to the Company Equipment. No Company Equipment will be released unless it is properly charged out to Contractor. Contractor's personnel operating any Company Equipment must be qualified to safely operate the specific equipment in question.
- Contractor must immediately report to a Company representative any damage to Company Equipment or Company Property.

7. Emergency Procedures.

- Any accident and/or incident occurring on Company Property must be immediately reported (day of occurrence) to Company and an investigation will follow. Company reserves the right to participate in investigations to the extent deemed appropriate by Company. A copy of any first report of injury completed by Contractor must be provided to Company within 24 hours of the accident or injury.
- Contractor must immediately stop all Services in areas where it is determined that a hazardous condition exists. Contractor may resume the Services only when authorized by Company to do so.
- If Contractor or any member of Contractor's Workforce believes they have been exposed to any Hazardous Materials, an incident report must be submitted to Company.
- Emergency drills will be conducted periodically while Contractor is on Company Property. Contractors are required to participate in the emergency drills. Contractor and Contractor's Workforce must familiarize itself with the emergency procedures and evacuation plans of the area in which Services will be performed.
- Contractor and Contractor Workforce are not allowed to use the manlifts.
- Company may provide first aid for minor injuries or medical response on a "Good Samaritan" basis only and not as a contractual obligation. Contractor assumes full and complete responsibility and liability for injuries and damages to Contractor, Contractor's Workforce and Contractor's visitors. Company is under no obligation to provide first aid, emergency medical treatment, or related services.

7. Violations. In the event of a Violation, Company may, in its discretion, (a) remove the individual(s) that are in Violation from the project or jobsite; (b)

deduct from Contractor's invoice, for each Violation, the greater of (i) $500 (the "*Fine*") or (ii) the amount equal to all fines and penalties assessed against Company by applicable Laws; and/or (c) immediately terminate Company's contracts with Contractor. The Fine shall increase by $500 for each subsequent Violation.

EXHIBIT 1

FLORIDA CRYSTALS CORPORATION AND SUBSIDIARIES

Visitor Release and Confidentiality Agreement

I am a contractor, subcontractor, materialman, or an employee of a contractor, subcontractor or materialman providing services or materials to Florida Crystals Corporation, Okeelanta Corporation, Osceola Farms Co., Sem-Chi Rice Products Corp., Sugar Farms Co-Op, Florida Crystals Food Corp., New Hope Power Company and/or any of their respective affiliated companies "*Company*"). In consideration of receiving permission from the Company to enter upon the Company's properties, which may include, but not limited to, refineries, mills, power plants, packaging, storage, and distribution facilities, labs, offices, kitchens and lounges, and other properties owned or controlled by the Company (the "*Properties*") during the period of services or work being performed by my employer or principal, I hereby release the Company, its employees, officers, directors, agents and assigns, from all liability, claims, demands, actions, and causes of action whatsoever, arising out of or related to any loss, damage or injury, including death, that may be sustained by me, or any of my property, while on the Properties even if caused by Company's negligence, negligent act and/or negligent condition.

I am aware that the Properties contain known and unknown inherent risks, dangers and hazards, including, but not limited to, risks involving vehicles, moving machinery, equipment, uneven or slippery surfaces, explosion, fire, smoke, gases, steam, chemicals, emissions and other conditions. Nonetheless, I elect to voluntarily enter upon the Properties, waive notice of any and all risks and hazardous or negligent conditions existing at the Properties, and voluntarily assume all risks of loss, damage or injury, including death, that may be sustained by me, or any property of mine, while on the Properties even if caused by Company's negligence, negligent act and/or negligent condition.

I acknowledge that, as a result of entering on the Properties, I may obtain information about the Company which is proprietary or confidential, such as manufacturing methods and processes, equipment, and designs (collectively, "*Confidential Information*"), and I agree that I will not disclose such Confidential Information to any other person or use it for any purpose adverse to the Company.

I agree not to photograph, tape, record, film, or create any drawings, sketches, notes, printed information, interior photographs, films, and tapes, or memoranda of, or regarding, the Properties or any Confidential Information without Company's prior written consent.

I agree to abide by the Company's hygiene, safety, and dress codes and the directions of Company representatives. This Agreement may be revoked at any time by the Company in its sole discretion. This Agreement shall be binding upon my heirs, next of kin, executors, administrators, personal representatives, agents and assigns.

In signing this Agreement, I hereby acknowledge and represent that I have read the foregoing Agreement, understand it, and have signed it voluntarily.

Signature: _____

Date: _____

Printed Name: _____

Address: _____

APPENDIX J

Fact Sheet

Burning Agricultural Waste: A Source of Dioxins

January 2014

cec.org

Commission for Environmental Cooperation

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BURNING AGRICULTURAL WASTE: A SOURCE OF DIOXINS

Background

Since 1994, Canada, Mexico and the United States have been cooperating on environmental matters of mutual concern through the Commission for Environmental Cooperation (CEC).[1] In 2000, the CEC's Sound Management of Chemicals (SMOC) Working Group established a task force on dioxins, furans and hexachlorobenzene, which had the mandate to develop and implement strategies for facilitating the effective management of chemicals in the region. One of its objectives has been to help Mexico develop public information materials that can provide the foundation for decision-making. This document is part of that effort. It is designed for the interested public and contains information that may be useful for decision-makers.

Burning Agricultural Waste

In many countries, burning agricultural waste, such as stalks, grasses, leaves and husks, continues to be the easiest and least expensive way to reduce or eliminate the volume of combustible materials produced by agricultural activities. Open-air burning is used to eliminate waste from the previous harvest in the quickest manner, and to clear fields to prepare them for planting. It is also used to release nutrients for the following growing season and to eliminate mosquitoes and other pests in crop-growing fields. Sugar cane fields are burned prior to harvesting to remove excess leaves and thus facilitate the harvesting and transporting of products to sugar refineries. It is also believed that burning the sugar cane fields reduces the risk of insect bites and cuts for workers.

It is estimated that burning biomass, such as wood, leaves, trees and grasses—including agricultural waste—produces 40% of carbon dioxide (CO_2), 32% of carbon monoxide (CO), 20% of particulate matter (PM), and 50% of polycyclic aromatic hydrocarbons (PAHs) released into the environment around the globe.[2] Although agricultural waste burning is not an environmentally acceptable form of agricultural management, it is a frequent practice and is worrisome from a public health viewpoint for a number of reasons:[3]

- Smoke from agricultural burning is released at or near ground level in areas that are generally populated, producing direct, intense exposure to pollutants for the nearby populations.
- This type of burning is generally carried out in stages, during specific times of the year, and may lead to very high concentrations of pollutants.
- Burning agricultural waste creates non-specific sources of pollutants for the atmosphere and takes place over very large areas. It is therefore difficult to measure and to regulate the resulting emissions.
- Combustibles and combustion conditions vary, and pesticides may be present.
- This type of burning contributes to climate change, since among the compounds released are greenhouse gases and short-lived climate-forcing pollutants like black carbon.
- Visibility in nearby areas and highways is affected.
- In addition, these incomplete combustion processes produce dioxins, which are highly toxic, carcinogenic pollutants.

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[1] See <www.cec.org>.
[2] A.D. Kambis and J.S. Levine. 1996. Biomass burning and the production of carbon dioxide: a numerical study. Chapter 17 in: *Biomass Burning and Global Change*, Joel S. Levine (comp.) 1: 170-177, Massachusetts Institute of Technology, Cambridge, MA (US).
[3] P.M. Lemieux, C.C. Lutes and D.A. Santoianni. 2004. Emissions of organic air toxics from open burning: a comprehensive review. *Progress in Energy and Combustion Science* 30: 1-32.

Agricultural Waste Burning Is a Significant Source Of Dioxins

Biomass burning as an agricultural practice is considered to be a significant source of dioxins. The factors that play a part in the emission of dioxins are the combustion conditions, chlorine content, and presence of pesticides absorbed into the leaves and stalks of agricultural waste.

It has been found in Taiwan, for example, that during the week of the most intense agricultural burning, the concentration of dioxins in the atmosphere is up to 17 times higher than in weeks when such burning is absent.[4] In China, larger amounts of dioxins are emitted in the provinces with more agricultural production—constituting between 10 and 20% of total emissions of dioxins[5]—than in provinces with less agricultural production. Results of studies indicate that dioxins can be formed during the combustion process due to the presence of chlorinated pesticides, such as pentachlorophenol (PCP) fungicide and the herbicide known as 2,4-dichlorophenoxyacetic acid (2,4-D). It has been demonstrated, for example, that dioxin emissions increase by 150 times when biomass treated with 2,4-D is burned.[6] Also, dioxins released to the soil through ash increase when agricultural waste contaminated with pesticides are burned. For example, dioxin emissions are between 35 and 270 times greater in the case of corn stalks treated with pesticides, compared to the amount of dioxins released when such waste has not been so treated.[7]

Dioxins and Their Toxicity

It is believed that even in very small amounts, dioxins constitute a health and environmental problem since they:

- Are persistent and remain in the environment for long periods of time before degrading into other chemical forms.
- Accumulate and are stored in the fatty tissue of animals and humans.
- Are able to be transported long distances in the atmosphere, and are thus sometimes generated in one area but ultimately can be found in another region far away.

Numerous studies link exposure to dioxins with a variety of harmful effects on health, such as:

- Lower sperm concentration in males who were exposed when they were infants or children.
- Changes in thyroid hormone levels.
- Neurological effects in the fetus due to exposure during pregnancy.
- Lower testosterone levels.
- Reproductive problems in women, such as prolonged menstruation and early menopause.
- Diabetes and harmful effects to the immune system.
- Chloracne, which can be severe when exposure is very high.
- Various types of cancer in humans.

Harmful Effects to Health Caused by Emissions from Agricultural Burning

Pollutants emitted from agricultural burning, such as polycyclic aromatic hydrocarbons and very small particulates ($PM_{2.5}$), can cause cancer in humans as well as severe respiratory illnesses, coughing, phlegm and asthma. For example, during the season in which sugar cane is burned, an increase in asthma attacks

[4] S. Shih, W. Lee, L. Lin, J. Huang, J. Su, and G. Chien. 2008. Significance of biomass open burning on the levels of the polychlorinated dibenzo-p-dioxins and dibenzofurans in the ambient air. *Journal of Hazardous Materials* 153: 276-84.
[5] Q. Zhang, J. Huang and G. Yu. 2008. Polychlorintaed dibenzo-p-dioxins and dibenzofurans emissions from open burning of crop residues in China between and 2004. *Environmental Pollution* 151: 39-46.
[6] M. Muñoz, B.K. Gullet, A. Touati, R. Font. 2012. Effect of 2,4-dichlorophenoxyacetic acid (2,4-D) on PCDD/F emissions from open burning of biomass. *Environmental Science & Technology* 46: 9308-14.
[7] T. Zhang, J. Huang and G. Yu. 2011. Influence of pesticides contamination on the emission of PCDD/PCDF to the land from open burning of corn straws. *Environmental Pollution* 159(6): 1744-48.

has been observed in the population living near sugar cane fields in southern Louisiana in the United States. In fact, hospital admissions due to various respiratory problems increase by 50% during this time of year.[8] In Brazil, the world's largest sugar cane producer, elevated PM levels have also been observed, as well as an increase in respiratory problems, during the season when sugar cane is burned.[9]

Agricultural Burning and the Burning of Agricultural Waste in Mexico

According to an inventory of dioxins and furans,[10] agricultural waste burning ranks third among the sources of dioxin emissions in Mexico. In the document cited, this source is identified as less serious than only two other diffuse sources: uncontrolled household trash burning and fires in sanitary landfills. The yearly emissions of dioxins and furans from agricultural burning in Mexico are estimated at 750.34 g i-TEQ_{DF}.[11]

In Mexico there is a widely held traditional practice of agricultural burning aimed at eliminating undesired waste after harvesting grains, such as rice, corn and wheat, and beans. It is also common to burn branches and weeds from fruit and walnut tree orchards and vineyards, and also poultry and livestock manure. Unfortunately, we find that plastic gunny sacks and other plastic materials used in greenhouses and ditches are also burned, as well as empty bags from insecticides and fertilizers, and paper and plastic materials used for protecting workers (used, for example, in banana and date fields), plus drying racks and other materials—combined with waste from harvested crops, contributing even more contamination.

"Slash and burn" agriculture has traditionally been used in Mexico for the purpose of rapidly incorporating certain nutrients into the soil, getting rid of weeds and eradicating pests. Nevertheless, this practice is highly contaminating and if adequate precautions are not taken, small, untended fires get out of control and become huge fires. Also, the use of fire in preparing agricultural fields has its consequences, including soil erosion, loss of nutrients and, in the long term, reduced productivity. When burning is first used, this practice appears to help promote new growth, but a decrease in fertility will be noted over the long term, together with deterioration of the plant cover and a loss of soil moisture. All of this translates into reduced crop yields and the eradication of beneficial microorganisms and insects.[12]

Sometimes agricultural burning is considered to be necessary, for example, in order to prevent the spreading of certain pests.[13] Other reasons are economic in nature, since if the straw left behind from harvesting is burned, costs can be reduced by not using machinery and diesel fuel, as it will not be necessary to incorporate the straw into the soil with a disc plough. Burning waste after harvest also saves time in preparing the soil for the next planting. However, over the long term, deterioration of plant cover and a diminishing of soil quality are inevitable.

Controls and Alternatives to Agricultural Waste Burning

- It is possible to decrease emissions by establishing a controlled, phased program for burning, designed to produce less smoke in a determined period of time. It is also important to assure that waste is as dry as possible before burning.

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[8] Raj Boopathy et al. 2002. Sugar cane (*Saccharum officinarum*) burning and asthma in southeast Louisiana, USA. *Bulletin of Environmental Contamination and Toxicology* 68: 173-79.
[9] See <www.burningissues.org>.
[10] Pablo Maíz Larralde. 2010. Mexico 2004 PCDD/F National Releases Inventory Revision: recent studies on quantification of emissions from specific sources. In: *Fourth Workshop on Sources and Measurements of Dioxins, Furans and HCB,* Commission for Environmental Cooperation, Mexico City, November 2010. <www.cec.org/Storage.asp?StorageID=4098>.
[11] International Toxic Equivalents (i-TEQs) for dioxins and furans (DF).
[12] United Nations Food and Agriculture Organization (FAO). Transición de la quema a la práctica de no quema, at: <www.fao.org/climatechange/34147-0513b607625cf6b489b18b544c3452038.pdf>.
[13] M. Quintero Núñez and A. Moncada Aguilas. 2008. Contaminación y control de las quemas agrícolas en Imperial, California y Mexicali, Baja California, *Región y Sociedad* 43(XX): 3-24.

- Collection of waste and its reuse for:
    - Fuels, such as ethanol and biogas (methane)
    - Compost production
    - Animal feed
    - Construction materials (adobe blocks made with straw, an excellent thermal insulator)
    - Growing mushrooms and other crops
    - Beds for stables (more information: <www.arb.ca.gov/smp/biomass/biomass.htm>)

- Reincorporation into cropland to increase soil fertility and improve its organic composition. It may be difficult at first, but in the long term, the agricultural characteristics of the soil will be improved. The advantage of using "stubble cultivation" is to spread out more than 50% of the waste (leaves, stalks and brush) from the previous harvest in the land plot, and in this way: reduce the use of agrochemicals, conserve soil moisture, diminish erosion and reduce the risk of fires. For more information regarding sustainable agriculture and stubble cultivation, see <www.inca.gob.mx/videoteca/> and <www.sagarpa.gob.mx/desarrolloRural/Documents/cambioclimatico/Tecnologias_mitigacion.pdf>.

- There are Mexican states in which, due to air quality problems, the burning of agricultural waste has been prohibited. In the state of Guanajuato, for example, a program has been implemented to provide agricultural producers with machinery for harvesting and reusing waste as livestock feed, thereby avoiding agricultural burning in rural areas.[14]

- If you have reached the conclusion that your only option is agricultural burning, consult <www.conafor.gob.mx:8080/biblioteca/ver.aspx?articulo=446> to learn how to use this practice in a responsible manner. For more information regarding agricultural burning, also visit <www.epa.gov/agriculture/tburn.html>.

- Energy extraction. There are a number of ways commonly used to extract energy from biomass, such as corn, sugar cane, wood, grasses and some agricultural waste. There are two basic alternatives to obtaining energy from biomass: burning it or converting it into fuel. Even though it is preferable for the environment, converting biomass into biofuel continues to be somewhat inefficient and particularly costly, and at this time burning biomass to produce energy is more affordable.

[14] See <www.guanajuato.gob.mx/noticia_detalle.php/7794>.

APPENDIX K

Growing Sugar Cane

GROWING SUGAR CANE

OUR NATURAL MILL PROCESS

THE HISTORY OF SUGAR

Sugar cane grows exceptionally well in the deep muck soils of Florida's Everglade Agricultural Area (EAA), south of Lake Okeechobee. Ample rainfall and tropical temperatures during summer months create ideal growing conditions, and the moderate winters are perfect for harvesting.

Planting takes place from September through January. Cane seeds, produced in white or reddish plumes at the tops of mature cane stalks are too small to be planted directly in the field. Instead, stalks are harvested from mature fields and cut into short 20-inch segments then placed in furrows and covered with soil. Sprouts from the cane stalks emerge two to three weeks later.

After 10-12 months, the sugar cane is ready for harvest. All of Florida Crystals' fields are mechanically harvested, using combine-like machines that slice the cane stalks just above root level. The cut cane is then transferred to trailers and taken to the mill for processing.

Sugar cane is typically grown for three years, then the fields are rotated with rice. Rotating sugar cane with rice restores the soils fertility, kills unfriendly burrowing nematodes and provides a rich habitat for many species of wading birds.

Chocolate Filled Valentine Sugar Cookies

APPENDIX L

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PAHs, carbonyls, VOCs and $PM_{2.5}$ emission factors for pre-harvest burning of Florida sugarcane

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ABSTRACT

Emission factors (EFs) for hazardous polycyclic aromatic hydrocarbons (PAHs), carbonyls, and volatile organic compounds (VOCs) as well as other species such as $PM_{2.5}$, elemental carbon (EC), organic carbon (OC), and tracer compounds (e.g., levoglucosan (LG) and other sugars) were investigated for sugarcane pre-harvest burning in Florida. A combustion chamber was used to simulate field burning conditions for determining EFs of both dry leaf and whole stalk biomass burning. Samples were collected from the chamber's exhaust duct following EPA sampling methods. The total PAH EFs were 7.13 ± 0.94 and 8.18 ± 3.26 mg kg^{-1} for dry leaf and whole sugarcane stalk burning, respectively. Carbonyl EFs were 201 ± 39 and 942 ± 539 mg kg^{-1} for dry leaf and whole stalk burning, respectively. PAH and carbonyl emissions were dominated by lower molecular weight compounds (e.g., naphthalene and formaldehyde, respectively). Of the aromatic VOCs studied, benzene was the predominant species. The $PM_{2.5}$ EF was 2.49 ± 0.66 g kg^{-1}, which is in range of the current published AP-42 EFs for particulate matter emissions from sugarcane burning and other sugarcane studies. The OC, EC and LG EFs were 0.16 ± 0.09 g kg^{-1}, 0.71 ± 0.22 g kg^{-1}, and 7.87 ± 5.42 mg kg^{-1}, respectively. EFs of gaseous pollutants were generally lower than EFs from studies of agricultural residue combustion, likely due to the high combustion efficiency observed in this study.

1. Introduction

Prescribed burning of sugarcane fields is practiced to facilitate harvesting by quickly and cheaply removing excess biomass, to reduce dangers from snakes and insects (Gullett et al., 2006), and to increase the sugar content of the stalk by water evaporation (Zamperlini et al., 2000). Current emission factors (EFs) for sugarcane pre-harvest burning, published by the United States Environmental Protection Agency (US EPA) in AP-42, are currently rated as unreliable ("category D") and are only available for total particulate matter, carbon monoxide (CO), and total hydrocarbons (USEPA, 1995). The current EFs are based on one study of Hawaiian sugarcane and it is possible that the EFs of other types of sugarcane may differ (Gullett et al., 2006). Additionally, there are no compound-specific EFs for hazardous air pollutants (HAPs) such as PAHs, VOCs, or carbonyls.

In Brazil, numerous studies have been conducted on the environmental impacts of sugarcane burning. Lara et al. (2005) used principal component analysis (PCA) to study the properties of aerosols from sugarcane burning and to assess their contribution to ambient particle concentrations in Piracicaba, Brazil. They concluded that sugarcane fires were the largest source of particulate matter in the area—specifically, 60% of the fine mode ($PM_{2.5}$) mass and 25% of the coarse mode ($PM_{10-2.5}$) mass. Kirchhoff et al. (1991) also observed elevated ambient CO and ozone (O_3) concentrations during sugarcane burning periods in Sao Paulo, Brazil.

Gullett et al. (2006) investigated the EFs of polychlorinated dibenzodioxins and dibenzofurans (PCDD/Fs) from Floridian and Hawaiian sugarcane burning in a combustion chamber. They found Hawaiian sugarcane EFs to be over 10 times higher than Florida sugarcane EFs. One proposed explanation for the difference was the

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significantly higher (13 times) chlorine content of the Hawaiian sugarcane. Meyer et al. (2004) also studied the EFs of PCDDs and PCDFs from Australian sugarcane in the field and in a combustion chamber. Meyer's results were comparable to the EFs from Gullett's research for Florida sugarcane. The differences between these two studies highlight the important effects of biomass source and burning conditions on pollutant EFs.

The objective of this study was to develop EFs for specific hazardous air pollutants (PAHs, carbonyls and VOCs) and $PM_{2.5}$ from sugarcane burning using a combustion chamber that simulated field burning. In addition to HAPs, elemental carbon (EC), organic carbon (OC) and several tracer compounds (levoglucosan, mannosan, galactosan, xylitol, xylose) were also investigated, which can be useful for developing source profiles for source apportionment studies and source impact assessment.

2. Methodology

Fig. 1 shows a schematic of an open burning combustion chamber built to simulate field burning. The chamber consisted of a cylindrical combustion section ($\sim$0.9 m^3) where the sugarcane was burned, a cone ($\sim$0.27 m^3) to funnel the exhaust gas from the combustion section to the exhaust stack, and a stack (diameter = 20 cm) where the exhaust gas was sampled. The floor of the combustion chamber consisted of an open grid that ensured adequate air availability for the combustion process. The chamber and stack surfaces were lined with aluminum foil to prevent catalytic formation of PAHs, which is associated with steel materials (Jenkins et al., 1996b).

To enhance mixing of the combustion gases before sampling, two baffles made of aluminum were placed at the entrance of the stack. Samples were collected from the stack approximately 8.5 duct diameters (i.e., 1.7 m) downstream of the baffles (USEPA, 2000a; USEPA, 2000c). A draft inducer was employed near the exit of the stack to stabilize the pressure as well as induce excess air into the chamber.

Sugarcane biomass was obtained from Florida fields in Belle Glade and Citra, FL and all collected biomass was treated and stored in a consistent manner. Most experiments used solely dry sugarcane leaves, which were fed into the chamber at a rate of approximately 100 g every 40 s to maintain near constant burning conditions. A few experiments were conducted using whole sugarcane stalks, which contained both dry and green leaves (with higher moisture contents), to investigate the effects of biomass composition and burning conditions on the EFs. When burning whole stalks, burning conditions were highly variable both during the experiment and across experiments due to the heterogeneous nature of the biomass.

Due to limitations in space, only one pollutant was sampled per experiment. Temperature and pressure were measured in the stack at regular time intervals in order to calculate the stack velocity and flowrate following EPA Method 2 (USEPA, 2000c). Temperature was monitored using a thermocouple, and the differential pressure was measured using an s-type pitot tube. Flue gases were monitored in select experiments to evaluate the combustion conditions of the chamber. Since pollutants form through incomplete combustion, the combustion efficiency (CE) is an important parameter to monitor when investigating emissions. To evaluate the combustion conditions of the experimental system, CO, CO_2, and O_2 concentrations were measured using a CO analyzer (48C, Thermo Electron Corporation), a CO_2 Analyzer (1400, Servomex) and an O_2 analyzer (Rapidox 3000, Cambridge Sensotec) in select experiments.

2.1. PAHs

PAH sampling and analysis was based on EPA Method TO-13A (USEPA, 1999a) adapted for stack sampling. Three experiments using solely dry leaves and two using whole stalks with a mixture of dry and green leaves were conducted. Two experiments collected duplicate samples; therefore, seven PAH samples were collected in total. Sampling times ranged from 30 to 70 min. On average, 4.5 kg of biomass was burned in dry leaf experiments and 8.8 kg in whole stalk experiments. Exhaust air was drawn isokinetically from the stack and passed through a quartz filter to collect particulate PAHs and then through a sorbent cartridge containing polyurethane foam (PUF) and XAD-2 resin to collect semivolatile organic compounds (SVOCs). Samples were sent to Columbia Analytical Services where they were Soxhlet extracted in a mixture of 10% diethyl ether in hexane, concentrated, and analyzed by gas chromatography–mass spectrometry (GC–MS) in accordance with EPA Method TO-13A (USEPA, 1999a). Method reporting limits (MRLs) ranged from 9.0 to 16 μg m^{-3} for naphthalene and 0.9 to 7.5 μg m^{-3} for all other PAH compounds.

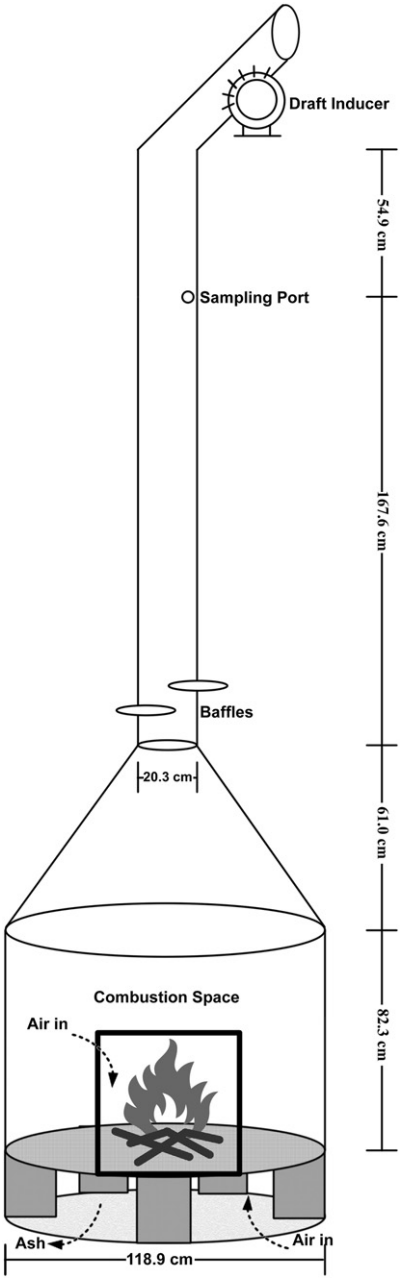

Fig. 1. Schematic of the combustion chamber.

2.2. Carbonyls

Carbonyl sampling and analysis followed EPA Method TO-11A (USEPA, 1999b) adapted for stack sampling. Five experiments were conducted—three for dry leaves and two for whole stalk biomass. Parallel duplicate samples were collected in four out of five experiments. Sample times ranged from three to ten minutes. On average, approximately 1 kg of biomass was burned in experiments, except one experiment where approximately 250 g was burned. Cartridges precoated with 2,4-dinitrophenylhydrazine (DNPH) (Supelco) were used to sample carbonyls. An LpDNPH ozone scrubber (Supelco) was applied to remove the interference of ozone during sampling. Following sampling, cartridges were shipped to Columbia Analytical Services where they were eluted with acetonitrile and analyzed by isocratic reverse-phase high-performance liquid chromatography (HPLC). All sample extraction and analysis procedures were performed in accordance with EPA Method TO-11A (USEPA, 1999b). MRLs ranged from 13 to 140 μg m^{-3} for target compounds.

2.3. VOCs

EPA Method 18 was applied for the sampling of VOCs (USEPA, 2000b). Gas samples were collected in Tedlar bags using a Vac-U-Chamber Kit (SKC). Four experiments were conducted for VOC sampling for dry leaves. Sampling time was approximately three minutes and 300 g of biomass was burned. Samples were analyzed by GC–MS for benzene, toluene, *o,m,p*-xylenes, ethylbenzene and styrene following EPA Method TO-15 (USEPA, 1999c). The MRL for *m,p*-xylenes was 10 μg m^{-3} and for all other target compounds was 5.0 μg m^{-3}.

2.4. $PM_{2.5}$

$PM_{2.5}$ sampling was based on EPA's Other Test Methods (OTM) 27 and 28 (USEPA, 2008a; USEPA, 2008b) with a few modifications to the impinger train. Exhaust gas was sampled isokinetically, passed through an in-stack, size selective cyclone (Sierra Instruments, Inc. Series 280 Cyclade™) to remove particles larger than 2.5 μm and then passed through a glass fiber filter (Type A/E, Gelman Sciences, Inc.) to collect the filterable particulate matter. In some experiments, an impinger train and Teflon filter (Zefluor, Pall Life Sciences) were added to cool the exhaust gas and collect the condensable particulate matter (CPM). Five PM sampling experiments were conducted. All experiments used dry leaves. $PM_{2.5}$ sampling times ranged from 1 to 18 min and mass loadings ranged from 0.2 to 2.4 kg.

The glass fiber filters were pre- and post-conditioned for at least 24 h for weighing. The front half of the filter holder and cyclone exit were rinsed with acetone, transferred to a pre-weighed weighing dish and also weighed as part of the filterable PM mass. For the CPM recovery, the impinger train was rinsed with deionized (DI) water, acetone, and methylene chloride. The rinses were carried out through procedures detailed in OTM 28 to determine the final CPM residue weight (USEPA, 2008b). A microbalance (Model MC 210 S, Sartorius Corp.; readability − 10 μg) was used for all weighing. Filters and dishes were weighed 3 times to determine the average mass and were repeatedly weighed at intervals of at least 6 h to ensure the weight was constant (defined as having a weight change of less than 0.5 mg).

2.5. EC and OC

Three $PM_{2.5}$ samples were collected on tissuquartz filters (Pall Life Sciences) to determine the emission factors of EC, OC and other tracer compounds. Prior to sampling, tissuquartz filters were baked at 550 °C for 12 h and allowed to cool for 12 h to remove any residual carbon in the filters. In these experiments, 0.5 kg of dry leaves was combusted and sampling times were, on average, 3.9 min.

Tissuquartz filters were sent cold to the Research Center for Environmental Changes, Academia Sinica in Taipei, Taiwan, where they were analyzed for EC and OC using a semi-continuous OCEC Carbon Aerosol Analyzer (Sunset Laboratory, Model 4) following the National Institute for Occupational Safety and Health (NIOSH) Method 5040 (NIOSH, 1999). As part of the analysis, a portion of each filter was heated at distinct intervals to 870 °C in a pure helium atmosphere to volatilize the organic carbon (OC). The sample was then cooled and reheated at discrete intervals again up to 870 °C in a 2% oxygen in helium atmosphere to evolve the elemental carbon (EC). The evolved carbon fractions were subsequently oxidized to CO_2 which was quantified by a non-dispersive infra-red (NDIR) detector. Correction for the pyrolized OC fraction was accomplished by monitoring the transmittance of a laser beam through the sample filter.

For the determination of selected polar organic compounds, including molecular tracers for biomass burning, a 2.2 cm^2 filter punch was extracted in 2 mL of ultrapure water under ultrasonic agitation for 1 h. The extract was filtered through a syringe filter (0.45 μm, PTFE, Pall Life Sciences) to remove insoluble materials. Anhydrosugars (levoglucosan, mannosan and galactosan), sugars (xylose) and sugar alcohols (xylitol), were quantified by high-performance anion exchange chromatography (HPAEC) on a Dionex ICS-3000 system equipped with an electrochemical detector (Dionex, USA). The MRL for this method is 0.1 ng. A more detailed description of the HPAEC analysis method can be found in Engling et al. (2009).

2.6. Calculations

CE is defined as the fraction of carbon released as CO_2. In this study, the modified combustion efficiency (MCE) was determined using Equation (1), which assumes all of the carbon is released as CO or CO_2.

$$\mathrm{MCE} = \Delta[CO_2]/(\Delta[CO_2] + \Delta[CO]) \quad (1)$$

where, Δ[CO] and Δ[CO_2] are the mass concentrations of CO and CO_2 in excess of the background. Previous studies have demonstrated that over 95% of carbon is released as CO or CO_2; therefore, it is reasonable to estimate the CE without hydrocarbons or particulate matter (Ward and Hardy, 1991; Gupta et al., 2001; Chen et al., 2007).

EFs were calculated using Equation (2) (Dhammapala et al., 2006), which assumes the chamber to be well mixed.

$$\mathrm{EF} = (\Delta C_x \times Q_{\mathrm{chamber}} \times t)/m_{\mathrm{burned}} \quad (2)$$

where ΔC_x is the measured pollutant concentration above the ambient level, Q_{chamber} is the flowrate through the chamber, t is the sampling time, and m_{burned} is the mass of biomass burned. In the cases where ambient concentrations were below detection limits, the background concentration was assumed to be zero for the EF calculations. All sample volumes and chamber flowrates were corrected to standard conditions (20 °C and 1 atm).

2.7. Quality assurance/quality control (QA/QC) measures

Ambient samples were obtained during each sampling campaign to determine the background concentrations of the compounds of interest. Field blank samples were also collected to ensure no contamination was introduced during sample storage and handling. Ambient and field blank samples were below analytical detection limits in all cases; therefore, the data was not blank corrected. Method blank samples were applied with each batch of samples analyzed to monitor for contamination and interferences in the analytical methods. Parallel duplicate samples were collected in select PAH, carbonyl, and VOC experiments to

Table 1
Summary of MCE, CO and CO_2 EFs.

	MCE (%)	Average [CO] (mg m^{-3})	Average [CO_2] (mg m^{-3})	CO EF (g kg^{-1})	CO_2 EF (g kg^{-1})
Present Study	98.5 ± 0.2	325	45,232	9.2 ± 3.3	1255 ± 287
AP-42[a]	NA	NA	NA	30–40	NA
Yokelson et al., 2008	97.6	NA	NA	28.3	1838

[a] USEPA, 1995.

measure the precision (as percent difference) of the sampling and analysis procedures.

For PAH analysis, a laboratory control sample (LCS) and a duplicate laboratory control sample (DLCS) were run with each batch of samples to monitor the extraction efficiency of target analytes. Additionally, isotopically labeled surrogate compounds (fluorene-d10 and pyrene-d10) were spiked onto the method blank, LCS, DLCS, and each sample as internal standards. Analyzed PAH concentrations were corrected to reflect compound specific (LCS and DLCS) and sample specific (surrogate compounds) extraction efficiencies.

For carbonyl sampling, a breakthrough test was conducted by placing two DNPH cartridges in series. The backup cartridge was analyzed to verify no sampling breakthrough occurred. Regarding VOCs, with each batch of samples analyzed, an LCS was prepared by spiking a Tedlar bag with known concentrations of the target analytes. The bag was analyzed to evaluate the analytical method's recovery. LCS recoveries for all compounds ranged from 79 to 98%. Additionally, surrogate compounds (1,2-dichloroethane-d4 and toluene-d8) were spiked into each sample analyzed to measure their recovery. Recoveries of 1,2-dichloroethane-d4 and toluene-d8 ranged from 94 to 111% and 95 to 103%, respectively. All recoveries were within the acceptable criteria of the analytical method.

A study was also conducted to determine the recovery efficiencies of target VOC compounds. A canister was spiked with compounds of known concentrations and then was sampled using the Tedlar bag and Vac-U-chamber system. The recovery efficiencies for the target compounds were, benzene: 80%, toluene: 79%, ethylbenzene: 80%, *m,p*-xylenes: 75%, styrene: 51%, and *o*-xylene: 71%. The recovery for higher molecular weight compounds, such as styrene, was rather low using this sampling method.

3. Results and discussion

3.1. Flue gases

Flue gases were monitored for three burning events where dry leaves were burned. Table 1 summarizes the average CO and CO_2 concentrations, the CO and CO_2 EFs, and the average MCE. The high MCE observed, 98.5 ± 0.21%, indicates the dominance of flaming combustion in this studied scenario.

3.2. PAHs

Species specific PAH EFs are presented in Table 2. The total PAH EF for dry leaf burning was 7.13 ± 0.94 mg kg^{-1}. Based on one sample pair, the precision was 5%. This EF does not include any emissions of higher molecular weight compounds, which were below detection limits in these experiments. The whole stalk EF was slightly higher than dry leaf experiments—8.18 ± 3.26 mg kg^{-1}. Based on one sample pair, the precision was 15%.

PAH emissions were dominated by low molecular weight compounds (i.e., two and three ring PAH compounds). In fact, naphthalene (2-ringed) contributed to 66% of the overall EF, on average. Three-ring PAHs (acenaphthylene, acenaphthene, fluorene, phenanthrene and anthracene) contributed to 27% of the total PAH EF and 4-ring PAHs (fluoranthene, pyrene, benzo[a]anthracene and chrysene) contributed to 8% of the total EF, on average.

PAH concentrations for the whole stalk burning (223–306 μg m^{-3}) were slightly higher than for dry leaf burning (119–136 μg m^{-3}). As other studies have demonstrated, moisture content (MC) has an important impact on emissions (Hays et al., 2005; McMeeking et al., 2009; Simoneit, 2002). Generally, higher MCs inhibit combustion by lowering the temperature and CE, leading to higher pollutant emissions. However, at very low moisture contents the biomass burns quickly, creating oxygen-limited conditions leading to a decrease in the CE.

In addition to CE, PAH formation is sensitive to temperature—formation is supported at high temperatures (in excess of 500 °C) (Conde et al., 2005). While higher MC fuels may exhibit lower CEs, they also likely have a lower combustion temperature, possibly inhibiting PAH formation (Korenaga et al., 2001). This may explain why whole stalk PAH EFs were only slightly higher than dry leaf EFs. It is apparent that there are

Table 2
Summary of PAH EFs (mg kg^{-1}).

	Sugarcane (present study)		Other references
	Dry leaves	Whole stalks	
Naphthalene	4.83 ± 0.72	5.24 ± 2.45	1.330–347.988[c,d]
Acenaphthylene	0.78 ± 0.09	0.80 ± 0.30	3.5[a], 6.9[b], 0.308–22.080[c]
Acenaphthene	ND	0.11	0.22[a], 0.60[b], 0.013–17.936[c]
Fluorene	0.26 ± 0.05	0.27 ± 0.20	0.83[a], 1.6[b], 0.046–3.277[c]
Phenanthrene	0.73 ± 0.10	0.87 ± 0.25	4.1[a], 4.0[b], 1.320–20.923[c]
Anthracene	0.14 ± 0.03	0.15 ± 0.06	1.4[a], 1.2[b], 0.061–3.977[c]
Fluoranthene	0.20 ± 0.02	0.30 ± 0.05	1.4[a], 1.2[b], 0.425–6.654[c]
Pyrene	0.18 ± 0.01	0.27 ± 0.05	1.3[a], 1.1[b], 0.337–5.031[c]
Benz[*a*]anthracene	ND	0.05 ± 0.01	0.87[a], 1.1[b], 0.060–2.276[c]
Chrysene	ND	0.08 ± 0.02	0.078–2.326[c]
Benzo[*b*]fluoranthene	ND	0.06 ± 0.00	0.47[a], 0.31[b], 0.018–9.311[c]
Benzo[*k*]fluoranthene	ND	0.03 ± 0.01	0.51[a], 0.44[b], 0.30–4.249[c]
Benzo[*a*]pyrene	ND	0.04 ± 0.01	0.39[a], 0.47[b], 0.006–14.336[c]
Indeno[1,2,3-*cd*]pyrene	ND	0.03 ± 0.00	0.032[a], 0.054[b], 0.070–4.836[c]
Benzo[*g,h,i*]perylene	ND	0.03 ± 0.00	0.083[a], 0.19[b], 0.001–1.046[c]

[a] Dhammapala et al. (2007), wheat.
[b] Dhammapala et al. (2007), Kentucky bluegrass.
[c] Jenkins et al. (1996b), agricultural and forest biomass.
[d] Data flagged as questionable by Jenkins et al. (1996b).

numerous factors that impact emissions and these relationships are not always straightforward.

As naphthalene consistently comprises a large portion of the PAH emissions, it serves as a good indicator of the total PAH concentration. Conde et al. (2005) found a strong relationship between the total PAH concentration and the naphthalene concentration. Naphthalene is one of the first PAH compounds formed and is one of the most stable. It also serves as a building block for further PAH formation, making it a good indicator of total PAH formation (Conde et al., 2005). In the present study, strong relationships were found between naphthalene, acenaphthylene, and phenanthrene concentrations and the total PAH concentration, as summarized in Fig. 2. The results suggest that measuring a few select PAH compounds could infer the total PAH emissions from a particular source.

Table 2 compares the sugarcane EFs to different agricultural residues from other studies. Sugarcane EFs are comparable, but are generally lower than EFs published for other agricultural materials. The relative abundance of acenaphthylene and phenanthrene is consistent for all fuel types. It addition to the obvious difference of fuel type, the lower EFs exhibited in this study can be attributed to the high MCE observed in this study ($\sim$99%).

3.3. Carbonyls

Five experiments were performed for carbonyl sampling—3 for dry leaves and 2 for whole sugarcane stalks. The mean total carbonyl EFs were 202 $\pm$ 39 mg kg^{-1} (average 25% precision based on 2 sample pairs) and 942 $\pm$ 539 mg kg^{-1} (average 11% precision based on 2 sample pairs) for dry leaf and whole stalk burnings, respectively. Table 3 summarizes the compound specific carbonyl EFs as well as carbonyl EFs from other studies. In all experiments, formaldehyde was the most dominant carbonyl compound accounting for over 50% (ranging 51–78%) of quantified carbonyl emissions. Acetaldehyde was the second most abundant compound followed by propionaldehyde.

As with the dominant PAH compounds, formaldehyde and acetaldehyde concentrations were highly correlated with the total carbonyl concentrations. Fig. 3 shows the correlations between formaldehyde and acetaldehyde concentrations and the total carbonyl concentration. Because of their strong correlation, formaldehyde and acetaldehyde could serve as predictor compounds for total carbonyl emissions.

Whole stalk burning exhibited considerably higher EFs than dry leaf experiments for all compounds except valeraldehyde. The ratio of whole stalk EFs to dry stalk EFs ranged from 0.8 (valeraldehyde) to 7.2 (acetaldehyde). The higher EFs from whole stalk experiments can be partially attributed to the high moisture content of the fuel. Whole stalk experiments also exhibited higher EF variability between experiments, which can be attributed to variable burning conditions and differences in the biomass source. Significant differences in the burning conditions were observed during the two whole stalk experiments. The first fire was much more intense—the average stack temperature was 315 °C and 1 kg of biomass burned in 3 min, whereas in the later experiment only 253 g of biomass burned in 3 min and the average temperature was around 63 °C. The different burning conditions were a result of different initial loading conditions and operator differences. The variation in the EFs can be attributed to these differences in burning conditions. The more intense fire was characterized by more flaming combustion as compared to the lower temperature fire, which exhibited more smoldering combustion. Therefore, in addition to the differences in burning conditions and MC, the biomass composition, condition, and treatment practices (which differed for the different growing areas) may have also influenced the EFs.

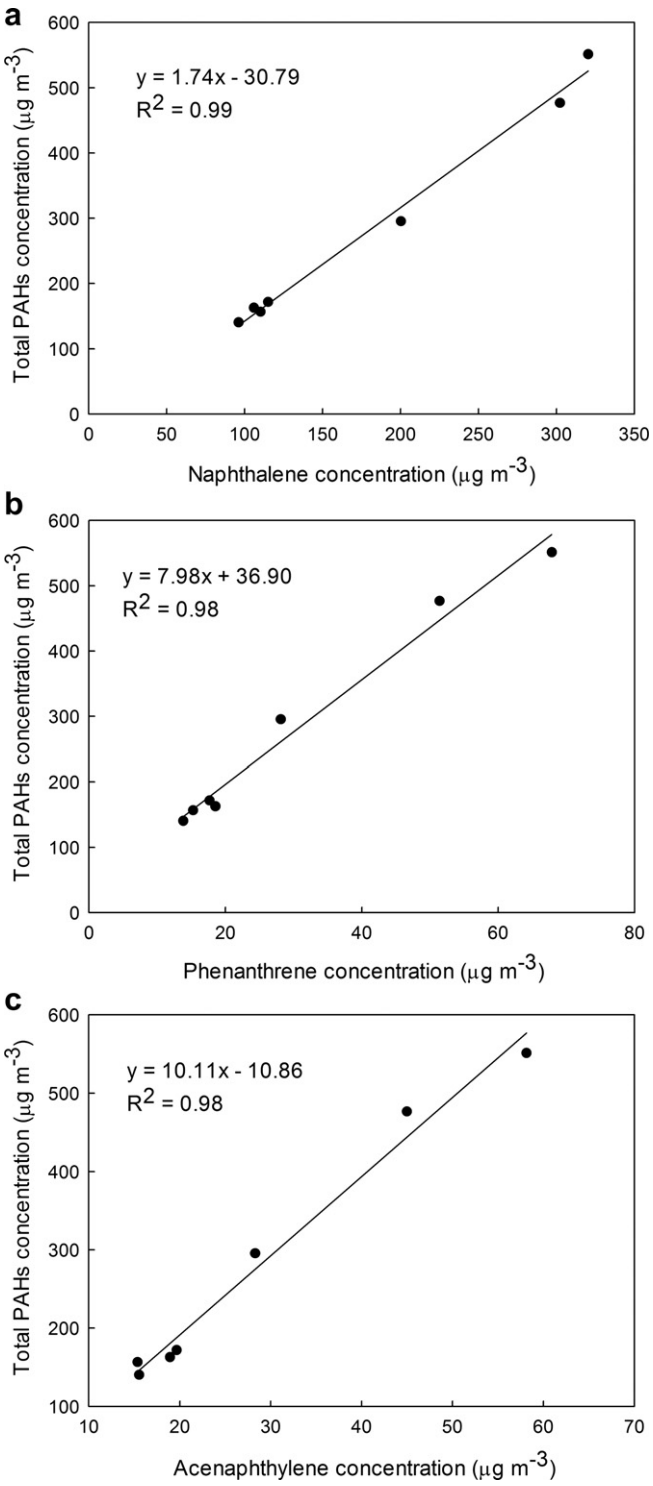

Fig. 2. Individual PAH concentrations vs. total PAH concentration (a) naphthalene (b) phenanthrene (c) acenaphthylene.

Compared to other studies, dry sugarcane EFs are lower than EFs determined for foliar fuels, but agree well with those of crop residue burned in a cookstove. The EFs for whole stalks agree well with those of the foliar fuels for formaldehyde, acetaldehyde and crotonaldehyde compounds. Formaldehyde followed by acetaldehyde and propionaldehyde were the most dominant compounds in all studies.

Yokelson et al. (2008) reported EFs for acetaldehyde and crotonaldehyde from sugarcane burning. It is obvious that the EFs

Table 3
Summary of carbonyl EFs (mg kg^{-1}).

	Sugarcane		Other references
	Dry leaves	Whole stalks	
Formaldehyde	150 ± 33.8	524 ± 316	304.9–1138.1[a], 78.3[b], 135[c], 422[d]
Acetaldehyde	44.8 ± 14.1	323 ± 198	358.0–1146.6[a], 85.1[b], 141[c], 86.3[d], 534[e]
Propionaldehyde	8.3 ± 5.9	51.0 ± 33.6	76.2–207.7[a], 21.6[b], 24.7[d]
Butyraldehyde	ND	3.7	43.0–123.4[a], 12.1[b], 25.6[c]
Benzaldehyde	2.2	9.6 ± 1.8	32.6–72.0[a], 22.2[b], 28.3[c], 12.2[d]
Valeraldehyde	2.5 ± 0.9	2.1 ± 0.5	0.6–175.8[a], 13.1[b], 9.9[c], 1.1[d]
2,5-dimethylbenzaldehyde	ND	33.0 ± 26.2	
Crotonaldehyde	ND	31.1 ± 5.5	39.4–74.9[a], 18.1[b], 32.3[c], 11.2[d], 91[e]

[a] Hays et al., 2002; range of mean EFs for various foliar fuels.
[b] Zhang and Smith, 1999; crop residue in a cookstove.
[c] Zhang and Smith, 1999, wood in a cookstove.
[d] Hedberg et al., 2002; birch wood in wood stove.
[e] Yokelson et al., 2008; sugarcane.

determined for dry sugarcane leaves are much lower than the EF reported by Yokelson et al. (2008). However, the upper range EF of our whole stalk experiments agrees well with that obtained by Yokelson et al. (2008). Yokelson et al. (2008) did not report the condition of sugarcane used (i.e., moisture content), but they reported a MCE of 97.6% (Table 1). They also used a different measurement technique—proton-transfer reaction mass spectrometry (PTR-MS). The differences between the biomass composition, burning conditions, and the measurement technique probably account for the observed differences in the EFs.

3.4. VOCs

VOC EFs are summarized in Table 4. The average precision was 15% based on four sample pairs. Benzene was the most abundant species followed by toluene. The abundance of benzene and toluene as the dominant aromatics is consistent with other biomass combustion studies (Hays et al., 2002).

The VOC EFs obtained in this study were significantly lower than those determined by Hays et al. (2002) for foliar fuels, but are similar with those reported by Jenkins et al. (1996a) for almond and walnut prunings. VOC EFs reported by Yokelson et al. (2008) are significantly higher than those determined in this study and are on par to Hays et al. (2002). As with the PAH and carbonyl EFs, differences in the sugarcane source, burning conditions, and measurement technique may explain the differences in the EFs obtained.

3.5. $PM_{2.5}$

The mean $PM_{2.5}$ EF was 2.49 ± 0.66 g kg^{-1}, based on dry leaf experiments. The EF is in excellent agreement with that determined by Yokelson et al. (2008) (2.17 g kg^{-1}) and is within the range of the current published PM EF (2.3–3.5 g kg^{-1}) for sugarcane pre-harvest burning (USEPA, 1995).

3.6. EC, OC, and tracer species

The average EFs for OC and EC were 0.16 ± 0.09 g kg^{-1} and 0.71 ± 0.22 g kg^{-1}, respectively. The high EC relative to OC ratio found in this study is unique for biomass burning. OC dominates EC in most biomass burning emissions; however, a few exceptions have been reported (McMeeking et al., 2009). Table 5 compares the EC and OC EFs determined in this study to other biomass fuels (which were analyzed by the same method—NIOSH 5040). Sugarcane OC EFs are on the low end of other reported EFs, whereas EC EFs are on the high end.

McMeeking et al. (2009) investigated a wide range of biomass materials and found a negative correlation between the MCE and OC EFs ($r^2 = 0.36$). They found that "leafy" fuels, which had lower MCEs, exhibited the highest OC EFs. EC EFs increased with increasing MCE, particularly for MCE > 93%; however, EC's dependence on MCE ($r^2 = 0.09$) was not as strong as in the case of OC. Instead, EC and other inorganic emissions were found to be a stronger function of the fuel type and composition. Nonetheless, the high EC relative to OC emissions from sugarcane burning can be

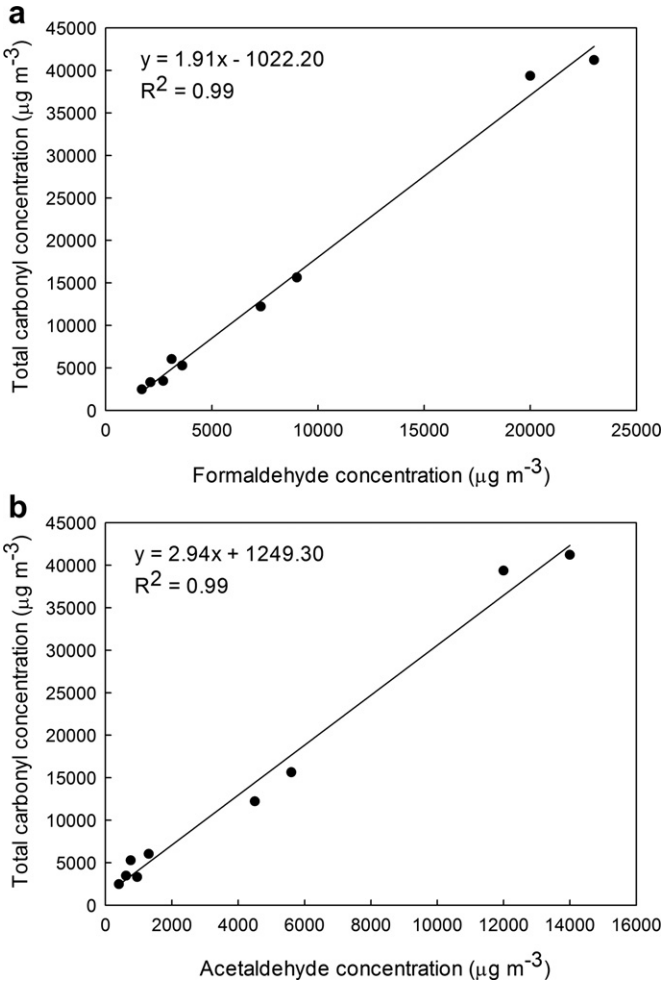

Fig. 3. Individual carbonyl concentrations vs. total carbonyl concentration (a) formaldehyde (b) acetaldehyde.

Table 4
Summary of VOC EFs (mg kg^{-1}).

	Present study	Other references
Benzene	16.5 ± 1.89	16–444[a], 207[b]
Toluene	5.2 ± 0.94	11–351[a], 120[b]
Ethylbenzene	0.81 ± 0.15	60[b]
m,p-Xylenes	0.94 ± 0.45	
Styrene	0.35 ± 0.25	7–652[a]
o-Xylene	0.29 ± 0.19	

[a] Jenkins et al., 1996a; agricultural materials.
[b] Yokelson et al., 2008; sugarcane.

partially attributed to the high MCE observed in this study and may also be a function of the biomass composition. The characteristic EC/OC ratio may be useful in future source apportionment studies to identify and quantify contributions from sugarcane burning.

Table 5 also reports EFs of several anhydrosugar compounds that are common products of the decomposition of cellulose. The EFs observed in this study are within the wide range of EFs reported by other studies, but are expected to be very dependent on the specific burning conditions (Engling et al., 2006). These compounds, particularly levoglucosan (LG) and mannosan (MN), can be used to develop source profiles to be used for source apportionment studies. Specifically, ratios of individual tracer species that are characteristic of certain biomass types can be utilized to provide additional insights into the actual biomass source. For instance, characteristic LG/MN ratios of 3–5 for softwood, 15–25 for hard wood and 40 for rice straw have been reported in previous studies (Engling et al., 2009), while the average LG/MN ratio for sugarcane was 10.

3.7. Emission estimates

In order to understand the magnitude of emissions produced from sugarcane burning, the total yearly emissions of the HAPs investigated in this study were estimated based on the EFs determined in this study. The emission estimates were then compared to the Palm Beach County (PBC) emission inventory from the 2005 national emission inventory and the state of Florida emissions inventory to estimate the relative importance of this practice (USEPA, 2010). To estimate the total emissions, the upper value of the 95% confidence interval for the whole stalk experiments were used when available. For VOC and PM experiments, where whole stalks were not tested, the upper value of the 95% confidence interval for dry leaves was used. EFs were multiplied by the published fuel loading for sugarcane, 7 tons per acre (USEPA, 1995), and an estimate of the sugarcane acreage burned, 335,650 acres, which is based on the 2008 agricultural year.

Table 5
Summary of $PM_{2.5}$, EC and OC EFs.

	Sugarcane	Other references[a]
$PM_{2.5}$ (g kg^{-1})	2.49 ± 0.66	3–12.1[b], 3.4–38.3[c], 10.8–28.4[d], 4.71–12.95[e]
OC (g kg^{-1})	0.16 ± 0.09	1.9–6.9[b], 0.5–26[c], 8–27.8[d], 1.23–8.94[e]
EC (g kg^{-1})	0.71 ± 0.22	0.35–0.63[b], 1.4–8.1[c], 0.2–1.3[d], 0.17–0.52[e]
Levoglucosan (mg kg^{-1})	7.87 ± 5.42	150–350[b], 0.14–350[f]
Mannosan (mg kg^{-1})	0.78 ± 0.12	0.011–10.5[f]
Galactosan (mg kg^{-1})	0.72 ± 0.33	0.005–66.5[f]
Xylitol (mg kg^{-1})	0.20 ± 0.14	
Xylose (mg kg^{-1})	0.51 ± 0.10	

[a] EF range is reported when several fuels are reported.
[b] Dhammapala et al., 2007; wheat and Kentucky bluegrass.
[c] McMeeking et al., 2009; various fuels.
[d] Hays et al., 2002; various foliar fuels.
[e] Hays et al., 2005; wheat and rice straw.
[f] Oros et al., 2006; grasses.

Table 6
Contribution of sugarcane burning to emissions inventories.

Compound	Fraction of inventory (%)	
	Palm Beach County	Florida
PAHs		
Naphthalene	1.7	1.3
Acenaphthylene	69	11
Acenaphthene	52	6.2
Fluorene	78	17
Phenanthrene	75	9.5
Anthracene	69	3.6
Fluoranthene	69	3.7
Pyrene	61	2.5
Benz[*a*]anthracene	50	1.0
Chrysene	67	1.5
Benzo[*b*]fluoranthene	77	11
Benzo[*k*]fluoranthene	61	1.4
Benzo[*a*]pyrene	62	2.1
Indeno[1,2,3-*cd*]pyrene	54	0.9
Benzo[*g,h,i*]perylene	23	0.6
Carbonyls		
Formaldehyde	86	16
Acetaldehyde	91	29
Propionaldehyde	89	37
VOCs		
Benzene	3.2	ND[a]
Toluene	0.5	ND[a]
Ethylbenzene	0.4	ND[a]
Styrene	1.6	ND[a]
o,m,p-xylene (mixture)	1.8	ND[a]

[a] Not determined because of insignificance.

Table 6 shows the potential contributions of sugarcane burning to the PBC and Florida state inventories for HAPs. Sugarcane field burning did not contribute substantially (<5%) to VOC compound or naphthalene emissions. VOC emissions are dominated by gasoline sources (on-road and non-road equipment) and naphthalene emissions are dominated by industrial boiler fuel combustion in PBC. However, sugarcane field burning plays an important role in the yearly emissions of other PAH (23–78%) compounds and carbonyls compounds (86–91%) in PBC. Additionally, sugarcane burning is estimated to contribute a notable amount at the state level for certain compounds (i.e., acenaphthylene, fluorene, benzo[*b*]fluoranthene, formaldehyde, acetaldehyde, propionaldehyde).

It should be emphasized that field burning is characterized by different burning phases (i.e., smoldering, flaming) and is influenced by a number of variables (meteorological conditions, plant conditions, plant treatment, etc.). EFs are expected to be highly variable during the field burning process as well as during the harvesting period, as the meteorological and plant conditions may change drastically throughout the harvesting season. The EFs reported in this study are most representative of the flaming phase of combustion and may be a conservative estimate of emissions, since previous studies have shown that smoldering combustion exhibits significantly higher emissions of pollutants (Jenkins et al., 1996b).

It should be noted that the ambient concentration levels will depend on many factors such as weather patterns. Ambient air sampling and analysis is, therefore, an important task for providing the necessary data for human health and environmental impact assessment.

4. Conclusions

This study characterized the emissions from the pre-harvest burning of sugarcane fields. EFs were determined for a number of HAPs including PAH, carbonyl and VOC compounds as well as $PM_{2.5}$

in a combustion chamber. Tracer compounds were also investigated and specific compound patterns were identified, which may be helpful for developing source profiles for future apportionment studies.

In general, EFs were consistent between experiments and comparable to other published emission factors for sugarcane burning and other agricultural materials considering the differences in biomass composition, biomass source, and burning conditions. PAH and carbonyl emissions were dominated by low molecular weight compounds, such as naphthalene, formaldehyde and acetaldehyde. The most abundant aromatic VOC compounds measured were benzene and toluene. Additionally, a number of unique compound ratios were observed that may be helpful for source apportionment studies.

Consistent with previous similar studies, our experiments show that EFs are strongly impacted by burning conditions (temperature, intensity, fuel density, combustion efficiency) and biomass properties (moisture content, composition). For example, experiments that used biomass with higher moisture content (i.e., whole stalks) exhibited higher EFs. Also, more intense fires (characterized by higher fuel loading and temperatures) produced lower EFs.

The data from this research will allow the EPA to validate and expand the EFs published in AP-42 for sugarcane pre-harvest burning. The EFs can be used to more accurately calculate the annual emissions from sugarcane pre-harvest burning and to evaluate the contribution of this source to local and state pollutant inventories. Ultimately, with more reliable data, regulatory agencies will be able to more accurately model human and environmental exposure and subsequently, make better management and regulatory decisions.

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APPENDIX M

Louisiana
Smoke Management Guidelines for Sugarcane Harvesting

Training Summary Report - Summer 2000

Louisiana Department of Agriculture and Forestry

LSU AgCenter Research & Extension

Louisiana Smoke Management Guidelines for Sugarcane Harvesting

Introduction

Prescribed burning as a harvest management tool in sugarcane is a widely used practice. There are numerous environmental and public issues associated with this practice, and the state is instituting a voluntary smoke and ash management program to assist growers in addressing these issues. Additionally a number of research projects are under way to address possible remedies to cane burning. Growers should have a responsible attitude toward environmental and public issues while attempting to be as efficient as possible in sugar production. There are several objectives which growers should always strive to achieve with regard to smoke and ash management:

- Minimize the adverse effect caused by open field burning of sugarcane.
- Prevent it from being blown across public highways and airports.
- Prevent it from affecting public areas, especially public health facilities such as hospitals, clinics, nursing homes and doctors' offices, etc.
- Prevent it from affecting schools during times when students and teachers are present.
- Prevent it from affecting subdivisions, individual homes and other housing facilities.
- Minimize ash fallout that may result from burning sugarcane.

Growers should practice smoke and ash management by using recommended prescribed burning practices. Prescribed burning can be defined as the controlled application of fire to sugarcane fields under weather conditions that allow the fire to be confined to a predetermined area, in a manner that will produce the desired result of reducing trash in the delivered cane supply. Smoke and ash management can be defined as conducting a prescribed burn under recommended weather conditions and with burning techniques to lessen the impact of smoke and ash generated from prescribed burning on the environment, public health and welfare.

Application of these guidelines will minimize concentrations of smoke and ash in sensitive areas and assist in maintaining air quality standards. The use of these voluntary guidelines will allow the industry to manage smoke and ash from sugarcane burning more effectively. Although voluntary, growers are strongly encouraged to incorporate these guidelines as routine harvesting practices.

Objective

The guidelines are intended to manage smoke and ash from sugarcane prescribed burning operations to lessen their impact on public health and welfare. In sugarcane prescribed burning, it is recognized that numerous variables affect the fire behavior and resulting smoke and ash. These guidelines do not attempt to consider all the variables affecting smoke and ash behavior, but only offer basic guidance. Nothing in these guidelines shall be construed as allowing any person to be in violation of any regulations, laws, ordinances or orders of the state of Louisiana or other governmental entity having jurisdiction or to relieve any person from the consequences of damages or injuries that may result from burning activities because of negligence.

Administration, Training and Communications

The Certified Prescribed Burn Manager (CPBM) program is administrated by the Louisiana Department of Agriculture and Forestry. The Louisiana Department of Agriculture and Forestry (LDAF), the American Sugar Cane League and the LSU AgCenter developed the *Louisiana Smoke Management Guidelines for Sugarcane Harvesting* and will provide training and program information for growers.

Growers should make every attempt to provide education and training to their employees who conduct burning operations. A thorough explanation of the goals and recommendations will help employees understand the importance of smoke and ash management. Additionally, it should be emphasized that prescribed burning burns should not go unattended. Proper equipment for controlling and confining fires, including a water tank, should be available at all burns.

The sugarcane industry's ability to burn sugarcane is a significant economic factor for the survival of the sugarcane industry. Until proven technology allows economically efficient harvesting without burning, it is critical that growers and processors do the best job possible with regard to smoke and ash management. Louisiana is not the only state, nor is sugar production the only industry, facing this challenge. Every industry that uses prescribed burning recognizes that reducing or eliminating open field burning is one of the most important research topics facing it.

Researchers are working to identify the most effective techniques for handling the trash layer. In the meantime, many growers concerned about stand loss as a result of the trash mat will likely continue to burn the trash layer later in the year. The same smoke and ash management practices need to be followed, regardless of the time of the year the trash layers are burned.

Procedure

When prescribed burning of sugarcane is to be used, the recommended procedures are:

Step 1. Identify Areas Sensitive to Smoke and Ash
Step 2. Obtain Fire Weather Forecast
Step 3. Develop a Prescribed Burn Plan
Step 4. Determine Smoke Category Day
Step 5. Determine Smoke and Ash Screening Distance
Step 6. Determine Trajectory of Smoke and Ash Plume
Step 7. Evaluate the Prescribed Burn Results

Note: In sugarcane prescribed burning, it is recognized that numerous variables affect the fire behavior and resulting smoke and ash. These guidelines do not attempt to consider all the variables, but they offer only basic guidance.

Step 1. Identify Smoke Sensitive Areas

Obtain a map that shows each farm or field to be burned. Each map should show sensitive areas that are within a 20-mile radius from each field. Also identify all sensitive areas adjacent to or within the farm and mark them on your map. The map will then show the fields to be burned and all potential sensitive areas within 20 miles in all directions from your farm or field.

The first step toward effective smoke and ash management is the awareness of where people, buildings, utility structures and highways are located that could be negatively affected by open field burning. People with health problems who live in areas potentially affected by open field burning should be identified before the harvest season begins. This requires effective communications between growers and the public. When burning cane heaps that are harvested by soldier machines, the smoke normally does not contain much ash. However, hotter fires from standing cane cause rising ash that later "falls out." This can create a problem long distances from the burn. Determination of downwind sensitive areas that could be affected by burning cane is important to help reduce the impact of ash "fallout." Smoke and ash sensitive areas include airports, highways, communities, recreation areas, schools, hospitals, utility substations, subdivisions, individual homes, factories and nursing homes, etc.

Knowledge of power lines, substations and gas lines. Open field burning, especially burning of uncut fields prior to chopper harvesting, should be carefully undertaken when power and gas lines or utility substations are present in the field or surrounding fields. Fire can destroy wood utility poles and cause disruption of electrical service when smoke and ash envelop utility lines. Areas around wooden poles should be kept free of weeds, and cane should not be grown immediately adjacent to the wooden poles. The area around the poles and under power lines should be cut green when practical or back burned from the side. As with any sensitive area, a water tank should be in the immediate area and the person responsible for the burn should remain on site until the burn is completed and declared safe.

When utility substations are adjacent to cane fields, ash from burning cane as well as green trash blown from combine extractor fans can cause serious problems. Communication with the utility company personnel and back burning around the facility should be practiced. When combine harvesting, extractor fan hoods should be turned in the opposite direction to avoid cane trash (burnt or green) from being blown into the station.

Classification of "no-burn" fields. Certain fields, because of their proximity to extremely sensitive areas, should never be burned. Growers using combine harvesters can cut them green and growers using soldier harvesters should make arrangements with the mill to accept cane from these fields unburned.

Communication with Fire Districts should be practiced. Growers should notify their local Fire Districts of daily burn plans and locations. Many motorist will call 911 to report a field on fire and by law the fire company must respond unless they have been notified prior to the prescribed burn.

Step 2. Obtain Fire Weather Forecast

Growers can obtain the fire weather forecast and smoke category day during harvest season from their sugar mills and the Internet. There are three weather variables that a burn plan must have: (1) surface winds, (2) transport winds and (3) category day. Two other variables, SILT and forecasted weather changes, will help the CPBM understand the weather for that particular day. These factors are used to determine the potential for fire, smoke and /or ash problems resulting from a prescribed burn.

The forecasts are transmitted on the Internet by the National Weather Service (NWS) from offices in Shreveport, Lake Charles and New Orleans. This information is available by 9:00 a.m. each day.

The National Weather Service will also broadcast the Category Day on the National Oceanic and Atmospheric Administration (NOAA) weather radio channel.

The Internet addresses are:

Lake Charles area: http://www.boi.noaa.gov/firewx/NEWFWFLCH.html
New Orleans area: http://www.boi.noaa.gov/firewx/NEWFWFNEW.html
Shreveport area: http://www.boi.noaa.gov/firewx/NEWFWFSHV.html
Statewide: http://forestry.ldaf.state.la.us/public/default.htm

Important Note: Occasionally, during periods of relatively stagnant air, the National Weather Service, at the request of the Louisiana Department of Environmental Quality, will issue an Air Stagnation Advisory (ASA). This advisory requires a smoke management Category Day 1 be issued. When an ASA is issued, growers are asked not to ignite new fires and to complete any fires burning at the time the Air Stagnation Advisory is declared.

NOTES:_____

Step 3. Develop a Prescribed Burn Plan

A Prescribed Burn Plan should be completed by each grower prior to the harvest season. One plan can be completed for an entire farm or for an individual field. All information needed to plan and conduct a burn and for comments concerning the burn is contained in this form.

Prescribed Burn Plan

Farm Operator _____

1 Address _____ Farm Location _____

City _____ _____

State & Zip _____ Parish _____

Field Identification: _____

_____

2 **Pre-Burn Considerations:**

Personnel & Equipment Needed _____

Special Precautions _____

Smoke and Ash Sensitive Areas _____

Notification List _____

Weather Information:	3 **Acceptable Range**	4 **Forecast**	5 **Actual**
Surface Winds			
Speed	_____	_____	_____
Direction	_____	_____	_____
Transport Winds			
Speed	_____	_____	
Direction	_____	_____	
Category Day:	_____	_____	

6 Surface Inversion Lifting Temperature (SILT) _____

7 Possible weather changes: _____

_____

Fire problems: _____

8 _____

Smoke or ash problems: _____

_____

9 Ignition Time _____ Completion Time _____

Plan Completed By _____ Signature _____ Date _____

10 Burn Completed By _____ Signature _____ Date _____

What to include in a Prescribed Burn Plan:

1. **Farm Information:** Space is provided for identifying the Farm Operator. Field Identification asks you to identify what fields will be covered by this plan. A simple statement as "see attached map" is acceptable, and an actual map or drawing is to be included with this plan if necessary.

2. **Pre-Burn Considerations:** In pre-burn considerations before the burn is started, the grower and/or the individual conducting the burn should think through the burn process and plan for possible problems. Some considerations to include:

   a. Are any special people or equipment needed before, during or after the burn to make it safer?

   b. Do any special precautions need to be taken before the field is ignited, such as, pre-cutting cane around utility poles and utility substations?

   c. Identify ALL sensitive areas, especially those that may have been built since the last burn.

   d. Identify sensitive areas and notify individuals of the upcoming burn.

3. **Acceptable Ranges in Weather:** Enter an acceptable range for surface winds, transport winds and category day. These values are determined by the grower **prior to harvest season**. Using the burn plan map, a grower can determine the best surface wind directions, as well as the acceptable category days for that farm.

4. **Weather Forecast:** Enter the forecasted values for surface winds, transport winds and category day as given by the fire weather forecast **on the day of the burn**.

5. **Actual Weather:** Enter the actual values for surface winds as determined by the CPBM on the day of the burn and **at the actual field location at the time of ignition**.

6. **Surface Inversion Lifting Temperature:** Enter the SILT (Surface Inversion Lifting Temperature) as given by the fire weather forecast on the day of the burn. Knowing this temperature, a grower can determine approximately at what temperature an inversion will start to rise.

7. **Possible weather changes:** Evaluate and record any predicted changes in the weather for the day of the burn, especially in wind direction. For instance, a fast-moving cold front will cause the wind to change direction and speed drastically.

8. **Fire Problems and Smoke or Ash Problems:** This area gives you a place to note any problems with fire, smoke or ash for future reference. Even the smallest event noted could prove to be important at some time in the future.

9. **Ignition Time and Completion Time:** Enter the time of day that the burn was started (Ignition Time) and the time of day that the burn was completed and declared safe (Completion Time).

10. **Plan Completed By and Burn Completed By:** Enter name of person who prepared the burn plan and the CPBM who actually conducted the burn. This requires a signature and date. Use the back of this form to record comments that can provide guidance for future burning or other beneficial information.

Step 4. Determine Smoke Category Day

During harvest season, a grower can obtain the smoke category day from the fire weather forecast from each sugar mill and/or the Internet.

Burn only during acceptable times and weather conditions. Wind direction, wind velocity and air temperature inversion layers (SILT) drastically affect smoke and ash management. Since cane fields are seldom burned during the early morning hours because of dew and wet leaves, early morning weather is usually not of great concern. However, many growers burn cane later in the day when afternoon temperature inversions (normally after 4:00 p.m.) often can occur. When a temperature inversion occurs, upper air temperatures prevent smoke and ash from rising, causing the smoke and ash to drift laterally. This can impact highways, residences and public areas. To reduce risk to sensitive areas, growers should avoid burning in the late afternoon. The ideal time to burn is often between 10:00 a.m. and 4:00 p.m.

Table 1 - Smoke Category Day

Category Day	Recommendation
1	NO BURNING
2	No burning until after 11 a.m. and not before surface inversion has lifted. Fire should be substantially burned out by 4 p.m.
3	Daytime burning after surface inversion has lifted.
4	Burning anytime.
5	"Unstable and Windy." Excellent smoke dispersal. Burn with caution.

NOTES:_____

Step 5. Determine Smoke and Ash Screening Distance

The type of burn, together with the category day, will determine the distance downwind that may be adversely impacted by smoke and ash. The grower must identify where people, buildings, utility structures and highways are located within the impact area that could be negatively affected by smoke and ash. The most important weather condition to consider for ash screening distance is the surface and transport wind direction.

Identify a planned burn as one of the three following categories: (1) backing fire, (2) head fire, (3) piles/windrows (see glossary for descriptions).

Using the following table, find the block that represents your **type of burn** and the forecast **category day**. The number in that block is the **minimum number of miles downwind** from the burn that smoke and ash may have an impact on sensitive areas. It is this distance downwind that the grower needs to examine for possible smoke and ash sensitive areas such as airports, highways, communities, recreation areas, schools, hospitals, utilities, subdivisions, residences, factories and nursing homes, etc.

Table 2 - Potential Impact Area from Burn Site

	Category Day				
Type of Burn	**1**	**2**	**3**	**4**	**5**
Backing fire	No Burn	10 miles	5 miles	3 miles	1 mile
Head fire	No Burn	20 miles	10 miles	5 miles	1 mile
Piles/windrows	No Burn	30 miles	15 miles	8 miles	1 mile

Important:
When **burning standing cane**, the ash produced potentially becomes the most important factor to consider. Unlike smoke, ash will travel further on a category 5 day than on a category 2 day. When considering the distance that ash will travel, the CPBM must understand that the screening distances for ash are somewhat the **reverse** of the distances predicted for smoke.

The best available information shows that the majority of ash will "fall out" within 3 - 5 miles from the burn site on a category 3 or 4 day with winds of 10 - 15 mph and a lesser amount of ash will continue to "fall out" for the next 15 - 20 miles.

Note: The most important weather condition to consider for **ash screening distance** is the surface and transport wind **direction.**

Step 6. Determine Direction of Smoke and Ash Plume

By using a template like the one shown in figure A, a CPBM can determine the direction smoke and ash will travel from a particular field based on the wind direction at ground level and the transport wind direction and speed. The distance smoke and ash will travel is determined in Step 5.

Use the template shown (Figure A) or you can draw the template directly on a map.

The following are the steps used in determining the trajectory of a smoke and ash plume:

1. Locate the burn site on a map.

2. Draw a line indicating the wind direction from the burn site. This centerline will represent the path of the smoke plume. Mark this centerline in miles from the burn site for the potential distance of smoke and ash impact (see Table 2). Every map will have a scale showing miles per inch. Use only the scale as shown on your map.

3. To allow for normal smoke and ash movement, as well as shifts in wind direction, draw two other lines from the burn site at an angle of 30 degrees from the centerline (see Figure A). The area contained within this 60 degree arc and the mileage indicated by the centerline are the potential smoke and ash impact areas.

Figure A, Smoke and Ash Impact Area

Step 7. Evaluate the Prescribed Burn Results

Evaluate the results and success of the burn. Make any necessary notations on your Prescribed Burn Plan for that particular farm or field. Keep your completed burn plan for future reference.

Glossary

Air Stagnation Advisory (ASA): A statement issued by a National Weather Service Forecast Office when atmospheric conditions are stable enough that the POTENTIAL exists for air pollutants to accumulate in a given area. The statement is initially issued when conditions are expected to last for at least 36 hours.

Backing Fire: The fire spreading against the wind. Flames tilt away from direction of spread. Less smoke results from a backing fire.

Category Day: A scale from 1 to 5 based on ventilation rates. For smoke dispersal, 1 is poor and 5 is good.

Certified Prescribed Burn Manager (CPBM): An individual who successfully completes (1) an approved certification training program, (2) passes a written test, (3) has performed five sugarcane burns successfully and (4) is certified by the Louisiana Department of Agriculture and Forestry (LDAF).

Head Fire: A fire spreading with the wind. Flames tilt in the direction of the spread.

Inversion: An increase of temperature with an increase of height in the atmosphere. Vertical motion in the atmosphere is inhibited, allowing for pollution buildup. A "normal" atmosphere has temperature decreasing with height.

Meter (m): Basic unit of length in the metric system. There are 39.37 inches/meter, 3.28 feet/meter, 1.1 yards/meter.

Meters Per Second (mps): Expression of meters traveled each second. One meter per second is equal to 2.2 miles per hour.

Mixing Height: Measured from the sea level upward, the height to which relatively vigorous mixing occurs because of convection. Same as mixing depth. Use of the term normally implies presence of an inversion and the base of the inversion is the top of the mixed layer and defines the mixing height. Minimum recommended mixing height is 500 meters (1,640 feet).

Particulate Matter: Any liquid or solid particles. "Total suspended particulates" as used in air quality are those particulates suspended in or falling through the atmosphere. They generally range in size from 0.1 to 100 microns.

Piles/windrows: Staking of cut cane. This is generally the slowest type of burn. The material is usually wetter and results in greater smoke emission and less ash.

Plume: The segment of the atmosphere occupied by the emission from a single source or a grouping of sources close together. A convection column, if one exists, forms a specific part of the plume.

Prescribed Burn: A prescribed burn means the controlled application of fire in a confined predetermined area to accomplish the harvest of sugarcane under specified smoke and ash management guidelines. It is used in sugarcane production to reduce vegetative trash during harvest.

Screening Distance: The area to examine for potential sensitive targets.

Sensitive Targets: Areas that can be adversely affected by smoke and ash. Examples: Airports, major highways, communities, recreation areas, schools, hospitals, nursing homes, subdivisions, residences, factories, etc.

Smoke Management: Conducting a prescribed burn under specific field and meteorological conditions, and with burning techniques that keep the smoke and ash impact on the environment within acceptable limits.

Surface Inversion Lifting Temperature (SILT): The ambient air temperature in which the surface inversion should lift.

Transport Wind Speed: A measure of the average rate of the horizontal transport of air within the mixing layer. May also be the wind speed at the final height of plume rise. Generally refers to the rate at which emissions will be transported from one area to another. Minimum recommended transport wind speed is 4 mps (8.8 mph).

Ventilation Rate: The mixing height times the transport wind speed gives a rate indicating the capability of the lower atmosphere to diffuse and disperse smoke. Ventilation rate is calculated by multiplying the afternoon mixing height in meters by the transport wind speed in meters per second. The minimum recommended mixing height (500 meters) and the minimum recommended transport wind speed (4 mps) provide the minimum recommended ventilation rate of 2000 (500 X 4 = 2,000).

Warm Front: The leading edge of a relatively warm air mass which moves in such a way that warm air displaces colder air. Winds associated with warm frontal activity are usually light, and mixing is limited. The atmosphere is relatively stable compared to cold-front activity.

Wind Shear: A variation in wind speed and/or direction in a layer of the atmosphere or between layers. The variation may be in the horizontal or vertical and may result in significant turbulence, depending on the magnitude of the wind speed/direction difference. A strong wind shear may act like an inversion and inhibit plume rise. It also may fracture the smoke plume, not allowing smoke and ash to rise much above terrain levels. A strong horizontal anticyclonic shear results in a downward motion and may bring smoke and ash aloft back to the surface.

LOUISIANA SUGAR FACTORIES

Alma Plantation
(225) 627-6666

Cajun Sugar Cooperative, Inc
(337) 365-3401

Caire & Graugnard
(504)-497-3351/497-8500

Caldwell Sugars Co-op, Inc.
(504) 447-4023

Cinclare Central Factory
(225) 749-2861

Cora-Texas Mfg. Co., Inc.
(225) 545-3679

Enterprise Factory
(337) 276-4592

Evan Hall Factory
(225) 473-8241

Glenwood Cooperative, Inc.
(504) 369-2941

Iberia Sugar Co-op., Inc.
(337) 364-1913

Jeanerette Sugar Co., Inc.
(337) 276-4238

Lafourche Sugar Corp.
(504) 447-3210

Louisiana Sugarcane Co-op
(337) 394-3255

Lula Sugar Factory
(225) 473-9293

Westfield Sugar Factory
(504) 369-6450

Raceland Raw Sugar Corp.
(504) 537-3533

St. James Factory
(225) 265-4057

St. Mary Sugar Co-op, Inc.
(337) 276-6761

Sterling Factory
(337) 828-0620

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Louisiana Smoke Management Guidelines for Sugarcane Harvesting

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APPENDIX N

Australian Government
Department of the Environment

Smoke from biomass burning

2005

Air quality fact sheet
Department of the Environment and Heritage, 2005

What is biomass burning?

Biomass burning is the combustion of organic matter. Burning can be from natural or manmade fires. Examples are the burning of crop stubble, forest residues and vegetation burnt for land clearing. Information about smoke from the combustion of wood in woodheaters is available in our factsheet on woodheaters and woodsmoke.

'Prescribed burning' is a term used to describe the deliberate use of fire for management purposes. Fire is used as a tool to reduce the risk of wild fires by clearing out highly flammable leaves and branches shed by native vegetation in parks, reserves, on farms and bush blocks.

Biomass burning is a major source of many air-borne particles and trace gases that influence the concentration of ozone at ground level. (See our fact sheet on ozone.)

What is smoke from biomass burning composed of?

When a fire is first lit the moisture is driven off. As it gets hotter, chemical reactions occur that produce gases. Smoke contains the unburnt portion of these gases. Smoke is a complex mixture of many chemicals including carbon dioxide, water vapour, carbon monoxide, particles, hydrocarbons, nitrogen oxides and thousands of other compounds. The actual composition of smoke depends on the type of wood and vegetation being burnt, the temperature of the fire and the wind conditions.

Particles from smoke tend to be very small – less than one micrometer in diameter. In comparison, a human hair is 70 micrometers in diameter. (See our fact sheet on particles.)

Biomass burning also produces carbon monoxide. (See our fact sheet on carbon monoxide.) The concentrations of carbon monoxide are highest when the fire is smouldering. Benzene and formaldehyde are present in smoke but at much lower levels than particles and carbon monoxide.

What are the health effects of smoke?

Thick smoke from biomass burning does not necessarily cause health problems for everyone exposed to it. Most healthy people recover quickly from exposure to smoke and do not suffer long-term effects. There are a number of factors that determine whether exposure to smoke results in health problems: the concentrations of the air pollutants, the length of exposure, age, individual susceptibility and whether or not there is pre-existing lung or heart disease.

Smoke has a range of health effects – from eye and respiratory tract irritation to serious disorders such as breathing problems, bronchitis, increased severity of asthma, cancer and premature death. The very fine particles in smoke can go deep into the lungs and fine particles, by themselves or in combination with other air pollutants, can make pre-existing diseases of the heart and lungs worse. Where there is short-term exposure to smoke, the particles are the most significant threat to public health.

High levels of carbon monoxide are poisonous to humans. However, carbon monoxide arising from smoke events does not usually reach levels that pose a risk to the general population, although firefighters and people with heart disease can be at risk.

Who are most susceptible to the health effects of smoke?

Most healthy people, including children, recover quickly from exposure to smoke and do not suffer long-term consequences. However, certain sensitive groups can experience more severe short-term and chronic effects. It appears that the same population groups that are susceptible to particles in cities are also susceptible to particles from biomass burning. These groups are: people with asthma and other respiratory disease, people with cardiovascular disease, children and the elderly. Pregnant women and unborn children are potentially susceptible, given that smoke from biomass burning contains many of the same compounds found in cigarette smoke.

How much of a problem is biomass burning in Australia?

Bushfires are a part of the Australian way of life. All States and Territories have experienced catastrophic fires over the years and observed the adverse effects on air quality as one of the consequences.

Smoke from prescribed burning is also common in Australia and the levels of smoke from this source vary depending on the nature of the vegetation and the extent of the fire. Smoke from prescribed burning can be minimised if fires are lit at the right time of year and managed correctly. Smaller hotter fires are preferred, as this minimises air pollution.

Fire is also a common land management practice in many agricultural areas of Australia where it is used to burn the by-products of some agricultural crops, eg sugar cane waste, wheat or rice stubble and forest residues. Extensive areas of crop residues are burnt each year across Australia which can create large amounts of smoke for extended periods.

What's being done to manage smoke from biomass burning?

There are many initiatives to reduce the use of fire in agriculture. Alternatives to burning are now encouraged because they have less impact on air quality and are more environmentally sustainable. Wheat stubble is used to produce straw board or it can be returned to the soil to reduce erosion and moisture loss. Ethanol production from a range of agricultural waste products such as wheat and rice straw and sugar cane waste is in the early stages. Land managers now routinely use tools to model dispersion of smoke in the atmosphere. This helps to manage and predict air quality outcomes from biomass burning in their areas.

For more information

Related publications are available from the Community Information Unit of the Department of the Environment and Heritage, phone 1800 803 772. These include Air Quality fact sheets on: lead, nitrogen dioxide, carbon monoxide, ozone, particles, sulfur dioxide, air toxics; woodheaters and woodsmoke; and National Standards for Criteria Air Pollutants in Australia.

See also our website at Air quality.

The Impact of Sugar Cane–Burning Emissions on the Respiratory System of Children and the Elderly

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We analyzed the influence of emissions from burning sugar cane on the respiratory system during almost 1 year in the city of Piracicaba in southeast Brazil. From April 1997 through March 1998, samples of inhalable particles were collected, separated into fine and coarse particulate mode, and analyzed for black carbon and tracer elements. At the same time, we examined daily records of children (< 13 years of age) and elderly people (> 64 years of age) admitted to the hospital because of respiratory diseases. Generalized linear models were adopted with natural cubic splines to control for season and linear terms to control for weather. Analyses were carried out for the entire period, as well as for burning and nonburning periods. Additional models were built using three factors obtained from factor analysis instead of particles or tracer elements. Increases of 10.2 μg/m^3 in particles ≤ 2.5 μm/m^3 aerodynamic diameter ($PM_{2.5}$) and 42.9 μg/m^3 in PM_{10} were associated with increases of 21.4% [95% confidence interval (CI), 4.3–38.5] and 31.03% (95% CI, 1.25–60.21) in child and elderly respiratory hospital admissions, respectively. When we compared periods, the effects during the burning period were much higher than the effects during nonburning period. Elements generated from sugar cane burning (factor 1) were those most associated with both child and elderly respiratory admissions. Our results show the adverse impact of sugar cane burning emissions on the health of the population, reinforcing the need for public efforts to reduce and eventually eliminate this source of air pollution. *Key words:* **air pollution, biomass burning, children, elderly people, health effects, Poisson regression, respiratory diseases, time series.** *Environ Health Perspect* **114:725–729 (2006). doi:10.1289/ehp.8485 available via *http://dx.doi.org/* [Online 13 January 2006]**

The impact of biomass and fossil fuel burning is felt throughout the world. Although studies have documented the impact of fossil fuel air pollution on health (Braga et al. 2001; Hoek and Brunekreef 1994; Hoek et al. 1997; Laden et al. 2000; Lin et al. 1999; Pope et al. 1995; Saldiva et al. 1994; Schwartz and Dockery 1992; Schwartz et al. 2001), there is a scarcity of information on biomass burning [Arbex et al. 2000; Brauer and Hisham-hashim 1998; Long et al. 1998; Phonboon et al. 1999; World Health Organization (WHO) 1998]. Most biomass burning is carried out in developing countries and is done to clear land for shifting cultivation, to convert forests to agricultural or pastoral lands, and to remove dry vegetation to promote agricultural productivity. Burning of agricultural wastes in the field, such as sugar cane and stalks from grain crops, is another important type of biomass burning (Artaxo et al. 1999; Crutzen and Andreae 1990).

Biomass burning emissions represent an important global source of particles and gases to the atmosphere, especially in the tropics where biomass burning is widespread. Annually, 7,500–8,600 Tg (teragrams) of dry material is emitted to the atmosphere around the world through the process of burning. About 43% of this dry material is derived from savannah burning, 23% from the burning of agricultural waste, 18% from rainforest burning, and 16% from wood burning for fuel (Levine et al. 1995). Biomass burning emits large amounts of carbon and particulate matter (PM). Estimates show that the annual carbon and PM released to the atmosphere because of biomass fires in the tropics is around 2,000–4,500 Tg and 36–154 Tg, respectively (Crutzen and Andreae 1990).

Brazil plays an important role in biomass burning emissions. Most of the fires in Brazil occur during the dry season, from May through October. An especially critical area in the country is the Amazon region, where every year approximately 17,000 km^2 of tropical forests are cut down and most of them burned. Until now, of the total Amazon area (5.5 million km^2), 14% has been deforested (Instituto Nacional de Pesquisas Espaciais 2002).

In São Paulo State, located in the southeastern region of Brazil, most of the fires are generated in agricultural fields, especially sugar cane crops, in which 20 tons/ha are burned every year to facilitate harvesting. Sugar cane fires also have significant effects on the composition and acidity of rainwater over large areas of southeastern Brazil because of the emissions of aerosol and trace gases (Lara et al. 2001). Aerosols can also cause injury to human health. Small PM, particularly those < 2.5 μm in aerodynamic diameter ($PM_{2.5}$), appear to have the greatest potential for damaging health because they can penetrate deep into the lungs and reach the lower respiratory system (American Thoracic Society 1996). Despite its importance, little information about the aerosol particles emitted from sugar cane burning is available (Lara et al. 2005; Martinelli et al. 2002).

Within this scenario, the Piracicaba region, located in São Paulo State, is especially interesting because the atmosphere of this region receives emissions not only from industrial and urban sources but also and primarily from sugar cane burning (Lara et al. 2001). In this study, we investigated the influences of PM generated from sugar cane burning on respiratory hospital admissions of children and the elderly.

Materials and Methods

Study area and sampling. The city of Piracicaba is located in the western part of the Piracicaba River basin, in São Paulo State, and has a population of approximately 320,000 inhabitants and a population density of 242 inhabitants/km^2 (Figure 1). The land use in this area is dominated by sugar cane plantations (80%), followed by pastures (11%), urban areas (6%), and forests (3%) (Centro de Energia Nuclear para a Agronomia 1997). Sugar cane is burned every year from May through October, during the dry season,

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whereas the wet season extends from November through April.

From April 1997 through March 1998, samples of inhalable particles (PM_{10}) were taken from four stacked filter units (SFU) in two separated size fractions: fine PM ($PM_{2.5}$) and coarse PM (2.5 < particle diameter < 10 μm). Each filter unit was a 47-mm diameter Nuclepore polycarbonate filter (Whatman International Ltd., Kent, UK) and was retrieved every 72 hr. Samplings were obtained at the meteorologic station of the Escola Superior de Agricultura Luiz de Queiroz, which is located about 4 km from downtown Piracicaba and < 1 km from the nearest sugar cane plantation. The SFU inlets were located 3 m above the ground to minimize direct influences of local resuspended soil dust. The dominant wind direction was from the sugar cane plantations to the sampling station. Gravimetric mass and black carbon (BC) were measured in the Nuclepore filters. BC was measured by a reflectance technique according to the method developed by Reid et al. (1998).

Elemental composition was measured by particle-induced X-ray emission (PIXE) (Johansson and Campbell 1998), a multielemental technique that allows the identification of 21 elements (aluminum, silicon, phosphorus, sulfur, chlorine, potassium, calcium, titanium, vanadium, chromium, manganese, iron, nickel, copper, zinc, selenium, bromine, rubidium, strontium, zirconium, lead). PIXE detection limits are about 1 ng/m^3 for elements with Z > 20 and about 10 ng/m^3 for elements from sodium to K. Details on PIXE technique and aerosol sampling have been reported by Artaxo et al. (1999) and Yamasoe et al. (2000).

To investigate the respiratory health effects on the Piracicaba population, we obtained daily records of hospital admissions due to respiratory diseases [categorized according to *International Classification of Diseases,* 9th revision, (ICD-9; WHO 1975) codes 460–519 and *International Classification of Diseases*, 10th revision (ICD-10; WHO 1994) codes J00–J99] for children (< 13 years of age) and the elderly (> 64 years of age) from the government health agency (DATASUS; Brasilia, Brazil) from April 1997 to March 1998. The Agricultural School of São Paulo University (Piracicaba, Brazil) provided daily records of minimum temperature and relative humidity.

Figure 1. Location of the city of Piracicaba (sampling site) in São Paulo State, southeastern Brazil.

Statistical analysis of air pollutant health effects. Counts of daily respiratory hospital admissions for children and elderly were modeled separately for the entire period in Poisson regressions. We used the generalized linear model (McCullagh and Nelder 1989) with natural cubic splines (Green and Silverman 1994) to control for season. Splines were used to account for the nonlinear dependence of the hospital admissions on that covariate.

The function of time was used to remove the basic seasonal pattern (and long-term trend) from the data. If each admission were an independent event, we would expect no serial correlation in the data. Seasonal patterns for each end point (child and elderly respiratory hospital admissions) were modeled. Because we assumed that seasonal patterns would vary according to the adopted end point, end point–specific time smoothing parameters were used.

It was not necessary to incorporate autoregressive terms (Brumback et al. 2000) in the models because autocorrelation plots showed there were no remaining serial correlations in the residual. Indicators for day of the week were included in order to control for short-term trends.

Respiratory diseases present an almost linear relationship with weather. Therefore, linear terms for temperature and relative humidity were adopted. To reduce sensitivity to outliers in the dependent variable, we used robust regression (M-estimation). Three-day moving averages of BC, PM, and their main tracer elements were used to estimate the effects on respiratory morbidity.

For each age group, using the coefficients of the pollutants that presented adverse effects on respiratory hospital admissions for the entire period, we estimated the effects in two different periods: burning (May through October) and nonburning (November through April).

Table 1. Factor analysis using varimax rotation for $PM_{2.5}$ tracer elements.

Tracer elements of $PM_{2.5}$	Factor (main sources)			Communalities
	Biomass burning	Industrial	Automotive	
Si	0.876	0.221	0.382	0.911
S	0.509	0.090	0.856	0.889
Cl	0.475	0.051	0.726	0.763
K	0.888	–0.045	0.385	0.898
V	0.295	0.288	0.665	0.973
Fe	0.619	0.671	0.377	0.874
Ni	0.283	0.317	0.686	0.982
Cu	0.256	0.678	0.200	0.910
Zn	0.039	0.836	0.086	0.879
Br	0.610	0.415	0.238	0.813
Pb	–0.089	0.640	–0.004	0.913
Percent variance	59	21	10	

The communality expressed for each variable represents the fraction of the respective variable that is explained by the retained factors. In this case, the communalities were typically higher than 82% ($PM_{2.5}$). This indicated that the factors could explain most of the data variability.

Table 2. Descriptive analyses of child and elderly respiratory hospital admissions, temperature, and humidity of Piracicaba during the study period.

	Daily mean	SD	Minimum	IQR	Maximum	*n* (days)
Hospital admissions						
Children	2.2	1.7	0.0	2.0	8.0	306
Elderly	0.9	1.0	0.0	1.0	5.0	306
Weather						
Minimum temperature (°C)	15.8	4.1	5.5	6.5	23.2	298
Relative humidity (%)	81.7	9.5	52.0	12.0	100.0	298

Table 3. Descriptive analysis of PM_{10}, $PM_{2.5}$, BC, Al, Si, S, K, and Mn in the entire study period and during burning and nonburning seasons.

Pollutant	Entire period		Burning period		Nonburning period	
	Mean ± SD	IQR	Mean ± SD	IQR	Mean ± SD	IQR
PM_{10} (μg/m^3)	56.1 ± 49.8	42.9	87.7 ± 57.9	89.5	28.9 ± 12.8	15.0
$PM_{2.5}$ (μg/m^3)	16.1 ± 12.4	10.2	22.8 ± 14.7	17.3	10.0 ± 4.6	5.5
BC (μg/m^3)	2.1 ± 2.0	1.9	4.2 ± 2.3	2.9	1.8 ± 0.7	1.0
Al (ng/m^3)	166.3 ± 260.7	193.7	370.8 ± 317.5	480.1	157.9 ± 149.7	124.9
Si (ng/m^3)	404.5 ± 369.1	275.7	545.3 ± 462.9	669.2	283.9 ± 201.8	234.6
S (ng/m^3)	1362.1 ± 1,049.2	1009.6	1922.9 ± 1,237.5	1370.5	881.4 ± 497.2	492.7
K (ng/m^3)	380.2 ± 359.0	383.5	626.6 ± 390.4	539.1	168.9 ± 113.4	114.2
Mn (ng/m^3)	12.6 ± 10.0	9.0	16.9 ± 12.4	12.3	8.8 ± 4.6	6.82

Additionally, because of the large number of elemental components, specific rotation factor analysis was performed to identify main factors that could represent the main sources of air pollution in Piracicaba (Weiss 1971), reducing the analysis to a small number of factors. Factor analysis is a multivariate technique that allows for the combination of multiple variables into few factors based on their degree of correlation, thereby reducing the number of elements included in the analysis. Using the varimax rotation technique, we identified three main factors from the 17 tracer elements mentioned above: biomass burning and soil dust (factor 1), industrial emissions (factor 2), and fuel or automotive emissions (factor 3) (Table 1). Additional models were built using the factors instead of tracer elements in single- and three-factor models.

Results were expressed in terms of percentage increases in respiratory hospital admissions for interquartile range (IQR) increases in air pollutant concentrations. To carry out the statistical analyses, we used S-Plus (version 4.536; Insightful Corporation, Seattle, WA, USA).

Results

At the time of the study, 26% of the Piracicaba population were children and adolescents < 13 years of age, and 7% were > 64 years of age. There were 673 hospital admissions due to respiratory diseases among children and 275 among the elderly. Table 2 shows the daily descriptive analysis of child and elderly respiratory hospital admissions and temperature and humidity of the city of Piracicaba during the study period. The average number of child respiratory hospital admissions was more than twice that of the elderly, although the child population was almost four times the elderly population. The weather in Piracicaba is warm (climate characterized as subtropical Cw; humid with dry winter), and low temperatures are very rare.

Table 3 presents the descriptive analysis of the PM and its main elements measured during the entire period of study and burning and nonburning periods. Concentrations of PM_{10}, $PM_{2.5}$, and BC presented large seasonal variability with higher concentrations during the burning period compared with the nonburning period. For the elements generated mainly by biomass burning and soil dust, their concentrations increased from 2-fold to 4-fold between nonburning and burning periods. During the entire period of study, the PM_{10} concentration surpassed the standard limit adopted for this pollutant (50 μg/m^3).

Daily variations in PM_{10} and $PM_{2.5}$ as well as in BC, Al, Si, and K, which are products of biomass burning and soil dust (Lara et al. 2005), were significantly associated with child respiratory hospital admissions even after controlling for season and weather (Figure 2). Mn and S, proxies of industrial and automotive emissions, respectively, also presented effects on respiratory hospital admissions.

Furthermore, we observed associations between daily variations in PM_{10}, BC, and K and elderly respiratory hospital admissions for the entire period (Table 4).

Figure 3 presents the estimated percentage increases in child respiratory hospital admissions shown by burning and nonburning periods. We observed that $PM_{2.5}$, PM_{10}, BC, and elements from $PM_{2.5}$ generated from biomass burning and soil dust promoted higher effects during the burning period than during the nonburning period. Because the burning period is also the period with lower humidity and temperature, which impair air pollution dispersion, increases were observed in the elements generated from other sources, and consequently, higher respiratory effects resulted.

Among the elderly during the sugar cane–burning period (Figure 4), we also observed significant increases in respiratory hospital admissions due to increases in PM_{10}, BC, and K, a proxy of biomass burning. Mn, a proxy of industrial emissions, presented significant associations with elderly hospital admissions during the nonburning period, showing a pattern of susceptibility different from those observed among children.

Models using factors (factor 1, biomass burning and soil dust; 2, industrial emissions; 3, automotive emissions) instead of pollutants and tracer elements were essential in specifying the source with the major impact on respiratory diseases among elderly and children. Factor 1 presented the highest effect on respiratory hospital admissions in both single- and three-factor models for children (Table 5) and the elderly (Table 6).

Discussion

In contrast to large cities, such as São Paulo; Santiago, Chile; New York, New York; Baltimore, Maryland; and cities in Pakistan and China, where the main sources of aerosol particles are emissions linked to fossil fuel combustion (Artaxo et al. 1999; Castanho and Artaxo 2001; Conner et al. 2001; Lena et al. 2002; Zhang et al. 2002), the main source of aerosol in Piracicaba is biomass burning (Martinelli et al. 2002). In Piracicaba, sugar cane burning contributed 60% of the fine-mode aerosol mass. The second major source was resuspended soil dust, representing 14% of the $PM_{2.5}$. Industry and oil combustion each contributed 12% of the total $PM_{2.5}$ mass (Lara et al. 2005).

Studies have already shown that land use can influence the atmospheric composition in the Amazon Basin (Artaxo et al. 1999; Yamasoe et al. 2000) and in the State of São Paulo (Lara et al. 2001; Martinelli et al. 2002). Particularly, in the Piracicaba basin, the main land use is sugar cane plantations, which cover almost 80% of the total area.

In the present study, increases in PM_{10} and $PM_{2.5}$ were strongly associated with respiratory hospital admissions. The magnitudes of those effects were slightly higher than that found in São Paulo, the largest Brazilian city (Braga et al. 1999, 2001; Lin et al. 1999). When the magnitude of the effects of PM_{10}

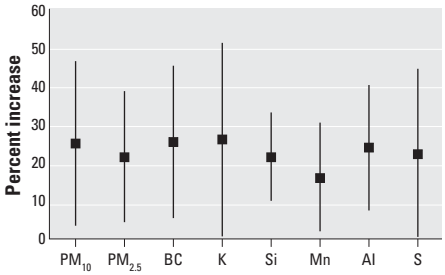

Figure 2. Percentage increases and 95% confidence intervals in child respiratory hospital admissions due to interquartile range increases in PM_{10}, $PM_{2.5}$, BC, Al, Si, Mn, K, and S during the period of study.

Table 4. Percentage increases and 95% confidence intervals (CIs) in elderly respiratory hospital admissions due to interquartile range increases in PM_{10}, BC, and K during the period of study.

Pollutant	Percentage increase (95% CI)
PM_{10}	31.03 (1.25–60.81)
BC	36.41 (11.14–61.68)
K	46.74 (11.67–81.82)

CI, confidence interval.

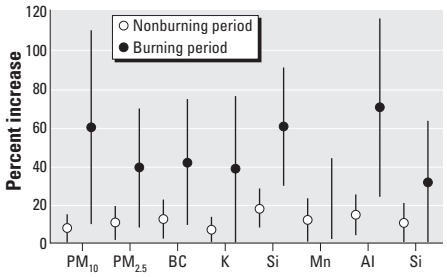

Figure 3. Percentage increases and 95% confidence intervals in child respiratory hospital admissions due to mean levels of PM_{10}, $PM_{2.5}$, BC, K, Si, Mn, Al, and S during burning and nonburning periods.

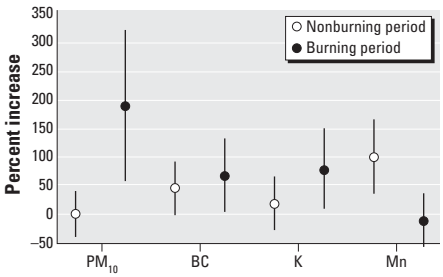

Figure 4. Percentage increases and 95% confidence intervals in elderly respiratory hospital admissions due to mean levels of PM_{10}, BC, K, and Mn during burning and nonburning periods.

and $PM_{2.5}$ were assessed for the same end point, there were no differences between them. This could be explained by the fact that we worked with total PM_{10} measurements instead of just the coarse-mode fraction. Hence, it is possible to assume that the effects observed for PM_{10} include $PM_{2.5}$ effects.

However, in a region with at least three important sources of PM air pollution—biomass burning and industrial and automotive emissions—it is necessary to identify the air pollution source responsible for most of the respiratory hospital admissions. In this context, the association of elemental components of $PM_{2.5}$ that are generated mainly from biomass burning and soil dust (BC, Al, Si, and K) and the results of factor analysis showing that factor 1 (biomass burning) was the most associated with respiratory hospital admissions reinforce the role of this source of air pollution on child and elderly respiratory diseases in this region, despite the contribution of industrial and automotive emissions.

The use of factor analysis to identify and apportion ambient concentrations to sources has been used in the past, and a workshop sponsored by the U.S. Environmental Protection Agency explored the use of resolved source contributions in health effects models (Thurston et al. 2005). For this workshop, multiple groups of investigators analyzed PM composition data sets from two U.S. cities, and although different factor analysis methods were used, similar source profiles were extracted from these sets, with a good agreement among the major resolved source types. The same investigators also found similar health effects associations (Hopke et al. 2005; Ito et al. 2005; Laden et al. 2000; Mar et al. 2005). In our data set, we had 1 year of daily data, which is less than that in the studies described above; the factor analysis chose only some factors, and in the model we adjusted for all potential confounders that have been shown to be the most relevant. The associations of health effects with the metals are still under investigation, and although some associations might be due to chance, evidence for them has been found in the past. We believe that our results are not due to chance, even though other studies are needed to confirm these findings.

Studies have shown that population exposure to high levels of pollutants can increase the risk of acute respiratory infections, chronic obstructive pulmonary disease, and lung cancer (Smith et al. 1999). Children are the most susceptible group. Studies performed in the city of São Paulo have shown adverse effects of PM on inflammatory and infectious diseases of both upper and lower respiratory tracts (Farhat et al. 2005; Lin et al. 1999). Indeed, acute lower respiratory infections are the single most important cause of mortality in children > 5 years of age (Bruce et al. 2000). Globally, about 43% of the total burden of disease due to environmental risks falls on children > 5 years of age, although they make up only 12% of the population (Smith et al. 1999).

This study adopted total respiratory hospital admissions of children and elderly instead of specific-respiratory disease diagnoses because of the small number of events observed in the city during the study period. Moreover, this approach avoided misclassification of disease-specific diagnoses.

Around Piracicaba, the percentage increase in child respiratory hospital admissions in relation to the mean levels of PM_{10}, $PM_{2.5}$, BC, Al, Si, Mn, K, and S was two to three times higher in the burning season than in the nonburning season, compromising the health of this population. Among the elderly, the same pattern of effects was observed between burning and nonburning periods. It undoubtedly shows that the months of the year when sugar cane is burned are the most dangerous for the inhabitants of the region.

In fact, the association between exposure to indoor and outdoor biomass smoke and health effects has already been reported in some areas of Asia and India (Behera et al. 1994; Phonboon et al. 1999). Deposition of carbon in the lungs occurred consistently in patients exposed to biomass burning (Bruce et al. 2000). However, different from most of the mentioned regions where outdoor biomass burning events are episodic, biomass burning is a common and scheduled activity in Piracicaba and other Brazilian sugar cane plantation areas, exposing the population on a regular basis.

The mechanisms whereby PM causes airway diseases have been studied. Oxidative stress, caused by the release of oxidant radicals by inflammatory cells, may be an important component in promoting inflammation and respiratory cell damage, compromising pulmonary function, increasing susceptibilities to allergens, and increasing the incidence of respiratory infections (Bernstein et al. 2004; Bruce et al. 2000).

The population of these areas of Brazil has been exposed to sugar cane burning for at least 6 months every year for the last four decades. The health effect is determined not just by acute exposures to high pollution levels but also, and more importantly, by the length of time that people spend breathing polluted air chronically. Similar to this study, the acute effect of sugar-cane–generated air pollution was assessed previously in São Paulo State (Arbex et al. 2000). However, the magnitude of chronic effects is not known.

Alcohol produced from sugar cane is a renewable fuel and is less pollutant than fossil fuel. Brazil is the largest producer of sugar and alcohol from sugar cane in the world. Plantations cover about 5 million hectares and surround a great number of cities, mainly in the southeastern region of the country. About 80% of the sugar cane crops are burned to facilitate harvesting every year (Brazilian Agriculture Ministry 2005).

The addition of alcohol to gasoline and national policies to reduce automotive emissions contributed to a decrease in air pollution in Brazilian urban centers in the last 20 years. Nowadays, the use of alcohol as automotive fuel is under study or has already been partially adopted in many countries. Hence, it is clear that ethanol production will tend to grow in this century. To avoid secondary health damages, the procedure of burning the crops before harvesting should be banished.

In summary, we showed that air pollution from biomass burning causes damage to the respiratory system, leading to an increase in respiratory hospital admissions. This effect is higher for children and the elderly, and it is similar to that observed in urban areas due to exposure to industrial and vehicle-emitted air pollutants. Finally, it is necessary to keep in mind the close relation between health effects and air pollution. This concern should be included in public efforts for sustainability and improving environmental health conditions.

Table 5. Regression coefficients, SEs, and statistical significance of the models for child respiratory hospital admissions using single or three factors as independent variables.

	Model					
	Single factor			Three factors		
	β	SE	*p*-Value	β	SE	*p*-Value
Biomass burning	0.1996	0.1138	0.0867	0.2138	0.1180	0.0775
Industrial	0.0722	0.0922	0.4378	0.0559	0.0921	0.5470
Automotive	0.0426	0.0949	0.6559	0.0832	0.0935	0.3791

Table 6. Regression coefficients, SEs, and statistical significance of the models for elderly respiratory hospital admissions using single or three factors as independent variables.

	Model					
	Single factor			Three factors		
	β	SE	*p*-Value	β	SE	*p*-Value
Biomass burning	0.4156	0.1522	0.0092	0.3527	0.1644	0.0380
Industrial	–0.0990	0.1380	0.4771	–0.0703	0.1356	0.6070
Automotive	–0.3009	0.1767	0.0961	–0.1753	0.1541	0.2622

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APPENDIX P

Agency for Toxic Substances and Disease Registry (ATSDR)
Case Studies in Environmental Medicine
Toxicity of Polycyclic Aromatic Hydrocarbons (PAHs)

Course: WB 1519
Original Date: July 1, 2009
Expiration Date: July 1, 2012

Table of Contents

Key Concepts	• Because of combustion of fossil fuels and organic waste, PAHs are ubiquitous in the environment • Studies show that certain PAH metabolites interact with DNA and are genotoxic, causing malignancies and heritable genetic damage in humans. • In humans, heavy occupational exposure to mixtures of PAHs entails a substantial risk of lung, skin, or bladder cancer.
About This and Other Case Studies in Environmental Medicine	This educational case study document is one in a series of self-instructional modules designed to increase the primary care provider's knowledge of hazardous substances in the environment and to promote the adoption of medical practices that aid in the evaluation and care of potentially exposed patients. The complete series of Case Studies in Environmental Medicine is located on the ATSDR Web site at http://www.atsdr.cdc.gov/csem/. In addition, the downloadable PDF version of this educational series and other environmental medicine materials provides content in an electronic, printable format, especially for those who may lack adequate Internet service.
How to Apply for and Receive Continuing Education Credit	See Internet address www.atsdr.cdc.gov/csem/conteduc.html for more information about continuing medical education credits, continuing nursing education credits, and other continuing education units.

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U.S. Department of Health and Human Services
Agency for Toxic Substances and Disease Registry
Division of Toxicology and Environmental Medicine
Environmental Medicine and Educational Services Branch

How to Use This Course

Introduction	The goal of *Case Studies in Environmental Medicine* (CSEM) is to increase the primary care provider's knowledge of hazardous substances in the environment and to help in evaluation and treating of potentially exposed patients. This CSEM focuses on the toxicity of polycyclic aromatic hydrocarbons.
Available Versions	Two versions of the *Toxicity of Polycyclic Aromatic Hydrocarbons* CSEM are available. • The HTML version http://www.atsdr.cdc.gov/csem/pah/ provides content through the Internet. • The downloadable PDF version provides content in an electronic, printable format, especially for those who may lack adequate Internet service. The HTML version offers interactive exercises and prescriptive feedback to the user.
Instructions	To make the most effective use of this course. • Take the Initial Check to assess your current knowledge about the toxicity of polycyclic aromatic hydrocarbons. • Read the title, learning objectives, text, and key points in each section. • Complete the progress check exercises at the end of each section and check your answers. • Complete and submit your assessment and posttest response online if you wish to obtain continuing education credit. Continuing education certificates can be printed immediately upon completion.
Instructional Format	This course is designed to help you learn efficiently. Topics are clearly labeled so that you can skip sections or quickly scan sections you are already familiar with. This labeling will also allow you to use this training material as a handy reference. To help you identify and absorb important content quickly, each section is structured as follows:

Section Element	Purpose
Title	Serves as a "focus question" that you should be able to answer after completing the section
Learning Objectives	Describes specific content addressed in each section and focuses your attention on important points
Text	Provides the information you need to answer the focus question(s) and achieve the learning objectives
Key Points	Highlights important issues and helps you review
Progress Check	Enables you to test yourself to determine whether you have mastered the learning objectives
Answers	Provide feedback to ensure you understand the content and can locate information in the text

Learning Objectives	Upon completion of the Toxicity of Polycyclic Aromatic Hydrocarbons (PAH) CSEM, you will be able to

Content Area	Objectives
Overview	• explain what PAHs are • describe the properties of PAHs • identify where PAHs are found
Exposure Pathways	• identify routes of exposure to PAHs
Who is at Risk	• identify the populations at high risk for exposure to PAHs
Standards and Regulations	• describe the Occupational Safety and Health Administration's permissible exposure limit (PEL) for PAHs • describe the U.S. Environmental Protection Agency's maximum contaminant level (MCL) for PAHs in drinking water
Biological Fate	• describe the biologic fate of PAHs in the body
Pathogenic Changes	• describe how PAHs are believed to induce pathogenic changes
Health Effects	• describe health effects associated with PAH exposure
Clinical Assessment	• describe typical signs and symptoms of patients with acute PAH exposure • describe typical signs and symptoms of patients with chronic PAH exposure • describe important elements of the exposure history • describe the focus of the physical examination • describe tests used to assist in evaluation of patients exposed to PAHs
Treatment and Management	• identify strategies for managing patients with chronic PAH exposure
Patient Education and Counseling	• describe care advice for the patient exposed to PAHs

Initial Check

Instructions	This Initial Check will help you assess your current knowledge about the toxicity of polycyclic aromatic hydrocarbons (PAH). To take the Initial Check, read the case below, and then answer the questions that follow.
Case Study	**Dyspnea, weight loss, and weakness in a 52-year-old male coal tar manufacturing plant worker** A 52-year-old man comes to your office for a health evaluation, his first in 3 years. While trying to assure you that he is in reasonably good health, he admits that his wife prompted this visit. She is concerned about his weight loss, lack of stamina, and weakness in the shoulders and arms. When you review his chart, you see that he has lost 30 pounds since his last visit. The patient also describes shortness of breath with moderate activity. He is a lifelong nonsmoker and drinks alcohol only occasionally. He is taking no medications. His past medical history is noncontributory. A review of systems reveals that the patient also has a chronic, intermittently productive cough, which has been ongoing for 1 month. The patient has worked at a coal tar manufacturing plant for the past 34 years. He has been a lifelong resident of an urban industrial neighborhood that is approximately 1 mile from where he works. He has been married for 25 years. His wife and adult daughter are in good health. A physical examination shows that his vital signs are normal. An inspection of his skin reveals multiple dry, scaly, hyperpigmented macules involving the forehead, temporoparietal areas, eyelids, and brows, and several hyperkeratotic papillomata on his face, neck, upper chest, forearms, and hands. Palpation of the right supraclavicular area reveals a firm, nontender, fixed lymph node 2 x 3 centimeters (cm) in size. Auscultation discloses intermittent, scattered, right-sided wheezes and dry bibasilar crackles. The remainder of the exam is unremarkable. The patient's laboratory results are remarkable for the following: 1. hemoglobin = 12.9 grams per deciliter (g/dL) (normal = 14–18 g/dL); 2. hematocrit = 36% (normal = 42%–52%); 3. leukocyte count = 2.9 x 10^3 per microliter (µL) (normal = 3.9–11 x 10^3/µL); 4. serum calcium = 12.9 milligrams per deciliter (mg/dL) (normal = 8.5–10.5 mg/dL); 5. alkaline phosphatase = 483 international units per liter (IU/L) (normal = 30–125 IU/L) with concomitant elevation of GGTP (GGT); 6. SGOT (AST) 121 IU/L (normal = 7–45); 7. SGPT (ALT) 129 IU/L (normal = 7–35 IU/L); 8. The chest radiograph reveals a 3.3-cm central, thick-walled, cavitating lesion with irregular, spicular margins in the right upper lobe, and atelectasis and prominence of the right hilar lymphatics.

Initial Check

1. What are likely sources of PAHs for the patient described in the case study?
2. Besides the patient, who in the case study might be at risk for PAH exposure?
3. The patient's daughter, who has lived in his household all of her life, recently gave birth to a daughter. Is the newborn at risk for PAH exposure? Why or why not?
4. How could you document that the work environment of the patient described in the case study contributed to his risk of lung cancer?
5. Before his present employment, the patient in the case study was employed as a laborer on a farm. He says that he was not exposed to pulmonary toxic agents such as asbestos or silica. What is the problem list and the differential diagnosis for this patient?
6. The diagnosis for the patient described in the case study is squamous cell carcinoma of the lung. In general, what can you do to decrease the risk for lung cancer among your patients?
7. Would you consider the patient described in the case study a sentinel case requiring notification of public health agencies? Explain.

Initial Check Answers

1. The patient may have been exposed to coal tar manufacturing pollutants at his work for more than 34 years. Moreover, if his home is in the prevailing downwind direction from the coal tar manufacturing plant, pollutants might contribute to ambient air contamination near his home. However, environmental studies related to air pollution are more complex and must separate out contaminants from indoor cooling/heating systems, environmental tobacco smoke, urban air pollution, and other sources. The patient might have been exposed to PAH mixtures by all three routes: inhalation, ingestion, and direct cutaneous contact.

   More information for this answer can be found in the "What Are Routes of Exposure to PAHs?" and "Who Is at Risk of Exposure to PAHs?" sections.

2. Workers at the coal tar manufacturing plant and residents in the community downwind from the plant might be exposed to PAH mixtures. However, other contributors to environmental ambient air contamination should be kept in mind, including environmental tobacco smoke, indoor cooking and heating practices, and urban air pollution. The patient's family members might be at risk for additional exposure if the patient carried these compounds home on his skin and work clothes.

   More information for this answer can be found in the "What Are Routes of Exposure to PAHs?" and "Who Is at Risk of Exposure to PAHs?" sections.

3. The patient's newborn granddaughter may be at risk for PAH exposure. If the patient's daughter breathed contaminated air in and around the house, then the baby could have been exposed *in utero*. This exposure could have occurred while the patient's daughter was

doing various household chores, such as laundering, dusting, and general cleaning of the contaminated home or her father's work clothing. Based on animal studies, PAH mixtures absorbed into the mother's system might continue to be transferred to the baby via breast milk. The baby might also be breathing contaminated air, thereby increasing her exposure.

More information for this answer can be found in the "Who Is at Risk of Exposure to PAHs?" section.

4. The role of the workplace in the patient's PAH mixture exposure can be determined by area sampling at the work site, individual monitoring, medical surveillance of coworkers, and air sampling within the immediate community. Industrial hygienists would typically perform these activities. Data may be available through sources at the coal tar manufacturing plant and at local, state, or federal agencies.

   More information for this answer can be found in the "Who Is at Risk of Exposure to PAHs" section.

5. The patient's problem list includes weight loss, fatigue, muscle weakness, skin lesions on exposed areas, exertional dyspnea, and a roentgenographically identified cavitating lesion in the right upper lobe with associated lymphadenopathy. The differential diagnosis includes carcinoma of the lung, tuberculosis, fungal lung infection, and lung abscess.

   More information for this answer can be found in the "What Health Effects are Associated with PAH Exposure" and "Clinical Assessment."" sections.

6. The main objective is to educate patients about cancer prevention. You should try to stimulate changes in their work habits and lifestyle that will decrease the risk for cancer. A risk assessment can identify elements in a person's workplace, family history, medical history, and lifestyle that might be controllable risk factors.

   For example, between 75% and 80% of all cases of bronchogenic carcinoma are due to cigarette smoking and are therefore preventable. Of the remaining 20%–25%, many are related to occupation or the environment and could therefore be prevented by appropriate workplace or environmental controls. The incidence of lung cancer might also be decreased through education efforts that focus on

   - smoking prevention,
   - improving working conditions,
   - substitution of less-hazardous materials in work processes and building materials, and
   - increased awareness of personal risk factors.

More information for this answer can be found in the "What Instructions Should Be Given to Patients Exposed to PAHs?" section.

7. In view of the patient's medical, social, occupational, and family history, the workplace and environmental factors emerge as the most likely causal factors in the development of his neoplastic disease. When the potential exists for others to be exposed, serious illness related to occupational or environmental factors should be reported to the appropriate state and federal authorities. For example, OSHA would have responsibility for PAHs in the workplace air at the coal tar manufacturing site. EPA would have responsibility for the level of emissions to the ambient air or water. Inclusion of this case in a tumor registry should also be considered.

   More information for this answer can be found in the "Clinical Assessment" and "How Should Patients Exposed to PAHs be Treated and Managed?" sections.

What Are Polycyclic Aromatic Hydrocarbons (PAHs)?

Learning Objective

Upon completion of this section, you will be able to

- explain what PAHs are, and
- describe the properties of PAHs.

Definition

PAHs are a class of organic compounds produced by incomplete combustion or high-pressure processes. PAHs form when complex organic substances are exposed to high temperatures or pressures.

Often, PAHs consist of three or more fused benzene rings containing only carbon and hydrogen (**Figure 1**). Differences in the configuration of rings may lead to differences in properties.

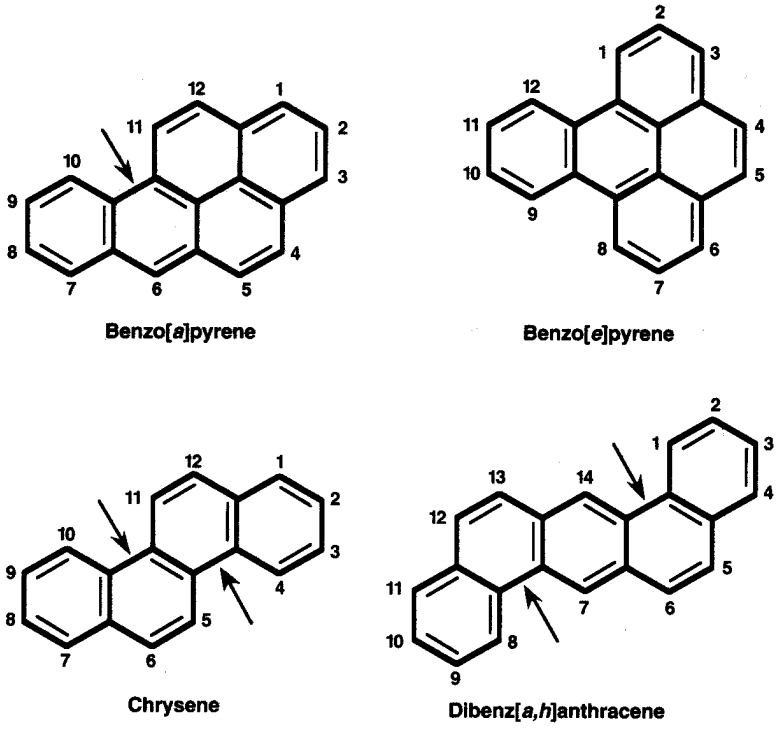

Figure 1. Structural formulas of selected polycyclic aromatic hydrocarbons (PAHs). The arrows indicate bay regions.

Synonyms

PAHs are known by several names:

- polycyclic organic matter (POM),
- polynuclear aromatic hydrocarbons,
- polynuclear aromatics (PNAs), and
- polynuclear hydrocarbons.

The more common PAHs include

- benzo(a)anthracene,

- benzo(a)pyrene,
- benzo(e)pyrene,
- benzo(g,h,i)perylene,
- benzo(k)fluoranthene,
- chrysene,
- coronene,
- dibenz(a,h)acridine,
- dibenz(a,h)anthracene, and
- pyrene.

Properties

PAHs:

- are solids with low volatility at room temperature,
- have relatively high molecular weights,
- are soluble in many organic solvents,
- are relatively insoluble in water, and
- most can be photo-oxidized and degraded to simpler substances.

Key Points

- PAHs are a class of organic compounds produced by incomplete combustion or high-pressure processes.
- Often, PAHs consist of three or more fused benzene rings containing only carbon and hydrogen.
- PAHs are solids with low volatility at room temperature. They are relatively insoluble in water, and most can be photo-oxidized and degraded to simpler substances.

Progress Check

1. Which of the following is (are) true regarding PAHs?

   A. PAHs comprise a class of organic compounds produced by high-pressure processes.
   B. Often, PAHs consist of three or more fused benzene rings containing only hydrogen and carbon.
   C. PAHs comprise a class of organic compounds produced from incomplete combustion.
   D. All of the above.

   To review relevant content, see "Definition" in this section.

2. Which of the following is (are) true regarding PAHs?

   A. They are water-soluble.
   B. They have relatively low molecular weights.
   C. They are solids with low volatility at room temperature.
   D. All of the above.

   To review relevant content, see "Properties" in this section.

Answers

1. The correct true answer is D. PAHs are a class of organic compounds produced from incomplete combustion or high-pressure processes and consist of three or more fused benzene rings containing only hydrogen and carbon.
2. The correct true answer is C. PAHs are solids with low volatility at room temperature. The remaining choices are false because PAHs are relatively insoluble in water and have relatively high molecular weights.

Where Are Polycyclic Aromatic Hydrocarbons (PAHs) Found?

Learning Objective	Upon completion of this section, you will be able to • identify where PAHs are found.
Introduction	PAHs are ubiquitous in the environment and are common byproducts of combustion processes. PAHs are a natural component of most fossil fuels. Although produced naturally by forest fires and volcanoes, most PAHs in ambient air are the result of man-made processes. Such processes include • burning fuels such as coal, wood, petroleum, petroleum products, or oil, • burning refuse, used tires, polypropylene, or polystyrene, • coke production, and • motor vehicle exhaust [Cherng *et al.* 1996; Lewitas 1997]. There are approximately 100 different known PAHs in air, soil, foodstuffs, and water [Zedeck 1980]. Diesel exhaust contains significant amounts of PAHs. Benzo(a)pyrene, a potent carcinogen, is commonly used as an environmental indicator for PAHs.
Industrial Use	PAHs are found in industries that produce or use coal tar, coke, or bitumen (asphalt). Coal tar pitch and creosote, which are complex mixtures of liquid and solid aromatic hydrocarbons produced in coke ovens, contain significant amounts of benzo(a)pyrene and other PAHs. PAHs are produced in • coal gasification plants, • municipal incinerators, • smokehouses, and • some aluminum production facilities.
Environmental Fate	Once emitted to the atmosphere, weight influences the fate of the gaseous PAH mixtures. Heavier PAHs (more than four rings) tend to adsorb to particulate matter, while lighter PAHs (less than four rings) tend to remain gaseous until removed via precipitation [Skupinska *et al.* 2004]. PAH concentrations in water tend to be low (around 100 ng/L) due to their weak solubility. The weak solubility leads to accumulation in sediments and aquatic organisms. PAHs can be absorbed by plants and can accumulate in soil.
Cigarettes	Cigarette smoke contains many PAHs; therefore, cigarette smoking and environmental tobacco smoke are additional sources of PAHs.
Environmental Indicator for PAHs	Benzo(a)pyrene, a potent carcinogen, is generally used as an environmental indicator for PAHs.

Key Points	• PAHs are ubiquitous in the environment. • Most PAHs in ambient air are the result of man-made processes. • PAHs are found in industries that produce or use coal tar, coke, or bitumen (asphalt). They are emitted by coal gasification plants, smokehouses, municipal incinerators, and some aluminum production facilities. • PAHs mostly accumulate in soils. • Benzo(a)pyrene is commonly used as an environmental indicator for PAHs.
Progress Check	3. Which of the following is true regarding PAHs? A. PAHs are found predominantly in water reservoirs. B. PAHS are found in relatively few geographic areas worldwide. C. PAHS are found in coal gasification plants and some aluminum production facilities. D. PAHs mostly result from natural processes. *To review relevant content, see "Introduction" and "Industrial Use" in this section.* 4. Which of the following industries or processes involve PAH production? A. Smokehouses. B. Municipal incinerators. C. Coal tar or coke production or use. D. All of the above. *To review relevant content, see "Industrial Use" in this section.*

Answers

3. The correct answer is C. PAHs are found in coal gasification plants and some aluminum production facilities.
4. The correct answer is D. Industries or processes that involve PAH production include smokehouses, municipal incinerators, and coal tar or coke production or use.

What Are the Routes of Exposure for Polycyclic Aromatic Hydrocarbons (PAHs)?

Learning Objective	Upon completion of this section, you will be able to • identify routes of exposure to PAHs.
Introduction	PAH exposure through air, water, soil, and food sources occurs on a regular basis for most people. Routes of exposure include ingestion, inhalation, and dermal contact in both occupational and non-occupational settings. Some exposures may involve more than one route simultaneously, affecting the total absorbed dose (such as dermal and inhalation exposures from contaminated air). All non-workplace sources of exposure such as diet, smoking, and burning of coal and wood should be taken into consideration.
Air	PAH concentrations in air can vary from less than 5 to 200,000 nanograms/cubic meter (ng/m^3) [Cherng *et al.* 1996; Georgiadis and Kyrtopoulos 1999]. Although environmental air levels are lower than those associated with specific occupational exposures, they are of public health concern when spread over large urban populations [Zmirou *et al.* 2000]. The background levels of seventeen of the Agency for Toxic Substances and Disease Registry's toxicological profile priority PAHs in ambient air are reported to be 0.02–1.2 $nanograms/m^3$ in rural areas and 0.15–19.3 ng/m^3 in urban areas [ATSDR 1995]. Cigarette smoking and environmental tobacco smoke are other sources of air exposure. Smoking one cigarette can yield an intake of 20–40 ng of benzo (a) pyrene [Phillips 1996; O'Neill *et al.* 1997]. Smoking one pack of unfiltered cigarettes per day yields 0.7 µg/day benzo(a)pyrene exposure. Smoking a pack of filtered cigarettes per day yields 0.4 µg/day [Sullivan and Krieger 2001]. Environmental tobacco smoke contains a variety of PAHs, such as benzo(a)pyrene, and more than 40 known or suspected human carcinogens. Side-stream smoke (smoke emitted from a burning cigarette between puffs) contains PAHs and other cytotoxic substances in quantities much higher than those found in mainstream smoke (exhaled smoke of smoker) [Jinot and Bayard 1996; Nelson 2001].
Water	PAHs can leach from soil into water. Water contamination also occurs from industrial effluents and accidental spills during oil shipment at sea. Concentrations of benzo(a)pyrene in drinking water are generally lower than those in untreated water and about 100-fold lower than the U.S. Environmental Protection Agency's (EPA) drinking water standard. (EPA's maximum contaminant level [MCL] for benzo(a)pyrene in drinking water is 0.2 parts per billion [ppb].)
Soil	Soil contains measurable amounts of PAHs, primarily from airborne fallout. Documented levels of PAHs in soil near oil refineries have been as high as 200,000 micrograms per kilogram (µg/kg) of dried soil. Levels in soil samples obtained near cities and areas with heavy traffic were typically less than 2,000 µg/kg [IARC 1973].

Foodstuffs	In non-occupational settings, up to 70% of PAH exposure for a non-smoking person can be associated with diet [Skupinska *et al.* 2004]. PAH concentrations in foodstuffs vary. Charring meat or barbecuing food over a charcoal, wood, or other type of fire greatly increases the concentration of PAHs. For example, the PAH level for charring meat can be as high as 10–20 µg/kg [Phillips 1999]. Charbroiled and smoked meats and fish contain more PAHs than do uncooked products, with up to 2.0 µg/kg of benzo(a)pyrene detected in smoked fish. Tea, roasted peanuts, coffee, refined vegetable oil, cereals, spinach, and many other foodstuffs contain PAHs. Some crops, such as wheat, rye, and lentils, may synthesize PAHs or absorb them via water, air, or soil [Grimmer 1968; Menzie *et al.* 1992; Shabad and Cohan 1972; IARC 1973].
Other Sources of Exposure	PAHs are found in prescription and nonprescription coal tar products used to treat dermatologic disorders such as psoriasis and dandruff [Van Schooten 1996]. PAHs and their metabolites are excreted in breast milk, and they readily cross the placenta. Anthracene laxative use has been associated with melanosis of the colon and rectum [Badiali *et al.* 1985].
Background Exposures	In the Third National Report on Human Exposure to Environmental Chemicals [CDC 2005], urinary levels of hydroxylated metabolites of PAHs were measured in a subsample of the National Health and Nutrition Examination Survey (NHANES) among participants aged 6 years and older during 1999–2002. Participants were selected within the specified age range to be a representative sample of the U.S. population. Measurements of the 22 metabolites reflect exposure to PAHs that occurred a few days prior to the urine samples being taken. Pyrene is commonly found in PAH mixtures, and its urinary metabolite, 1-hydroxypyrene, has been used as an indicator of exposure to PAH chemicals [Becher and Bjorseth 1983; Granella and Clonfero 1993; Popp 1997; Santella *et al.* 1993, CDC 2005]. The American Conference of Governmental Industrial Hygienists recommends measurement of 1-hydroxypyrene in the end-of-shift, end-of-work-week urine samples as a biological exposure index (BEI) for assessment of exposure to mixtures containing PAHs [ACGIH 2005; Heikkila *et al.* 1995]. The geometric mean of urine concentrations (in nanograms/grams creatinine) of 1-hydroxypyrene for the U.S. population aged 6 years and older for survey years 1999–2000 was 74.2, and in survey years 2001–2002 it was 46.4 [CDC 2005]. The geometric mean levels of 1-hydroxypyrene in a NHANES 2001–2002 subsample is similar to that of other general populations residing in an urban setting [Goen *et al.* 1995; Chuang *et al.* 1999; Heudorf and Angerer 2001; Roggi *et al.* 1997; Yang *et al.* 2003]. Higher levels have been noted for residents of industrialized urban areas than in rural or suburban settings [Adonis *et al.* 2003; Kanoh *et al.* 1993; Kuo *et al.* 2004]. Many-fold higher levels can be found in workers from certain occupations [Jacob and Seidel 2002], including aluminum smelting [Alexandrie *et al.* 2000]; diesel engine

	mechanics [Adonis *et al.* 2003; Kuusimaki *et al.* 2004]; taxi, bus, and truck drivers [Chuang *et al.* 2003; Hansen *et al.* 2004; Kuusimaki *et al.* 2004]; painters [Lee *et al.* 2003], boilermakers [Mukherjee *et al.* 2004]; toll booth operators [Tsai *et al.* 2004]; traffic police [Merlo *et al.* 1998] and coke oven plant workers [Lu *et al.* 2002; Serdar *et al.* 2003; Siwinska *et al.* 2004]. Tobacco smoking leads to higher levels in smokers [Chuang *et al.* 2003; Adonis *et al.* 2003; Heudorf and Angerer 2001b] as well as in the non-smoking children of smokers [Tsai *et al.* 2003]. Coal stove exposure or consumption of broiled, fried, or grilled meat contribute to higher levels of 1-hydroxypyrene [Siwinska *et al.* 1999; Scheepers *et al.* 2002; Yang *et al.* 2003; [CDC 2005].
Key Points	• PAH exposure occurs on a regular basis for most people. • In non-occupational settings, most PAH exposures for a non-smoking person can be associated with diet • Routes of exposure include inhalation, ingestion, and dermal • Exposure may also occur via placental transfer, breast milk, and coal tar-containing products.
Progress Check	5. In non-occupational settings, PAH exposure in a non-smoking individual mostly comes from which of the following? A. Foodstuff ingestion. B. Inhalation route. C. Dermal route. D. Water. *To review relevant content, see "Foodstuffs" in this section.* 6. Exposure to PAHs may occur as a result of which of the following? A. Eating roasted peanuts. B. Eating charbroiled meats. C. Inhaling second hand tobacco smoke. D. All of the above. *To review relevant content, see "Air" and "Foodstuffs" in this section.*

Answers

5. The correct answer is A. In non-occupational settings, the majority of PAH exposure in a non-smoking individual comes from foodstuff ingestion.
6. The correct answer is D. Exposure to PAHs may occur as a result of eating roasted peanuts, eating charbroiled meats, or inhaling secondhand tobacco smoke.

Who Is at Risk of Exposure to Polycyclic Aromatic Hydrocarbons (PAHs)?

Learning Objective	Upon completion of this section, you will be able to • identify the populations at high risk for exposure to PAHs.
Introduction	Persons working with coal and coal products have a greater likelihood of exposure to PAHs. Awareness of historical occupational and environmental exposures can aid the clinician in not only assessing potential sources of exposure but also in recognizing those populations who may be at higher risk of exposure.
Historical Occupational Exposures	Percival Pott, an English surgeon, was the first to report a connection between occupational exposure and cancer. In 1775, he described an unusually high incidence of scrotal cancer among London chimney sweeps and suggested this was due to their exposure to soot and ash. Since then, other coal tar-related cancers have been induced in laboratory animals and found in humans [Kennaway 1995; Kjaerheim 1999]. For example, the PAH benzo(a)pyrene, which was isolated from coal tar in the 1930s, was determined to be carcinogenic when applied to the skin of test animals. In 1947, the relationship between lung cancer and working conditions of gas industry workers and those working with coal tar was established [Kenneway 1995]. An increased incidence of cancers, particularly of the lung, was shown in epidemiologic studies of gas workers [Doll *et al.* 1965, 1972]. Several epidemiologic studies have shown increased cancer mortality in workers exposed to PAH mixtures. Exposure to other potentially carcinogenic substances often occurred in these studies [Lloyd 1971; Mazumdar *et al.* 1975; Redmond *et al.* 1972; Redmond and Strobino 1976; Hammond *et al.* 1976].
Current Occupational Exposures	Workers in industries or trades using or producing coal or coal products are at highest risk for PAH exposure. Those workers include, but are not limited to • aluminum workers, • asphalt workers, • carbon black workers, • chimney sweeps, • coal-gas workers, • coke oven workers, • fishermen (coal tar on nets), • graphite electrode workers, • machinists, • mechanics (auto and diesel engine), • printers, • road (pavement) workers, • roofers, • steel foundry workers, • tire and rubber manufacturing workers, and • workers exposed to creosote, such as

	○ carpenters, ○ farmers, ○ railroad workers, ○ tunnel construction workers, and ○ utility workers. A small increased risk of cancer in workers exposed to diesel exhaust has been suggested by some epidemiologic studies [Bhatia *et al.* 1998; Boffetta *et al.* 1988, 1990, 1997; Garshick *et al.* 1987, 1988; Steenland *et al.* 1990, 1992]. Exposure is almost always to mixtures that pose a challenge in developing conclusions [Samet 1995].
Historical Environmental Exposures	Historically, in locations where gas for lighting and heating was manufactured from coal or oil, large amounts of PAHs existed and may still exist as waste deposits. Before World War II, more than 1,000 coal gasification plants are estimated to have existed throughout the midwestern and eastern United States [Environmental Research and Technology 1984]. These plants began to phase out in the early 1950s when the use of interstate natural gas pipelines became more prominent.
Susceptible Populations	Fetuses may be at risk for PAH exposure. PAH and its metabolites have been shown to cross the placenta in various animal studies [ATSDR 1995]. Because PAHs are excreted in breast milk, nursing infants of exposed mothers can be secondarily exposed.
Key Points	• PAH and metabolites cross the placenta and are excreted in breast milk. • Occupations that entail exposure to PAH include workers exposed to coal and coal products.
Progress Check	7. Which of the following groups of workers is (are) at most risk of heavy exposure to PAHs on the job? A. Workers exposed to creosote. B. Steel foundry workers. C. Roofers. D. All of the above. *To review relevant content, see "Current Occupational Exposures" in this section.* 8. Which of the following is true regarding PAH exposure? A. Those most at risk are workers in the auto emission testing industry. B. Health endpoints can often be directly traced to a specific PAH metabolite. C. PAHs cross the placenta. D. None of the above. *To review relevant content, see "Current Occupational Exposures" and "Susceptible Populations" in this section.*

Answers

7. The correct answer is D. Steel foundry workers, roofers, and workers exposed to creosote have jobs that put them at risk of heavy exposure to PAHs.
8. 8.The correct answer is C. PAHs do cross the placenta. Persons working with coal and coal products have a greater likelihood of exposure to PAHs than workers in the auto emission testing industry. Exposures are typically to mixtures of PAHs, making it difficult to attribute a health end point to a specific PAH metabolite.

What Are the Standards and Regulations for Polycyclic Aromatic Hydrocarbons (PAH) Exposure?

Learning Objectives

Upon completion of this section, you will be able to

- describe the Occupational Safety and Health Administration's (OSHA) Permissible Exposure Level (PEL) for PAH, and
- describe the U.S. Environmental Protection Agency's (EPA) Maximum Contaminant Level (MCL) for PAH in drinking water.

Introduction

U.S. government agencies have established standards that are relevant to PAHs exposures in the workplace and the environment. There is

- a standard relating to PAH in the workplace, and
- a standard for PAH in drinking water.

Workplace

OSHA has not established a substance-specific standard for occupational exposure to PAHs. Exposures are regulated under OSHA's Air Contaminants Standard for substances termed coal tar pitch volatiles (CTPVs) and coke oven emissions. Employees exposed to CTPVs in the coke oven industry are covered by the coke oven emissions standard.

The OSHA coke oven emissions standard requires employers to control employee exposure to coke oven emissions by the use of engineering controls and work practices. Wherever the engineering and work practice controls that can be instituted are not sufficient to reduce employee exposures to or below the permissible exposure limit, the employer shall nonetheless use them to reduce exposures to the lowest level achievable by these controls and shall supplement them by the use of respiratory protection. The OSHA standard also includes elements of medical surveillance for workers exposed to coke oven emissions.

Air

The OSHA PEL for PAHs in the workplace is 0.2 milligram/cubic meter (mg/m^3).

The OSHA-mandated PAH workroom air standard is an 8–hour time-weighted average (TWA) permissible exposure limit (PEL) of 0.2 mg/m^3, measured as the benzene-soluble fraction of coal tar pitch volatiles. The OSHA standard for coke oven emissions is 0.15 mg/m^3. The National Institute for Occupational Safety and Health (NIOSH) has recommended that the workplace exposure limit for PAHs be set at the lowest detectable concentration, which was 0.1 mg/m^3 for coal tar pitch volatile agents at the time of the recommendation. Table 1 summarizes relevant exposure criteria for PAHs.

Workplace Standards

Table 1. Standards and Regulations for Polycyclic Aromatic Hydrocarbons (PAHs)

Agency	Focus	Level	Comments
American Conference of Governmental Industrial Hygienists	Air: workplace	0.2 milligrams per cubic meter (mg/m^3) for benzene-soluble coal tar pitch fraction	Advisory: TLV* (8-hour TWA†)
National Institute for Occupational Safety and Health	Air: workplace	0.1 mg/m^3 for coal tar pitch volatile agents	Advisory: REL‡ (8-hour TWA)
Occupational Safety and Health Administration	Air: workplace	0.2 mg/m^3 for benzene-soluble coal tar pitch fraction	Regulation: (benzene soluble fraction of coal tar volatiles) PEL§ (8-hour workday)
U.S. Environmental Protection Agency	Water	0.0001 milligrams per liter (mg/L)	MCL¶ for benz(a)anthracene
		0.0002 mg/L	MCL for benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene
		0.0003 mg/L	MCL for dibenz(a,h)anthracene
		0.0004 mg/L	MCL for indenol(1,2,3-c,d)pyrene

*TLV: threshold limit value.

†TWA (time-weighted average): concentration for a normal 8-hour workday and a 40-hour workweek to which nearly all workers may be repeatedly exposed.

‡REL (recommended exposure limit): recommended airborne exposure limit for coal tar pitch volatiles (cyclohexane-extractable fraction) averaged over a 10-hour work shift.

§PEL (permissible exposure limit): the legal airborne permissible exposure limit (PEL) for coal tar pitch volatiles (benzene soluble fraction) averaged over an 8-hour work shift.

¶MCL: maximum contaminant level.

Environ-mental Standards	***Water*** The maximum contaminant level goal for benzo(a)pyrene in drinking water is 0.2 parts per billion (ppb). In 1980, EPA developed ambient water quality criteria to protect human health from the carcinogenic effects of PAH exposure. The recommendation was a goal of zero (nondetectable level for carcinogenic PAHs in ambient water). EPA, as a regulatory agency, sets a maximum contaminant level (MCL) for benzo(a)pyrene, the most carcinogenic PAH, at 0.2 ppb. EPA also sets MCLs for five other carcinogenic PAHs (see Table 1). For more information on EPA rules and regulations regarding PAH, visit EPA's Web site at www.epa.gov. ***Food*** The U.S. Food and Drug Administration has not established standards governing the PAH content of foodstuffs.
Key Points	• OSHA's PEL for PAH in the workplace is 0.2 mg/m^3 for benzene-soluble coal tar pitch fraction of air (8-hour TWA). • OSHA requires workers to be trained in the proper use of appropriate personal protective equipment (PPE) and safety. • Workers must receive medical surveillance if exposed above the PEL. • EPA's maximum contaminant level (MCL) for PAH in drinking water is 0.2 ppb of drinking water.

Progress Check

9. Which of the following is true regarding OSHA's role in PAH exposure limits?

   A. OSHA is a regulatory agency that has a permissible exposure level (PEL) established for PAHs in the workplace.
   B. OSHA requires all workers to be provided with appropriate personal protection equipment (PPE) and receive safety training.
   C. OSHA requires workers to receive medical surveillance if exposed above the permissible exposure limit PEL.
   D. All of the above.

   To review relevant content, see "Workplace Standards" in this section.

10. Which of the following is true regarding EPA's role in PAH exposure limits?

    A. The maximum contaminant levels (MCLs) for PAHs are set to protect human health against the carcinogenic effects of PAH.
    B. EPA regulates PAH levels in foodstuffs.
    C. EPA is a regulatory agency that has established MCLs for several PAHs in air.
    D. MCLs are based on an 8–hour time weighted average.

    To review relevant content, see "Environmental Standards" in this section.

Answers

9. The correct answer is D. OSHA is a regulatory agency which has established a PEL established for PAHs in the workplace. OSHA requires all workers to be provided with appropriate PPE and receive safety training. OSHA requires workers to receive medical surveillance if exposed above the PEL.
10. The correct answer is A. The MCLs for PAHs are set to protect human health against the carcinogenic effects of PAHs. The FDA, not EPA, regulates contaminant levels in foodstuffs and currently has no standard set for PAHs in foodstuffs. EPA is a regulatory agency that has established MCLs for several PAHs in water, not air. OSHA's PELs are based on an 8–hour time-weighted average, not EPA's MCLs.

What Is the Biologic Fate of PAHs in the Body?

Learning Objective

Upon completion of this section, you will be able to

- describe the biologic fate of the PAHs in the body.

Introduction

Once PAHs enter the body, several things occur:

- PAHs are metabolized in a number of organs and excreted in bile and urine
- PAHs are excreted in breast milk and stored to a limited degree in adipose tissue.

Not much data for humans exists regarding the metabolic fate of PAHs. However, information on absorption, distribution, and elimination of these substances is available from animal studies.

Pyrene is commonly found in PAH mixtures, and its urinary metabolite, 1-hydroxypyrene, has been used as an indicator of exposure to PAH chemicals [Becher and Bjorseth 1983; Granella and Clonfero 1993; Popp 1997; Santella *et al.* 1993, CDC 2005]. The ACGIH recommends measurement of 1-hydroxypyrene in the end-of-shift, end-of-work-week urine samples as a biological exposure index (BEI) for assessment of exposure to mixtures containing PAHs.

Measurements of 22 PAH hydroxylated urinary metabolites were taken as part of the Third National Report on Human Exposure to Environmental Chemicals from a subsample of the National Health and Nutrition Examination Survey (NHANES) from participants aged 6 years and older during 1999–2002. These data provide physicians with a reference range so that they can determine whether people have been exposed to higher levels of PAHs than are found in the general population [CDC 2005].

Absorption

PAHs are absorbed through ingestion, inhalation, and dermal contact, according to animal study data. The percent absorbed varies in these studies for several reasons, including the vehicle (transport medium) in which the PAHs are found [Kawamura *et al.* 1988]. In general, PAHs not bound to particulate matter may be absorbed in the lungs better than the same dose found on the surface of airborne particulate matter [Cresia *et al.* 1976; Seto 1993].

Distribution

Once absorbed, PAHs

- enter the lymph,
- circulate in the blood, and
- are metabolized primarily in the liver and kidney.

PAHs differ with respect to distribution patterns and lipophilic properties [Busbee *et al.* 1990]. Because of their lipophilic nature, PAHs can accumulate in breast milk and adipose tissue. However, biliary and urinary excretion of PAHs is relatively efficient because of the wide distribution of enzymes that transform PAHs into polar metabolites.

Metabolism and Excretion	PAHs are predominantly metabolized via CYP enzymes (enzymes in the P-450 mixed-function oxidase system) in the liver [Kapitulnik *et al.* 1977; Keifer *et al.* 1988; Monteith *et al.* 1987]. In addition to the liver and kidneys, metabolism of PAHs occurs in the adrenal glands, testes, thyroid, lungs, skin, sebaceous glands, and small intestines [ATSDR 1995]. PAHs are transformed initially to epoxides, which are converted to dihydrodiol derivatives and phenols. Glucuronide and sulfate conjugates of these metabolites are excreted in the bile and urine. Glutathione conjugates are further metabolized to mercapturic acids in the kidney and are excreted in the urine. The hydroxylated metabolites of the PAHs are excreted in human urine both as free hydroxylated metabolites and as hydroxylated metabolites conjugated to glucuronic acid and sulfate [CDC 2005]. A commonly measured urinary metabolite is 1-hydroxypyrene [Becher and Bjorseth 1983; Granella and Clonfero 1993; Popp 1997; Santella 1993]. Metabolism is a prerequisite for hepatobiliary excretion and elimination through the feces, regardless of route of entry. Excretion half-lives in feces and urine have been reported in animal studies as 22 hours and 28 hours, respectively [Becher and Bjorseth 1983].
Key Points	• Absorption by route varies in animal studies. • PAH distribution patterns vary due to differences in lipophilic properties. • Metabolism via CYP liver enzymes (enzymes in the P-450 mixed-function oxidase system) is the predominant mechanism of PAH metabolism. • PAHs are transformed to epoxides, then to dihydrodiol derivatives and phenols. • Excretion occurs via the bile or urine after metabolite conjugation to glucuronides and sulfates. • 1- Hydroxypyrene is a commonly measured urine metabolite.

Progress Check

11. Which of the following is true regarding PAHs?

    A. PAHs are mostly excreted unchanged in the feces.
    B. PAHs are stored in adipose tissue to a large extent.
    C. PAHs have varied distribution patterns.
    D. PAHs become lodged and are retained in lung tissue.

    To review relevant content, see "Absorption", "Distribution", and "Metabolism and Excretion" in this section.

12. Which of the following is true of PAHs?

    A. All PAH metabolites are formed via CYP liver enzymes (enzymes in the P-450 mixed function oxidase system).
    B. PAHs can accumulate in breast milk and adipose tissue.
    C. Most PAHs have similar distribution patterns and lipophilic properties.
    D. PAHs bound to particulate matter are better absorbed in the lungs than are unbound PAHs.

    To review relevant content, see "Absorption", "Distribution", and "Metabolism and Excretion" in this section.

Answers

11. The correct answer is C. PAHs have varied distribution patterns due to differences in lipophilic properties. Because of their lipophilic nature, PAHs can accumulate in breast milk and adipose tissue. However, biliary and urinary excretion of PAHs is relatively efficient because of the wide distribution of enzymes that transform PAHs into polar metabolites.
12. The correct answer is B. PAHs can accumulate in breast milk and adipose tissue. Not all PAH metabolites are formed via CYP liver enzymes (enzymes in the P-450 mixed function oxidase system). PAHs do differ in their distribution patterns and lipophilic properties. Unbound PAHs are better absorbed in the lungs, not bound PAHs.

How Do PAHs Induce Pathogenic Change?

Learning Objective	Upon completion of this section, you will be able to • describe how PAHs are believed to induce pathogenic changes.
Introduction	A key factor in PAH toxicity is the formation of reactive metabolites. Not all PAHs are of the same toxicity because of differences in structure that affect metabolism. Another factor to consider is the biologic effective dose, or the amount of toxics that actually reaches the cells or target sites where interaction and adverse effects can occur. CYP1A1, the primary cytochrome P-450 isoenzyme that biologically activates benzo (a) pyrene, may be induced by other substances [Kemena *et al.* 1988; Robinson *et al.* 1975]. The mechanism of PAH-induced carcinogenesis is believed to be via the binding of PAH metabolites to deoxyribonucleic acid (DNA).
Carcinogenicity	Some parent PAHs are weak carcinogens that require metabolism to become more potent carcinogens. Diol epoxides—PAH intermediate metabolites—are mutagenic and affect normal cell replication when they react with DNA to form adducts. A theory to explain the variability in the potency of different diol epoxides, "the bay theory," predicts that an epoxide will be highly reactive and mutagenic if it is in the "bay" region of the PAH molecule (**Figure 1**) [Jerina *et al.* 1976 and 1980; Weis 1998]. The bay region is the space between the aromatic rings of the PAH molecule. PAH-induced carcinogenesis can result when a PAH-DNA adduct forms at a site critical to the regulation of cell differentiation or growth. A mutation occurs during cell replication if the aberration remains unrepaired. Cells affected most significantly by acute PAH exposure appear to be those with rapid replicative turnover, such as those in bone marrow, skin, and lung tissue. Tissues with slower turnover rates, such as liver tissue, are less susceptible. Benzo(a)pyrene diol epoxide adducts bind covalently to several guanine positions of the bronchial epithelial cell DNA p53 gene, where cancer mutations are known to occur from exposure to cigarette smoke. This is one possible genotoxic mechanism of cancer causation by tobacco [Denissenko 1996].
Genetic Susceptibility	CYP1A1 inducible persons might be at greater risk for the effects of PAHs. Persons with a high degree of CYP1A1 inducibility may be more susceptible to PAH health risks. Genetic variation in CYP1A1 inducibility has been implicated as a determining factor for susceptibility to lung and laryngeal cancer.

	CYP1A1, the primary cytochrome P-450 isoenzyme that biologically activates benzo (a) pyrene, may be induced by other substances [Kemena *et al.* 1988; Robinson *et al.* 1975]. Glutathione transferase deficiencies may result in elevated cancer risk. Several studies have focused on breast cancer risk and metabolism of PAHs [Ambrosone *et al.* 1995; Calaf and Russo 1993; Davis *et al.* 1993; Hecht *et al.* 1994].
Oncogene Activation	Several animal studies have implicated the *ras* oncogene in PAH tumor induction [Chakravarti *et al.* 1995; DiGiovanni *et al.* 1993; Ronai *et al.* 1994].
Key Points	• The formation of reactive metabolites and the biologically effective dose are key to PAH toxicity. • Diol epoxides—PAH intermediate metabolites—are mutagenic and affect normal cell replication when they react with DNA to form adducts. • The location of epoxides in the bay region of a PAH predicts reactivity and mutagenicity. • DNA adducts, as markers of exposure used in research, can be measured in various biologic media. • The ability of CYP1A1 to biologically activate PAHs may be heritable and thus point to genetically susceptible populations at risk of PAH carcinogenesis.
Progress Check	13. The mechanism of PAH-induced carcinogenesis is believed to be which of the following? A. Binding of PAH metabolites to DNA. B. Generation of active oxygen species. C. Cell-mediated inflammatory mechanisms. D. All of the above. *To review relevant content, see "Carcinogenicity" in this section.* 14. Which of the following is (are) true regarding PAHs? A. Tissues with rapid cell turnover are most vulnerable to the carcinogenic effects from some PAHs. B. The degree to which CYP1A1 biologically activates PAHs may be heritable. C. The location of epoxides in the bay region of a PAH predicts reactivity and mutagenicity. D. All of the above are true. *To review relevant content, see "Carcinogenicity" and "Genetic Susceptibility" in this section.*

Answers

13. The correct answer is A. The mechanism of PAH induced carcinogenesis is believed to be the binding of PAH metabolites to DNA.
14. The correct answer is D. All of the statements are true. Tissues with rapid cell turnover are most vulnerable to the carcinogenic effects from some PAHs; the degree to which CYP1A1 biologically activates PAHs may be heritable; and the location of epoxides in the bay region of a PAH predicts reactivity and mutagenicity.

What Health Effects Are Associated With PAH Exposure?

Learning Objective

Upon completion of this section, you will be able to

- describe health effects associated with PAH exposure.

Introduction

The most significant endpoint of PAH toxicity is cancer.

PAHs generally have a low degree of acute toxicity to humans. Some studies have shown noncarcinogenic effects that are based on PAH exposure dose [Gupta *et al.* 1991].

After chronic exposure, the non-carcinogenic effects of PAHs involve primarily the

- pulmonary,
- gastrointestinal,
- renal, and dermatologic systems.

Many PAHs are only slightly mutagenic or even nonmutagenic in vitro; however, their metabolites or derivatives can be potent mutagens.

Carcinogenicity

The carcinogenicity of certain PAHs is well established in laboratory animals. Researchers have reported increased incidences of skin, lung, bladder, liver, and stomach cancers, as well as injection-site sarcomas, in animals. Animal studies show that certain PAHs also can affect the hematopoietic and immune systems and can produce reproductive, neurologic, and developmental effects [Blanton 1986, 1988; Dasgupta and Lahiri 1992; Hahon and Booth 1986; Malmgren *et al.* 1952; Philips *et al.* 1973; Szczeklik *et al.* 1994; Yasuhira 1964; Zhao 1990].

It is difficult to ascribe observed health effects in epidemiological studies to specific PAHs because most exposures are to PAH mixtures.

Increased incidences of lung, skin, and bladder cancers are associated with occupational exposure to PAHs. Epidemiologic reports of PAH-exposed workers have noted increased incidences of skin, lung, bladder, and gastrointestinal cancers. These reports, however, provide only qualitative evidence of the carcinogenic potential of PAHs in humans because of the presence of multiple PAH compounds and other suspected carcinogens. Some of these reports also indicate the lack of quantitative monitoring data [Hammond *et al.* 1976; Lloyd 1971; Mazumdar 1975; Redmond *et al.* 1972; Redmond and Strobino 1976].

The earliest human PAH-related epidemiologic study was reported in 1936 by investigators in Japan and England who studied lung cancer mortality among workers in coal carbonization and gasification processes. Subsequent U.S. studies among coke oven workers confirmed an excess of lung cancer mortality, with the suggestion of excessive genitourinary system cancer mortality. Later experimental studies showed that PAHs in soot were probably responsible for the increased incidence of scrotal cancer noted by Percival Pott among London chimney sweeps in his 1775 treatise [Zedeck 1980].

Research	Continued research regarding the mutagenic and carcinogenic effects from chronic exposure to PAHs and metabolites is needed. The following table indicates the carcinogenic classifications of selected PAHs by specific agencies.

Agency	PAH Compound(s)	Carcinogenic Classification
U.S. Department of Health and Human Services (HHS)	• benz(a)anthracene, • benzo(b)fluoranthene, • benzo(a)pyrene, • dibenz(a,h)anthracene, and • indeno(1,2,3-c,d)pyrene.	Known animal carcinogens
International Agency for Research on Cancer (IARC)	• benz(a)anthracene and • benzo(a)pyrene.	Probably carcinogenic to humans
	• benzo(a)fluoranthene, • benzo(k)fluoranthene, and • ideno(1,2,3-c,d)pyrene.	Possibly carcinogenic to humans
	• anthracene, • benzo(g,h,i)perylene, • benzo(e)pyrene, • chrysene, • fluoranthene, • fluorene, • phenanthrene, and • pyrene.	Not classifiable as to their carcinogenicity to humans
U.S. Environmental Protection Agency (EPA)	• benz(a)anthracene, • benzo(a)pyrene, • benzo(b)fluoranthene, • benzo(k)fluoranthene, • chrysene, • dibenz(a,h)anthracene, and • indeno(1,2,3-c,d)pyrene.	Probable human carcinogens
	• acenaphthylene, • anthracene, • benzo(g,h,i)perylene, • fluoranthene, • fluorene, • phenanthrene, and pyrene.	Not classifiable as to human carcinogenicity

Key Points

- PAHs generally have a low degree of acute toxicity to humans.
- The most significant endpoint of PAH toxicity is cancer.
- Increased incidences of lung, skin, and bladder cancers are associated with occupational exposure to PAHs. Data for other sites is much less persuasive.
- It is difficult to ascribe observed health effects in epidemiological studies to specific PAHs because most exposures are to PAH mixtures.
- Animal studies show that certain PAHs affect the hematopoietic, immune, reproductive, and neurologic systems and cause developmental effects.

Progress Check

15. Which of the following is (are) true?

    A. PAHs generally have a high degree of acute toxicity in humans.
    B. PAHs have been associated with increased incidences of lung, skin, and bladder cancers from occupational exposures.
    C. Specific PAHs can easily be linked to observed health effects in epidemiologic studies.
    D. All of the above.

    To review relevant content, see "Introduction" and "Carcinogenicity" in this section.

16. According to IARC and EPA, which of the following PAHs are probable human carcinogens?

    A. Benzo(a)pyrene.
    B. Benz(a)anthracene.
    C. Anthracene.
    D. Both A and B.

    To review relevant content, see "Carcinogenicity" in this section.

Answers

15. The correct answer is B. PAHs have been associated with increased incidences of lung, skin, and bladder cancers from occupational exposures. PAHs, however, do not have a high degree of acute toxicity in humans. It also is difficult to ascribe observed health effects in epidemiological studies to specific PAHs because most exposures are to PAH mixtures.
16. The correct answer is D. According to IARC and EPA, both benzo(a)pyrene and benz(a)anthracene are probable human carcinogens. Anthracene is not classifiable as to its carcinogenicity to humans by either agency.

Clinical Assessment

Learning Objectives	Upon completion of this section, you will be able to • describe typical signs and symptoms of patients with acute PAH exposure, • describe typical signs and symptoms of patients with chronic PAH exposure, • describe important elements of the exposure history, • describe the focus of the physical examination, and • describe tests used to assist in evaluation of patients exposed to PAHs.
Introduction	In addition to the standard clinical approaches to patient evaluation, clinicians should take an appropriate PAH exposure history. They should know what to look for during the physical exam and how to test for PAH exposure.
Signs and Symptoms—Acute Exposure	Acute effects attributed to PAH exposure, such as headache, nausea, respiratory and dermal irritation, are probably caused by other agents. Since PAHs have low acute toxicity, other more acutely toxic agents probably cause the acute symptoms attributed to PAHs. Hydrogen sulfide in roofing tars and sulfur dioxide in foundries are examples of concomitant, acutely toxic contaminants. Naphthalene, the most abundant constituent of coal tar, is a skin irritant, and its vapors may cause headache, nausea, vomiting, and diaphoresis [Rom 1998].
Signs and Symptoms—Chronic Exposure	Effects reported from occupational exposure to PAHs include • chronic bronchitis, • chronic cough irritation, • bronchogenic cancer, • dermatitis, • cutaneous photosensitization, and • pilosebaceous reactions. Reported health effects associated with chronic exposure to coal tar and its by-products (*e.g.*, PAHs). • Skin: erythema, burns, and warts on sun-exposed areas with progression to cancer. The toxic effects of coal tar are enhanced by exposure to ultraviolet light. • Eyes: irritation and photosensitivity. • Respiratory system: cough, bronchitis, and bronchogenic cancer. • Gastrointestinal system: leukoplakia, buccal-pharyngeal cancer, and cancer of the lip. • Hematopoietic system: leukemia (inconclusive) and lymphoma. • Genitourinary system: hematuria and kidney and bladder cancers.

Exposure History	Exposure is most often determined based on the patient exposure history. A relevant patient history might include the following information: • occupational history, • occupation of the spouse and other household members, • use of medications, including coal tar-containing dermatologic preparations, • diet, especially charbroiled meats, • alcohol consumption; and • smoking habits. Hobbies and recreational activities might reveal additional evidence of exposure to PAH-containing mixtures. In general, risk increases with total dose. For more information on the exposure history, see the *Taking an Exposure History* CSEM at www.atsdr.cdc.gov/csem/exphistory/
Physical Examination	Physical examination is important. Physical examination should include a review of all systems, with the knowledge that cancer is the most significant endpoint of chronic PAH toxicity. If PAH exposure is suspected, the clinician should be alert to malignant transformation of actinic skin lesions. The buccal mucosa and oropharynx should be inspected for malignant changes. Inspection of sun-exposed areas for evidence of hyperpigmentation in response to sunlight is advised.
Direct Biological Measurement	Direct biologic measurement of PAHs is neither cost-effective nor clinically useful. Direct measurement refers to testing directly for the parent compound (or specific PAHs exposed to), not the metabolites. Although researchers have examined PAHs directly in the blood and tissues of experimental animals, these methods have not been widely used for human samples. The high costs of testing and limited knowledge of the significance of background levels in humans limit the clinical usefulness of such tests.
Indirect Biological Measurement	The most common tests for determining exposure to PAHs involve examining tissues, blood, and urine for the presence of metabolites. Pyrene is commonly found in PAH mixtures, and its urinary metabolite, 1-hydroxypyrene, has been used as an indicator of exposure to PAH chemicals [Becher and Bjorseth 1983; Granella and Clonfero 1993; Popp 1997; Santella *et al. 1*993, CDC 2005]. The ACGIH recommends measurement of 1-hydroxypyrene in the end-of-shift, end-of-work-week urine samples as a biological exposure index (BEI) for assessment of exposure to mixtures containing PAHs. This practice may help identify workplaces requiring improved industrial hygiene measures [ACGIH

2005; Heikkila *et al.* 1995].

In the Third National Report on Human Exposure to Environmental Chemicals, urinary levels of hydroxylated metabolites of PAHs were measured in a subsample of the National Health and Nutrition Examination Survey (NHANES) participants aged 6 years and older during 1999–2002. The geometric mean for 1-hydroxypyrene (ng/g of creatinine) for the U.S. population aged 6 years and older during 1999–2002 was 74.2, with a 95% confidence interval of (61.6–89.3).

Note that finding a measurable amount of one or more metabolites in the urine does not mean that the levels of the PAH metabolites cause an adverse health effect. Whether levels of PAH metabolites at the levels reported are cause for health concern is not known, and more research is needed. These data provide physicians with a reference range so that they can determine whether people have been exposed to higher levels of PAHs than are found in the general population. As well, the data help scientists plan and conduct research on exposure to PAHs and health effects.

Deoxyribonucleic acid (DNA) adducts may be used as an indicator of exposure in research settings and can be measured in a variety of biologic media [Popp 1997; Ross *et al.* 1991; Santella *et al.* 1993; Weyand and La Voie 1988]. For example, tissue in culture can be labeled with radioactive phosphorus and analyzed by thin-layer chromatography and scintillation to identify and quantify the DNA adducts formed. Also, an immunoassay technique, ELISA, has been developed to detect antibodies to the PAH-DNA adducts in blood. These tests are not readily available for routine clinical use.

PAH diol epoxides form adducts with hemoglobin in the red blood cells. These adducts can be quantified by use of fluorescence spectroscopy. This technique is limited in its potential usefulness, however, because of individual differences in PAH metabolism and the limited specificity of the technique itself.

In general, indirect biologic monitoring can be useful in determining whether exposure to PAHs has occurred. However, it is not clinically useful for evaluating individual patients because normal or toxic levels have not been determined. Arterial blood gases, a chest radiograph, and other monitoring might be indicated. Individual variability, confounding effects of drugs or cigarettes, and nonspecificity of techniques are likely to complicate the interpretation of the results, especially in low-level environmental exposures.

Employees exposed to CTPVs in the coke oven industry are covered by the coke oven emissions standard. This OSHA standard includes elements of medical surveillance for workers exposed to coke oven emissions. It should be noted that OSHA recommended surveillance set at the time of the standard might not necessarily be consistent with current evidenced based medical practice.

Key Points	• Acute effects attributed to PAH exposure are probably caused by other agents. • Exposure is most often determined based on the patient exposure history. • Pertinent exposure history should include past and current occupational, recreational, hobbies, dietary, and smoking assessments. • Physical examination is important, including a review of all systems. • Direct biologic measurement of PAHs is neither cost-effective nor clinically useful. • A commonly measured urinary metabolite used to assess PAH exposure is 1-hydroxypyrene. • DNA adducts may be used as an indicator of exposure in research settings and can be measured in a variety of biologic media.
Progress Check	17. Which of the following is (are) true regarding the evaluation of a patient exposed to PAHs? A. Urinary 1-hydroxypyrene levels are used as prognostic indicators. B. Direct biologic measurement of PAHs is clinically useful. C. Exposure is most often determined based on the patient exposure history. D. All of the above. *To review relevant content, see "Exposure History", "Direct Biological Measurement" and "Indirect Biological Measurement" in this section.* 18. Key features of the physical examination of PAH-exposed patients include which of the following? A. Inspection of the buccal mucosa and oropharynx. B. Inspection of skin, especially sun-exposed areas. C. Auscultation of the lungs. D. All of the above. *To review relevant content, see "Physical Examination" in this section.*

Answers

17. The correct answer is C. Exposure is most often determined based on the patient exposure history. Urinary 1-hydroxypyrene levels are used as exposure indicators, not prognostic indicators. Direct biologic measurement of PAHs is not clinically useful.
18. The correct answer is D. Physical examination of PAH-exposed patients includes a complete review of systems for carcinogenic endpoints, which includes the inspection of the buccal mucosa and oropharynx, inspection of skin (especially sun-exposed areas), and auscultation of the lungs.

How Should Patients Exposed to PAHs Be Treated and Managed?

Learning Objectives	Upon completion of this section, you will be able to • identify strategies for managing patients with acute high dose PAH exposure, and • identify strategies for managing patients with chronic low level PAH exposure.
Introduction	The management and treatment focus of individuals exposed to PAHs differs for high dose acute and low dose chronic exposures. Decontamination and supportive measures are the primary objectives after acute high dose PAH exposure. Treatment of chronic PAH toxicity is symptomatic and supportive. Health education and risk communication are important aspects of patient care. Some clinicians recommend periodic pulmonary function tests and chest x-rays for PAH-exposed individuals (inhalation exposures).
Acute High Dose Exposure	Decontamination and supportive measures are the primary objectives after acute high dose PAH exposure. Acute symptoms are generally from co-exposures to other substances. Contaminated clothing should be removed from the victim as soon as possible. The victim's skin should be decontaminated by gently scrubbing with soap and water. Ocular contamination should be treated with irrigation and a complete eye examination. Supportive care should be administered as clinically necessary.
Chronic Exposure and Toxicity	Effective risk communication and education for patients at risk for PAH-related disease is an important part of patient care. Persons exposed to potentially significant levels of PAHs should be aware of the increased risk for bronchogenic cancer and the additive effect of cigarette smoke and other toxic agents. Periodic evaluations of healthy patients who have been significantly exposed to PAHs, even in the absence of symptoms, may facilitate early diagnosis and intervention if a malignancy develops. The OSHA coke oven emissions standard includes elements of medical surveillance for workers exposed to coke oven emissions. It should be noted that OSHA recommended surveillance (set at the time of the standard) might not necessarily be consistent with current evidenced based medical practice.
Risk Communication in Patient Care	Because estimation of additional risk due to PAH exposure is often impossible, the challenge to the clinician is to maintain a balance between appropriate concern and undue alarm. Predicting the carcinogenicity of a complex chemical mixture based on one or several of its components is difficult because of possible interactions among the components. Effective risk communication takes this into account and can be important in prevention or management of disease.

Key Points	• Decontamination and supportive measures are the primary objectives after acute high dose PAH exposure. • Treatment of chronic PAH toxicity is generally symptomatic and supportive. • Effective risk communication and health education are important aspects of patient care. • Periodic evaluations of healthy patients who have been significantly exposed to PAHs, even in the absence of symptoms, is recommended by some clinicians to facilitate early diagnosis and intervention if a malignancy develops.
Progress Check	19. Primary strategies for managing patients with acute high dose exposure to PAH include(s) which of the following? A. Removal of contaminated clothing. B. Cleansing of skin with soap and water. C. Supportive care as clinically indicated. D. All of the above. *To review relevant content, see "Acute High Dose Exposure" in this section.* 20. When managing patients with chronic low-level exposure to PAH, strategies aimed at reducing patient risk of overexposure to PAHs and PAH related disease include (s) which of the following? A. Smoking cessation. B. Patient health education. C. Periodic evaluations for those significantly exposed. D. All of the above. *To review relevant content, see "Risk Communication in Patient Care" in this section.*

Answers

19. The correct answer is D. Since symptoms from acute PAH exposures are mainly from co-exposure to other substances, the primary strategies for managing patients with acute high dose exposure to PAH include removal of contaminated clothing, decontamination of skin with soap and water, and supportive care as clinically indicated.
20. The correct answer is D. When managing patients with chronic low level exposure to PAH, strategies aimed at reducing patient risk of overexposure to PAHs and PAH related disease include smoking cessation, patient health education, and periodic evaluations for those significantly exposed.

What Instructions Should Be Given to Patients to Prevent Overexposure to PAHs?

Learning Objective

Upon completion of this section, you will be able to

- describe care advice the clinician can provide to patients to prevent overexposure to PAHs.

Introduction

By utilizing effective risk communication techniques, the clinician can promote patient behaviors that may reduce the risk of PAH overexposure and PAH related disease. The clinician can provide advice on

- self-care, so that patients can minimize risk of PAH overexposure and
- when to follow-up with a health care provider.

ATSDR has developed patient education care instruction sheets for use in clinical settings; a list of these can be found at:

www.atsdr.cdc.gov/emes/health_professionals/instruction_sheets.html

Self-Care Advice

Self-care advice creates awareness and suggests actionable behaviors that may reduce the risk of PAH overexposure and PAH related disease.

Sample Advice	Rationale
Stop smoking and avoid exposure to smoke.	Smoking and exposure to second hand smoke increase the risk of lung cancer. Cigarette smoke contains PAHs and other carcinogenic substances. Exposure to PAHs by smoking or second hand smoke may increase the risk of overexposure to PAHs and PAH related disease.
Minimize dietary PAH exposures.	The FDA has not published PAH "safe levels" for foodstuffs. However, given that PAHs in food increase the exposure dose and risk of adverse health effects, efforts to minimize dietary contributions would be prudent. Foods that may contain PAHs include • charbroiled, chargrilled, and smoked meats and fish, • tea, • roasted peanuts, • coffee, • refined vegetable oil, • cereals, • spinach, • wheat, • rye, and • lentils.

Minimize hobby, recreational, and home/outdoor PAH exposures.	Awareness of potential PAH exposure through hobbies, recreational, and home/outdoor scenarios and taking action to minimize or avoid exposure may decrease the risk of PAH overexposure. Wearing gloves when working with cutting oils (as well as other PAH-containing substances encountered in hobbies, recreational, and home/outdoor scenarios) and avoiding outdoor burning practices are some examples of behaviors that would decrease total PAH exposure dose.

Advice on when to Follow up with a Health Care Provider

Patients should be advised to consult their physician if they develop signs or symptoms to include

- a new cough or chronic cough with or without hemoptysis,
- unexplained weight loss,
- shortness of breath, and
- other applicable health changes associated with cancer or other serious health condition such as increased fatigue and weakness, recurring respiratory infections, etc.

Key Points

- The clinician can promote patient behaviors that may reduce the risk of PAH overexposure and PAH related disease by providing advice on
    - self-care, so that patients can minimize risk of PAH overexposure and
    - when to follow-up with a health care provider.

Progress Check

21. Clinicians can help their patients reduce the risk of overexposure to PAHs and PAH related disease by

    A. Offering information and assistance with smoking cessation.
    B. Providing information on PAH related health effects.
    C. Providing information on behaviors that can reduce the risk of PAH overexposure and PAH related disease.
    D. All of the above.

    To review relevant content, see "Self Care Advice" in this section.

Answers

21. The correct answer is D. Clinicians can help their patients reduce the risk of overexposure to PAHs and PAH related disease by offering information and assistance with smoking cessation, providing information on PAH related health effects, and providing information on behaviors that can reduce the risk of PAH overexposure and PAH related disease.

Sources of Additional Information

Polycyclic Aromatic Hydrocarbons (PAHs) Specific Information

Please refer to the following Web resources for more information on the adverse effects of PAH toxicity, the treatment of PAH-associated diseases, and management of persons exposed to PAHs.

- Agency for Toxic Substances and Disease Registry
  www.atsdr.cdc.gov
  - For chemical, emergency situations
    - **CDC Emergency Response: 770-488-7100 and request the ATSDR Duty Officer**
  - For chemical, non- emergency situations
    - CDC-INFO (www.bt.cdc.gov/coca/800cdcinfo.asp)
    - 800-CDC-INFO (800-232-4636) TTY 888-232-6348 - 24 Hours/Day
    - E-mail: cdcinfo@cdc.gov

      PLEASE NOTE
      ATSDR cannot respond to questions about individual medical cases, provide second opinions or make specific recommendations regarding therapy. Those issues should be addressed directly with your health care provider.
  - Toxicological Profile for Polycyclic Aromatic Hydrocarbons
    www.atsdr.cdc.gov/toxprofiles/tp69.html
  - ToxFAQs™ for Polycyclic Aromatic Hydrocarbons
    www.atsdr.cdc.gov/tfacts69.html
  - Agency for Toxic Substances and Disease Registry Toxic Substances and Your Health - Polycyclic Aromatic Hydrocarbons (www.atsdr.cdc.gov/substances/PAHs/

Clinical Resources

- American College of Occupational and Environmental Medicine (ACOEM) www.acoem.org
  - ACOEM is the nation's largest medical society dedicated to promoting the health of workers through preventive medicine, clinical care, research, and education.
  - Its members are a dynamic group of physicians encompassing specialists in a variety of medical practices is united via the College to develop positions and policies on vital issues relevant to the practice of preventive medicine both within and outside of the workplace.
- American College of Medical Toxicologists (ACMT) www.acmt.net
  - ACMT is a professional, nonprofit association of physicians with recognized expertise in medical toxicology.
  - The College is dedicated to advancing the science and practice of medical toxicology through a variety of activities.
- Association of Occupational and Environmental Clinics www.aoec.org
  - The Association of Occupational and Environmental Clinics (AOEC) is a network of more than 60 clinics and more than 250 individuals committed to improving the practice of occupational and environmental medicine through information sharing and collaborative research.
- Pediatric Environmental Health Specialty Units (PEHSUs) www.pehsu.net
  - Each PEHSU is based at an academic center and is a collaboration between the pediatric clinic and the (AOEC) occupational and environmental clinic at each site.
  - The PEHSU's have been developed to provide education and consultation for health professionals, public health professionals and others about the topic of children's environmental health.
  - The PEHSU staff is available for consultation about potential pediatric environmental health concerns affecting both the child and the family. Health care professionals may contact their regional PEHSU site for clinical advice.
- Poison Control Center
  - The American Association of Poison Control Centers may be contacted for questions about poisons and poisonings. The web site provides information about poison centers and poison prevention. AAPC does not provide information about treatment or diagnosis of poisoning or research information for student papers.
  - American Association of Poison Control Centers (1-800-222-1222 or www.aapcc.org).

General Environmental Health Information

Please refer to the following Web resources for general information on environmental health.

- Agency for Toxic Substances and Disease Registry www.atsdr.cdc.gov/
    - To view the complete library of CSEMs www.atsdr.cdc.gov/csem/.
    - Taking an Exposure History CSEM www.atsdr.cdc.gov/csem/exphistory/
- Centers for Disease Control and Prevention (CDC) www.cdc.gov
    - CDC works to protect public health and the safety of people, by providing information to enhance health decisions, and promotes health through partnerships with state health departments and other organizations.
    - The CDC focuses national attention on developing and applying disease prevention and control (especially infectious diseases), environmental health, occupational safety and health, health promotion, prevention and education activities designed to improve the health of the people of the United States.
- National Center for Environmental Health (NCEH) www.cdc.gov/nceh/
    - NCEH works to prevent illness, disability, and death from interactions between people and the environment. It is especially committed to safeguarding the health of populations that are particularly vulnerable to certain environmental hazards - children, the elderly, and people with disabilities.
    - NCEH seeks to achieve its mission through science, service, and leadership.
- National Institute of Health (NIH) www.nih.gov
    - A part of the U.S. Department of Health and Human Services, NIH is the primary Federal agency for conducting and supporting medical research.
- National Institute of Occupational Safety and Health (NIOSH) www.cdc.gov/niosh/
    - NIOSH is in the U.S. Department of Health and Human Services and is an agency established to help assure safe and healthful working conditions for working men and women by providing research, information, education, and training in the field of occupational safety and health.

- U.S. Department of Labor, Occupational Safety and Health Administration (OSHA) www.osha.gov

  - The mission of OSHA is to assure safe and healthful working conditions for working men and women

- U.S. Environmental Protection Agency www.epa.gov

  - EPA leads the nation's environmental science, research, education and assessment efforts.
  - The mission of the Environmental Protection Agency is to protect human health and the environment.
  - Since 1970, EPA has been working for a cleaner, healthier environment for the American people

Assessment and Posttest

Introduction	ATSDR seeks feedback on this course so we can asses its usefulness and effectiveness. We ask you to complete the assessment questionnaire online for this purpose. In addition, if you complete the assessment and posttest online, you can receive continuing education credits as follows.

Accrediting Organization	Credits Offered
Accreditation Council for Continuing Medical Education (ACCME)	**CME**: The Centers for Disease Control and Prevention is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians. The Centers for Disease Control and Prevention designates this educational activity for a maximum of **2** AMA PRA Category 1 Credit(s)™. Physicians should only claim credit commensurate with the extent of their participation in the activity.
American Nurses Credentialing Center (ANCC), Commission on Accreditation	**CNE**: The Centers for Disease Control and Prevention is accredited as a provider of Continuing Nursing Education by the American Nurses Credentialing Center's Commission on Accreditation. This activity provides **2** contact hours.
National Commission for Health Education Credentialing, Inc. (NCHEC)	**CHES**: The Centers for Disease Control and Prevention is a designated provider of continuing education contact hours (CECH) in health education by the National Commission for Health Education Credentialing, Inc. This program is a designated event for the Certified Health Education Specialist (CHES) to receive **2** Category I contact hours in health education, CDC provider number GA0082.
International Association for Continuing Education and Training (IACET)	**CEU**: The CDC has been approved as an Authorized Provider by the International Association for Continuing Education and Training (IACET), 1760 Old Meadow Road, Suite 500, McLean, VA 22102. The CDC is authorized by IACET to offer **0.2** IACET CEU's for this program.

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Instructions	To complete the assessment and posttest, go to www2.cdc.gov/atsdrce/ and follow the instructions on that page. You can immediately print your continuing education certificate from your personal transcript online. No fees are charged.

Posttest

There may be more than one correct answer per question.

1. Other names for PAHs include which of the following?

   A. Polynuclear aromatics (PNAs).
   B. Polynuclear hydrocarbons.
   C. Polynuclear aromatic hydrocarbons.
   D. Polycyclic organic matter (POM).

2. Which of the following statements regarding PAHs is (are) true?

   A. Heavier PAHs (more than 4 rings) tend to adsorb to particulate matter.
   B. Lighter PAHs (less than 4 rings) tend to remain gaseous until removed via precipitation.
   C. PAH concentration in water tends to be low (around 100 ng/l) due to their weak solubility.
   D. PAHs can be absorbed by plants.
   E. PAHs mostly accumulate in soil.

3. Potential sources of PAH exposure include

   A. Passive inhalation of cigarette smoke.
   B. Motor vehicle exhaust.
   C. Alcoholic beverages.
   D. Inhalation of paint vapors in poorly ventilated area.
   E. Wood stoves for home heating.

4. Which of the following statements regarding PAHs are **FALSE**?

   A. PAHs are found only in a small number of industrial settings.
   B. PAHs mostly accumulate in soils.
   C. Benzo[a]pyrene is generally used as an environmental indicator for PAHs.
   D. Most PAHs in ambient air are the result of man-made processes.

5. In non-occupational settings, the majority of PAH exposure in a nonsmoking individual comes from which of the following?

   A. Foodstuff ingestion.
   B. Inhalation route.
   C. Dermal route.
   D. Water.

6. Persons with potentially increased PAH exposure include

   A. Hunters.
   B. Coke oven workers.
   C. Roofing asphalt applicators.
   D. Chimney sweeps.

E. Breastfeeding mothers.

7. Which of the following statements are true about PAHs?

A. Exposure is most often determined based on patient history.
B. Direct assays in the body are not clinically useful.
C. Exposure can cause pancreatitis.
D. Acute exposure can cause convulsions or unexplained loss of consciousness.
E. The prognosis for most acutely exposed patients is poor.

8. Which of the following statements regarding OSHA's standards for PAHs is/are **FALSE**?

A. OSHA requires workers who are exposed to PAH above the PEL to be under medical surveillance.
B. OSHA requires all workers to be trained in proper use of appropriate personal protective equipment and safety.
C. Purchase of personal protective equipment is the responsibility of individual employees.
D. The OSHA PEL is a legally enforceable standard.

9. Which of the following statements regarding PAHs in water is/are true?

A. The EPA maximum contaminant level is a legally enforceable standard.
B. The maximum contaminant level is an 8-hour time-weighted average.
C. EPA has set maximum contaminant levels for PAHs in foodstuffs.
D. EPA developed ambient water quality criteria to protect human health from the carcinogenic effects of PAH exposure.

10. Which of the following regarding the biologic fate of PAHs in the body are true?

A. PAHs are metabolized in a number of organs and excreted in bile and urine.
B. Information on the absorption, distribution, and elimination of PAHs in the human body is derived primarily from animal studies.
C. Generally, PAHs bound to airborne particulate matter are not absorbed as well in the lungs as the same dose of PAHs that are unbound to particulate matter.
D. The liver P-450 mixed-function oxidase system is the predominant mechanism of PAH metabolism.
E. 1-Hydroxypryrene is a commonly measured urine metabolite for PAH exposure.

11. The following signs and symptoms can be found in patients

chronically exposed to PAHs

A. Chloracne.
B. Bronchitis.
C. Vertigo.
D. Exotropia.
E. Cutaneous photosensitization.

12. The mechanism of PAH-induced carcinogenesis is believed to be which of the following?

A. Covalent binding of PAH metabolites to DNA.
B. Generation of active oxygen species.
C. Cell-mediated inflammatory mechanisms.
D. All of the above.

13. Reported health effects associated with chronic exposure to coal tar and its by-products (*e.g.*, PAH) include

A. Warts on sun-exposed areas of the skin, with progression to cancer.
B. Irritation of the eyes.
C. Bronchogenic cancer.
D. Leukoplakia.
E. Lymphoma.

14. In the treatment of patients with PAH exposure, which of the following is/are true?

A. Education and future avoidance of exposure are important.
B. Continued use of tobacco products should be discouraged.
C. Treatment of acute exposure is largely symptomatic.
D. The specific PAH should be determined so that an antidote can be prescribed.
E. A fat biopsy is integral to medical surveillance of PAH-exposed patients.

15. Which of the following should be included in the differential diagnosis of a patient suffering from the chronic effects of PAH exposure?

A. Pancytopenia.
B. Hepatic angiosarcoma.
C. Pancreatitis.
D. Tuberculosis.
E. Lung abscess.

16. Regarding PAH distribution, metabolism, and excretion, which are true?

A. The liver and kidney are both involved in metabolism.

	B. Binding of PAH metabolites to DNA is believed to be the mechanism of PAH-induced carcinogenesis. C. Metabolized PAHs cannot be eliminated by hepatobiliary excretion. D. Excretion is through bile and urine. E. Calcium EDTA chelation enhances PAH excretion. 17. Which of the following statements is (are) true? A. Management of a worker exposed to PAHs includes bone marrow aspiration. B. PAH metabolites can cross the placental barrier. C. Acutely exposed skin should be decontaminated by gently scrubbing with a 10% iodine solution. D. Hair analysis can reveal past PAH exposure. E. The bay region theory attempts to explain why PAHs are found in bay waters. 18. What steps can patients take to reduce the risk of overexposure to PAHs? A. Minimize hobby and recreational PAH exposures. B. Avoid exposure to all forms of smoke. C. Stop smoking. D. Minimize dietary PAH exposure. E. All of the above.
Relevant Content	To review content relevant to the post-test questions, see:

Question	Location of Relevant Content
1	What are PAHs?
2	What are PAHs? Where are PAHs found?
3	Where are PAHs found?
4	Where are PAHs found?
5	What are routes of exposure for PAHs?
6	Who is at risk of PAH exposure?
7	Clinical Assessment
8	What are standards and regulations for PAH exposure?
9	What are standards and regulations for PAH exposure?
10	What is the biologic fate of PAHs in the body?
11	Clinical assessment
12	How do PAHs induce pathogenic changes?

13	What health effects are associated with PAH exposure?
14	How should patients exposed to PAHs be treated and managed?
15	Clinical assessment
16	What is the biologic fate of PAHs? How do PAHs induce pathogenic changes?
17	What are routes of exposure for PAHs?
18	What instructions should be given to patients exposed to PAHs?

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APPENDIX Q

TECHNICAL PAPER

Airborne polycyclic aromatic hydrocarbons in a medium-sized city affected by preharvest sugarcane burning and inhalation risk for human health

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Polycyclic aromatic hydrocarbons (PAHs) in air were measured in a municipality where sugarcane plantations are extensive, at three sites, one in the city center and two in rural localities. Twenty-four-hour sampling was done using PS1 PUF samplers from Andersen Instruments Inc., at least 1 day per month per site, from June 2009 to October 2009. The chemical analyses were performed by gas chromatography–mass spectrometry (GC/MS) for the 16 most toxic PAHs. The incremental lifetime cancer risk (ILTR) by inhalation was determined by the Monte Carlo method for the urban population using Crystal Ball software. The total concentration of the 16 PAHs at all sites varied from 6.2 to 65.7 ng m^{-3}, with an average of 25.9 ± 18.2 ng m^{-3}. The average concentrations per site were 14.1 ± 13.0 ng m^{-3} at rural site B, 20.7 ± 11.5 ng m^{-3} at rural site A, and 36.1 ± 22.7 ng m^{-3} at the central site. The cancer risk for infants, children, and adults was approximately 14%, 25%, and 61% of the total IRLT, respectively. The mean (95% upper probability limit [95% UPL]) values were 1.2 × 10^{-7} (2.2 × 10^{-7}) for infants, 2.2 × 10^{-7} (4.1 × 10^{-7}) for children, and 8.9 × 10−7 (1.1 × 10^{-6}) for adults. Although the three most abundant PAHs found were phenanthrene, fluoranthene, and pyrene, the three most important contributions to the incremental risk of cancer came from benzo[a]pyrene, benzo[b] fluoranthene, and naphthalene. Compared with the risks in big cities such as São Paulo, this would be low, but not negligible. Analysis of ratios of PAHs according to the literature showed that vehicle exhaust and biomass burning, including sugarcane burning, seem to be the most important contributors to PAH concentrations in the central area of Araraquara City.

Implications: The growth of biofuel use worldwide, especially ethanol, together with preharvesting burning practice, is cause of concern with regard to possible health effects, due to increased air pollution levels in cities in regions where sugarcane plantation and processing are intensive. This paper shows that the risk of cancer from PAH inhalation in an urban area surrounded by sugarcane agriculture was of the same order of magnitude as the tolerable risk value of 10^{-6}. As other classical and hazardous pollutants are also present, care should be taken to keep pollution as low as possible to protect human health.

Introduction

Approximately 92% of the light-duty cars currently produced in Brazil are flex-fuel vehicles, and the majority of these vehicles use hydrated ethanol as fuel (UNICA [Brazilian Sugarcane Industry Association], 2010). The USA (38% share) and Brazil (31% share) are the two largest producers of ethanol in the world, and there is a trend towards increasing production because of the rising global demand for biofuel (Market Research Analyst, 2008). According to a U.S. Environmental Protection Agency (EPA) (2010b) announcement, ethanol from sugarcane complies with the applicable 50% greenhouse gas (GHG) reduction threshold for the advanced fuel category. It is foreseen that Brazil will continue to increase its production of ethanol and that the area planted with cane will increase by nearly the same proportion. Currently, 8.5 million ha have been planted with sugarcane and are primarily located in the southeast region of Brazil (UNICA, 2010).

The large area of sugarcane plantation and the preharvest practice of burning are a cause of concern because of the potential health effects and environmental impact that result from increased air pollution in municipalities where sugarcane is a monoculture. This concern is related to the toxic substances that arise from sugarcane production such as polycyclic aromatic hydrocarbons (PAHs) because of both preharvest burning practices and biomass burning at sugar and ethanol production plants.

In the traditional method, sugarcane is burned prior to harvesting to help protect workers who perform manual cut and to facilitate the task of cane cutting. The preharvest burning has enabled daily increased productivity by almost 3-fold, as it reduces the physical effort expended at work. However, the emissions from field burning create health and environmental

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impacts. As a consequence of a state law and an agreement with producers, this practice is being replaced by mechanical cut of the green sugarcane in order to prevent preharvest burning, although at a higher cost.

In previous studies on a sugarcane plantation, Ribeiro (2009) and Ribeiro and Pesquero (2010) identified indications that pollution from the sugarcane harvesting processes that are in current use represents a respiratory health risk factor for children, even when the pollution is below the recommended limits. Further study is needed to assess these effects, especially when sugarcane plantations are expanding in Brazil.

In Brazil, few studies on the contents of PAHs in urban environments have been reported, and they are presented in Table 1. The majority of these studies have been restricted to the states of Bahia, Rio de Janeiro, and São Paulo.

Studies conducted in areas where preharvest sugarcane burning occurs have found important differences in the concentrations of PAHs during the harvesting and nonharvesting periods (Andrade et al., 2010; Cristale et al., 2012). Umbuzeiro et al. (2008) found that the mutagenic activity of inhalable particulate matter (aerodynamic diameter <10 μm; PM_{10}) was higher in samples from regions with sugarcane harvesting than it was in samples collected in São Paulo itself.

The data in Table 1 show that the areas with higher PAH levels were those where sugarcane plantations coexist with sugar and ethanol production plants, specifically the municipalities of Araraquara (maximum level of 1794 ng m^{-3} of total PAHs in 1995) and Campos dos Goytacazes (maximum level of 342 ng m^{-3} in 2002).

The carcinogenic effects from exposure to PAHs in populations living in sugarcane-producing regions are poorly known. Predictive risk assessment methods have been recommended to assess the increased cancer risk from outdoor concentrations of PAHs and other toxic pollutants when the epidemiological studies for identifying very low increments of risk are limited or do not provide information on the important sources or pollutants (Meneses et al., 2004; Woodruff et al., 2000). Chen and Liao (2006) and Bai et al. (2009) used quantitative health risk assessments for environmental exposure to PAHs.

This study estimated the incremental cancer risk in Araraquara, which is an important municipality in Brazil in terms of sugarcane production and processing, where several

Table 1. Summary of PAH measurement studies performed in Brazil

Municipality (State)	Location	Date	Media/Particle Size	PAH	Concentration (ng m^{-3})	Reference
Salvador (BA)	Urban—bus station	April and August 1991	QFF/TSP	Chrysene	26.6	Pereira et al. (2002)
	Urban tunnel			Pyrene	79.4 ± 11.5	
				Fluoranthene	39.0 ± 5.2	
Rio de Janeiro (RJ)	Urban	Summer 1999	GFF/TSP	16 PAHs	23.4	Pereira Netto et al. (2001, 2002)
Niterói (RJ)		Summer 1999			6.78	
		Winter 1999			6.94	
Campos de	Urban	December 1996 to November 1997	QFF/TSP	Total PAHs	9.31–342	Azevedo et al. (2002)
Goytacazes	Suburban				1.03–312	
(RJ)[a]	Rural				0.12–165	
São Paulo (SP)	Urban	October 1995	Two PUF samplers in series; TSP and gas phase	15 PAHs	38–136	CETESB (2002)
Araraquara (SP)[a]		October 1995			9–110	
	Rural	Apr 1996, July 1996			413–1794	
Cubatão (SP)	Urban and Industrial	November and December 1995			33–167	
São Paulo (SP)	Urban	Winter 2000	QFF/TSP	Total PAHs	3.10	Vasconcellos et al. (2003)
	Urban forest				1.92	
	Urban	February to May 2000	PTFE-GFF/PM_{10}	17 PAHs	82.3	De Martinis et al. (2002)
Araraquara (SP)[a] urban	Harvest season	August 2002, September 2003	PTFE-GFF/PM_{10}	13 PAHs	11.6	Andrade et al. (2010)
	Nonharvest season	Mar 2003, January 2004			2.5	
	Indoor, harvest season	August 2007	PFTE/PM_{10}	13 PAHs	22.9	Cristale et al. (2012)
	Indoor, nonharvest season	January 2008			2.35	

Notes: [a]Sugarcane burning. QFF = quartz fiber filter; TSP = total suspended particulate; GFF = glass fiber filter; PUF = polyurethane foam; PTFE-GFF = polytetrafluoroethlytene filter–coated GFF; PM_{10} = particulate matter less than 10 μm in diameter; PTFE = polytetrafluoroethlytene filter.

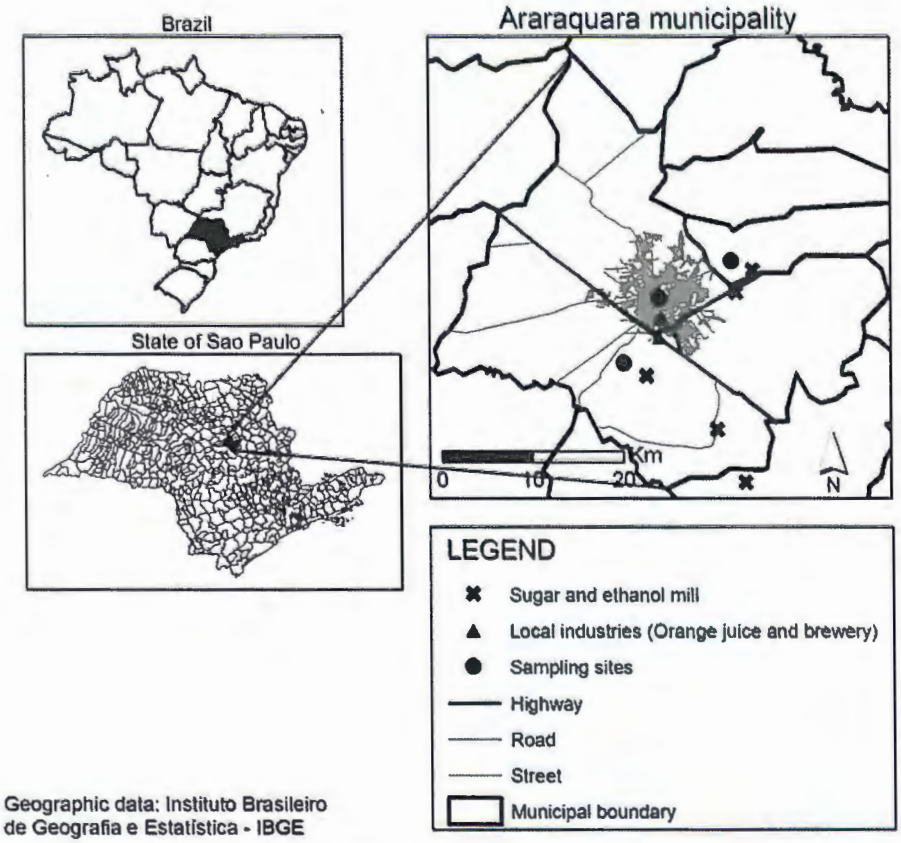

Figure 1. Municipality of Araraquara: Geographical position, and location of point sources and sampling sites.

studies on air pollution have been conducted and where the preharvest burning of sugarcane crops is still practiced.

The municipality of Araraquara is located in the central region of the state and is 254 km from the city of São Paulo. Its location is shown in Figure 1. It has a total area of 1006 km^2 and lies in the transition zone between the Peripheral Depression and Western Paulista Upland, with altitudes ranging from 600 to 715 m. Its climate can be characterized as high-lying tropical, with a rainy summer and dry winter. The average temperature is 21.7 °C, the average of the minimum temperature of the coolest month is 10.8 °C, and the average maximum temperature in the hottest month is 29.6 °C. The average annual rainfall is 1430 mm, and 70% of this rain falls between October and March.

Araraquara had 208,662 inhabitants in 2010; most of them (97.16%) lived in urban areas, with a fleet of 74,533 vehicles (CETESB [Sao Paulo State Environmental Agency], 2010). Six factories, which include four sugar and ethanol production plants, one brewery, and a concentrated orange juice plant, are the most important emission sources of particulate matter considered in the source inventory produced by the state environmental agency (CETESB, 2010), which did not consider emissions from biomass burning in the open air. For the year 2009, 379 million square meters of land were cultivated with sugarcane in Araraquara, and 38% of this land was subject to preharvest burning. The production cycle of sugar and ethanol mills spans 24 hr a day and 7 days a week during the harvesting season, which usually lasts from April to November and is more intensive from May to October. In the nonharvesting season, the work is only performed during the day 5 days a week, and it usually lasts from 8 a.m. to 6 p.m.

Materials and Methods

Sampling sites

Air samples were collected at three sites: one downtown (Central station), where traffic is heavier, and two in rural areas (Rural A and Rural B stations) to obtain a better average for the rural concentrations. The rural stations were placed near areas of intense agricultural activity that were exposed to emissions from sugarcane activities, including emissions from preharvest burning and sugarcane processing plants (producing sugar and ethanol). The Rural A site is south of the city, and Rural B is east of the city. The Araraquara urban area and rural sampling points are surrounded by sugarcane plantations. Preharvest burning events occur randomly throughout the area. In addition, the rural sampling points were placed in areas that are more favorably affected by emissions from sugarcane activities according to the prevailing winds. Two sugar and ethanol mills are located northeast of the city at distances of 7 and 8 km from the city center, a sugar alcohol mill is located south of the city and 8 km from the center, and two mills are located south-southeast at distances of 12 and 13.5 km from the city center.

Sample collection

Sample collecting was performed in accordance with EPA method TO-13A (EPA, 1999) using a PS1 polyurethane foam (PUF) sampler (Andersen Instruments Inc.; Smyrna, GA, USA) that was assembled with 10.16-cm-diameter quartz microfiber filters (Whatman Inc.; Clifton, NJ, USA), followed by a 6-cm-diameter and 7.62-cm-length PUF sampler (Tisch Environmental, Inc.; Cleveland, OH, USA). Samplings were conducted for 24 hr at least 1 day per month from June 2009 to October 2009 during the sugarcane harvesting period. The sampling flow rate varied from a minimum of 0.241–0.272 $m^3\ min^{-1}$ and produced an average flow rate of 0.257 $m^3\ min^{-1}$ (at 25 °C and 1 atm).

The time period was chosen to contain the harvesting season and the worst meteorological conditions because the meteorological conditions in the region during late autumn (April and May) and winter (June to September) favor an increase in pollution as a result of lower wind speeds, more frequent thermal inversions of low altitude, and decreased precipitation levels. At the same time, the number of outbreaks of fire spots increased. The environmental contour could be traced by the higher PM_{10} concentrations, as shown in Figure 2, using the data provided by the environmental agency (CETESB, 2010).

A field blank was analyzed in each sampling campaign. Thursday was the customary sampling day, and holidays and weekends were excluded. In the first campaign in June 2009, samples were collected only at the Central and Rural A sites because of a lack of permission from the industry. In the fourth sampling campaign in October 2009, no samples were collected at the Rural A site because of operating problems with one of the samplers.

Extraction and analysis

The extraction and chemical analysis of PAHs was undertaken by gas chromatography–mass spectrometry (GC/MS) based on EPA method 8270C (EPA, 1996). Filters and PUF samplers were combined for the extraction, and the examined chemical species were those of toxicological interest that, according to the current information, constituted a set of 16 in the group of PAHs.

Quality assurance/quality control (QA/QC)

Field blanks were used to determine the contamination as a result of transport and manipulation, and the results from field blanks were subtracted from the sample concentrations. Blank contamination was higher for naphthalene, which was found in all blanks; acenaphthylene, acenaphthene, fluorene, phenanthrene, and fluoranthene were found in 75% of the blanks; anthracene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, and benzo[*a*]pyrene were found in 50% of the blanks; and benzo[*a*]anthracene, chrysene, indeno[1,2,3-*c,d*]pyrene, dibenz[*a,h*]anthracene, benzo[*g,h,i*]perylene were found in 25% of the blanks.

In addition, surrogate standards (2-fluorobiphenyl, and *p*-terphenyl-d14) were added to all of the samples to monitor the performance of the laboratory procedures. The recovery efficiency of 2-fluorobiphenyl ranged from 51% to 73% (average 60%), and that of *p*-terphenyl-d14 ranged from 47% to 126% (average 76%). The PAH concentrations in the samples were not corrected for recovery factors.

The detection limit of GC/MS for the 16 PAHs was 8.0 ng. The laboratory responsible for the chemical analyses holds an International Organization For Standardization/International Electrotechnical Commission (ISO/IEC) 17025:2001 certificate for PAH analysis in air samples.

Risk analysis

The incremental lifetime cancer risk (ILTR) from the inhalation of PAH was calculated only for the urban population because less than 3% of the total Araraquara municipality population resided in rural areas in this municipality. The rural risk would be lower than the urban risk according to the results of the PAH concentrations, and the ILTR was estimated by considering the average PAH concentrations measured in the Central area for the PAHs that were classified as carcinogenic or most likely/possibly carcinogenic for humans by the International Agency For Research On Cancer (IARC) or the EPA. Cancer risk was estimated for three specific age groups: adults, children, and infants. For each PAH, the mean concentration and minimum and maximum values for the 95%

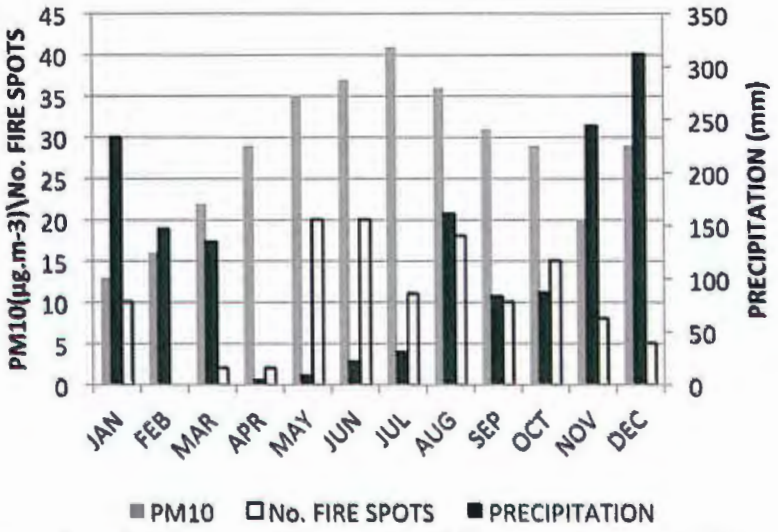

Figure 2. Average PM_{10} concentrations (central station), number of fire spots and rainfall in Araraquara, by month, in 2009.

Table 2. Exposure parameters adopted in the risk analysis

Parameter	Unit	Infants	Children	Adults	Distribution Function
Age	yr	0–5	6–19	20–70	
Body weight (BW)[a]	kg	14.5 ± 4.9	43.8 ± 12.3	62.8 ± 2.7	Log-normal
Exposure duration	yr	5	14	51	
Averaging time	day	25550	25550	25550	
Inhalation rate (IR_{ar})[b]	m^3/day	8.3 ± 2.2	15.6 ± 4.0	16.6 ± 4.0	Log-normal
Exposure frequency	day/yr	335; 350; 365	335; 350; 365	335; 350; 365	Triangular
Conversion factor		1×10^{-6}	1×10^{-6}	1×10^{-6}	
PAH concentration	$\mu g\ m^{-3}$	min; mean; max	min; mean; max	min; mean; max	Triangular

Notes: [a]IBGE (2010); [b]EPA (2010a).

confidence interval were estimated. For exposure frequency, it was assumed that people can leave town for up to 30 days per year for vacation. In Brazil, however, this is not common for everyone. The triangular distribution function was assumed for the PAH concentrations and for the exposure frequency because it is a conservative estimate of the actual distribution when there is great uncertainty regarding the available data. For the other exposure factors, log-normal distributions (Finley et al., 1994) were considered. The inhalation slope factors were obtained from the Office of Environmental Health Hazard Estimates (OEHHA) (2010), which considered the toxicity equivalency factor (TEF) values presented by Collins et al. (1998). The Monte Carlo method was utilized to estimate the risk and sensitivity analysis with the Crystal Ball software. The number of simulations was 100,000, and the adopted exposure factors are shown in Table 2.

The ILTR was calculated for *i*th PAHs and *j*th age group using eq 1:

$$ILTR_{ij} = CSF_i \frac{C_{ai} \cdot IR_j \cdot EF_j \cdot ED_j \cdot CF}{LT \cdot BW_j} \quad (1)$$

where

C_{ai} is the concentration of *i*th PAH in the air (mg m^{-3}),
IR_j is the air inhalation rate of the *j*th age group (m^3 day^{-1}),
EF_j is the exposure frequency of the *j*th age group (day yr^{-1}),
ED_j is the exposure duration of the *j*th age group (yr),
BW_j is the body weight for the *j*th age group (kg),
CSF_i is the inhalation cancer slope factor of *i*th PAH (kg day mg^{-1}),
LT is the averaging time (equal to 70 years for carcinogens), and
CF is a unity conversion factor.

The total ILTR was determined using eq 2:

$$ILTR = \sum_{i=1}^{n} \sum_{j=1}^{3} ILTR_{ij} \quad (2)$$

Results

The results for 16 PAHs (Table 3) showed that the total concentration of the sixteen PAHs at all of the sites varied from 6.2 to 65.7 ng m^{-3}, with an average of 25.9 ± 18.2 ng m^{-3}. The averages per site were 14.1 ± 13.0 ng m^{-3} at the Rural B site, 20.7 ± 11.5 ng m^{-3} at the Rural A site, and 36.1 ± 22.7 ng m^{-3} at the Central site.

The three most abundant PAHs were phenanthrene, fluoranthene, and pyrene, in that order, for all sites, as shown in Figure 3.

The ILTR values resulting from the inhalation of carcinogenic PAHs calculated for the urban population of Araraquara are shown in Table 4.

The cancer risk for infants, children and adults was approximately 14%, 25%, and 61% of the total IRLT, respectively. The mean (95% upper probability limit [95% UPL]) values were 1.2×10^{-7} (2.2×10^{-7}) for infants, 2.2×10^{-7} (4.1×10^{-7}) for children, and 8.9×10^{-7} (1.1×10^{-6}) for adults. Comparison was made between the contributions of each PAH to the total concentration of PAH used to calculate risk and the contribution to the total IRLT (Figure 4). Although the three most abundant of the 16 PAHs analyzed were phenanthrene, fluoranthene, and pyrene, the three most important contributions to the incremental risk of cancer were benzo[*a*]pyrene, benzo[*b*] fluoranthene, and naphthalene.

To analyze the possible sources of PAHs in Araraquara, the PAH ratios were used in accordance with the information from several sources, as shown in Table 5. The benzo[*a*]pyrene/benzo[*g,h,i*]perylene (BaP/B(ghi)P) ratios obtained at the Araraquara Central site indicate that the source of PAHs was primarily vehicles of all types, and diesel vehicles were specifically mentioned. The phenanthrene/anthracene (PHE/AN), fluoranthene/pyrene (FLT/PYR), fluoranthene/(fluoranthene+pyrene) (FLT/(FLT+PYR)), and indeno[1,2,3-*c,d*]pyrene/(indeno[1,2,3-*c,d*]pyrene+benzo[*g,h,i*]perylene) (IcDP/(IcDP+BghiP)) ratios show that the PAH emission sources were coal and biomass combustion. Because coal is not used in the region, biomass combustion was a possible source of PAHs. The benz[*a*]anthracene/chrysene (BaA/CRY), fluorene/(fluorene+pyrene) (FL/(FL+PYR)), and indeno[1,2,3-*c,d*]pyrene/(indeno[1,2,3-*c,d*]pyrene+benzo[*g,h,i*]perylene) (IcDP/(IcDP+BghiP)) ratios show that gasoline combustion was also an important PAH source. In conclusion, vehicle exhaust and biomass burning, which includes sugarcane and urban vegetation burning, were the most important contributors to PAH concentrations in the central area of the city of Araraquara.

Table 3. Results of the average, minimum, and maximum PAH concentrations in the municipality of Araraquara (ng m^{-3})

	Rural A			Rural B			Central		
PAH	Average	Min.	Max.	Average	Min.	Max.	Average	Min.	Max.
Naphthalene	0.49	0.02	1.21	0.90	0.02	1.69	1.14	0.02	1.8
Acenaphthylene	0.36	0.02	0.73	0.45	0.02	1.23	0.73	0.26	0.96
Acenaphthene	0.03	0.02	0.03	0.02	0.02	0.02	0.10	0.02	0.27
Fluorene	0.31	0.02	0.49	0.14	0.02	0.27	0.60	0.26	0.87
Phenanthrene	5.8	2.2	8.6	4.4	2.0	9.0	13.0	2.9	18.4
Anthracene	0.28	0.10	0.46	0.20	0.02	0.49	1.16	0.21	1.6
Fluoranthene	4.9	3.6	7.3	3.1	1.2	6.4	7.5	1.6	9.3
Pyrene	4.7	3.3	7.6	2.6	0.81	5.7	6.7	1.4	9.3
Benzo[*a*]anthracene	0.35	0.16	0.49	0.13	0.05	0.27	0.64	0.08	1.1
Chrysene	0.64	0.42	0.79	0.36	0.19	0.63	1.06	0.24	1.6
Benzo[*b*]fluoranthene	0.79	0.37	1.29	0.32	0.14	0.66	1.07	0.26	1.5
Benzo[*k*]fluoranthene	0.34	0.02	0.65	0.41	0.08	0.72	0.42	0.08	0.69
Benzo[*a*]pyrene	0.42	0.13	0.44	0.26	0.14	0.36	0.53	0.18	0.64
Indeno[1,2,3-*c,d*]pyrene	0.33	0.08	0.51	0.16	0.05	0.22	0.62	0.05	1.1
Dibenz[*a,h*]anthracene	0.02	0.02	0.02	0.02	0.02	0.02	0.04	0.02	0.05
Benzo[*g,h,i*]perylene	0.83	0.37	1.56	0.54	0.08	0.77	1.06	0.13	1.9
Total	20.7			14.1			36.1		
Standard deviation	11.5			13.0			22.7		

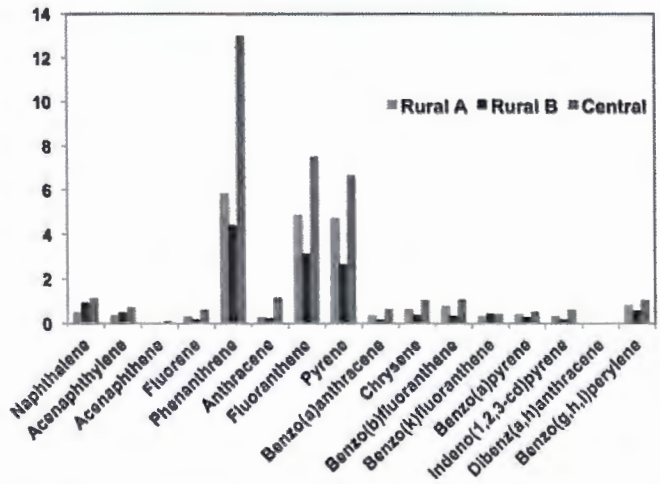

Figure 3. Average concentrations of individual PAH by sampling site (ng m^{-3}).

Discussion

Godoi et al. (2004) collected samples in the suburban area of Araraquara in August 2002 and found high average values of the 16 PAHs (49.3 ± 2.8 ng m^{-3}). Andrade et al. (2010) also found great differences between the levels of PAHs measured in the harvest season (August 2002 and September 2003) and nonharvest season (March 2003 and January 2004), which were 11.6 and 2.5 ng m^{-3}, respectively. The average concentration for the total PAHs in this study for the Central site resulted in 36.1 ng m^{-3}, which were a little lower than the results obtained by Godoi et al. (2004) for the harvest season and can be explained by less preharvest burning in 2009 as a result of the practice being eliminated and because of unusually high precipitation rates in 2009, which could have resulted in better air quality. Indeed, the annual average concentration of PM_{10} in 2010 in Araraquara was 24% greater than the 2009 annual average (CETESB, 2011).

The average level of PAHs in the Araraquara Central site was much lower than the average in the São Paulo city central region (36.1 against 77.8 ng m^{-3}), according to a study conducted by De Assuncao et al. (2007) that used the same sampling and analytical methods as the present study. The city of Sao Paulo has approximately 11 million inhabitants and a fleet of approximately 6 million vehicles that are the predominant emission source in the city. Impacts from industrial, commercial, and service sources are also present. Therefore, the risk of cancer in Sao Paulo city would be higher than that in Araraquara city. The concentrations of PM_{10} were similar in both cities and do not explain the difference in PAH concentrations.

The risk analysis indicates that the median value of the ILTR for inhalation is 1.07×10^{-6} and that the upper 95% percentile is 1.6×10^{-6}, which is on the same order of magnitude as the tolerable risk value of 10^{-6} and indicates that this exposure does not currently result in significant public health problems. The ILTR for adults is higher, and the sensitivity analysis indicates that the input variables of body weight (61%) and inhalation rate (25%) for adults and the inhalation rate for children (12%) exercise a greater impact on the ILTR. Compared with a large city such as Sao Paulo/Brazil, the risks in Araraquara are much lower, although they are not negligible.

Pollution from agricultural activities, especially sugarcane burning, appears to affect the air quality in urban areas, but the PAH ratio analysis indicated that combustion from vehicles was

Table 4. Results of the incremental lifetime cancer risk (ILTR) for carcinogenic PAH inhalation

HPA	Classification (EPA, IARC)	Inhalation Slope Factor[a] (mg kg^{-1} day^{-1})$^{-1}$	Risk for Infants		Risk for Children		Risk for Adults		ILTR	
			Mean	(95% UPL*)	Mean	(95% UPL)	Mean	(95% UPL)	Mean	(95% UPL)
Benzo[*a*]anthracene	2B, B2	0.39	1.02E-8	(2.59E-8)	1.84E-8	(4.18E-8)	4.63E-8	(8.80E-8)	7.56E-8	(1.35E-7)
Benzo[*a*]pyrene	1, B2	3.9	9.02E-8	(1.98E-7)	1.59E-7	(3.11E-7)	3.85E-7	(6.67E-7)	6.25E-7	(9.81E-7)
Benzo[*b*] fluoranthene	2B, B2	0.39	1.83E-8	(4.02E-8)	3.08E-8	(6.50E-8)	7.74E-8	(1.39E-7)	1.16E-7	(2.08E-7)
Benzo[*k*] fluoranthene	2B, B2	0.39	7.17E-9	(1.68E-8)	1.20E-8	(2.68E-8)	3.03E-8	(5.75E-8)	4.94E-8	(8.77E-8)
Chrysene	2B, B2	0.039	1.80E-9	(4.15E-9)	3.04E-9	(6.55E-9)	7.62E-9	(1.39E-8)	1.25E-8	(2.12E-8)
Dibenz[*a,h*] anthracene	2A, B2	4.1	7.72E-9	(1.76E-8)	1.30E-8	(2.81E-8)	3.27E-8	(6.00E-8)	5.30E-8	(9.05E-8)
Indeno[1,2,3-*c,d*] pyrene	2B, C	0.39	1.06E-8	(2.56E-8)	1.78E-8	(4.05E-8)	4.47E-8	(8.72E-8)	7.31E-8	(1.34E-7)
Naphthalene	2B, C	0.12	7.42E-9	(1.63E-8)	1.25E-8	(2.56E-8)	3.15E-8	(5.48E-8)	5.14E-8	(8.05E-8)
Total			1.54E-7	(3.30E-7)	2.60E-7	(5.16E-7)	6.53E-7	(1.96E-6)	1.07E-6	(1.59E-6)

Notes: [a]OEHHA (2010); *Upper Probability Limit for 95% of confidence.

Figure 4. Participation of carcinogenic PAHs in their total mass concentration and in the ILTR.

also an important PAH emission source. Because sugarcane burning is being eliminated, combustion from vehicles will be an increased pollution source in the future.

The concentrations of PAHs were higher in the past, according to the literature review presented in this paper. The cancer risk from PAH inhalation had a higher long-term effect for people who have lived in the region for a long time, as indicated by Cristale et al. (2012), who estimated the influence of sugarcane burning on indoor/outdoor PAH concentrations for the same city. They found significant differences in the PAH concentrations during the harvesting and nonharvesting seasons and showed that the average concentration of PAHs in the harvesting season exceeded the guide values of some European countries.

It would also be of concern if sugarcane burning were to increase, although this should not occur because regulations are forcing a decrease in the percentage of the area dedicated to sugarcane cultivation that may be burnt before harvesting. However, there has been an increase in the area of agricultural land used for sugarcane planting in that region because of market forces. In addition, urban development, population growth, and an increase in industrial activity could contribute to increasing pollution in the future and might increase health risks; the control of such factors depends on pollution control measures.

Demand for the production of biofuels has accelerated the expansion of areas under sugarcane cultivation in other regions of the country where well-established regulations have not been enacted and where enforcement is less effective; therefore, this increased demand could result in higher levels of toxic pollutants and increased health risks.

Finally, in Brazil biomass has an outstanding potential for energy, and its use provide an important contribution to the national energy matrix. Biofuels are renewable, and they are part of a planetary effort to reduce climate impacts. Because biofuels may be equally or more dangerous than fossil fuels, adequate measures must still be taken to reduce all of the environmental impacts resulting from their production and use. Besides PAHs, other classical and hazardous pollutants are also present in the air of the municipality of Araraquara, so care should be taken to keep pollution as low as possible to protect human health.

Table 5. PAH ratios from the Araraquara Central site data and selected ratios from literature

Condition	BaP/B(ghi)P	PHE/AN	BaA/CRY	FLT/PYR	FL/(FL+PYR)	FLA/(FLA+PYR)	BaA/(BaA+CHR)	IcDP/(IcDP+BghiP)
Results in Araraquara Central site								
Collection campaign 1	0.88	11.70	0.63	1.03	0.09	0.52	0.40	0.42
Collection campaign 2	0.38	11.04	0.59	1.48	0.08	0.60	0.37	0.29
Collection campaign 3	0.32	9.72	0.50	1.0	0.05	0.50	0.33	0.4
Collection campaign 4	1.4	14.00	0.33	1.1	0.16	0.53	0.25	0.3
Average	**0.74**	**11.62**	**0.51**	**1.17**	**0.09**	**0.54**	**0.34**	**0.34**
Ratio from literature								
Diesel combustion	**0.46–0.81**[a,b]	7.6–8.8[a,b]	0.17–0.36[a,b]		>0.5[h]			
Gasoline exhaust	0.3–0.4[a]	3.4–8[a]	**0.28–1.2**[a,c]		**<0.5**[h]			**0.2–0.5**[f]
Vehicles	**0.3–0.78**[d,e]	(2.7)[e]	0.63[e]	0.9–1.1[f,g]			>0.35[j]	
	>0.6[k]	**3–12**[f,g]						
Firewood fire[c]		3	0.93					
Oil burning[d]	>2							
Pyrogenic source[f,g]		<10*		<1*				
Coal and wood combustion[f,g]		**>10***		**>1***				
Fossil fuel combustion[i]						0.4–0.5		
Grass, wood and coal combustion						**>0.5**[i]		**0.2–0.5**[f]
Coal combustion[j]							**0.2–0.35**	

Notes: *Both ratios must be considered concomitantly. Ratios from literature highlighted in bold were those in agreement with result ratios. [a]Rogge et al. (1993); [b]Westerholm et al. (1991); [c]Gschwend and Hites (1981); [d]Daisey et al. (1979); [e]Smith and Harrison (1996); [f]Yunker er al. (2002); [g]Wang (2007); [h]Ravindra et al. (2008); [i]De La Torre-Roche et al. (2009); [j]Akyüz and Çabuk (2010); [k]Katsoyiannis et al. (2007).

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APPENDIX R

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Properties of aerosols from sugar-cane burning emissions in Southeastern Brazil

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Abstract

The influences of biomass burning emissions in the composition of aerosol have been studied during 1 year around the city of Piracicaba (Southeastern Brazil). Inhalable particles, separated in $PM_{2.5}$ and coarse particulate mode (CPM, with size in the range ($2.5 < d_p < 10\,\mu m$)), were sampled from April 1997 to March 1998 and analyzed for BC, Al, Si, P, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Se, Br, Rb, Sr, Zr, Pb. The average concentrations of $PM_{2.5}$, CPM, BC and chemical elements were statistically higher in the dry season than in the wet season. The results of absolute principal component analysis showed four and three different sources for $PM_{2.5}$ and CPM, respectively. Sugar-cane burning is the main source of $PM_{2.5}$ representing 60% of $PM_{2.5}$, soil dust accounted for 14%, and industries and oil combustion contributed with 12% each one. Resuspended soil is the main source of CPM followed by industrial emissions and sugar-cane burning. The sampling and analytical procedures applied in this study showed that sugar-cane burning and agricultural practices are the main sources of inhalable particles, possibly altering the aerosol composition around the city of Piracicaba.

Keywords: Aerosol composition; Biomass burning; Black carbon; Aerosol source apportionment; Brazil

1. Introduction

In the Northern Hemisphere the main source of aerosols is linked to fossil fuel burning, whose emissions have been well documented, while in tropical areas biomass burning may be one of the most important global source of atmospheric particles (Crutzen and Andreae, 1990; Oglesby et al., 1999). Aerosol particles from biomass burning are important in the radiative balance of the atmosphere, through a complex micro-physical process, possibly affecting the ground-based radiation budget (Oglesby et al., 1999; Ramanathan et al., 2001). The carbonaceous aerosols (black carbon and organic aerosol) are identified as playing a major role in the absorption of solar radiation. These aerosols, together with the resulting reflection of solar radiation to space, lead to large changes (global mean radiative aerosol forcing range from -0.5 to $+0.2\,W\,m^{-2}$) in the solar radiation absorbed on the surface (Ramanathan et al., 2001; Streets et al., 2001). Additionally, the particles emitted by biomass burning can affect the concentration of cloud condensation nuclei, probably changing the hydrological cycles and cloud albedo (Roberts et al., 2001; Rosenfeld, 1999).

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Due to the large fire catastrophes of anthropogenic origin in recent years and the intensive scientific efforts over the last decade, the general public as well as the scientific community are now aware that emissions from biomass burning represent a large perturbation to global atmospheric chemistry, particularly in the tropics. Biomass burning includes the burning of forests, savannas, and agricultural lands. Brazil plays an important role in biomass burning emissions, with the detection by AVHRR of about 235,000 fire spots in 2004 (www.cptec.inpe.br/queimadas). Most of these fires occurred in the southern part of the Amazon basin during the dry season (from August to November). Emissions from these fires have significant impacts on the concentration of trace gases and aerosols over a large area (Artaxo et al., 2002; Yamasoe et al., 2000). From Southeastern through Northeastern Brazil sugar-cane burnings are also key contributors in terms of emissions, as already highlighted a decade ago by Crutzen and Andreae (1990). Brazil owns 25% of the global sugar-cane areas, and is the major sugar-cane producer in the world. Currently, it has 4,500,000 ha of sugar-cane crops and more than 1,100,000 rural workers are involved in the sugar-cane industry (Zamperlini et al., 2000). The area cultivated with sugar-cane in the state of São Paulo in Southeastern Brazil, reaches approximately 24,000 km^2. In order to facilitate harvesting, which also occurs in others places such as Hawaii and Africa, the sugar-cane is burned and most fires occur every year during the dry season (from May to November). About 20 t of sugar-cane dry matter per hectare is burned, contributing to a global emission with approximately 0.48 Tg of carbon per year. As a result, aerosol and trace gases emitted from sugar-cane fires have significant effects on the composition and acidity of rainwater over large areas of Southern Brazil (Lara et al., 2001). Despite its importance, little information is available about the effects of sugar-cane burnings on aerosol particles (Azevedo et al., 2002; Martinelli et al. 2002).

The focus of this paper was the investigation of the chemical and physical characteristics of sugar-cane burning aerosol emissions, looking especially at the elemental composition, and source apportionment of fine and coarse mode aerosols. The use of real-time aerosol monitors (TEOM and Aethalometer) allowed obtaining of diurnal patterns, while stacked filter units (SFU) separated fine and coarse mode aerosol.

2. Methodology

The city of Piracicaba is located in the western part of the Piracicaba River basin. It has approximately 320,000 inhabitants and a population density of 242 inhabitants km^{-2} (Fig. 1). In an area encompassed by a circumference centered by the city and with a radius of 20 km (area of approximately 1200 km^2), the land use is dominated by sugar-cane plantations (80%), followed by pastures (11%), urban areas (6%) and forests (3%) (Lara et al., 2001; Krushe et al., 2002). Additionally, more than 1000 small industries were found (Lara et al., 2001).

Aerosol sampling was performed day and night from April 1997 through March 1998 using the SFU (Hopke et al., 1997). The inhalable particles (PM_{10}) have been sampled in the SFU on 47 mm diameter nuclepore polycarbonate filters in two separated size fractions: fine particulate mode, $PM_{2.5}$ ($d_p < 2.5\,\mu m$) and coarse mode, CPM ($2.5 < d_p < 10\,\mu m$). Each sample was taken over a period of 48 h. The sampling site was located in the meteorological station of the "Escola Superior de Agricultura Luiz de Queiroz—ESALQ". This site lies about 4 km from the Piracicaba downtown and less than 1 km from the nearest sugar-cane plantation. The SFU inlets were located 3 m above the ground to minimize direct influences of local resuspended soil dust. The dominant wind direction was from the sugar-cane plantations to the sampling station.

In order to identify the daily variability of the sugar-cane emissions in the concentration of PM_{10} and BC, from 3rd October until 13th November, we employed a real time aerosol mass monitor—the Tapered Element Oscillating Microbalance (TEOM), series 1400a operated with a time resolution of 10 min, and a heating of the inlet at 50 °C. The organic carbon (OC) concentration was measured with 10 min time resolution, in the $PM_{2.5}$ fraction by a direct automatic thermal-CO_2 technique, performed by the Ambient Carbon Particulate Monitor, series 5400. During the analysis the collected sample is heated first to 340 1 °C and then to 750 1 °C determining the concentrations of organic and total carbon, respectively. Black carbon (BC) was also determined during this period using an Aethalometer with the same time resolution. During this time the samples were classified in two different conditions: days with and without sugar-cane burning impacts. Parameters such as precipitation, weekends, and holidays were used in separating these periods. Gravimetric mass and BC were measured in the nuclepore filters. BC was measured by a reflectance technique according to the method developed by Reid et al. (1998). Elemental composition was measured by particle-induced X-ray emission (PIXE), a multi-elemental technique that allows the identification of about 21 elements (Al, Si, P, S, Cl, K, Ca, Ti, V, Cr, Mn, Fe, Ni, Cu, Zn, Se, Br, Rb, Sr, Zr, and Pb). PIXE detection limits are about 1 ng m^{-3} for elements with $Z > 20$, and about 10 ng m^{-3} for elements from Na to K. More details about PIXE technique and aerosol sampling can

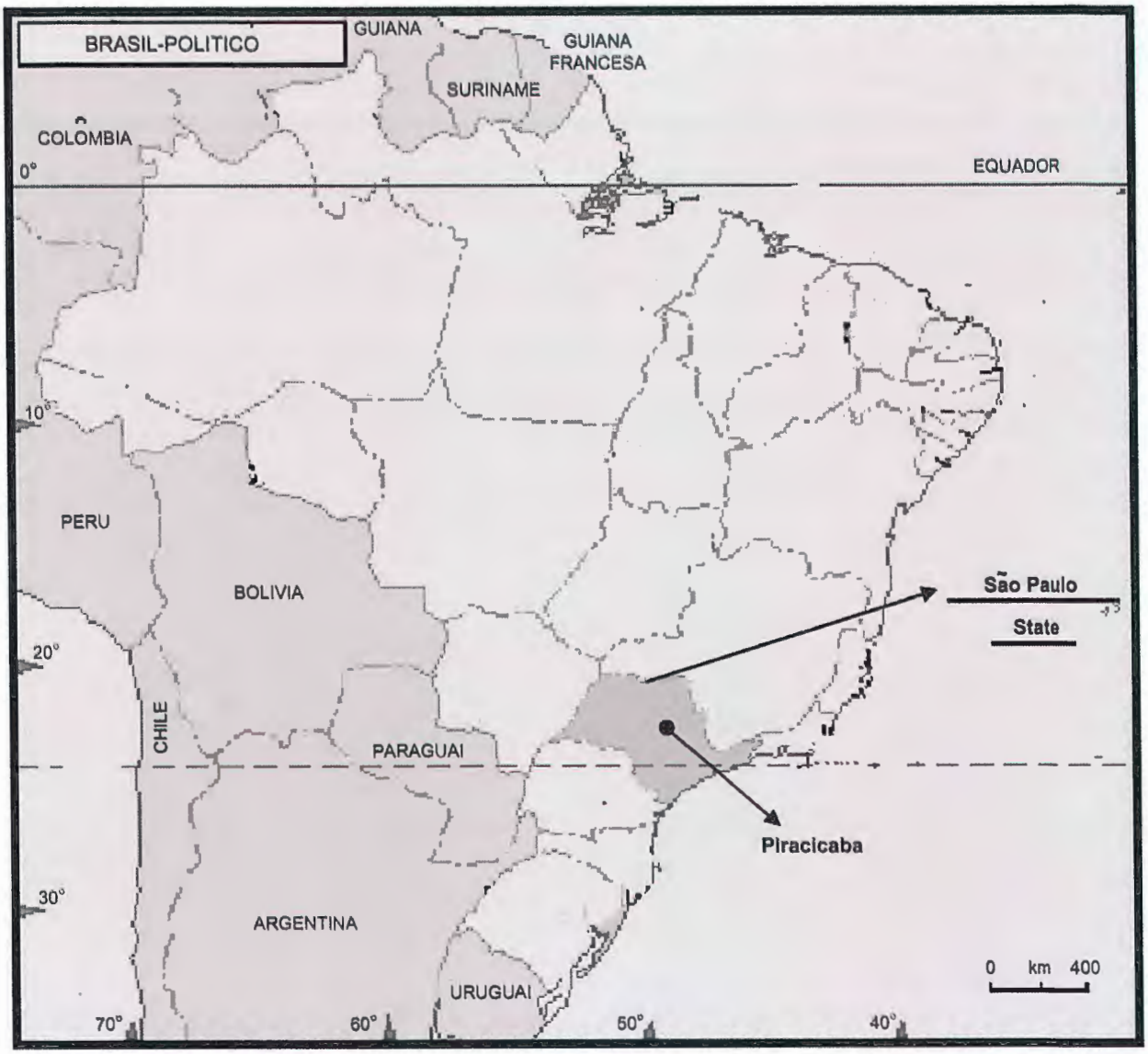

Fig. 1. Map of Brazil with divisions of the states showing the sampling site (Piracicaba in the State of São Paulo). Names of states are underlined.

be obtained from Artaxo et al. (1999) and Yamasoe et al. (2000).

Multivariate analyses have been successful when applied to studies of environmental problems (Hopke, 1991). In order to help the identification of possible aerosol sources and quantify the source apportionment we performed principal component analysis (PCA) and absolute principal component analysis (APCA) (Thurston and Spengler, 1985; Swietlicki et al., 1996). ANOVA tests (Tukey honest significant test) were used to evaluate the seasonal differences in the concentrations of the elements.

3. Results

Sugar-cane is burned and harvested from May to November. This period is coincident with the dry season in Southern Brazil. From December to April the wet pattern prevails and there are fewer burning activities in the area of Piracicaba and in all State of São Paulo (Fig. 2). According to Fig. 2, the peak of the burning season (August) is just after the minimum of precipitation. Based on this fact and for statistical purposes we grouped samples according to the occurrence of fire events: dry season (May–November), coincident with

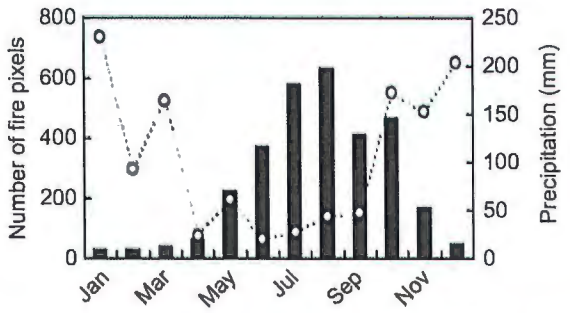

Fig. 2. Number of fires pixels (black column) in São Paulo State (www.cptec.inpe.br/queimadas) during 1997–1998 and mean monthly precipitation (dashed line) in Piracicaba City (www.esalq.br).

the sugar-cane burning season and wet season (December–April), coincident with non-burning season.

Fig. 3 shows the aerosol mass and BC concentrations for the whole sampling period. The PM_{10} concentration had a large seasonal variability. Higher concentrations were found during the dry season and smaller concentrations during the wet season. The highest concentration ($238\,\mu g\,m^{-3}$) was found in early September and the smallest concentrations ($9.3\,\mu g\,m^{-3}$) in middle October, just after the first rains (Fig. 3a). BC concentration also had the same pattern of seasonal variability of the inhalable particulate (Fig. 3b). The highest concentration ($10.7\,\mu g\,m^{-3}$) was observed in middle August and the lowest one in the end of December ($0.8\,\mu g\,m^{-3}$). By grouping particles and chemical elements in the wet and dry seasons, several important differences emerge between these two groups. The average concentrations of $PM_{2.5}$, CPM, BC and chemical elements were statistically higher ($P<0.01$) during the dry season than in the wet season (Table 1). For instance, the PM_{10} wet season average concentration was equal to $34.1\,\mu g\,m^{-3}$, and increased to $90.7\,\mu g\,m^{-3}$ during the dry season. Coarse particulate mode represented the largest fraction of PM_{10}, consisting of 75.0% and 66.1% of the PM_{10} mass during dry and wet seasons, respectively.

Since the sugar-cane biomass burning happens mostly around 5:00–6:00 p.m., we investigated the diurnal pattern of BC and PM_{10}. Hourly PM_{10} and BC concentrations and BC/OC ratio are shown in Fig. 4. Further, we grouped the concentrations according to burning and non-burning days collected from 3rd October until 13th November. There was a peak in the PM_{10} concentration between 7:00 and 10:00 a.m. This occurred in both situations (Fig. 4a). A second peak in the PM_{10} concentration occurred between 6:00 and 11:00 p.m., but only during burning days (Fig. 4a). A similar peak occurred for BC in the same time of the day and also only during burning days (Fig. 4b). In the rest of the daytime, the BC concentration was quite constant and with no significant difference between burning and non-burning days (Fig. 4b). The diurnal cycle of OC measured by the 5400 Ambient Carbon Particulate Monitor presents a similar pattern as the BC concentration. The OC aerosol can be originated from direct emissions of particles, such as biomass burning (Azevedo et al., 2002), and also from atmospheric secondary formation from gaseous precursors. The ratio of BC/OC varies significantly along the day (Fig. 4c). The maximum value of the ratio BC/OC occurred, again only during burning days, between 6:00 and 11:00 p.m., coincident with the time of the sugar-cane fires. During the day and before the burning time, it is possible to observe a sharp decrease in the ratio (BC/OC <40%), indicating two processes: a reduction in the direct emission of BC by the sugar-cane fires, and the secondary production of OC particulate in the atmosphere. Chow et al. (1996) have pointed out that the limit of 50% of (BC/OC) indicates the presence of secondary organic aerosols in the urban atmosphere. The non-burning days did not show a strong gradient in the ratio, presenting a BC/OC ratio <25%. This fact points to the possibility of changes in the production of OC related to sugar-cane burning emissions.

In order to study the aerosol sources in the region of Piracicaba, PCA was performed including the chemical composition of fine and coarse mode particulates. The communality for each variable, which represents the fraction of each variable that is explained by the retained factors, was typically higher than 82% (fine mode) and 93% (coarse mode). This indicated that the factors could explain most of the data variability. For the fine mode, four factors have been retained. They explained 92% of the variance of the data set (Table 2). The first one is loaded with $PM_{2.5}$, BC, K, S, and Br representing biomass burning ($PM_{2.5}$, BC, K) and it explained 27.6% of the variance of the data. The second factor is associated with Al, Si, Ca, Fe, and Ti, characteristics elements of resuspended soil; it explained 24.5% of the variance. The third factor (22.3% of the variance) is associated with heavy metals due to the significant loads for Zn, Pb, Cu, and Mn. The association of these elements with Fe can be related to industrial process involving ferrous smelter emissions. The last factor has significant loads for V and Ni, representing aerosols from residual oil combustion and explained 17.6% of the variance of the data. In order to quantify the relative importance of each identified source in the fine fraction, the APCA has been used to derive the source apportionment in the fine mode aerosol. Standard procedures for the APCA calculations were applied (Thurston and Spengler, 1985). Table 3 shows the absolute elemental source profile for $PM_{2.5}$. Elements like S, Cl, and K and $PM_{2.5}$ were 2–10 times higher in the biomass burning source profile. The source

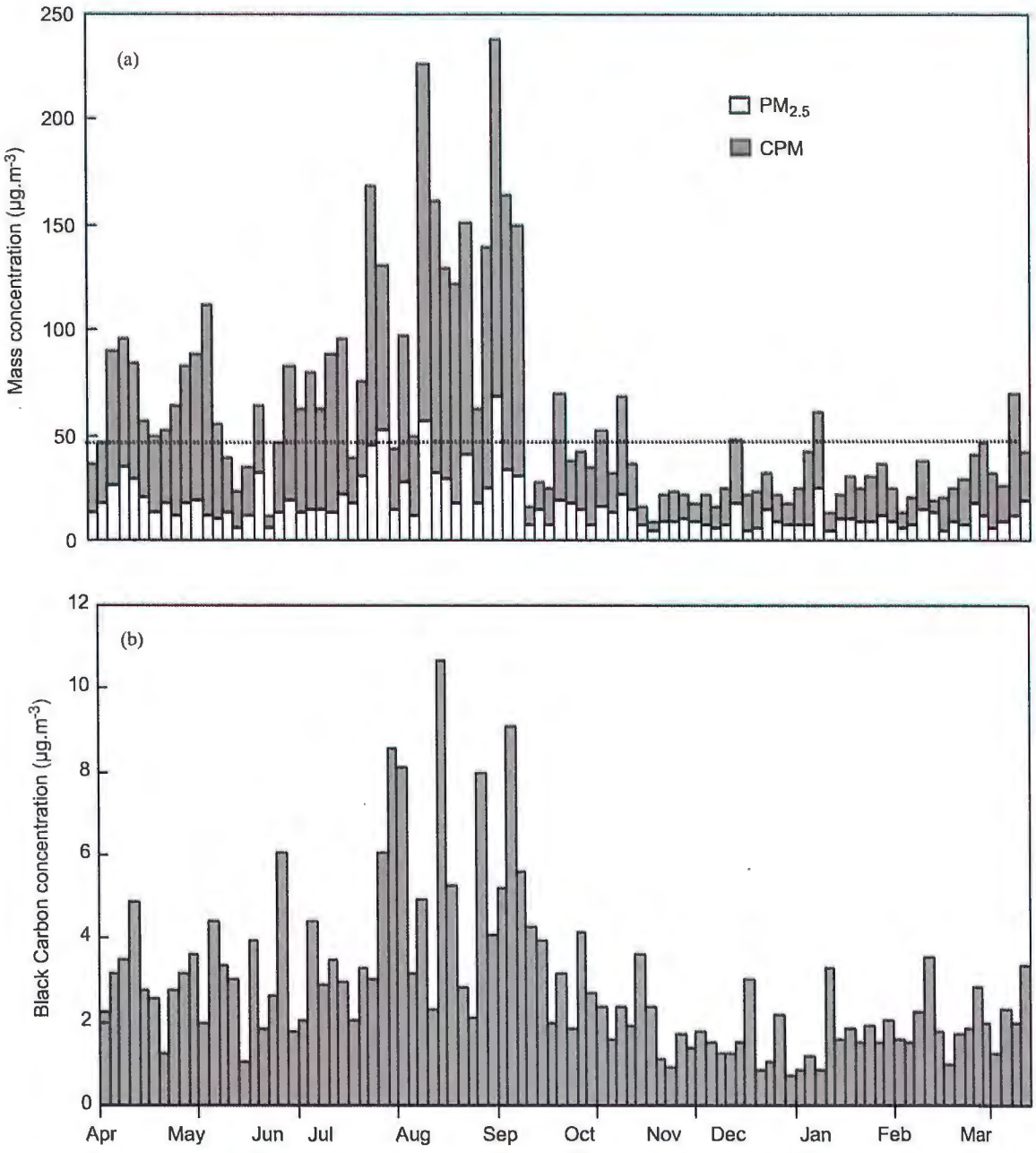

Fig. 3. Temporal variability of (a) fine and coarse mode mass concentrations. The sum of the two fractions results in the PM_{10} concentrations (inhalable particles), and (b) black carbon mass concentration. Aerosol sampling was performed from April 1997 through March 1998.

apportionment of $PM_{2.5}$ obtained using APCA is shown in Fig. 5a. Sugar-cane burning is the major source of $PM_{2.5}$, contributing with 60% of the fine mode aerosol mass. The second major source is resuspended soil dust representing 14% of the $PM_{2.5}$. Heavy metals and oil combustion contribute each one with 12% of the total fine particulate. The model could apportion 98% of the fine mode mass concentration.

For the coarse mode, the PCA factor loading matrix after Varimax rotation showed three components (Table 4). Communalities were always higher than 93% and three factors explained 96% of the variance of the coarse mode data. The first factor is clearly associated with soil dust, since it is loaded with CPM, Al, Si, Ti, Ca, and Fe and could explain 62.1% of the variance of the data. The second factor explained 20.8% of the

Table 1
Concentration averages and standard deviation of the elements (expressed in ng m^{-3}), BC, $PM_{2.5}$ and CPM (expressed in μg m^{-3}), during the dry and wet season and annual period, correspondent to the sampling period from April 1997 through March 1998

	Fine particulate mode, $PM_{2.5}$, (d_p < 2.5 μm)							Coarse particulate mode, CPM, (2.5 < d_p < 10 μm)					
	Dry season		Wet season		Annual			Dry season		Wet season		Annual	
	Mean	σ	Mean	σ	Mean	σ		Mean	σ	Mean	σ	Mean	σ
$PM_{2.5}$[a]	22.7	14.5	11.6	5.9	16.1	11.6	CPM[a]	68.1	43.2	22.6	14.4	41.1	37.1
BC[a]	4.2	2.2	2.1	0.9	2.9	1.9	—						
Al	342.7	315.6	166.1	145.3	237.9	244.7	Al	3097	2354	880.2	801.2	1782	1948
Si	556.2	447.1	306.1	197.7	407.8	343.8	Si	5486	3806	1522	1314	3134	3263
P	5.9	3.1	5.1	3.9	5.4	4.8	P	73.7	38.8	52.5	26.8	61.1	33.7
S	1898	1271	1087	689	1417	1042	S	1046	737.9	364.3	263.1	641.4	610.0
Cl	18.1	20.3	5.6	5.9	12.0	9.23	Cl	140.4	118.7	33.7	58.1	84.8	106.1
K	601.7	389.0	196.6	145.7	361.4	336.3	K	806.6	508.5	228.7	134.7	463.6	442.2
Ca	109.6	107.5	51.5	37.3	75.1	71.1	Ca	1241	799.1	309.7	283.1	688.7	717.3
Ti	44.8	37.8	23.9	19.3	32.4	29.9	Ti	485.0	370.6	136.3	146.8	278.1	311.9
V	86.6	84.2	33.1	30.8	54.8	64.0	V	99.9	97.2	18.2	22.3	57.9	80.4
Cr	3.8	4.3	2.3	2.2	2.9	3.3	Cr	3.5	2.7	—	—	3.5	2.7
Mn	15.4	12.0	10.1	6.2	12.3	9.3	Mn	61.0	37.4	19.0	17.8	36.1	34.3
Fe	447.6	314.1	260.6	150.5	336.7	247.7	Fe	3646	2733	1119	1071	2147	2285
Ni	17.1	15.4	7.3	6.1	11.3	9.4	Ni	20.3	10.1	4.3	4.8	12.1	9.4
Cu	5.6	4.7	3.6	1.9	4.4	3.5	Cu	8.9	7.1	2.4	2.1	5.2	3.6
Zn	105.8	172.8	76.7	59.6	88.5	119.3	Zn	77.0	58.7	45.1	36.2	58.1	53.8
Br	7.4	5.0	3.1	1.9	4.9	4.1	Br	3.5	2.3	2.8	1.8	3.1	2.2
Pb	23.9	27.7	18.1	10.7	20.5	19.6	Pb	15.1	10.9	9.9	7.9	12.0	9.9

[a]Expressed in μg m^{-3}, σ = standard deviation.

variance. It represents heavy metals emissions with high factor loadings for Pb and Zn. The third factor (13.6% of the variance) is loaded with K and S and it is related to biomass burning emissions. Fig. 5b shows the source profile apportionment of CPM. The contribution of resuspended soil dust to CPM represents 51%, biomass burning 25%, and industrial emissions 20%. The APCA model could explain 96% of the coarse mode mass.

4. Discussion

The PM_{10} concentration during the dry season in the Piracicaba region is similar to the values observed in Rondônia, Amazon Basin, where extensive deforestation and associated fires have occurred in the last years (Artaxo et al., 2000, 2002) and higher than that observed in São Paulo metropolitan area (Castanho and Artaxo, 2001). However, during the wet season the PM_{10} values are higher than Rondônia and similar to those found in São Paulo metropolitan area. The annual average PM_{10} (Table 1) around Piracicaba is slightly higher than the Brazilian air pollution standard limit for PM_{10} of 50 μg m^{-3}. Coarse particulate mode dominates the PM_{10} concentration which is possibly associated with biomass burning and the large area of bare soils around Piracicaba. The biomass burning during the harvest period emits coarse particulate originated from the burning itself to the atmosphere and allows the soil dust resuspension. The soil cultivation for sugar-cane or other crops causes a strong soil resuspension during all year due to the large area of bare soils exposed (Cerri et al., 2001). This increases the magnitude of the coarse mode aerosols compared to the fine mode.

In large urban areas, such as São Paulo and Santiago, Chile, the main sources of aerosol particles are emissions linked to fossil fuel combustion (Artaxo et al., 1999; Castanho and Artaxo, 2001), while in the area around Piracicaba biomass burning is the strongest source. It is not surprising that agricultural related factors can influence the composition of aerosols in the State of São Paulo so much. Ten percent of the total area of this State is used for sugar-cane plantations and biomass burning is one of the major contributors to the aerosol in South America. There are several evidences showing that sugar-cane burning has a large impact on the atmospheric composition of the region of Piracicaba. Lara et al. (2001) have reported acid rain in four different sites at the Piracicaba River Basin. Although there is an industrial area in this region, the main source of acid rain is associated with sugar-cane burning (Lara et al., 2001).

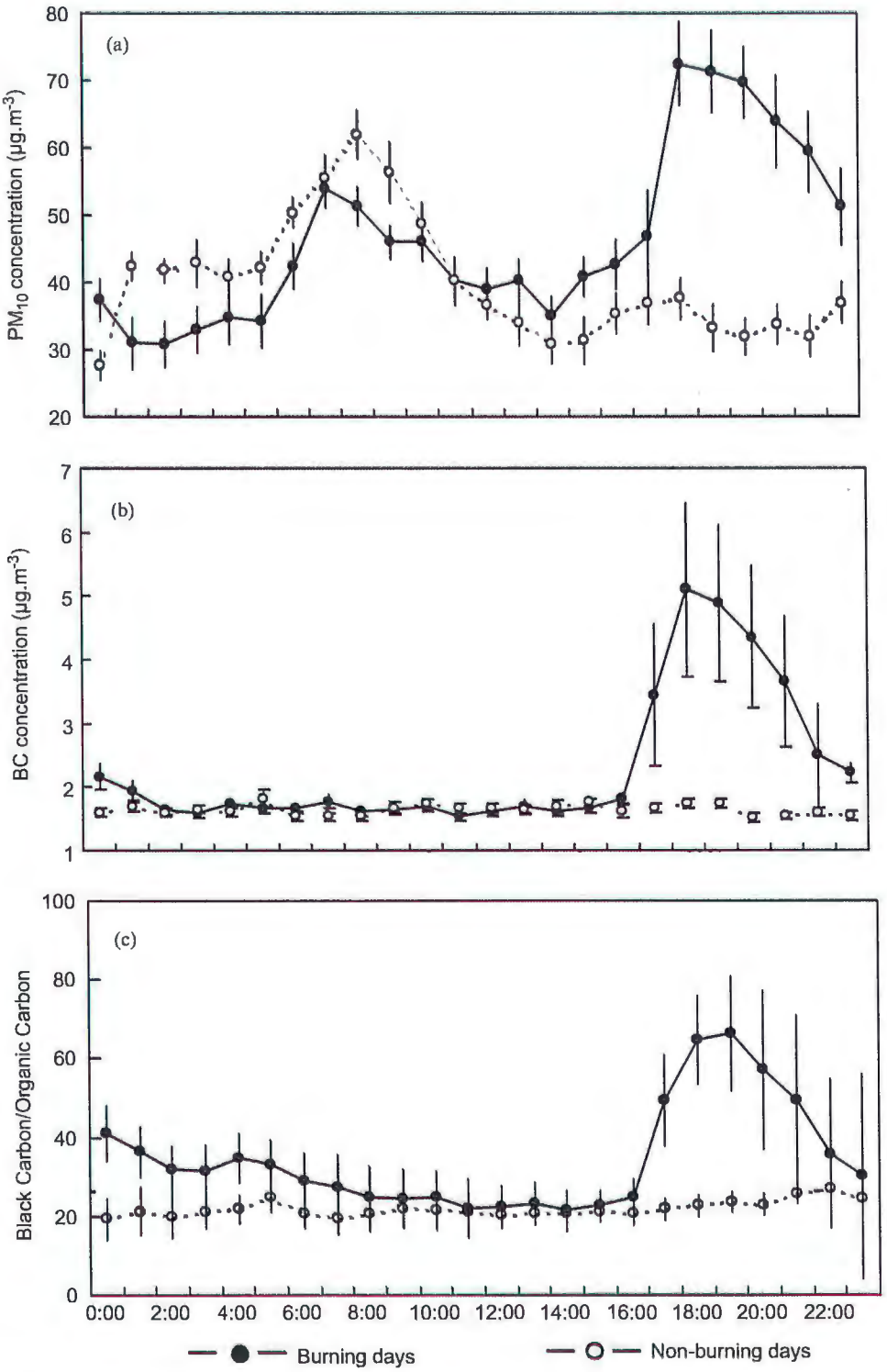

Fig. 4. Diurnal cycle during the burning and non-burning days for (a) mass concentration of PM_{10}, (b) mass concentration of BC, and (c) black carbon/organic carbon ratio.

Factor analysis has showed the large influence of sugar-cane burning emissions in the composition of fine particulate mode. Four factors explain most of the composition of fine mode aerosol. The most important source is sugar-cane burning, which represents 60% of the total fine mode mass. Additional statistical analysis

Table 2
Varimax rotated factor loadings for the $PM_{2.5}$, BC and elemental concentrations of the fine particulate mode sampled from April 1997 through March 1998 at the city of Piracicaba

Elemental	Factor 1	Factor 2	Factor 3	Factor 4	Communalities
	Biomass burning	Soil	Heavy metals	Oil combustion	
$PM_{2.5}$	0.79	0.44	0.20	0.33	0.96
BC	0.88	0.29	0.20	0.27	0.96
Al	0.47	0.83	—	0.20	0.96
Si	0.50	0.82	0.20	—	0.96
S	0.64	0.36	0.20	0.58	0.88
K	0.79	0.45	—	0.20	0.89
Ca	0.44	0.60	—	0.48	0.82
Ti	0.43	0.84	—	0.30	0.98
V	0.21	0.23	0.20	0.92	0.98
Mn	0.20	0.37	0.76	0.27	0.82
Fe	0.33	0.67	0.56	0.27	0.94
Ni	0.23	0.22	0.24	0.92	0.99
Cu	0.35	—	0.86	—	0.88
Zn	—	—	0.94	—	0.90
Br	0.86	0.40	0.20	—	0.93
Pb	—	—	0.91	0.20	0.89
var. (%)	27.6	24.5	22.3	17.6	

Only factor loadings larger than 0.2 are shown.

Table 3
Absolute aerosol source apportionment for the $PM_{2.5}$, BC (expressed in $\mu g\,m^{-3}$) and elemental concentrations (expressed in $ng\,m^{-3}$) of the $PM_{2.5}$

Elemental	Factor 1	Factor 2	Factor 3	Factor 4	Concentration model/measured
	Biomass burning	Soil	Heavy metals	Oil combustion	
$PM_{2.5}$	9.8	2.2	1.9	1.9	0.98
BC	1.9	0.3	0.4	0.3	0.96
Al	117	84.9	—	22.6	0.94
Si	195	120	40.4	—	0.87
P	1.4	1.4	1.7	0.3	0.88
S	761.4	167.5	115.1	312.6	0.96
Cl	6.8	2.2	—	3.4	1.03
K	260.4	60.2	—	28.8	0.97
Ca	36.3	19.8	—	18.7	0.99
Ti	14.1	10.6	—	4.6	0.90
V	12.1	5.9	9.7	28.6	1.03
Cr	0.8	0.3	1.1	0.3	0.85
Mn	2.3	1.5	6.8	1.3	0.97
Fe	92.0	70.5	133	34.3	0.98
Ni	2.6	1.1	2.5	5.3	1.01
Cu	1.3	—	2.8	—	0.93
Zn	—	—	97.5	—	1.10
Br	3.5	0.7	0.6	—	0.99
Pb	—	—	17.6	2.1	0.96

with elements that were not included in the factor model emphasizes this point. The Spearman correlation ($P<0.01$) of the factor scores 1 identified in the fine mode (sugar-cane burning) with Cl ($r=0.72$), Mg ($r=0.59$), and P ($r=0.54$) are statistically significant ($P<0.01$), supporting the identification of this source by

the factor model. These elements were identified in biomass burning emission studies (Levine et al., 1995; Echalar et al., 1995; Yamasoe et al., 2000). In terms of soil dust mass apportionment in the fine mode, the high value obtained in this study (14%) can be explained by the fact that the sugar-cane burning process generates significant convection and raises soil dust by the convective plume, mixing up biomass burning aerosols with fine soil dust. Besides, the resuspended soil is also associated with bare soils around Piracicaba region. Additionally, emissions from sugar-cane burning are responsible for 64% of the fine mode BC concentration in Piracicaba, a comparable value (55%) found for Piracicaba region using isotopic techniques in aerosol (Martinelli et al., 2002). A similar value was obtained in other biomass burning studies (Ferek et al., 1998; Yamasoe et al., 2000; Chankina et al., 2001). The ratio of BC to $PM_{2.5}$ is about 19% higher than the values observed in forest fires ranging from 13% to 18% (Echalar et al., 1995; Yamasoe et al., 2000). This is because the fires in the sugar-cane plantations occur mainly in the flaming phase, responsible for higher ratio of BC emissions, in a contrary way of the forest fires which occur mainly in smoldering phase (Yamasoe et al., 2000).

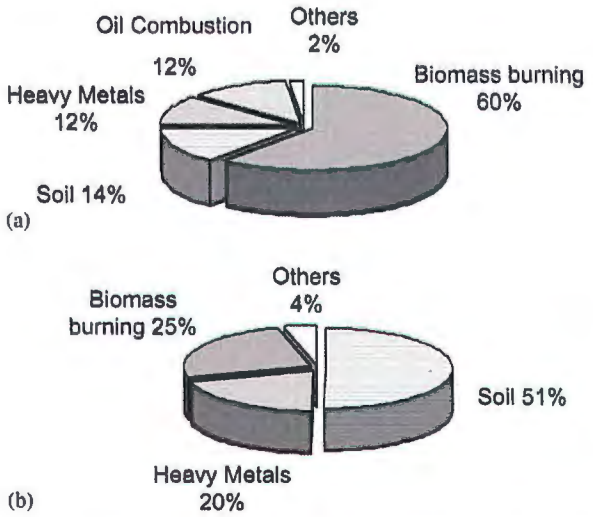

Fig. 5. Source apportionment of (a) fine mode particulate, and (b) coarse mode particulate. The quantification of the contribution of the sources has been done using absolute principal component analysis (APCA) for each factor identified in the principal component analysis.

As expected, resuspended soil is the main source of CPM. It represents 51% of the total coarse mode mass. The identification of the third factor as sugar-cane burning is due to the strong presence of S and K in this factor (Yamasoe et al., 2000). In addition, the significant correlation of the factor 1 (biomass burning) identified in the fine particulate mode with this factor 3 in the coarse mode is also an evidence of the biomass burning origin in this third coarse mode factor (Table 5).

The significant correlations between soil dust in the coarse mode and sugar-cane burning in the fine mode (Table 5) emphasizes the relation between the sugar-cane burning emissions with soil dust resuspension. The significant correlation between the fine and coarse soil dust factors indicates the adequacy of the identification of the sources, as well as it occurs to fine and coarse heavy metals emission factors.

The averages of PM_{10} concentration over 1 h obtained from TEOM equipment are significantly higher during the period when the sugar-cane is burnt. The biomass burning usually starts around 5:00–6:00 p.m. The

Table 4
Varimax rotated factor loadings for the CPM and elemental concentrations of the coarse particulate mode sampled from April 1997 through March 1998 at the city of Piracicaba

Elemental	Factor 1	Factor 2	Factor 3	Communalities
	Soil	Heavy Metals	Biomass burning	
CPM	0.89	0.24	0.38	0.99
Al	0.94	—	0.25	0.99
Si	0.91	0.23	0.34	0.98
S	0.66	—	0.71	0.99
K	0.76	0.22	0.57	0.99
Ca	0.83	0.29	0.41	0.97
Ti	0.94	0.22	0.22	0.94
Mn	0.84	0.43	0.26	0.96
Fe	0.94	0.26	—	0.94
Zn	0.36	0.89	—	0.93
Pb	—	0.95	—	0.96
Var (%)	62.1	20.8	13.6	

Only factor loadings larger than 0.2 are shown.

Table 5
Spearmans correlation of factor scores identified by PCA between fine and coarse mode particulate[a]

Coarse particulate mode factors	Fine particulate mode factors			
	Biomass burning	Soil dust	Heavy metals	Oil combustion
Soil dust	0.54	0.37	—	—
Heavy metals	—	—	0.38	—
Biomass burning	0.39	—	—	—

[a]Only statistically significant ($P<0.01$) correlations are shown.

morning peak in PM_{10} concentrations at 7:00–10:00 a.m. in the days with and without biomass burning (Fig. 4a) is characteristic of the forming and growing of the diurnal boundary layer, when wind speeds start resuspended soils nearby the sampling site. It is interesting to note the significant difference between the burning and non-burning periods around 6:00 and 10:00 p.m. This is the time when sugar-cane is burned, emitting inhalable particles and increasing significantly the PM_{10} concentrations to the atmosphere. The same trend was observed for BC concentrations (Fig. 4b), suggesting that the sugar-cane burning is the main responsible for the increasing of the BC concentrations along the diurnal cycles, as well as for the PM_{10} concentration. These changes in the aerosol properties such as concentration values and chemical composition is even more important if we call attention to the fact that the large population who has faced these biomass burning episodes during the last four decades. Aerosols, particularly those less than 2.5 μm in diameter ($PM_{2.5}$), appear to have the greatest potential for damaging health because they can penetrate deep into the lungs and reach the lower respiratory system (Hamilton and Mansfield, 1991; EPA, 1996).

5. Conclusions

High levels of pollutants have been measured during the sugar-cane burning season in the city of Piracicaba. A complex system of air pollution sources modulates the concentration of pollutants in the air around Piracicaba. As there is intense biomass burning in other regions in Brazil possible influences of medium and long-range transport of upwind fires emissions also could contribute to the air pollution in this area. During the entire year, the influence of the sugar-cane burning can be seen in the composition of fine and coarse particulate modes and in the variations of BC concentration. With this study it was possible to identify the influence of sugar-cane burning in the composition of aerosol in an urban center, and until now this was completely unknown. The results of APCA have showed that there is a large contribution of sugar-cane fires to the structure of the aerosol, 60% of the fine mode mass, 64% of the BC mass and 25% of the coarse mode mass can be attributed to emissions from sugar-cane burning. The aerosols and BC released from biomass burning can act as CCN. It affects cloud albedo by altering the hygroscopic properties of CCN and the solar heating caused by BC can supposedly reduce cloudiness. Through this process, the cloud microphysical and radiative process in tropical rain and cloud systems can hypothetically be affected with potential climatic and hydrological consequences.

Sugar-cane burning altering the structure of air composition has already taken place over the city of Piracicaba. What happens around the city of Piracicaba could also be experimented by other regions in Southeast Brazil. In the state of São Paulo the main land use is pasture and sugar-cane crops. They cover an area around 50 and 10%, respectively, of the total area of the state. In both cases biomass burning is a common feature. Although these aerosols from biomass burning have an atmospheric residence time of a few days to several weeks, they can be widespread over long distances (hundred to thousands of kilometers). They also could be interfering in the quality of the air, regional climate changes, and in hydrological cycles.

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APPENDIX S

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Particulate Matter Air Pollution and Cardiovascular Disease: An Update to the Scientific Statement From the American Heart Association

Robert D. Brook, Sanjay Rajagopalan, C. Arden Pope, III, Jeffrey R. Brook, Aruni Bhatnagar, Ana V. Diez-Roux, Fernando Holguin, Yuling Hong, Russell V. Luepker, Murray A. Mittleman, Annette Peters, David Siscovick, Sidney C. Smith, Jr, Laurie Whitsel, Joel D. Kaufman and on behalf of the American Heart Association Council on Epidemiology and Prevention, Council on the Kidney in Cardiovascular Disease, and Council on Nutrition, Physical Activity and Metabolism

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AHA Scientific Statement

Particulate Matter Air Pollution and Cardiovascular Disease

An Update to the Scientific Statement From the American Heart Association

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***Abstract*—**In 2004, the first American Heart Association scientific statement on "Air Pollution and Cardiovascular Disease" concluded that exposure to particulate matter (PM) air pollution contributes to cardiovascular morbidity and mortality. In the interim, numerous studies have expanded our understanding of this association and further elucidated the physiological and molecular mechanisms involved. The main objective of this updated American Heart Association scientific statement is to provide a comprehensive review of the new evidence linking PM exposure with cardiovascular disease, with a specific focus on highlighting the clinical implications for researchers and healthcare providers. The writing group also sought to provide expert consensus opinions on many aspects of the current state of science and updated suggestions for areas of future research. On the basis of the findings of this review, several new conclusions were reached, including the following: Exposure to PM <2.5 μm in diameter ($PM_{2.5}$) over a few hours to weeks can trigger cardiovascular disease–related mortality and nonfatal events; longer-term exposure (eg, a few years) increases the risk for cardiovascular mortality to an even greater extent than exposures over a few days and reduces life expectancy within more highly exposed segments of the population by several months to a few years; reductions in PM levels are associated with decreases in cardiovascular mortality within a time frame as short as a few years; and many credible pathological mechanisms have been elucidated that lend biological plausibility to these findings. It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality. This body of evidence has grown and been strengthened substantially since the first American Heart Association scientific statement was published. Finally, $PM_{2.5}$ exposure is deemed a modifiable factor that contributes to cardiovascular morbidity and mortality. **(*Circulation*. 2010;121:2331-2378.)**

Key Words: AHA Scientific Statements ■ atherosclerosis ■ epidemiology ■ prevention ■ air pollution ■ public policy

In 2004, the American Heart Association (AHA) published its first scientific statement regarding air pollution and cardiovascular disease (CVD).[1] The rationale was to provide researchers, healthcare providers, and regulatory agencies with a comprehensive review of the evidence linking air pollution exposure with cardiovascular morbidity and mor-

The American Heart Association makes every effort to avoid any actual or potential conflicts of interest that may arise as a result of an outside relationship or a personal, professional, or business interest of a member of the writing panel. Specifically, all members of the writing group are required to complete and submit a Disclosure Questionnaire showing all such relationships that might be perceived as real or potential conflicts of interest.

This statement was approved by the American Heart Association Science Advisory and Coordinating Committee on February 22, 2010. A copy of the statement is available at http://www.americanheart.org/presenter.jhtml?identifier=3003999 by selecting either the "topic list" link or the "chronological list" link (No. KB-0038). To purchase additional reprints, call 843-216-2533 or e-mail kelle.ramsay@wolterskluwer.com.

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Expert peer review of AHA Scientific Statements is conducted at the AHA National Center. For more on AHA statements and guidelines development, visit http://www.americanheart.org/presenter.jhtml?identifier=3023366.

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tality. There was also an explicit aim to educate clinicians about the importance of this issue, because the cardiovascular health consequences of air pollution generally equal or exceed those due to pulmonary diseases.[1–4] Finally, a list of key remaining scientific questions and strategic avenues for investigation were provided to help foster and guide future research.

The first AHA writing group concluded that short-term exposure to particulate matter (PM) air pollution contributes to acute cardiovascular morbidity and mortality[1] and that exposure to elevated PM levels over the long term can reduce life expectancy by a few years. Although some mechanistic details remained incompletely described, the existing science was deemed adequate to substantiate several plausible biological pathways whereby PM could instigate acute cardiovascular events and promote chronic disease.

There is mounting evidence from a rapid growth of published data since the previous statement related to the harmful cardiovascular effects of air pollution.[3,4] Most, but not all, epidemiological studies corroborate the elevated risk for cardiovascular events associated with exposure to fine PM <2.5 μm in aerodynamic diameter ($PM_{2.5}$). $PM_{2.5}$ generally has been associated with increased risks of myocardial infarction (MI), stroke, arrhythmia, and heart failure exacerbation within hours to days of exposure in susceptible individuals. Several new studies have also demonstrated that residing in locations with higher long-term average PM levels elevates the risk for cardiovascular morbidity and mortality. Some recent evidence also implicates other size fractions, such as ultrafine particles (UFPs) <0.1 μm, gaseous copollutants (eg, ozone and nitrogen oxides [NO_x]), and specific sources of pollution (eg, traffic). In addition, there have been many insights into the mechanisms whereby PM could prove capable of promoting CVDs.[2–4] Air pollutants have been linked with endothelial dysfunction and vasoconstriction, increased blood pressure (BP), prothrombotic and coagulant changes, systemic inflammatory and oxidative stress responses, autonomic imbalance and arrhythmias, and the progression of atherosclerosis. In the interim, the US Environmental Protection Agency (EPA) completed its updated "Air Quality Criteria for Particulate Matter"[5] and afterward strengthened the National Ambient Air Quality Standards (NAAQS) for daily $PM_{2.5}$ levels starting in 2006 (down from 65 to 35 $\mu g/m^3$).[6] The most recent scientific review coordinated by the EPA, the final report of the Integrated Science Assessment for Particulate Matter (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546), has also been made available publicly. These numerous changes and advances provide the rationale for the present updated AHA scientific statement on PM air pollution and CVD. This updated statement is similar in scope, content, and overall structure to the first document; however, it provides many additional conclusions and recommendations that can now be made because of the expanded number and quality of studies.

Objectives and Methods

The primary objective of this scientific statement is to provide a comprehensive updated evaluation of the evidence linking PM exposure with CVDs. The focus of this review is explicitly on PM because the majority of air pollution studies have centered on its cardiovascular effects, and the strength of the evidence makes it possible to provide consensus opinions and recommendations. Except for in a few circumstances, such as when copollutants have been shown to (or not to) modify the responses to PM exposure or to have independent cardiovascular effects in epidemiological studies of major importance, a detailed discussion of other air pollutants (eg, ozone and NO_2) is beyond the scope of this document. Additional objectives are to provide expert consensus opinions on aspects related to the current state of science, to specifically highlight the health and clinical implications of the reviewed findings, and to provide prudent and practical recommendations for measures to reduce PM exposure that might thereby lower the associated cardiovascular risk. This updated scientific statement is structured to first provide a clinical perspective on the cardiovascular risks posed by PM exposure and then briefly review the components of air pollution. The following sections highlight the major findings from epidemiological studies, including mortality, morbidity, and surrogate outcome results. Next, the animal and human mechanistic studies are reviewed, and an overall framework whereby PM exposure could cause CVDs is outlined. Finally, updated consensus opinions and conclusions are provided, followed by suggestions for areas of future research and policy considerations.

Members of the current writing group were selected from across a broad range of disciplines, including cardiovascular and environmental epidemiology and statistics, atmospheric sciences, cardiovascular and pulmonary medicine, basic science research, and public policy. The writing group identified studies published in the English language between January 1, 2004, and March 31, 2009, by a World Wide Web–based literature search using Medline, PubMed, and Google search engines. Key terms included *air pollution* or *particulate matter* plus any of the following: *cardiovascular, myocardial, heart, cardiac, stroke, heart failure, arrhythmia, heart rate variability, autonomic, sympathetic, atherosclerosis, vascular, blood pressure, hypertension, diabetes, metabolic, thrombosis,* and *coagulation*. Additional studies were identified within the references of these publications and by the personal knowledge of the writing group members. A few studies published after March 31, 2009, were added during the review process. All of the identified epidemiological studies that provided mortality data or hard cardiovascular outcomes (eg, MIs) and controlled human exposure protocols were included. In a few circumstances, studies before 2004 were included briefly in the discussion or tables when it was believed that they provided contextual background and/or relevant findings from earlier analyses of ongoing studies (eg, Harvard Six Cities and American Cancer Society [ACS] cohorts) from which new results after 2004 have been published. It is a limitation of the present review that it was not possible to cite all surrogate outcome human studies because of the enormous number of publications. Some were not included, without intentional bias with regard to results, when multiple referenced studies demonstrated similar findings. In such a situation (eg, heart rate variability [HRV]), this

limitation was noted within the specific section. A main theme of the present statement is to provide clinical context and recommendations for healthcare providers, and thus, it was beyond the scope and not the intent of this document to include all animal, ex vivo, or toxicological studies. A number of these publications were also not included, without intentional bias with regard to results. The writing group included publications that were believed to have relevant implications for human cardiovascular health, those that formed the foundation of the mechanistic hypotheses, and studies that were deemed of major importance. Finally, the "evidence summary" statements and all points in the conclusions and recommendations represent consensus expert opinions agreed on by all members of the writing group during formal discussions. It is explicitly stated when no such agreement was reached. These statements and the points within Tables 6 and 7 do not represent the result of applying the standard AHA criteria (ie, level and class) to the sum findings of the present review, because those do not apply, but rather the qualitative consensus opinions agreed on by the writing group. The purpose is to provide expert opinions on the comparative relative ranking and the strength of the overall evidence regarding different areas within this field of science.

Perspective on the Air Pollution–Cardiovascular Risk Association

Traditional cardiovascular risk factors account for the major portion of the risk for ischemic cardiac events within a population.[7] Individuals with optimal levels of all risk factors have been shown to have a low lifetime cardiovascular event rate.[8] Thus, control of the traditional risk factors is recognized to be of paramount importance to prevent CVDs. In this context, there has been some debate about the overall clinical relevance and utility of adding novel risk factors to risk-prediction models to incrementally improve their overall predictive value, even when assessed by multiple methodologies.[9] On the other hand, the ability to predict future events by existing models remains imperfect. In addition to several mathematical and statistical explanations for this shortcoming,[10,11] it is important to recognize that the development of vascular or atherosclerotic disease (the factor predicted by most statistical models) is usually a necessary but insufficient cause of future ischemic events in and of itself. Cardiovascular events must also be triggered by an additional factor at some unknowable future time, and therefore, they transpire as a stochastic process within a population.[12] This is one of several reasons why PM air pollution is a uniquely important public health issue among the list of novel risk factors; PM inhalation is an established trigger of cardiovascular events that occur within hours to days after exposure.[12] Because of the ubiquitous and involuntary nature of PM exposure, it may continuously enhance acute cardiovascular risk among millions of susceptible people worldwide in an often inconspicuous manner. Moreover, beyond serving as a simple trigger, PM elicits numerous adverse biological responses (eg, systemic inflammation) that, in premise, may further augment future cardiovascular risk over the long term after months to years of exposure.

Effects of Short-Term Exposure

Time-series studies estimate that a 10-$\mu g/m^3$ increase in mean 24-hour $PM_{2.5}$ concentration increases the relative risk (RR) for daily cardiovascular mortality by approximately 0.4% to 1.0%.[3] Despite theoretical statistical risks ascribed to all individuals, this elevated risk from exposure is not equally distributed within a population. At present-day levels, $PM_{2.5}$ likely poses an acute threat principally to susceptible people, even if seemingly healthy, such as the elderly and those with (unrecognized) existing coronary artery or structural heart disease.[13] Therefore, the absolute risk rather than the RR of exposure may more effectively convey the tangible health burden within a population. A 10-$\mu g/m^3$ increase during the preceding day contributes on average to the premature death of approximately 1 susceptible person per day in a region of 5 million people (based on annual US death rates in 2005).[3,14] Although the dangers to 1 individual at any single time point may be small, the public health burden derived from this ubiquitous risk is enormous. Short-term increases in $PM_{2.5}$ levels lead to the early mortality of tens of thousands of individuals per year in the United States alone.[1,3,5]

Effects of Long-Term Exposure

Cohort studies estimate that the RR associated with living in areas with higher PM levels over the long term is of greater magnitude than that observed from short-term exposure increases (RR between 1.06 and 1.76 per 10 $\mu g/m^3$ $PM_{2.5}$).[3] In this context, the World Health Organization estimated that $PM_{2.5}$ contributes to approximately 800 000 premature deaths per year, ranking it as the 13th leading cause of worldwide mortality.[15] Hence, PM air pollution appears to be an important modifiable factor that affects the public health on a global scale.

Air Pollution

The first AHA statement on air pollution reviewed the size fractions, sources, and chemical constituents of PM and the main gaseous air pollutants: Nitrogen oxides (NO_x; ie, $NO+NO_2$), carbon monoxide (CO), sulfur dioxide (SO_2), and ozone (O_3).[1] Therefore, this section within the updated statement focuses on several other contemporary aspects of air pollution characterization and exposure assessment, particularly in relation to their potential influences on cardiovascular health. In brief, PM is broadly categorized by aerodynamic diameter: All particles <10 μm (thoracic particles [PM_{10}]), all particles <2.5 μm (fine particles [$PM_{2.5}$]), all particles <0.1 μm (UFP), and particles between 2.5 and 10 μm (coarse particles [$PM_{10-2.5}$]). Hence, PM_{10} contains within it the coarse and $PM_{2.5}$ fractions, and $PM_{2.5}$ includes UFP particles. The concentrations of PM_{10} and $PM_{2.5}$ are typically measured in their mass per volume of air ($\mu g/m^3$), whereas UFPs are often measured by their number per cubic centimeter (Table 1). The major source of $PM_{2.5}$ throughout

Table 1. Ambient Air Pollutants

Pollutant	US Average Range	US Typical Peak*	Most Recent NAAQS for Criteria Pollutants (Averaging Time)
O_3†	0–125 ppb	200 ppb	75 ppb (8 h)‡
NO_2†	0.5–50 ppb	200 ppb	100 ppb (1 h)§ 53 ppb (Annual mean)
NO†	0–100 ppb	200 ppb	
SO_2†	0.1–50 ppb	150 ppb	140 ppb (24 h)‖ 30 ppb (Annual mean)
CO†	0.1–5 ppm	20 ppm	35 ppm (1 h)‖ 9 ppm (8 h)‖
PM_{10}¶	10–100 $\mu g/m^3$	300 $\mu g/m^3$	150 $\mu g/m^3$ (24 h)#
$PM_{2.5}$¶	5–50 $\mu g/m^3$ (Mean=13.4±5.6)	100 $\mu g/m^3$	15 $\mu g/m^3$ (Annual mean) 35 $\mu g/m^3$ (24 h)**
$PM_{2.5}$ lead¶	0.5–5 ng/m^3	150 ng/m^3	0.15 $\mu g/m^3$ (Rolling 3-month average)††
NH_3†	0.1–20 ppb	100 ppb	
HNO_3†	0–5 ppb	10 ppb	
Methane†	1–2 ppm	5 ppm	
Formaldehyde†	0.1–10 ppb	40 ppb	
Acetaldehyde†	0.1–5 ppb	20 ppb	
NMHC (VOC)¶	20–100 $\mu g/m^3$	250 $\mu g/m^3$	
Propane¶	2–20 $\mu g/m^3$	500 $\mu g/m^3$	
Benzene¶	0.5–10 $\mu g/m^3$	100 $\mu g/m^3$	
1,3-Butadiene¶	0.1–2 $\mu g/m^3$	10 $\mu g/m^3$	
Total suspended particles¶	20–300 $\mu g/m^3$	1000 $\mu g/m^3$	
$PM_{10-2.5}$¶	5–50 $\mu g/m^3$	200 $\mu g/m^3$	
Sulfate¶	0.5–10 $\mu g/m^3$	30 $\mu g/m^3$	
Nitrate¶	0.1–5 $\mu g/m^3$	20 $\mu g/m^3$	
Organic carbon¶	1–20 $\mu g/m^3$	30 $\mu g/m^3$	
Elemental carbon¶	0.1–3 $\mu g/m^3$	10 $\mu g/m^3$	
PAH¶	2–50 ng/m^3	200 ng/m^3	
UFP†	1000–20 000/cm^3	100 000/cm^3	

ppb Indicates parts per billion; ppm, parts per million; and PAH, polycyclic aromatic hydrocarbon.

*Generally not in concentrated plumes or locations of direct source emission impact.

†Typical hourly average concentrations reached in US cities.

‡The 8-hour standard is met when the 3-year average of the 4th highest daily maximum 8-hour average is less than or equal to the indicated number. In January 2010, the EPA proposed a more stringent 8-hour standard within the range of 60 to 70 ppb (http://www.epa.gov/air/ozonepollution/actions.html).

§To attain this standard, the 3-year average of the 98th percentile of the daily maximum 1-hour average at each monitor within an area must not exceed this value.

‖The level is not to be exceeded more than once per year.

¶Typical 24-hour average concentrations.

#The level is not to be exceeded more than once per year on average over 3 years.

**The daily standard is met when the 3-year average of the 98th percentile of 24-hour PM level is less than or equal to the indicated number.

††Although the typical concentrations shown in the table are for $PM_{2.5}$, the lead standard continues to be based on measurements in total suspended particulate.

the world today is the human combustion of fossil fuels from a variety of activities (eg, industry, traffic, and power generation). Biomass burning, heating, cooking, indoor activities, and nonhuman sources (eg, fires) may also be relevant sources, particularly in certain regions.

Common air pollutants and those designated as EPA criteria pollutants (ie, specifically targeted in regulations through limits on emissions or government standards such as the NAAQS) are listed in Table 1. The World Health Organization also provides ambient guidelines (http://www.euro.who.int/Document/E90038.pdf). As a result, many pollutant concentrations are tracked in the United States by nationwide monitoring networks, with up to approximately 1200 sites for O_3 and $PM_{2.5}$. Data are archived by the EPA and are available to the public (http://www.epa.gov/ttn/airs/airsaqs/). O_3 levels exceed the national standard in many areas, and thus, daily information is provided to assist the public in reducing their exposure. A lower standard for ozone concentrations was proposed recently, which will lead to more frequent occurrences of outdoor exposures deemed to be excessive (Table 1). The reporting of $PM_{2.5}$ is also becoming common because of its impact on public health and frequent violations of standards. Current and forecast air quality indices and information on both $PM_{2.5}$ and ozone are available (http://airnow.gov/). At the end of 2008, 211 US counties (or portions of counties) were in nonattainment of the 2006 daily $PM_{2.5}$ NAAQS (http://www.epa.gov/pmdesignations/2006standards/state.htm). On a positive note, the various regulations that have been established have led to substantial reductions in PM and other pollutant levels over the past 40 years in the United States and contributed toward similar improvements in other countries. However, reducing the levels of some pollutants, such as O_3, remains a challenge because of the complex chemical processes that lead to their formation in the atmosphere.[16] The population of many developing nations (China, India, Middle Eastern countries) continues to be exposed to high levels, particularly of PM, which can routinely exceed 100 $\mu g/m^3$ for prolonged periods (http://siteresources.worldbank.org/DATASTATISTICS/Resources/table 3_13.pdf).

Air Pollution Mixtures, Chemistry, and Sources

Detailed information regarding PM sizes, composition, chemistry, sources, and atmospheric interactions is beyond the scope of this document but can be found in the 2004 US EPA Air Quality Criteria for Particulate Matter final report (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=87903). The source for much of the information provided in this brief summary is this document, unless otherwise specifically referenced. The typical range of ambient concentrations for several air pollutants in the United States, including the latest US NAAQS for the criteria pollutants, is given in Table 1. Classification of air quality according to 1 single pollutant and by size or mass provides an incomplete picture, because ambient air pollution is a complex mixture of gases, particles, and liquids that are continually changing and interacting with each other and natural atmospheric gases. Although $PM_{2.5}$ mass has rightfully attracted considerable attention as a target for regulation and epidemiological study, more than 98% of

the air pollutant mass in the mixture we breathe in urban settings is from gases or vapor-phase compounds such as CO, nonmethane hydrocarbons or volatile organic carbons (VOCs), NO_2, NO, O_3, and SO_2. Each of these can have independent and potentially synergistic or antagonistic effects with each other and with PM; however, at present, the cardiovascular health impact of exposure to combinations of air pollutants is not well understood.

Most of the studies linking CVDs with PM exposures have focused on particle mass; thus, this association is evaluated and reported in the majority of epidemiological and toxicological studies reviewed. Although PM is regulated by mass concentration, the aspect of PM most harmful to cardiovascular health may not be best quantified by mass measurement alone. The sum effect of many features related to chemical composition and size/morphology (eg, oxidative stress potential, solubility, charge, surface area, particle count, lung deposition, and stability within the atmosphere and biological tissues) is important to consider. With regard to specific "toxic" compounds within PM, several lines of existing evidence support the idea that transition metals, organic compounds, semiquinones, and endotoxin are likely relevant in relation to promoting CVDs. In addition, certain characteristics of UFPs (eg, high surface area, particle number, metal and organic carbon content) suggest that they may pose a particularly high cardiovascular risk after short-term exposure.[17] Both the additional characterization of "criteria" pollutants and the measurement of several other pollutants (discussed below) are important to inform air quality management practices that involve air quality modeling, as well as epidemiological studies and risk assessment, which ultimately aim to improve risk-reduction strategies.

In addition to their mass concentration, pollutants can be characterized on the basis of their origin or chemical and physical properties. In terms of origin, nitrogen oxides ($NO+NO_2$), CO, SO_2, and $PM_{2.5,}$ as well as carbon dioxide (CO_2), are mainly associated with combustion of fuel or other high-temperature industrial processes. Combustion PM is composed of many chemical compounds, including organic carbon species, elemental or black carbon, and trace metals (eg, lead and arsenic). They range in size from molecular clusters a few nanometers in diameter to light-scattering particles that peak on a mass contribution basis in the diameter range of 200 to 1000 nm (0.2 to 1 μm). UFP numbers are also strongly linked to fresh combustion and traffic-related pollution. Ammonia, methane, pesticides (persistent organic pollutants), reduced sulfur compounds, resuspended dust, and natural coarse particles ($PM_{10-2.5}$) are associated with noncombustion surface or fugitive releases that arise from a variety of human (eg, agriculture) and natural (eg, erosion) activities. Agricultural emissions and releases from a range of industrial processes and waste management are also important sources. Road and wind-blown dust from agricultural practices and from certain industrial facilities (eg, mineral industry) also contribute to these particles, which are typically in the coarse ($PM_{10-2.5}$) or even larger ($>PM_{10}$) range.

In addition to pollutants formed directly by combustion, many others are produced primarily through chemical reactions in the atmosphere among directly emitted pollutants. These are known as secondary pollutants. Sunlight, water vapor, and clouds are often involved in this atmospheric chemistry, which leads to greater oxidation of the pollutants. Examples include PM-associated sulfate, nitrate, and ammonium and many of the organic compounds within $PM_{2.5}$. Besides O_3, which is the most prevalent secondary gaseous oxidant, a number of inorganic and organic acids and VOCs form in the atmosphere. Examples are the hydroxyl radical, peroxyacetyl nitrate, nitric acid, formic and acetic acid, formaldehyde, and acrolein.

VOCs and semivolatile organic compounds (SVOCs), the latter of which are found in both the gas and particle phase, are an additional large class of pollutants. They are associated with both combustion and fugitive emissions, as well as with secondary formation. Key examples are benzene, toluene, xylene, 1,3-butadiene, and polycyclic aromatic hydrocarbons. VOCs are among the 188 hazardous air pollutants listed by the EPA, and their main emission sources have been identified and are regulated (http://www.epa.gov/ttn/atw/mactfnlalph.html). VOCs can undergo reactions that convert toxic substances to less toxic products or vice versa. Many VOCs contribute to the formation of O_3 and are oxidized in the atmosphere, becoming SVOCs, and subsequently partition within particles and contribute to the composition of $PM_{2.5}$, as well as to its mass. A great deal of research has focused on $PM_{2.5}$ in the past decade, which has led to advances in measurement technologies[18] and greater understanding of its chemistry and atmospheric behavior.[19] Nonetheless, understanding is incomplete, particularly with regard to formation of the secondary organic fraction, the relative role of anthropogenic and biogenic emissions to organics, surface chemistry, oxidative potential,[20] and gas-to-particle partitioning.

An alternative to attempting to identify one by one which pollutant(s) or chemical compounds are most harmful is to focus on identifying the sources, which typically emit mixtures of pollutants, of greatest concern. It may be the mixture of pollutants (along with the source from which it is derived, which determines its characteristics) that is most pertinent to human health outcomes. Such information may actually be more relevant for aiding the development of effective air quality policies. One important example reviewed in the epidemiology section is that the evidence continues to grow regarding the harmful cardiovascular effects of traffic-related pollution. Traffic is ubiquitous in modern society, with a sizeable proportion of the population, particularly persons disadvantaged by low socioeconomic status, living close enough (within 500 m) to a major road or a freeway to be chronically exposed to elevated concentrations. Additionally, daily behavior brings most people close to this source, with the average US citizen over 15 years of age spending 55 minutes each day traveling in motor vehicles.[21] However, despite the consistent epidemiological findings, these studies have yet to elucidate which of the many pollutants or other associated risks (ie, noise) produced by traffic are responsible for the increase in risk for CVD. Until the most harmful agents are identified, the only practical manner to potentially reduce health consequences would be to reduce overall traffic and related emissions and to configure cities and lifestyles

such that there is greater separation between the people and the source, so that we could spend less time in traffic (a major source of personal exposures in our society). There are also a myriad of other important pollutant sources of known toxic pollutants that have been implicated in health-effect studies (eg, power generation, industrial sources, steel mills, and wood smoke). A better understanding of the factors that influence population exposure to these sources, of how their emissions and mixtures of different sources affect health, and about the factors that make individuals more susceptible will aid in the development of more effective environmental health policies.

Determinants of Air Pollution Exposure

Many aspects of air pollution play a role in the characteristics of population- and individual-level exposures. Pollutants vary on multiple time scales, with emission rates, weather patterns, and diurnal/seasonal cycles in solar radiation and temperature having the greatest impact on concentrations. The temporal behavior of a pollutant is also governed by its formation rate and the length of time it remains in the atmosphere. As such, the concentrations of many air pollutants tend to co-vary. For example, NO_x and CO are emitted during combustion, as are some particle constituents (eg, elemental carbon) and VOCs, and thus, their concentrations peak during rush hour. On the other hand, O_3 and other photochemical oxidants, including secondary $PM_{2.5}$ and secondary VOCs, peak in the afternoon, particularly given certain meteorologic conditions (eg, more sunshine). Among the common air pollutants, O_3 and $PM_{2.5}$ have the longest atmospheric lifetime and thus can build up over multiple days and spread, by the prevailing winds, over large geographic regions. This can lead to similarities in their temporal and spatial patterns over broad regions and to greater numbers of people being exposed to similar levels, thus lessening interindividual variability in exposure.

Periods of suppressed horizontal and vertical mixing in the lower atmosphere lead to the buildup of multiple pollutants. These situations are most common under slow-moving or stationary high-pressure systems, which bring light winds, a stable atmosphere, and more sunshine. The frequency and seasonality of these meteorologic conditions and how they affect concentrations vary geographically, which leads to differences in the characteristics of pollution episodes from the western to the eastern United States, as well as within these regions.

The commonality of meteorology and emission sources leads to covariation in pollutant concentrations on multiple temporal and spatial scales, which makes it more challenging for epidemiological studies to identify the health effects of individual pollutants and the effects of copollutants or mixtures. Studies that depend on daily counts of mortality or morbidity events have difficulties separating the effects of the different pollutants in the urban mix. Even prospective panel studies measuring specific end points on a subdaily time scale are hindered by pollutant covariation. Some of these challenges could potentially be addressed by undertaking studies covering multiple geographic locations with differences in the structure of pollutant covariation due to different meteorology and source mixes. Indeed, this has been done, at least in part, by several existing multicity studies. Consistency in the findings in individual studies conducted in different cities also helps isolate the pollutants that may be more responsible for the health effects. The consistent positive findings with certain pollutants (eg, PM mass concentration) have helped strengthen the evidence regarding PM_{10} and $PM_{2.5}$ effects, but regardless of location, there remains the strong underlying commonality of fossil fuel combustion for many pollutants.

A final issue to consider is the cardiovascular health effects of exposures that occur at the personal level because of the different microenvironments or activities an individual experiences (eg, time in traffic, indoor sources, secondhand tobacco smoke, occupational exposure, and degree of indoor penetration of ambient PM into homes) versus the effects of exposures from less variable urban- to regional-scale ambient concentrations (ie, background pollution that most individuals encounter more uniformly). Personal monitoring demonstrates substantial variations among individual pollution exposures or characteristics among those living within the same metropolitan area and even the same neighborhood.[22,23] However, the differing additive, synergistic, and/or confounding effects on cardiovascular health of these 2 contrasting components of a person's overall exposure have not been well described. For the most part, the magnitude of the findings reported by the major epidemiological studies (see next section) are indicative of the effects of the urban- to regional-scale ambient concentrations. Actual exposures to all pollutants also vary at the personal level. The cardiovascular health importance of these individual-level variations (above and beyond the effect of urban/regional levels) remains largely unknown, in part because it has been difficult to quantify. The degree to which measurement of personal exposures or more precise exposure assessment (eg, use of geographic information systems, land-use regression models, spatial-temporal models, and adjustments for indoor penetration) can reduce the effects of exposure misclassification in epidemiological studies also remains to be fully elucidated.[24–26]

Epidemiological Studies of Air Pollution

Epidemiological studies of air pollution have examined the health effects of exposures observed in real-world settings at ambient levels. Associations between relevant health end points and measures of air pollution are evaluated while attempting to control for effects of other pertinent factors (eg, patient and environmental characteristics). Despite substantial study and statistical improvements and the relative consistency of results, some potential for residual confounding of variables and publication bias[27] of positive studies are limitations to acknowledge. Probably the most relevant, well-defined, and extensively studied health end points include mortality (all-cause and cause-specific), hospitalizations, and clinical cardiovascular events. This section reviews the results of the epidemiological research with a focus on new studies since the first AHA statement was published,[1] as well as on the cardiovascular health implications. In sum, numerous studies of varied design have been published in the interim that significantly add to the overall weight of evi-

Table 2. Comparison of Pooled Estimated of Percent Increase (and 95% CI or Posterior Interval or *t* Value) in RR of Mortality Estimated Across Meta-Analyses and Multicity Studies of Daily Changes in Exposure

			Percent Increases in Mortality (95% CI)		
	Primary Source	Exposure Increment	All-Cause	Cardiovascular	Respiratory
Meta-estimate with and without adjustment for publication bias	Anderson et al[27] 2005	20 $\mu g/m^3$ PM_{10}	1.0 (0.8–1.2) 1.2 (1.0–1.4)	…	…
Meta-estimates from COMEAP report to the UK Department of Health on CVD and air pollution	COMEAP[31] 2006	20 $\mu g/m^3$ PM_{10} 10 $\mu g/m^3$ $PM_{2.5}$	… …	1.8 (1.4–2.4) 1.4 (0.7–2.2)	… …
NMMAPS, 20 to 100 US cities	Dominici et al[34] 2003	20 $\mu g/m^3$ PM_{10}	0.4 (0.2–0.8)	0.6 (0.3–1.0)*	…
APHEA-2, 15 to 29 European cities	Katsouyanni et al[35] 2003 Analitis et al[36] 2006	20 $\mu g/m^3$ PM_{10}	1.2 (0.8–1.4)	1.5 (0.9–2.1)	1.2 (0.4–1.9)
US, 6 cities	Klemm and Mason[37] 2003	10 $\mu g/m^3$ $PM_{2.5}$	1.2 (0.8–1.6)	1.3 (0.3–2.4)†	0.6 (−2.9, 4.2)‡
US, 27 cities, case-crossover	Franklin et al[38] 2007	10 $\mu g/m^3$ $PM_{2.5}$	1.2 (0.3–2.1)	0.9 (−.1, 2.0)	1.8 (0.2, 3.4)
California, 9 cities	Ostro et al[39] 2006	10 $\mu g/m^3$ $PM_{2.5}$	0.6 (0.2–1.0)	0.6 (0.0, 1.1)	2.2 (0.6, 3.9)
France, 9 cities	Le Tertre et al[40] 2002	20 $\mu g/m^3$ BS	1.2 (0.5–1.8)§	1.2 (0.2–2.2)§	1.1 (−1.4, 3.2)§
Japan, 13 cities, age >65 y	Omori et al[41] 2003	20 $\mu g/m^3$ SPM	1.0 (0.8–1.3)	1.1 (0.7–1.5)	1.4 (0.9–2.1)
Asia, 4 cities	Wong et al[42] 2008	10 $\mu g/m^3$ PM_{10}	0.55 (0.26–0.85)	0.59 (0.22–0.93)	0.62 (0.16–1.04)
US, 112 cities	Zanobetti et al[43] 2009	10 $\mu g/m^3$ $PM_{2.5}$	0.98 (0.75–1.22)	0.85 (0.46–1.24)	1.68 (1.04–2.33)
		10 $\mu g/m^3$ $PM_{10-2.5}$‖	0.46 (0.21–0.71)	0.32 (0.00–0.64)	1.16 (0.43–1.89)
		10 $\mu g/m^3$ $PM_{2.5}$¶	0.77 (0.43–1.12)	0.61 (0.05–1.17)	1.63 (0.69–2.59)
		10 $\mu g/m^3$ $PM_{10-2.5}$¶	0.47 (0.21–0.73)	0.29 (−0.04, 0.61)	1.14 (0.043–1.85)

CI indicates confidence interval or posterior interval.
*Cardiovascular and respiratory deaths combined.
†Ischemic heart disease deaths.
‡Chronic obstructive pulmonary disease deaths.
§Includes general additive model–based analyses with potentially inadequate convergence.
‖Results for $PM_{10-2.5}$ are from 47 cities.
¶Results of 2 pollutant models controlling for alternate PM size in 47 cities.

dence that exposure to air pollutants at present-day levels contributes to cardiovascular morbidity and mortality.

Mortality and Air Pollution

Time-Series and Related Studies

Time-series and case-crossover studies explore associations between short-term changes in air pollution and daily changes in death counts. The sum of current evidence supports the findings of an earlier review[28] that demonstrated that short-term elevations in daily PM levels lead to a greater absolute risk for CVD-related mortality than for all other causes. Even if similar acute RR elevations (≈1.01) are estimated between cardiovascular and pulmonary mortality, CVDs account for 69% of the increase in absolute mortality rates compared with 28% for pulmonary diseases attributable to short-term PM exposure. Recently, more rigorous modeling techniques have been used in attempts to better estimate pollution-mortality associations while controlling for other time-dependent confounding covariables.[29,30] There have been well over 100 published daily time-series studies reporting small but statistically significant PM-mortality associations that have been the subject of quantitative reviews or meta-analyses.[3,27,31–33] Table 2 summarizes recent multicity analyses and studies published since 2004.

To address concerns about city selection bias, publication bias, and influences of copollutants, several large, multicity, daily time-series studies have been conducted worldwide. One of the largest was the National Morbidity, Mortality, and Air Pollution Study (NMMAPS). Published reports from this study included as few as 20 US cities,[44,45] as many as 100 cities,[46,47] and more recently, data for hundreds of counties (Table 2).[48] The observed relationship between PM exposure and excess mortality remained independent of several gaseous copollutants (NO_2, CO, or SO_2). Recent analyses suggest that O_3 may also independently contribute to cardiopulmonary mortality risk[49,50]; however, coexposures to secondary particle pollutants may be responsible in part for this latter association.[51]

Several studies have also been conducted outside the United States, including the Air Pollution and Health: A European Approach (APHEA and APHEA-2) projects, which examined daily PM-related mortality effects in multiple cities.[36,52] PM air pollution was significantly associated with daily mortality counts for all-cause, cardiovascular, and respiratory mortality (Table 2). Further analyses of the European data suggest that CVD deaths are also associated with exposure to NO_2[53] and CO.[54] A few new time-series studies have also confirmed similar increases in cardiovascular mortality related to short-term PM exposure in China[55–57] and Bangkok, Thailand.[42] Additional multicity studies have been conducted worldwide with analyses of CVD deaths (Table 2).[38–42,58–60] Finally, in a recent analysis that included several Asian

cities, SO_2, NO_2, O_3, and PM_{10} were all associated with excess cardiovascular mortality.[42]

In an attempt to evaluate the coherence of multicity studies across continents, the Air Pollution and Health: A Combined European and North American Approach (APHENA) study analyzed data from the APHEA, NMMAPS, and Canadian studies.[61] The combined effect on all-cause mortality ranged from 0.2% to 0.6% for a 10-$\mu g/m^3$ elevation in daily ambient PM_{10}, with the largest effects observed in Canada. Among individuals older than 75 years, the effects were greater for cardiovascular mortality than for overall and pulmonary mortality (0.47% to 1.30%). Older age (>75 years) and higher rates of unemployment were related to greater PM mortality risks in both continents. Higher NO_2 levels were associated with larger PM_{10} effects on mortality, particularly in Europe. Finally, there appeared to be no lower-limit threshold below which PM_{10} was not associated with excess mortality across all regions.

Evidence Summary

The overall evidence from time-series analyses conducted worldwide since publication of the first AHA statement[1] confirms the existence of a small, yet consistent association between increased mortality and short-term elevations in PM_{10} and $PM_{2.5}$ approximately equal to a 0.4% to 1.0% increase in daily mortality (and cardiovascular death specifically) due to a 10-$\mu g/m^3$ elevation in $PM_{2.5}$ during the preceding 1 to 5 days (Table 2).

Cohort and Related Studies

Although short-term changes in PM concentrations have deleterious health effects, longer-term exposures may have a more pertinent clinical health effect on cardiovascular morbidity and mortality given that individuals are typically exposed to higher air pollution levels over extended periods of time. An additional source of exposure variability that has been exploited in epidemiological studies is spatial variability, which includes differences in average ambient concentrations over extended periods of time across metropolitan areas or across smaller communities within local areas. Recent emphasis has been on prospective cohort studies that control for individual differences in multiple confounding variables and cardiovascular risk factors. A summary of these studies is presented in Table 3 and Figure 1. These cohort studies generally demonstrate larger overall mortality effects than the results of time-series analyses.

Harvard Six Cities and ACS Studies

Two landmark cohort-based mortality studies, the Harvard Six Cities[62] and the ACS studies,[66] were reported in the mid 1990s and were discussed previously.[1] In both, $PM_{2.5}$ and sulfate particulate pollution were associated with increases in all-cause and cardiopulmonary disease (Table 3). In addition, intensive independent reanalyses[63] corroborated the original findings of both studies and resulted in innovative methodological contributions that demonstrated the robustness of the results to alternative modeling approaches. In both the Harvard Six Cities[62,64] and the ACS[67] studies, PM air pollution–related mortality was substantially higher for cardiovascular- than for pulmonary-related causes.

Since 2004, there have been further analyses of both studies. Laden et al[64] extended the mortality follow-up of the Harvard Six Cities cohort for an additional 8 years. $PM_{2.5}$ associations, similar to those found in the original analysis, were observed for all-cause and CVD mortality (Table 3). Furthermore, reductions in $PM_{2.5}$ concentrations for the extended follow-up period were associated with reduced mortality risk. Further analysis suggested that the health effects of changes in exposure were seen primarily within 2 years.[84] In addition to confirming the earlier mortality relationship, the recent observations suggest that the adverse health effects mediated by longer-term PM air pollution exposure can be estimated reasonably accurately by the previous few years of particle levels.

Extended analyses of the ACS cohort that emphasize efforts to control for the effects of other covariates and risk factors have corroborated the previously reported mortality associations with particulate and sulfur oxide pollution.[68] Elevated mortality risks were most strongly associated with $PM_{2.5}$. Coarse particles ($PM_{10-2.5}$) and gaseous pollutants, except for SO_2, were generally not significantly related to mortality. In another extended analysis,[67] the death certificate classifications of underlying causes of death due to $PM_{2.5}$ exposures were observed to be principally ischemic heart disease, arrhythmias, heart failure, and cardiac arrest. Finally, recent additional analyses attempted to control for the fact that variations in exposure to air pollution across cities or within cities may correlate with socioeconomic or demographic gradients that influence health and susceptibility to environmental exposures.[85,86] When controlled for individual risk factor data, the mortality associations for intrametropolitan $PM_{2.5}$ concentration differences within the Los Angeles, Calif, area were generally larger than those observed in the full cohort across metropolitan areas.[69] However, the results were somewhat sensitive to the inclusion of zip code–level ecological variables, which suggests potential contextual neighborhood confounding. Krewski et al[70] subsequently observed that full adjustments for multiple ecological covariates did not reduce the estimated $PM_{2.5}$-related mortality effect. The association for ischemic heart disease mortality in particular was highly robust across various study areas and modeling strategies and after controlling for both individual and ecological covariates.

An additional recent analysis of the ACS cohort evaluated the health effects of ozone compared with $PM_{2.5}$.[87] The findings reconfirmed the independent cardiovascular mortality increase related to fine-particle exposure. However, after adjustment for $PM_{2.5}$, ozone was associated solely with an elevated risk of death due to respiratory causes; there was no independent risk of ozone exposure on CVD-related mortality. This suggests that the positive findings reported in NMMAPS[50] regarding cardiopulmonary mortality and short-term ozone exposure could be explained at least in part by the enhanced risk of mortality due to lung disease categories.

Table 3. Summary of Cohort Study Results

Study	Size of Cohort (000s)	Follow-Up Period	Covariates Controlled for	Percent Increases in Mortality (95% CI) Associated With 10 $\mu g/m^3$ $PM_{2.5}$ (or Other When Indicated)			
				All-Cause	Cardiopulmonary	Cardiovascular	Ischemic Heart Disease
Harvard Six Cities, original (Dockery et al[62] 1993)	≈8	1974–1991	Individual (smoking+others)	13 (4.2–23)	18 (6.0–32)	…	…
Harvard Six-Cities, HEI reanalysis, Krewski et al[63] 2004	≈8	1974–1991	Individual (smoking+others)	14 (5.4–23)	19 (6.5–33)	…	…
Harvard Six-Cities, extended, Laden et al[64] 2006	≈8	1974–1998	Individual (smoking+others)	16 (7–26)	…	28 (13–44)	…
Six-Cities Medicare cohort, Eftim et al[65] 2008	≈340	2000–2002	Individual (age, sex)	21 (15–27)	…	…	…
ACS, Original, Pope et al[66] 1995	≈500	1982–1989	Individual (smoking+others)	6.6 (3.5–9.8)	12 (6.7–17)	…	…
ACS, HEI reanalysis, Krewski et al[63] 2004	≈500	1982–1989	Individual (smoking+others) +ecological	7.0 (3.9 10)	12 (7.4–17)	13 (8.1–18)	…
ACS, extended I, Pope et al[67,68] 2002, 2004	≈500	1982–1998	Individual (smoking+others)	6.2 (1.6–11)	9.3 (3.3–16)	12 (8–15)	18 (14–23)
ACS, intrametro Los Angeles, Jerrett et al[69] 2005	≈23	1982–2000	Individual (smoking+others) +ecological	17 (5–30)	12 (−3–30)	…	39 (12–73)
ACS, extended II, Krewski et al[70] 2009	≈500	1982–2000	Individual (smoking+others) +ecological	5.6 (3.5–7.8)	13 (9.5–16)	…	24 (20–29)
ACS, Medicare cohort, Eftim et al[65] 2008	7333	2000–2002	Individual (age, sex)+ecological +COPD	11 (9–13)	…	…	…
US Medicare cohort, east/central/west, Zeger et al[71] 2008	13 200	2000–2005	Individual (age, sex)+ecological +COPD	6.8 (4.9–8.7),* 13 (9.5–17) −1.1 (−3 to 0.8)	…	…	…
Women's Health Initiative, Miller et al[72] 2007	≈66	1994–2002	Individual (smoking+others)	…	…	76 (25–147), 24 (9–41)†	…
Nurses' Health Study, Puett et al[73] 2008	≈66	1992–2002	Individual (smoking+others) ecological	7.0 (−3.0 to 18)‡	…	30 (0–71)‡	…
AHSMOG, males only, McDonnell et al[74] 2000	≈4	1977–1992	Individual (smoking+others)	8.5 (−2.3 to 21)	23 (−3 to 55)	…	…
AHSMOG, females only, Chen et al[75] 2005	≈4	1977–2000	Individual (smoking+others)	…	…	42 (6–90)	…
VA hypertensive male I study, Lipfert et al[76] 2006	≈42	1989–1996	Individual (smoking+others) +ecological	15 (5–26)§	…	…	…
VA hypertensive male II study, Lipfert et al[77] 2006	≈30	1997–2001	Individual (smoking+others) +ecological	6 (−6 to 22)	…	…	…
11 CA county, elderly, Enstrom[78] 2005	≈36	1973–2002	Individual (smoking+others) +ecological	4 (1–7)‖, 1 (−0.6 to 2.6)	…	…	…
French PAARC, Filleul et al[79] 2005	≈14	1974–2000	Individual (smoking+others)	7 (3–10)‡	5 (−2 to 12)‡	…	…
German women, Gehring et al[80] 2006	≈5	1980s, 1990s–2003	Individual smoking and socioeconomic status	12 (−8 to 38)	52 (9–115)	…	…

(*Continued*)

Table 3. Continued

Study	Size of Cohort (000s)	Follow-Up Period	Covariates Controlled for	Percent Increases in Mortality (95% CI) Associated With 10 $\mu g/m^3$ $PM_{2.5}$ (or Other When Indicated)			
				All-Cause	Cardiopulmonary	Cardiovascular	Ischemic Heart Disease
Oslo, Norway, intrametro, Naess et al[81] 2007	≈144	1992–1998	Individual age, occupational class, education	…	…	10 (5–16),¶ 14 (6–21), 5 (1–8), 3 (0–5)	…
Dutch cohort, Beelen et al[82] 2008	≈121	1987–1996	Individual (smoking+others) +ecological	6 (−3 to 16)	…	4 (−10 to 21)	…
Great Britain, Elliott et al[83] 2007	≈660	1966–1998	Socioeconomic status	1.3 (1.0–1.6)‡#	1.7 (1.3–2.2)‡#	1.2 (0.7–1.7)‡#	

HEI indicates Health Effects Institute; VA, Veterans Affairs; COPD, chronic obstructive pulmonary disease; and CA, California.

*Three estimates are for the East, Central, and West regions of the United States, respectively.

†Any cardiovascular event.

‡Associated with 10 $\mu g/m^3$ British Smoke (BS) or PM_{10}.

§Estimates from the single-pollutant model. Effect estimates were smaller and statistically insignificant in analyses restricted to counties with nitrogen dioxide data. County-level traffic density was a strong predictor of survival, and stronger than $PM_{2.5}$ when included with $PM_{2.5}$ in joint regressions.

||Two estimates are for the follow-up period 1973–1982 and the follow-up period 1983–2002, respectively.

¶Four estimates are for men 51–70 y old, women 51–70 y old, men 71–90 y old, and women 71–90 y old, respectively.

#Using last 0- to 4-year exposure window.

Additional Cohort Studies

Several additional cohort studies have been published in the past few years (Table 3). Eftim and colleagues[65] studied 2 very large "cohorts" of US Medicare participants who lived in locations included in the Harvard Six Cities and ACS studies. Effects of $PM_{2.5}$ exposure on mortality for the period 2000 to 2002 were estimated after controlling for multiple factors, although not at the individual patient level. For all-cause mortality, the $PM_{2.5}$-mortality associations were larger than those observed in the Harvard Six Cities or ACS cohorts. In an additional analysis of 13.2 million US Medicare participants for the time period 2000 to 2005,[71] $PM_{2.5}$-mortality associations were shown to be similar to those observed in the Harvard Six Cities and ACS studies in the East and Central regions of the United States (and when the data were pooled for the entire United States). However, $PM_{2.5}$ was not associated with mortality in the Western United States or for the oldest age group (>85 years old). These findings generally corroborate the earlier cohort studies and add evidence that aspects of exposure (PM sources or composition) and patient susceptibility might play important roles in determining the health risks.

In a cohort of postmenopausal women without prior CVD from the Women's Health Initiative Observational Study,[72] an association between longer-term $PM_{2.5}$ exposure (median follow-up of 6 years) and cardiovascular events (primary end point) was observed. After adjustment for age and other risk factors, an incremental difference of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with a 24% (95% confidence interval [CI]9% to 41%) increase in all first cardiovascular events (fatal and nonfatal, with a total of 1816 cases). Notably, an incremental difference of 10 $\mu g/m^3$ $PM_{2.5}$ was also associated with a large 76% (95% CI 25% to 147%) increase in fatal cardiovascular events, based on 261 deaths. The risks for both coronary heart disease and strokes were found to be similarly elevated. Interestingly, within-city $PM_{2.5}$ gradients appeared to have larger cardiovascular effects than those between cities, although this difference was not statistically significant. Finally, overweight women (body mass index >24.8 kg/m^2) were at relatively greater cardiovascular risk due to particulate air pollution than leaner women. Noteworthy aspects of this study were improved assessment of the end points by medical record review (rather than by death certificate) and long-term particle exposure estimation. The control for individual-level confounding variables was also superior to that of previous cohort studies.

In another cohort of women, a subset of the Nurses' Health Study from the northeastern United States,[73] an increase of 10 $\mu g/m^3$ modeled estimates of PM_{10} exposures was associated with an approximately 7% to 16% increased risk of all-cause mortality and a 30% to 40% increase in fatal coronary heart disease, depending on the level of adjustment for covariates. This study found that the strongest health risks for all-cause and cardiovascular mortality were seen in association with the average PM_{10} exposure during the previous 24 months before death. Similar to the findings of the Women's Health Initiative, the cardiovascular mortality risk estimates were larger than those of previous cohort studies. In addition, obese women (body mass index >30 kg/m^2) were at greater relative risk, and the increases in mortality (all-cause and cardiovascular) were larger than the effects on nonfatal events. The results were also in accordance with the latest Harvard Six Cities analyses[64] that show that exposure over the most recent preceding 1 to 2 years can accurately estimate the majority of the health risks due to longer-term PM air pollution exposures.

The pollution-mortality association has also been assessed in several other cohort studies in the United States and Europe (Table 3).[76–83] In a recent analysis of the Adventist Health Study of Smog (AHSMOG) cohort with a much

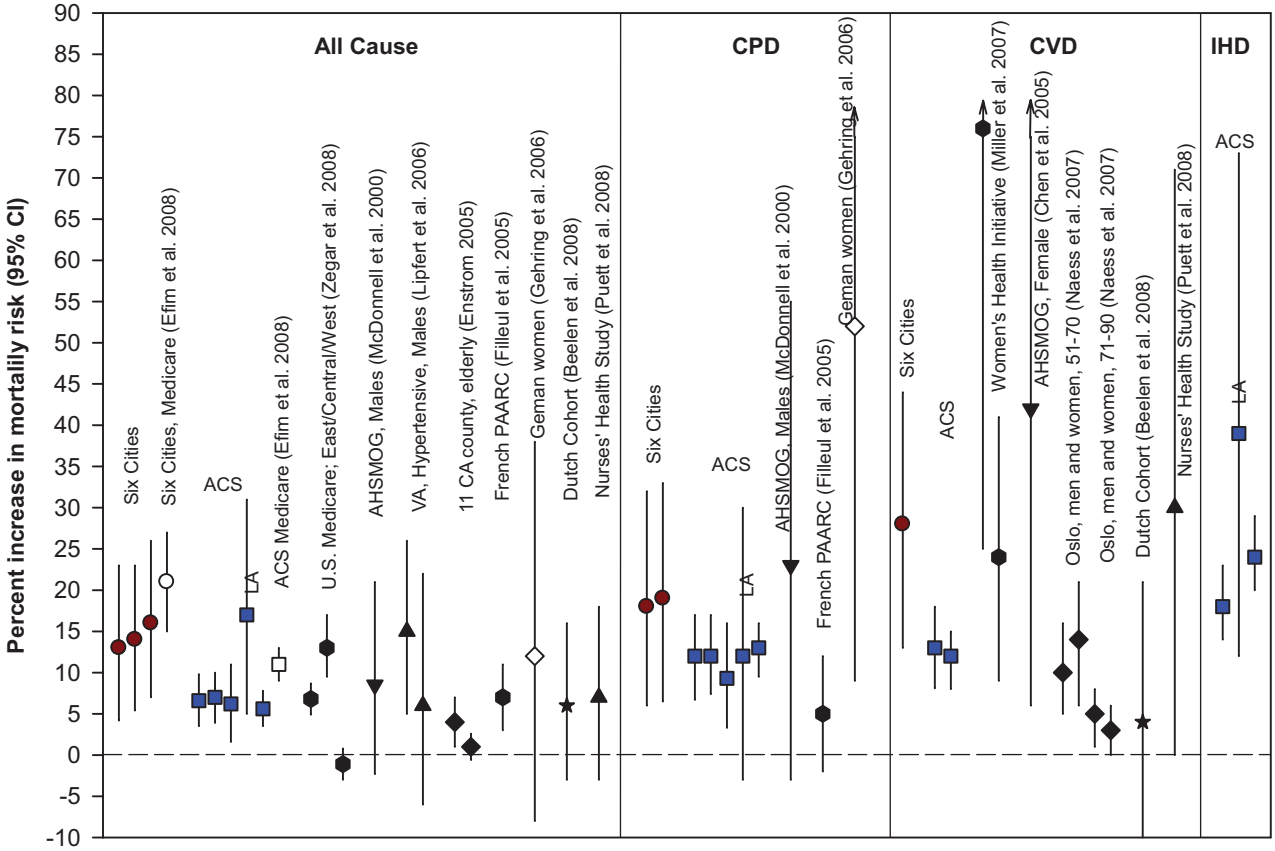

Figure 1. Risk estimates provided by several cohort studies per increment of 10 $\mu g/m^3$ in $PM_{2.5}$ or PM_{10}. CPD indicates cardiopulmonary disease; IHD, ischemic heart disease.

longer follow-up than the original studies,[74,88] fatal coronary heart disease was significantly associated with $PM_{2.5}$ among females but not males.[75] These observations along with the remarkably robust health effects in the Women's Health Initiative Observational Study and Nurses' Health Study suggest that women may be at special risk from PM exposure. The overall cohort study evidence demonstrates that a 10-$\mu g/m^3$ increase in $PM_{2.5}$ exposure is in general positively associated with excess mortality, largely driven by increases in cardiopulmonary or cardiovascular deaths (Figure 1). Independent results from the Women's Health Initiative Study,[72] the US Medicare cohorts,[71] the German women cohort,[80] and the intracity Oslo (Norway) study[81] contribute substantially to this evidence. Although the Dutch cohort,[82] AHSMOG,[74,75] French PAARC (Pollution Atmosphérique et Affections Respiratoires Chroniques [air pollution and chronic respiratory diseases]),[79] Veterans Affairs hypertensive male study,[77] and 11 CA county[78] studies observed increased mortality risks associated with higher $PM_{2.5}$ exposure that were statistically significant in some analyses, the observed health risks were less robust. A finding that is somewhat consistent across the Veterans Affairs hypertensive male study,[77] 11 CA county,[78] Oslo,[81] and US Medicare cohorts[71] is that the $PM_{2.5}$-mortality effect estimates tend to decline with longer periods of follow up or in a substantially older cohort. These studies also often observed elevated mortality risks according to alternative indicators of air pollution exposure, especially metrics of traffic-related exposure.

Evidence Summary

The overall evidence from the cohort studies demonstrates on average an approximate 10% increase in all-cause mortality per 10-$\mu g/m^3$ elevation in long-term average $PM_{2.5}$ exposure. The mortality risk specifically related to CVD appears to be elevated to a similar (or perhaps even greater) extent, ranging from 3% to 76% (Table 3). This broader estimated range in risk compared with the short-term effects observed in time series is due to several recent cohort studies[72,73] that demonstrated larger cardiovascular mortality risks (eg, >30%) than in earlier cohort observations. This may reflect superior aspects of these studies that allowed for a better characterization of the cardiovascular risk of long-term exposure, the fact that these cohorts consisted of only women, or other unclear reasons. Compared with cardiovascular mortality, there is less existing evidence to support an increase in the risk for nonfatal cardiovascular events related to $PM_{2.5}$ exposure among the existing cohort studies, because many of them did not specifically investigate nonfatal outcomes, and several of the more recent studies reported nonsignificant relationships.[72,73]

Natural Experiment and Intervention Studies

Several studies have shown improvements in health outcomes in association with exposures using well-defined natural experiments or interventions, such as abrupt reductions in air pollution[89–91] or changes over a longer period of time.[64,92]

Table 4. Comparison of Pooled Estimated of Percent Increase in Risk of Hospital Admission for CVD Estimated Across Meta-Analyses and Multicity Studies of Daily Changes in Exposure

	Primary Source	Exposure Increment	% Increase (95% CI)
Cardiac admissions, meta-analysis of 51 estimates	COMEAP[31] 2006	20 $\mu g/m^3$ PM_{10}	1.8 (1.4–1.2)
Cardiac admissions, 8 US cities	Schwartz[96] 1999	20 $\mu g/m^3$ PM_{10}	2.0 (1.5–2.5)
Cardiac admissions, 10 US cities	Zanobetti et al[97] 2000	20 $\mu g/m^3$ PM_{10}	2.6 (2.0–3.0)
Cardiac admissions, 14 US cities	Samet et al[98] 2000	20 $\mu g/m^3$ PM_{10}	2.0 (1.5–2.5)
	Schwartz et al[99] 2003		
Cardiac admissions, 8 European cities	Le Tertre et al[40] 2002	20 $\mu g/m^3$ PM_{10}	1.4 (0.8–2.0)
Cardiovascular admissions, 14 Spanish cities	Ballester et al[100] 2006	20 $\mu g/m^3$ PM_{10}	1.8 (7–3.0)
Cardiovascular admission, 8 French cities	Larrieu et al[101] 2007	20 $\mu g/m^3$ PM_{10}	1.6 (0.4–3.0)
Cardiovascular admissions, 202 US counties	Bell et al[102] 2008	20 $\mu g/m^3$ PM_{10}	0.8 (0.6–1.0)
Medicare national claims history files	Dominici et al[103] 2006	10 $\mu g/m^3$ $PM_{2.5}$	
Ischemic heart disease,			0.44 (0.02–0.86)
Cerebrovascular disease			0.81 (0.30–1.32)
Heart failure			1.28 (0.78–1.78)
Heart rhythm			0.57 (−0.01 to 1.15)

Small but statistically significant drops in mortality were associated with an 8½-month copper smelter strike that resulted in sharp reductions in sulfate PM and related air pollutants across 4 Southwest states, even after controlling for other factors.[93] Data from US Medicare enrollment files were used to estimate the association between changes in monthly mortality rates for US counties and average $PM_{2.5}$ concentrations for the previous 12 months.[94] $PM_{2.5}$-mortality associations were observed at the national scale but not the local scale, which raises concerns about possible statistical confounding due to unmeasured individual and ecological variables as a cause for any positive findings in this study. However, a recent large study found that reductions in PM air pollution exposure on a local scale (across US counties) over a 2-decade period (1980s and 1990s) were associated with increased life expectancy even after controlling for changes in socioeconomic, demographic, and proxy smoking variables.[95] Indeed, a decrease of 10 $\mu g/m^3$ in the long-term $PM_{2.5}$ concentration was related to an increase in mean life expectancy of 0.61 ± 0.20 years.

Hospitalization Rates

There are many daily time-series or case-crossover studies that have evaluated associations between cardiovascular hospitalizations and short-term changes in air pollution. Because of the great number of publications, all studies (particularly those focusing on nonparticulate air pollutants) cannot be discussed individually. Nevertheless, Table 4 presents a comparison of pooled estimates of percent increase in RR of hospital admission for general cardiac conditions across a previous meta-analysis of 51 published estimates (COMEAP [Committee on the Medical Effects of Air Pollutants]) and results from many selected multicity studies published after 2004. Several studies before 2004 are included in Table 4 only to demonstrate the consistency of effect.

Because of its comparatively large size and importance, the results of a recent analysis of Medicare files in 204 US urban counties with 11.5 million individuals older than 65 years merit discussion. Daily changes in $PM_{2.5}$ levels were associated with a variety of cardiovascular hospital admission subtypes.[103] A 10-$\mu g/m^3$ increase in $PM_{2.5}$ exposure was related to increases in hospitalizations for cerebrovascular disease by 0.81% (95% CI 0.3% to 1.32%), peripheral vascular disease by 0.86% (95% CI −0.06% to 1.79%), ischemic heart disease by 0.44% (95% CI 0.02% to 0.86%), arrhythmias by 0.57% (95% CI −0.01% to 1.15%), and heart failure by 1.28 (95% CI 0.78% to 1.78%). The most rapid effects, which occurred largely on the same day of $PM_{2.5}$ elevation, were seen for cerebrovascular, arrhythmia, and heart failure admissions. Ischemic heart disease events tended to increase to a greater extent 2 days after exposures. A consistent finding was that the cardiovascular effects of pollution were much stronger in the Northeast than in other regions. In fact, there were few significant associations in Western US regions. It was speculated that these differences reflected variations in particle composition (eg, greater sulfate in the East and nitrate components in the West) and pollution sources (eg, power generation in the East and transportation sources in the West). In a follow-up analysis by Peng et al,[104] $PM_{10-2.5}$ levels were not statistically associated with cardiovascular hospitalizations after adjustment for $PM_{2.5}$. This suggests that the smaller particles (ie, $PM_{2.5}$) are principally responsible for the cardiovascular hospitalizations attributed in prior studies to the combination of both fine and coarse particles (ie, PM_{10}). Given the differences between the size fractions, the results imply that particles and their components derived from combustion sources (ie, $PM_{2.5}$) are more harmful to the cardiovascular system than larger coarse particles. Finally, there is some evidence that gaseous pollutants may also instigate hospitalizations. Hospital admissions for cardiovascular causes, particularly ischemic heart disease, were found to rise in relation to the previous-day and same-day level of SO_2, even after adjustment for PM_{10} levels.[105]

Table 5. Comparisons of Estimated Percent Increase in Risk of Ischemic Heart Disease Events due to Concurrent or Recent Daily PM Exposure

Event/Study Area	Primary Source	Exposure Increment	% Increase (95% CI)
MI events–Boston, Mass	Peters et al[110] 2001	10 $\mu g/m^3$ $PM_{2.5}$	20 (5.4–37)
MI, 1st hospitalization–Rome, Italy	D'Ippoliti et al[112] 2003	30 $\mu g/m^3$ TSP	7.1 (1.2–13.1)
MI, emergency hospitalizations–21 US cities	Zanobetti and Schwartz[113] 2005	20 $\mu g/m^3$ PM_{10}	1.3 (0.2–2.4)
Hospital readmissions for MI, angina, dysrhythmia, or heart failure of MI survivors–5 European cities	Von Klot et al[114] 2005	20 $\mu g/m^3$ PM_{10}	4.2 (0.8–8.0)
MI events–Seattle, Wash	Sullivan et al[115] 2005	10 $\mu g/m^3$ $PM_{2.5}$	4.0 (−4.0–14.5)
MI and unstable angina events–Wasatch Front, Utah	Pope et al[13] 2006	10 $\mu g/m^3$ $PM_{2.5}$	4.8 (1.0–6.6)
Tokyo metropolitan area	Murakami et al[109] 2006	TSP >300 $\mu g/m^3$ for 1 h vs reference periods <99 $\mu g/m^3$	40 (0–97)*
Nonfatal MI, Augsburg, Germany	Peters et al[111] 2004	Exposure to traffic 1 h before MI (note: not PM but self-reported traffic exposure)	292 (222–383)
Nonfatal MI, Augsburg, Germany	Peters et al[116] 2005	Ambient UFP, $PM_{2.5}$, and PM_{10} levels	No association with UFP or $PM_{2.5}$ on same day. Positive associations with $PM_{2.5}$ levels on 2 days prior

TSP indicates total suspended particulate matter.
*Adjusted rate ratio for MI deaths.

Evidence Summary

Excess cardiovascular mortality and increased rates of hospitalizations are similarly associated with day-to-day changes in PM air pollution (Tables 2 and 4). However, significant differences between geographic regions in the risk relationships have been observed, and more investigation is required to explain this heterogeneity.

Specific Cardiovascular Events/Conditions

Ischemic Heart Disease

Among the cohort studies that provided relevant results, the ACS study found a relationship between increased risk for ischemic heart disease death and long-term exposure to elevated $PM_{2.5}$ levels (Table 3).[67,69,106] Indeed, ischemic cardiac events accounted for the largest relative (RR 1.18, 95% CI 1.14 to 1.23) and absolute risk for mortality per 10-$\mu g/m^3$ elevation in $PM_{2.5}$.[67] A survival analysis of US Medicare data for 196 000 survivors of acute MI in 21 cities showed the risk of an adverse post-MI outcome (death, subsequent MI, or first admission for congestive heart failure) was increased with higher exposure to PM_{10}.[107] Data from the Worcester Heart Attack study also found that long-term exposure to traffic-related air pollution was associated with significantly increased risk of acute MI.[108] However, in the Women's Health Initiative[72] and the Nurses' Health Study,[73] only disease categories that included fatal coronary events, but not nonfatal MI alone, were statistically elevated in relation to $PM_{2.5}$. The effect size for cardiovascular mortality was much larger and much more statistically robust than for nonfatal events such as MI in both studies.

Various time-series and case-crossover studies have also reported increased ischemic heart disease hospital admissions associated with short-term elevated concentrations of inhalable and/or fine PM air pollution.[31,40,103] In the US Medicare study, a reduction of $PM_{2.5}$ by 10 $\mu g/m^3$ was estimated to reduce ischemic heart disease admissions in 204 counties by 1523 (95% posterior interval 69 to 2976) cases per year.[103] Several studies have also found positive associations between elevated PM or traffic exposures over a period as brief as a few hours[109–111] or a few days and an elevated risk for MI (Table 5).[13,110,112–115] In general, acute increases in risk for ischemic heart disease events have been observed consistently, even as rapidly as 1 to 2 hours after exposure to elevated PM, in case-crossover analyses.[109–111] Other studies have reported an increased risk for MI shortly after exposure to traffic. Peters et al[111] reported in 691 subjects in Augsburg, Germany, a strong association (odds ratio 2.92, 95% CI 2.22 to 3.83) between onset of MI and traffic exposure within the past hour, although whether this was a result of the air pollution or a combination of other factors (eg, noise and stress) is not certain. Additional analyses did not report an association between recent UFP exposures and MI onset; however, the levels of $PM_{2.5}$ and several gaseous pollutants 2 days earlier were related to MI risk.[116] The lack of relationship between MI and UFPs may be due to the fact that the levels were measured regionally and remote from the localized source and may therefore reflect exposure misclassification. Finally, in the only study in which participating subjects had coronary angiograms performed previously, ischemic cardiac events were found to occur in relation to PM air pollution exposure solely among individuals with obstructive coronary atherosclerosis in at least 1 vessel.[13] This finding suggests the importance of patient susceptibility (eg, the presence of preexisting coronary artery disease) for PM to trigger an acute ischemic event within hours to days after exposure.

Heart Failure

In the ACS cohort study, it appeared that deaths due to arrhythmias, heart failure, and cardiac arrest (RR 1.13, 95% CI 1.05 to 1.21 per 10 $\mu g/m^3$) were also associated with

prolonged exposure to $PM_{2.5}$, although not as strongly as ischemic heart disease mortality,[67] although potential mortality misclassification on death certificates makes the actual cause of death not entirely certain in all circumstances. Heart failure rates or mortality associations were not reported in the other cohort studies.

Daily hospitalizations for heart failure have also been associated with short-term changes in PM exposure.[31] Heart failure associations with PM were observed in a large daily time-series analysis of $PM_{2.5}$ and cardiovascular and respiratory hospitalizations by use of a national database constructed from US Medicare files.[103] A 10-$\mu g/m^3$ increase in concurrent-day $PM_{2.5}$ was associated with a 1.28% (95% CI 0.78% to 1.78%) increase in heart failure admissions, the single largest cause for hospitalization in this cohort. A reduction of $PM_{2.5}$ by 10 $\mu g/m^3$ was estimated to reduce heart failure admissions in 204 counties by 3156 (95% posterior interval 1923 to 4389) cases per year.[103] Another analysis in Medicare recipients in 7 US cities found a 10-$\mu g/m^3$ increase in concurrent-day PM_{10} was associated with a 0.72% (95% CI 0.35% to 1.10%) increase in heart failure admissions.[117] Traffic-related air pollution has also been shown to be significantly associated with increased mortality risk after acute heart failure.[118] Finally, a study from Utah's Wasatch Front area explored longer lagged-exposure periods and found that a 14-day lagged cumulative moving average of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with a 13.1% (95% CI 1.3% to 26.2%) increase in heart failure admissions.[119]

Cerebrovascular Disease

Among the cohort studies that provided pertinent results, the Women's Health Initiative reported significant increases in both nonfatal stroke (hazard ratio 1.28, 95% CI 1.02 to 1.61) and fatal cerebrovascular disease (hazard ratio 1.83, 95% CI 1.11 to 3.00) per 10-$\mu g/m^3$ elevation in prolonged exposure to $PM_{2.5}$.[72] However, no significant association between stroke mortality and PM air pollution was found in the ACS study.[67]

Several studies have also reported small but statistically significant associations between short-term PM exposure and cerebrovascular disease. Daily time-series studies of stroke mortality in Seoul, Korea,[120,121] observed that elevated air pollution (including measures of PM, NO_2, CO, and O_3) was associated with increases in stroke mortality. When analyzed separately by stroke type,[121] the pollution association was associated with ischemic but not hemorrhagic stroke. Risk of stroke mortality was also associated with daily increases in PM_{10} and NO_2 in Shanghai, China.[56] A daily time-series study in Helsinki, Finland,[122] found that $PM_{2.5}$ and CO were associated with stroke mortality in the warm but not the cold seasons. Several studies have also observed increased stroke or cerebrovascular hospital admissions associated with increased exposure to PM or related pollutants.[31,38,40,46,123–125] For example, a study of hospital admissions for Medicare recipients in 9 US cities[125] found that several measures of air pollution (PM_{10}, CO, NO_2, and SO_2) 0 to 2 days before admission were associated with ischemic but not hemorrhagic stroke. Studies of ischemic stroke and transient ischemic attacks based on population-based surveillance have also been conducted in Dijon, France,[126] where O_3 exposure (but not PM_{10}) was associated with ischemic stroke, and in Corpus Christi, Tex,[127] where both $PM_{2.5}$ and O_3 were associated with ischemic strokes and transient ischemic attacks.

Peripheral Arterial and Venous Diseases

There have been only a few studies that have explored a relationship between air pollution and peripheral vascular diseases. Studies using Medicare data for 204 US counties observed nearly statistically significant positive associations between daily changes in measures of PM pollution and hospitalizations for peripheral vascular diseases.[103,104] The ACS cohort found no association between other atherosclerotic and aortic aneurysm deaths and long-term $PM_{2.5}$ exposure.[67]

Recently, a case-control study from the Lombardy region of Italy found a 70% increase in risk of deep vein thrombosis per 10-$\mu g/m^3$ elevation in long-term PM_{10} level.[128] This is the first observation that particulate air pollution can enhance coagulation and thrombosis risk in a manner that adversely affects the venous circulation in addition to the arterial cardiovascular system.

Cardiac Arrhythmias and Arrest

Several studies have observed associations between fine PM and related pollutants and cardiac arrhythmias, often based on data from implanted cardioverter-defibrillators.[129–136] However, no clear pollution-related associations were observed in studies from a relatively clean metropolitan area, Vancouver, British Columbia, Canada,[137,138] or from a relatively large study in Atlanta, Ga.[139] Similarly, pollution-related associations have been observed with cardiac arrest in Rome, Italy,[140] and Indianapolis, Ind,[141] but not in Seattle, Wash.[142,143] The mixed results may reflect different PM compositions due to different sources or variations among the methods used.

Evidence Summary

On the basis of the available epidemiological studies that have reported the associations between PM exposures with specific subsets of cardiovascular outcomes (morbidity, mortality, or hospitalizations), the existing level of overall evidence is strong for an effect of PM on ischemic heart disease, moderate (yet growing) for heart failure and ischemic stroke, and modest or mixed for peripheral vascular and cardiac arrhythmia/arrest (Table 6).

Ambient Air Pollution and Subclinical Pathophysiological Responses in Human Populations

It is likely that many subclinical physiological changes occur in individuals in response to $PM_{2.5}$ exposures that do not become overtly manifest as a cardiovascular event (eg, death or MI). The illustration of these more subtle responses bolsters the plausibility of the observable outcome associations and provides insight into the pathways whereby air

Table 6. Overall Summary of Epidemiological Evidence of the Cardiovascular Effects of $PM_{2.5}$, Traffic-Related, or Combustion-Related Air Pollution Exposure at Ambient Levels

Health Outcomes	Short-Term Exposure (Days)	Longer-Term Exposure (Months to Years)
Clinical cardiovascular end points from epidemiological studies at ambient pollution concentrations		
Cardiovascular mortality	↑ ↑ ↑	↑ ↑ ↑
Cardiovascular hospitalizations	↑ ↑ ↑	↑
Ischemic heart disease*	↑ ↑ ↑	↑ ↑ ↑
Heart failure*	↑ ↑	↑
Ischemic stroke*	↑ ↑	↑
Vascular diseases	↑	↑†
Cardiac arrhythmia/cardiac arrest	↑	↑
Subclinical cardiovascular end points and/or surrogate measures in human studies		
Surrogate markers of atherosclerosis	N/A	↑
Systemic inflammation	↑ ↑	↑
Systemic oxidative stress	↑	
Endothelial cell activation/ blood coagulation	↑ ↑	↑
Vascular/endothelial dysfunction	↑ ↑	
BP	↑ ↑	
Altered HRV	↑ ↑ ↑	↑
Cardiac ischemia	↑	
Arrhythmias	↑	

The arrows are not indicators of the relative size of the association but represent a qualitative assessment based on the consensus of the writing group of the strength of the epidemiological evidence based on the number and/or quality, as well as the consistency, of the relevant epidemiological studies.

↑ ↑ ↑ Indicates strong overall epidemiological evidence.

↑ ↑ Indicates moderate overall epidemiological evidence.

↑ Indicates some but limited or weak available epidemiological evidence.

Blank indicates lack of evidence.

N/A indicates not applicable.

*Categories include fatal and nonfatal events.

†Deep venous thrombosis only.

pollutants mediate CVDs. The "Biological Mechanisms" section discusses the hypothesized global pathways and reviews the studies related to the fundamental cellular/molecular mechanisms elucidated by controlled human and animal exposures and toxicological/basic science experiments. The following section reviews the recent evidence that ambient exposure to air pollution can mediate potentially harmful subclinical cardiovascular effects. In general, many positive associations are found (Table 6). Numerous complex interactions between variations in the characteristics, sources, and chemistry of the particles, coupled with diversity in time frames, mixtures of exposures, and degrees of individual susceptibility, likely explain some of the disparity among findings.

Systemic Inflammation

There is evidence that under some circumstances, exposure to ambient PM can be associated with elevated circulating proinflammatory biomarkers that are indicative of a systemic response after PM air pollution inhalation that is not limited to the confines of the lung. Early reports found associations with day-to-day variation in acute-phase proteins, such as C-reactive protein (CRP), fibrinogen, or white blood cell counts,[144–147] as reviewed previously.[1] Limited evidence on the association between cumulative PM exposures and fibrinogen levels and counts of platelets and white blood cells was also available.[148]

A number of more recent studies have reported positive associations with short-term ambient PM exposure and day-to-day elevations in inflammatory markers. These include increases in CRP in an elderly population[149] and individuals with coronary atherosclerosis[150]; CRP and fibrinogen in young adults[151] and elderly overweight individuals[152]; and CRP, tumor necrosis factor-α (TNF-α), and interleukin (IL)-1β in children.[153] Recent evidence has also been found for an upregulation of circulating soluble adhesion molecules (eg, intercellular adhesion molecule-1) in 92 Boston, Mass–area individuals with diabetes[154] and 57 male subjects with coronary artery disease in Germany.[150] In a larger analysis of 1003 MI survivors, also in Germany, CRP was not related to PM exposure; however, ambient particle number concentration and PM_{10} were associated with increased IL-6 and fibrinogen, respectively.[155] Short-term levels of in-vehicle $PM_{2.5}$ have also been linked to increases in CRP among healthy highway patrol troopers.[156] In a follow-up analysis, elevations in certain particulate components of traffic pollution (eg, chromium) were associated with increased white blood cell counts and increased IL-6 levels.[157] Short-term changes in ambient PM levels have also been linked to acute (1 to 3 days later) alterations in biomarkers of inflammation, oxidative stress, and platelet activation among elderly adults with coronary artery disease living in retirement communities in Los Angeles, Calif.[158,159] Pollutants associated with primary combustion (eg, elemental and black carbon, primary organic carbon) and UFPs rather than $PM_{2.5}$ appeared to be strongly associated with adverse responses in this population.

Regarding more long-term exposures,[160] a positive association between white blood cell count and estimated long-term 1-year exposure to PM_{10} was reported in the Third National Health and Nutrition Examination Survey. Among 4814 adults in Germany, small increases in annual mean $PM_{2.5}$ (3.9 $\mu g/m^3$) were associated with increases in high-sensitivity CRP by 23.9% and in fibrinogen by 3.9% among men only. Estimated long-term traffic exposure was not related to inflammatory changes in either sex.[161]

Several studies, including some with improved exposure assessment,[162] some that included analyses of large population cohorts,[163,164] and a recent evaluation of long-term annual PM_{10} levels in England,[165] have not found a relationship between particulate exposure and inflammation. It is

conceivable that differences in the magnitude or character of the inflammatory response will occur because of variations in the particulate chemistry and duration/intensity of exposures. Certain individuals may also be more susceptible. The evidence suggests that subjects with underlying cardiovascular risk factors and the metabolic syndrome may exhibit stronger associations.[152,160,166] Conversely, antiinflammatory medications such as statins may mitigate the actions of ambient particles.[152,155] All together, there is some evidence for a positive association between recent and long-term PM exposure and a systemic proinflammatory response; nevertheless, there is variation in the strength and consistency of changes among the variety of biomarkers and patient populations evaluated (Table 6).

Systemic Oxidative Stress

A state of oxidative stress refers to a condition in which levels of free radicals or reactive oxygen/nitrogen species (eg, O_2^-, H_2O_2, $ONOO^-$) are higher than normal (eg, healthy individuals in whom they are countered by homeostatic processes such as antioxidants) and thus are capable of exerting many adverse biological effects (eg, lipid/protein/deoxyribonucleic acid [DNA] oxidation, initiation of proinflammatory cascades). Although many biomarkers of differing systemic responses are available (eg, lipid or protein oxidation products), oxidative stress may occur at the local cellular/tissue level and not be directly observable by circulating markers. In addition, oxidative stress is often induced by and elicits inflammatory processes. The 2 processes are biologically linked. Therefore, human studies investigating the effect of PM on oxidative stress per se are difficult to perform. Only a few studies have directly investigated the occurrence of systemic oxidative stress in humans in relation to ambient PM exposure. Three studies of young adults conducted in Denmark demonstrated elevations in biomarkers of protein, lipid, or DNA oxidation in relation to PM exposure from traffic sources.[167–169] In a study of 76 young adults from Taipei, Taiwan,[151] the investigators found evidence of increased levels of 8-hydroxy-2′-deoxyguanosine adducts in DNA in relation to short-term elevations in ambient PM. Two studies have also demonstrated increases in plasma homocysteine, evidence that exposure to ambient PM can elevate this circulating mediator of oxidative stress.[170,171] Finally, Romieu et al[172] found that dietary supplementation with omega-3 polyunsaturated fatty acids might be capable of altering the systemic oxidative stress response (reduction in copper/zinc superoxide dismutase and glutathione) induced by air pollutants among residents living in a nursing home in Mexico City, Mexico. Because of the relatively small number of studies, more investigation is required to make firm conclusions and to understand the nature of the systemic oxidative stress response potentially induced by ambient PM (Table 6).

Thrombosis and Coagulation

Early reports indicated that increased plasma viscosity[144] and elevated concentrations of fibrinogen[146] are associated with short-term changes in ambient PM concentrations. More recent evidence was found for an upregulation of circulating von Willebrand factor in 57 male subjects with coronary artery disease in Germany[150] and 92 Boston-area individuals with diabetes.[154] Riediker[157] found that components of in-vehicle $PM_{2.5}$ were also related to increased von Willebrand factor and decreased protein C among highway patrol troopers. In the Atherosclerosis Risk in Communities study, a 12.8-$\mu g/m^3$ elevation in ambient PM_{10} was associated with a 3.9% higher von Willebrand factor level,[173] but only among those with diabetes. There was no linkage between PM_{10} exposure and fibrinogen or white blood cell levels.

Alterations in other markers that indicate changes in thrombosis, fibrinolysis, and global coagulation have also been reported. An immediate elevation in soluble CD40-ligand concentration, possibly reflecting platelet activation, recently was found to be related to ambient UFP and accumulation-mode particle ($PM_{0.1–1.0}$) levels in patients with coronary artery disease.[155] Ambient PM_{10} levels have also been associated with augmented platelet aggregation 24 to 96 hours after exposure among healthy adults.[174] In this study, there were no concomitant observable changes in thrombin generation, CRP, or fibrinogen induced by PM_{10}. Increases in plasminogen activator inhibitor-1 and fibrinogen levels have been noted in healthy subjects,[151] as well as elevated plasminogen activator inhibitor-1 in patients with coronary artery disease only,[175] in association with ambient PM levels in Taipei. Chronic indoor pollution exposure to biomass cooking in rural India has also been associated with elevated circulating markers of platelet activation.[176] Recently, Baccarelli et al[128,177] demonstrated in healthy subjects and among individuals with deep venous thrombosis living in the Lombardy region of Italy that prothrombin time was shortened in relation to recent and long-term ambient PM_{10} concentrations. Nevertheless, some studies found no effects of ambient pollution,[178] nor have significant changes been reported among all the biomarkers or subgroups of individuals investigated.[150,154,170,173] Similar to the study on systemic inflammation, the results related to thrombosis/coagulation are quite variable given the differences in study designs, patients, biomarkers evaluated, and pollutants; however, these adverse effects appear somewhat more consistent among higher-risk individuals (Table 6).

Systemic and Pulmonary Arterial BP

Several studies have reported that higher daily PM levels are related to acute increases in systemic arterial BP (approximately a 1- to 4-mm Hg increase per 10-$\mu g/m^3$ elevation in PM).[179–184] In a small study of patients with severe heart failure,[185] pulmonary artery and right ventricular diastolic BP were found to increase slightly in relation to same-day levels of PM. Chronic exposure to elevated $PM_{2.5}$ was associated with increased levels of circulating endothelin (ET)-1 and elevated mean pulmonary arterial pressure in children living in Mexico City.[186] These results may explain in part the risk for heart failure exacerbations due to PM

exposure; however, not all studies of systemic arterial BP have been positive.[187–189]

Recently, Dvonch et al[190] demonstrated significant associations between increases in systolic BP and daily elevations in $PM_{2.5}$ across 347 adults living in 3 distinct communities within metropolitan Detroit, Mich. Much larger effects were observed 2 to 5 days after higher $PM_{2.5}$ levels within a specific urban location of southwest Detroit (8.6 mm Hg systolic BP increase per 10-$\mu g/m^3$ $PM_{2.5}$) than throughout the entire region or cohort (3.2 mm Hg). This suggests that specific air pollution sources and components contribute significantly to the potential for PM exposure to raise BP. Interestingly, it was recently reported in a crossover study of 15 healthy individuals that systolic BP was significantly lower (114 versus 121 mm Hg) during a 2-hour walk in Beijing, China, while the subjects were wearing a high-efficiency particulate-filter facemask than when they were not protected.[191] Wearing the facemask was also associated with increased HRV, which suggests that the rapid BP-raising effects of particle inhalation may be mediated through the autonomic nervous system (ANS). In a similar fashion,[192] reducing exposure to particulate pollution from cooking stoves was shown to be associated with lower systolic (3.7 mm Hg, 95% CI −8.1 to 0.6 mm Hg) and diastolic (3.0 mm Hg, 95% CI −5.7 to −0.4 mm Hg) BP among Guatemalan women than among control subjects after an average of 293 days. These findings demonstrate that indoor sources of PM (eg, cooking, biomass) may have important cardiovascular health consequences and that reductions in particulate exposure are capable of lowering BP, and they suggest that chronic exposure to PM air pollution may alter long-term basal BP levels. Even given the rapid variability of BP on a short-term basis and the numerous factors involved in determining individual responses (eg, patient susceptibility, PM composition, and time frames of exposure), overall, it appears that ambient PM can adversely affect systemic hemodynamics, at least under certain circumstances (Table 6).

Vascular Function

In the first ambient PM study related to changes in vascular function, O'Neill et al[193] reported that both endothelium-dependent and -independent vasodilation were blunted in relation to air pollution levels in Boston. The largest changes occurred in association with sulfate and black carbon, suggestive of coal-burning and traffic sources, respectively. Significant adverse responses were observed within 1 day yet were still present and slightly more robust up to 6 days after exposure. Moreover, the adverse responses occurred solely among diabetic individuals and not in patients at risk for diabetes mellitus. Two other studies[184,194] also demonstrated impaired vascular function due to short-term changes in ambient PM among diabetic patients. In the study by Schneider et al,[194] endothelium-dependent vasodilation was blunted during the first day, whereas small-artery compliance was impaired 1 to 3 days after elevated ambient PM levels. Interestingly, higher concentrations of blood myeloperoxidase were related to a greater degree of endothelial dysfunction, which suggests that white blood cell sources of reactive oxygen species (ROS) may be involved.

In healthy adults, very short-term exposure to elevated levels of ambient PM from traffic sources while exercising for 30 minutes near roadways[195] and when resting by bus stops for 2 hours[196] has been related to impaired endothelium-dependent vasodilation. Daily changes in ambient gaseous pollutants (SO_2 and NO_x) in Paris, France, have also been associated with impaired endothelium-dependent vasodilation among nonsmoking men.[197] Finally, indoor particulate air pollution may also be harmful to vascular function. Bräuner and colleagues[198] recently reported that reductions in 48-hour $PM_{2.5}$ levels due to filtering of air in subjects' homes resulted in improved microvascular vascular function among elderly subjects. Nevertheless, changes in short-term ambient PM levels have not been linked with impaired conduit[197] or microvascular[178] endothelial function in all studies. Even when the few negative studies are considered, the overall evidence supports the concept that ambient PM is capable of impairing vascular function, particularly among higher-risk individuals (eg, those with diabetes) and after traffic-related exposure (Table 6).

Atherosclerosis

A few cross-sectional studies have reported an association between measures of atherosclerosis in humans and long-term exposures to ambient air pollution levels. The first study to demonstrate this relationship was an analysis of data from 798 participants in 2 clinical trials conducted in the Los Angeles area. A cross-sectional contrast in exposure of 10 $\mu g/m^3$ $PM_{2.5}$ was associated with an adjusted nonsignificant 4.2% (95% CI −0.2% to 8.9%) increase in common carotid intima-media thickness[199]; however, in certain subgroups of patients, such as women, the effect was much larger (13.8%, 95% CI 4.0% to 24.5%). In a population-based sample of 4494 subjects from Germany,[200] it was found that residential proximity to major roadways was associated with increased coronary artery calcification. A reduction in distance from a major road by half was associated with a 7% (95% CI 0.1% to 14.4%) higher coronary artery calcium score. Proximity to traffic was also related to an increased risk for peripheral artery disease in women but not men.[201] In an analysis of 3 measures of subclinical disease (carotid intima-media thickness, coronary calcium, and ankle-brachial index) among 5172 adults from the Multi-Ethnic Study of Atherosclerosis, only common carotid intima-media thickness was modestly (yet significantly) associated with 20-year exposure to $PM_{2.5}$.[202] In a related study from the same cohort, abdominal aortic calcium was associated with long-term $PM_{2.5}$ exposure, especially for residentially stable participants who resided near a $PM_{2.5}$ monitor.[203] Although it appears that long-term exposure to higher levels of ambient PM might accelerate the progression of atherosclerosis, more investigations are needed (Table 6).

Heart Rate Variability

Numerous studies have continued to explore associations between daily changes in PM air pollution exposure and alterations (typically reductions) in HRV metrics, putative markers of cardiac autonomic balance.[129,149,156,204–242] Recent observations in the Normative Aging Study cohort have shown strong effect modification of the PM-HRV relationship by obesity and genes that modulate endogenous oxidative stress or xenobiotic metabolism, such as glutathione S-transferase M1, methylenetetrahydrofolate reductase, and the hemochromatosis gene.[207,243,244] Additional findings suggest protective effects of statins, dietary antioxidants, and B vitamins, as well as omega-3 polyunsaturated fatty acids.[205,207,215,243,244] These results suggest that pathways that reduce endogenous oxidative stress have a protective effect that mitigates reductions in HRV due to ambient PM exposure.

However, the overall results are not entirely consistent. Some studies have reported increases in HRV mediated by PM, specifically among younger healthy people and patients with chronic obstructive lung disease.[156,208,216] Nevertheless, the general pattern suggests that PM exposure is associated with increased heart rate and reductions in most indices of HRV among older or susceptible individuals, such as those with obesity and the metabolic syndrome. Typically, time-domain measures (eg, standard deviation of normal RR intervals) and total power are reduced within hours after exposure. Most, but not all, pertinent studies have also found that the largest reduction in power is within the high-frequency domain. In sum, these observations provide some evidence that ambient PM air pollution exposure rapidly reduces HRV, a surrogate marker for a worse cardiovascular prognosis (Table 6). Although studies corroborating changes in autonomic activity by other methods (eg, microneurography or norepinephrine kinetics) have not been performed, the HRV findings are perhaps reflective of the instigation of a generalized cardiovascular autonomic imbalance due to relatively greater parasympathetic than sympathetic nervous system withdrawal.

Cardiac Ischemia and Repolarization Abnormalities

There has been limited direct evidence for the actual induction of cardiac ischemia or repolarization abnormalities in the electrocardiogram (ECG) by exposure to ambient levels of PM.[223,245] Recent follow-up analyses from the initial ULTRA study (Exposure and Risk Assessment for Fine and Ultrafine Particles in Ambient Air)[245] suggested that traffic-related combustion pollutants were most strongly related to the promotion of ST-segment depression among elderly nonsmokers during exercise stress testing.[246] Moreover, even very acute $PM_{2.5}$ exposure within the past 1 or 4 hours has been associated with cardiac ischemia during exercise.[247] New findings support these associations in elderly subjects[248] and in patients with coronary artery disease in Boston.[249] In the latter study, traffic-related PM was most strongly related to the incidence of ST-segment depression during 24-hour Holter monitoring, and the risk for ischemia was greatest within the first month after a cardiac event among patients with diabetes. Overall, there is a modest level of evidence that PM exposure can promote cardiac ischemia in susceptible individuals (Table 6).

Epigenetic Changes

There have been relatively few studies examining gene–air pollution exposure interactions, and most have done so while investigating a small number of loci for genetic polymorphisms. Although some studies have suggested greater air pollution susceptibility with one or another genomic polymorphism,[207,243,244] few have evaluated the potential for epigenetic changes after exposures. Reduced levels of DNA methylation have been linked to aging, oxidative stress, and CVD. Recently, Baccarelli et al[250] have shown among 718 elderly participants in the Normative Aging Study that short-term exposures (over 1 to 7 days) to $PM_{2.5}$ and black carbon are associated with decreased "global" DNA methylation in long interspersed nucleotide elements. It was posited that oxidative stress from air pollution exposure could have interfered with the capacity for methyltransferases to interact with DNA or altered the expression of genes involved in the methylation process. This observed effect of pollution exposure was analogous to changes seen with 3.4 years of aging in the cohort. Additional findings among workers in a furnace steel plant support these observations.[251] Nevertheless, the mechanisms involved and the cardiovascular implications of these preliminary, although provocative, epigenetic changes require more investigation.

Traditional Cardiovascular Risk Factors

In addition to the fact that individuals with traditional risk factors are likely to be at higher risk for cardiovascular events due to PM exposure, air pollutants may also promote the development of these risk factors over a prolonged period of time. Few published studies have investigated this possibility. A report from the Multi-Ethnic Study of Atherosclerosis has demonstrated that residential proximity to major roadways was associated with a higher left ventricular mass index as measured by cardiac magnetic resonance imaging.[252] The degree of increase was analogous to a 5.6-mm Hg increase in systolic BP among the study participants. This suggests that traffic-related exposures may have increased left ventricular mass by chronically elevating systemic arterial BP, a common cause of left ventricular hypertrophy. However, other mechanisms cannot be excluded, such as systemic inflammation and oxidative stress, which could potentially activate neurohormonal pathways (eg, ANS imbalance, renin-angiotensin system) that could directly mediate such a finding. In addition, a recent study of adults older than 30 years of age (n=132 224) participating in the National Health Interview Survey reported a significant association between self-reported hypertension and estimated annual $PM_{2.5}$ exposure using US EPA monitoring data.[253] A 10-$\mu g/m^3$ elevation in $PM_{2.5}$ was associated with an

adjusted odds ratio of 1.05 (CI 1.00 to 1.10) for the presence of hypertension. The increase in risk was found only among non-Hispanic whites. These studies provide some initial evidence that longer-term PM exposures may augment the risk for developing chronically elevated BP levels or even overt hypertension.

Brook et al[254] have also demonstrated a novel relationship between a metric of long-term traffic exposure (NO_2 level by residence) and the odds of having the diagnosis of diabetes mellitus among patients in 2 respiratory clinics in Ontario, Canada. In women only, the odds ratio of diabetes was 1.04 (95% CI 1.00 to 1.08) for each increase of 1 parts per billion (ppb) of NO_2. Across the interquartile range (4 ppb NO_2), exposures were associated with nearly a 17% increase in odds for diabetes mellitus. The first biological support for this finding comes from a study in Iran that demonstrated that the previous 7-day-long exposure to PM_{10} was independently associated with worse metabolic insulin sensitivity among 374 children 10 to 18 years of age.[255] These findings suggest that the systemic proinflammatory and oxidative responses due to long-term PM air pollution exposure could potentially increase the risk for developing clinically important aspects of the metabolic syndrome, such as hypertension and diabetes mellitus. Further studies in this regard are warranted.

Evidence Summary

Table 6 provides a consensus qualitative synopsis based on the expert opinions of the writing group members of the overall level of existing support, linking each surrogate or intermediate cardiovascular outcome with exposures to PM at ambient concentrations, based solely on the database of observational studies.

Additional Epidemiological Findings and Areas of Continued Research

Responsible Sources and Pollution Constituents

Although PM concentration (mass per cubic meter) has been associated with cardiovascular events in numerous studies, the specific particulate constituents and the sources responsible remain less clear. Despite the fact that it is a difficult undertaking, several epidemiological studies have attempted to identify the culprit components within the PM mixtures. With regard to PM-associated inorganic ions (nitrate and sulfate), it has been suggested that the overall toxicological data do not clearly implicate these compounds as responsible for mediating the cardiovascular health effects of $PM_{2.5}$.[256] Nevertheless, sulfate particles have been associated with cardiopulmonary mortality in the ACS and Harvard Six Cities studies.[62,68] A recent time-series analysis among 25 US cities found that cardiovascular risk was increased when PM mass contained a higher proportion of sulfate, as well as some metals (aluminum, arsenic, silicon, and nickel).[257] It is possible that these positive findings represent sulfate serving as a marker for an effect mediated by a toxic PM mixture derived from commonly associated sources (eg, coal combustion). Nevertheless, a direct role for particle sulfate in causing cardiovascular events cannot be excluded entirely.[256]

In California, short-term exposures to several different PM constituents that likely reflect combustion-derived particulates, including organic and elemental carbon and nitrates, were most strongly associated with higher cardiovascular mortality.[258] Certain metals (zinc, titanium, potassium, and iron) and sulfate levels in the winter months were also positively related. Similarly, ambient levels of organic and elemental carbon have been most strongly linked among PM constituents with hospitalizations for CVDs in multipollutant models in a study among 119 US cities.[259] Finally, $PM_{2.5}$ composed of higher levels of elemental carbon, along with the metals nickel and vanadium,[48] has also been linked with greater risks for cardiovascular hospitalizations.[260] These results support that the chemistry or composition of the $PM_{2.5}$ (eg, organic/elemental carbon and certain metals) along with the responsible source from which these mixtures are derived (eg, fresh combustion, traffic) may play important roles in determining the risk for cardiovascular events. However, the extent to which these constituents mediate specific responses, alone or together, and their importance beyond the concentration of $PM_{2.5}$ mass alone represent an area of active research that requires more investigation to reach firm conclusions.

Many experiments have demonstrated the especially toxic properties and strong oxidizing potential of the smallest particle sizes (eg, UFP) and of the specific chemical species typically rich within this size fraction (eg, transition metals, organic compounds, and semiquinones).[261] Although some epidemiological evidence suggests that exposure to ultrafine compounds[17] may be associated with higher cardiovascular risk (eg, an elevation of UFP count by 9748/cm^3 has been associated with an increase in cardiovascular mortality of approximately 3% within 4 days in Erfurt, Germany[262]) and adverse responses,[158,159] there have been few such studies because they are challenging to conduct, for numerous reasons. Moreover, there are few UFP monitors, and the levels measured at regional sites may not accurately reflect an individual person's exposure because of marked spatial heterogeneity, because the concentrations are dominated by local point sources of fresh combustion (eg, roadways). This could help explain some of the previously negative study findings.[116]

Similarly, coarse particulates between 0.25 and 1.0 μm in diameter may affect the cardiovascular system,[221,264,265] and although the available data related to hard events and cardiovascular mortality have suggested a relationship,[265,266] recent findings have been less consistent.[104] In the most recent time-series analysis of 112 US cities, coarse PM was independently associated with elevated all-cause, stroke, and pulmonary, but not cardiovascular, mortality after controlling for $PM_{2.5}$.[43] Coarse PM was also not associated with either fatal or nonfatal cardiovascular events after controlling for $PM_{2.5}$ levels in the Nurses' Health Study[267] or the Women's Health Initiative cohort analyses.[72] Additional research is required to establish whether there are independent health effects of the other

particulate size fractions beyond those posed by fine particles. On the other hand, $PM_{2.5}$ mass concentration is the metric most consistently associated with cardiovascular morbidity and mortality. It remains to be determined whether this reflects limitations of available data, the long-lived and regionally homogenous atmospheric nature of $PM_{2.5}$, that few studies have investigated the independent effects of the other sizes, difficulties in performing epidemiology studies with adequate UFP exposure estimates, or that specific constituents within the fine PM fraction (or another unidentified agent correlated with that fraction) are actually responsible for causing cardiovascular events. Although particles <0.1 μm (ie, UFPs) do make up a small fraction of $PM_{2.5}$ mass, the correlation between UFP particle number and total $PM_{2.5}$ mass concentration is often weak. Because of their minute size, UFPs make up only a small portion of the total $PM_{2.5}$ mass, even though they represent the largest actual number of particles within fine PM. They also have the highest surface area and a differing surface chemistry. Therefore, changes in the underlying UFP concentration do not likely account for or explain the linkages between $PM_{2.5}$ mass concentration and cardiovascular events observed in large multicity studies. The overall epidemiological evidence thus indicates that fine PM poses an independent cardiovascular risk and that any putative effects of these other size fractions cannot fully explain the observed $PM_{2.5}$–cardiovascular morbidity/mortality relationship.

On the other hand, there is mounting evidence for a distinctive role played by motor vehicle traffic–related exposures in elevating cardiovascular risk.[108,111,268,269] Lipfert et al[76,77] interpreted the results of their analysis of the Veterans Affairs hypertensive male cohort as suggesting that traffic density was a more "significant and robust predictor of survival in this cohort" than $PM_{2.5}$. Analyses of the Oslo,[81] Dutch,[82] AHSMOG,[74,75,88] French PAARC,[79] and German women cohorts[80] and related studies from areas in the United Kingdom,[270] Canada,[271] Norway,[272] and Rome[273] found that measures that often indicate traffic-related exposure (NO_2, NO_x, traffic density, and living near major roads) were also associated with increased mortality. Long-term 5-year average traffic-generated air pollution exposure has been associated with an increased risk of fatal MI (odds ratio 1.23, 95% CI 1.15 to 1.32 per 31-$\mu g/m^3$ increase in NO_2) but not nonfatal MI in Stockholm County, Sweden.[274] The results mirror the results of several cohort studies[72,73] that found that air pollution exposures appeared to be more strongly linked with cardiovascular mortality than nonfatal events. Recently, an analysis from a cohort in the Netherlands demonstrated that several metrics of traffic-related air pollution exposure remained significantly associated with increased risk for cardiovascular events even after adjustment for higher levels of traffic noise.[275]

The effect of long-term traffic-related exposure on incidence of fatal and nonfatal coronary heart disease was recently assessed after adjustment for background air pollutants and cardiovascular risk factors in 13 309 adults in the Atherosclerosis Risk in Communities study.[276] Interestingly, background chronic ambient $PM_{2.5}$ concentrations were not related to the interpolated traffic exposure levels or to heart disease outcomes, which supports the highly localized nature of traffic sources of exposure. After 13 years of follow-up in 4 US communities, individuals residing within the highest quartile of traffic density had a relative risk of 1.32 (95% CI 1.06 to 1.65) for fatal and nonfatal heart disease events. Despite multiple statistical adjustments, the investigators also acknowledged the possibility for residual confounding as a potential source of bias. The specific traffic-related pollution components, such as UFP or gaseous-phase chemicals (eg, SVOCs), that are responsible for the positive findings among these studies remain unknown. The close proximity to roadways within these epidemiological studies (eg, 400 m) required to observe an association with elevated cardiovascular risk, however, matches the atmospheric fate of these shorter-lived pollutants. The findings may thus suggest the existence of cardiovascular health effects mediated by specific air pollutants rather than $PM_{2.5}$ per se. There is room for improvement in assessment of traffic exposures in epidemiological research, and better approaches are now being incorporated into research projects, such as accounting for associated factors (eg, noise or spatial autocorrelation with socioeconomic status).[275,277]

Geographic differences in cardiovascular risk due to PM have also been observed across US regions, with more consistent or stronger effects observed in Eastern versus Western states.[71,103,257] Differences between North American and European cities have also been reported.[61] PM exposures are typically, but not always,[258] associated with larger effects during warmer months (spring through fall) than in the winter.[45,103,257] Variations in pollution characteristics (eg, sulfate), time spent outdoors, air conditioning usage and particle penetration indoors, ambient temperature and meteorology, and mobile (eg, diesel) or stationary (eg, coal combustion) sources of exposure may help explain these differences. Finally, variations in the cardiovascular risk posed by PM may also occur because of heterogeneity in the metric of exposure, such as personal versus background regional,[25] indoor versus outdoor sources, and differences in intracity versus intercity gradients.[69] A better understanding of the responsible constituents and sources is important and could potentially lead to more targeted and effective regulations. On the other hand, finding continued evidence that the adverse cardiovascular health effects cannot be linked conclusively to a particular or specific chemical species or source of pollution but rather that they occur in response to a variety of exposure types or mixtures would support the present-day policy of reducing exposure to overall fine particulate mass to achieve public health benefits.

Time Course and Concentration-Response Relationships

Many studies have demonstrated that PM air pollution exposure does not simply advance the mortality by a few days of critically ill individuals who would have otherwise died (eg, mortality displacement or "harvesting").[278,279] There also appears to be a monotonic (eg, linear or log-linear) concentration-response relationship between $PM_{2.5}$ and mor-

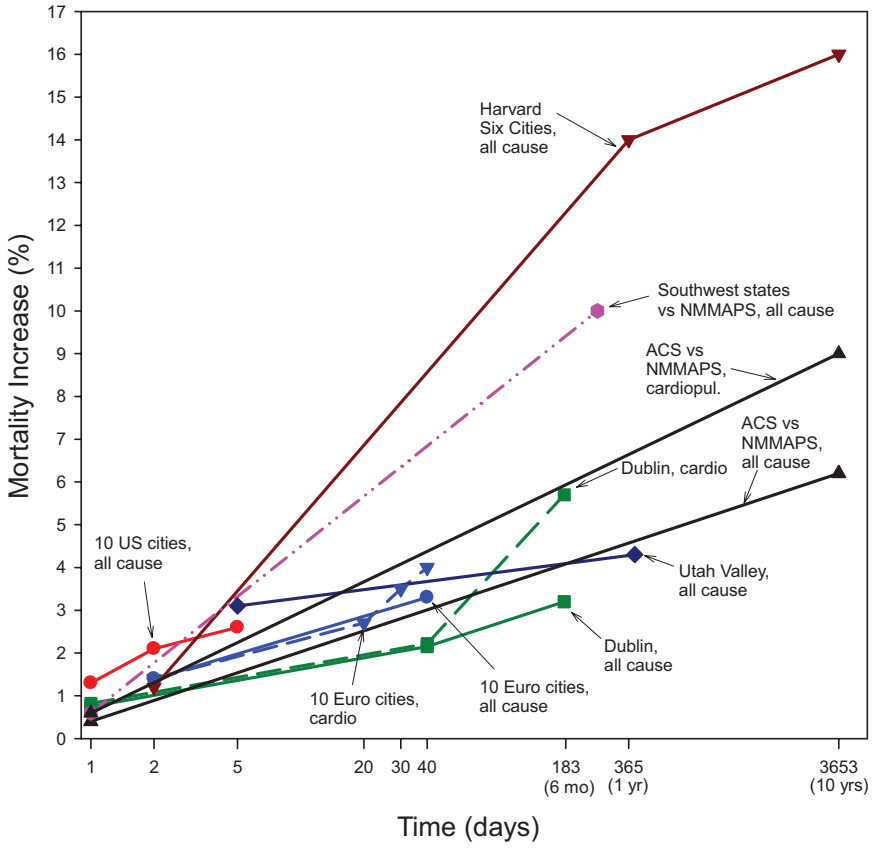

Figure 2. Comparison of estimates of percent change in mortality risk associated with an increment of 10 $\mu g/m^3$ in $PM_{2.5}$ or 20 $\mu g/m^3$ of PM_{10} or British Smoke (BS) for different time scales of exposure (log scale of approximate number of days, updated and adapted from Pope[281a]). Euro indicates European; cardio, cardiovascular disease; and cardiopul, cardiopulmonary.

tality risk observed in cohort studies that extends below present-day regulations of 15 $\mu g/m^3$ for mean annual levels, without a discernable "safe" threshold.[67,70,84] Cardiovascular risk due to particle exposure was also shown to extend below 15 $\mu g/m^3$ in the recent analysis of the Women's Health Initiative Observational Study.[72] This monotonic association supports the idea that any reduction in particulate pollution will translate into health benefits within a population of people, each with their own individual level of susceptibility. It also suggests that a larger decrease in $PM_{2.5}$ exposures will produce a greater reduction in mortality. Finally, a recent analysis of the literature provided important new insights into the nature of the PM exposure-response relationship.[280] The risk for cardiovascular mortality was shown to increase in a linear fashion across a logarithmically increasing dosage of inhaled fine-particle levels that ranged from ambient PM air pollution (≈0.2 mg/d), through secondhand smoke (≈1 mg/d), to active smoking (200 mg/d). This means that the exposure response is extremely steep at very low PM levels (ie, ambient air pollution) and flattens out at higher concentrations (ie, active smoking). This may help explain the seemingly incongruent and comparatively very high degree of cardiovascular risk posed by the much lower levels of PM exposure from ambient pollution and secondhand smoke versus the much higher doses due to active smoking. Thus, the cardiovascular system may be extremely sensitive to very low levels of PM inhalation as encountered with ambient pollution.

At present, the underlying nature and full scope of the temporal-risk relationship posed by longer-term PM exposures remain uncertain.[2,281] The writing group members did concur that the available epidemiological studies demonstrate larger cardiovascular risks posed by more prolonged exposures to higher PM levels than observed over only a few days (Figure 2). Cohort studies using Cox regression survival analyses (over months to years) are capable of evaluating a more complete portion of the temporal-risk relationship than time-series analyses over only a few days that use Poisson regression. However, given the lack of complete information, no conclusions could be drawn on the full magnitude of the augmented risk posed by chronic exposures, the time window (a few months versus decades) required to exhibit this enhanced risk, the underlying biological causes, the extent to which statistical differences between study types explain the variations in risk, and whether clinically relevant chronic CVDs are precipitated by chronic exposures. Some writing group members believe it is important to differentiate as 2 distinct issues the potentially greater effect of long-term exposures on increasing the risk for acute events (eg, cardiovascular mortality) compared with the putative effect on initiating or accelerating the development of chronic CVD processes per se (eg, coronary atherosclerosis). As such, it is possible that the greater risks observed in cohort studies could be capturing the fact that repetitive exposures over months or years augment the risk for sudden cardiovascular events in susceptible people, without actually worsening an underlying "chronic" disease process.

On the one hand, the available studies demonstrate that the majority of the larger risk-effect sizes posed by longer-term versus short-term exposures appear to be manifested within

only 1 to 2 years of follow-up. Extending the duration of follow-up increases cardiovascular risk, but to a progressively smaller degree over time (Figure 2). The discrepancy in the effect sizes among study types (eg, cohort versus time-series studies) could also reflect differences in statistical methodologies or population susceptibilities.[282–284] Recent attempts to investigate this matter[64,84] suggest that the risk for acute events associated with chronic exposures may be reasonably well estimated by only the most proximal 1 to 2 years of PM levels. The most recent time frames of exposure also explain a substantial portion of the excess cardiovascular risk observed in several cohort studies.[70,72,73,83] These findings bolster the argument that relatively rapid and pliable (and potentially reversible) biological responses, such as the instigation of plaque instability or the enhanced thrombotic potential caused by PM-mediated inflammation or endothelial dysfunction (which can occur and abate over only a few weeks to months), could explain the biology responsible for this greater relative risk.

On the other hand, cogent alternative arguments can be made to explain the differences in relative risk between the cohort and time-series studies. The likely high correlation of a recent year's exposure levels with exposures over many years, as well as the uniform rank ordering of exposure severity over time among cities, can explain why only a short period of PM exposure assessment is required to understand the risk of longer-term exposures. In addition, no studies have evaluated the potential risks of exposure over decades or a lifetime. PM augments the ability of traditional risk factors to accelerate the development of atherosclerosis in experimental settings. As such, it is also plausible that long-term exposures may enhance cardiovascular risk to an even greater extent by increasing an individual's susceptibility for future cardiovascular events or acute exposures. In addition, the full extent of this possibility may not be illustrated by the limited follow-up period (4 to 5 years) of the majority of cohort studies. The writing group thus agreed that this important issue requires more investigation.

It is also possible that these 2 explanations are not mutually exclusive. Furthermore, it cannot be concluded from available information that a long period of time is required for reductions in PM levels to translate into a decrease in cardiovascular risk. On the contrary, reductions in secondhand smoke[285] and PM air pollution levels[64,84,90,95] appear to produce fairly rapid decreases in cardiovascular event rates, within a few months to years.[284] At present, the available data do not allow for firm conclusions regarding the underlying biology and the full extent of the potentially nonuniform PM exposure–to–cardiovascular risk temporal relationship.

Susceptibility to Air Pollution Exposure

Susceptibility refers to a heightened risk for a particular cardiovascular end point or event to occur compared with the general population at the same concentration of PM exposure. Typically, this is indicative of an underlying medical condition (eg, diabetes) or personal characteristic (eg, old age) that causes this enhanced risk. This is in contrast to the term "vulnerability," which refers to a population of individuals at greater risk for more frequent or high levels of exposures.

Earlier studies reviewed in the first AHA scientific statement[1] suggested that susceptible populations include the elderly; individuals with diabetes; patients with preexisting coronary heart disease, chronic lung disease, or heart failure; and individuals with low education or socioeconomic status. In the ACS study, current and previous smokers appeared to be at the same or greater degree of risk.[67] Among more recent studies, the Women's Health Initiative also reported positive findings among active smokers and an elevated risk for cardiovascular mortality induced by $PM_{2.5}$.[72] Conversely, current smokers were found to be at no increased risk for cardiovascular mortality in response to $PM_{2.5}$ exposure in the Nurses' Health Study.[73] Thus, the effect modification of smoking status requires more investigation. The APHENA study of European and North American cities recently confirmed that elderly and unemployed individuals are at higher risk of short-term PM exposure.[61] In a multicity time-series study in Asia, women, the elderly, and individuals with lower education and socioeconomic status were also shown to be at elevated risk.[286] A few additional studies have reported some evidence of susceptibility to short-term PM exposures among older individuals, people with diabetes, and those with a lower level of education.[287–289] Finally, a recent study illustrated that present-day levels of $PM_{2.5}$ likely increase the risk for a cardiac event within a few days of exposure principally (or even solely) among individuals with preexisting significant coronary artery disease, even if they are seemingly healthy (eg, without anginal symptoms). Patients without obstructive lesions on heart catheterization were not at any risk for $PM_{2.5}$-induced myocardial events over the short term.[13] This is not surprising, because most acute cardiovascular events occur among individuals with underlying vulnerable substrate (eg, unstable plaques) and not in individuals with normal coronary arteries.

Obesity has been newly recognized as a possible susceptibility factor. Two cohort studies have shown that a greater body mass index enhances the susceptibility for PM-induced cardiovascular mortality, at least among women.[72,73] Although individuals with diabetes showed a trend toward greater risk in the Women's Health Initiative,[72] hypertension, high cholesterol, smoking, elderly age, education, and income did not alter the risk association. Overall, there appears to be little effect modification by race, hypercholesterolemia, or BP among the studies. Finally, sex may also be a risk-effect modifier. The particularly robust risk estimates of the 2 cohort studies that included only women,[72,73] the fact that PM increased cardiovascular risk in female but not male participants of the AHSMOG study,[75] and the multicity time-series findings in Asia[286] suggest that women may be at greater risk for cardiovascular mortality related to PM. Further studies are needed to clarify whether obese individuals and women are indeed susceptible populations.

Biological Mechanisms

There has been substantial improvement in our understanding of the biological mechanisms involved in PM-mediated

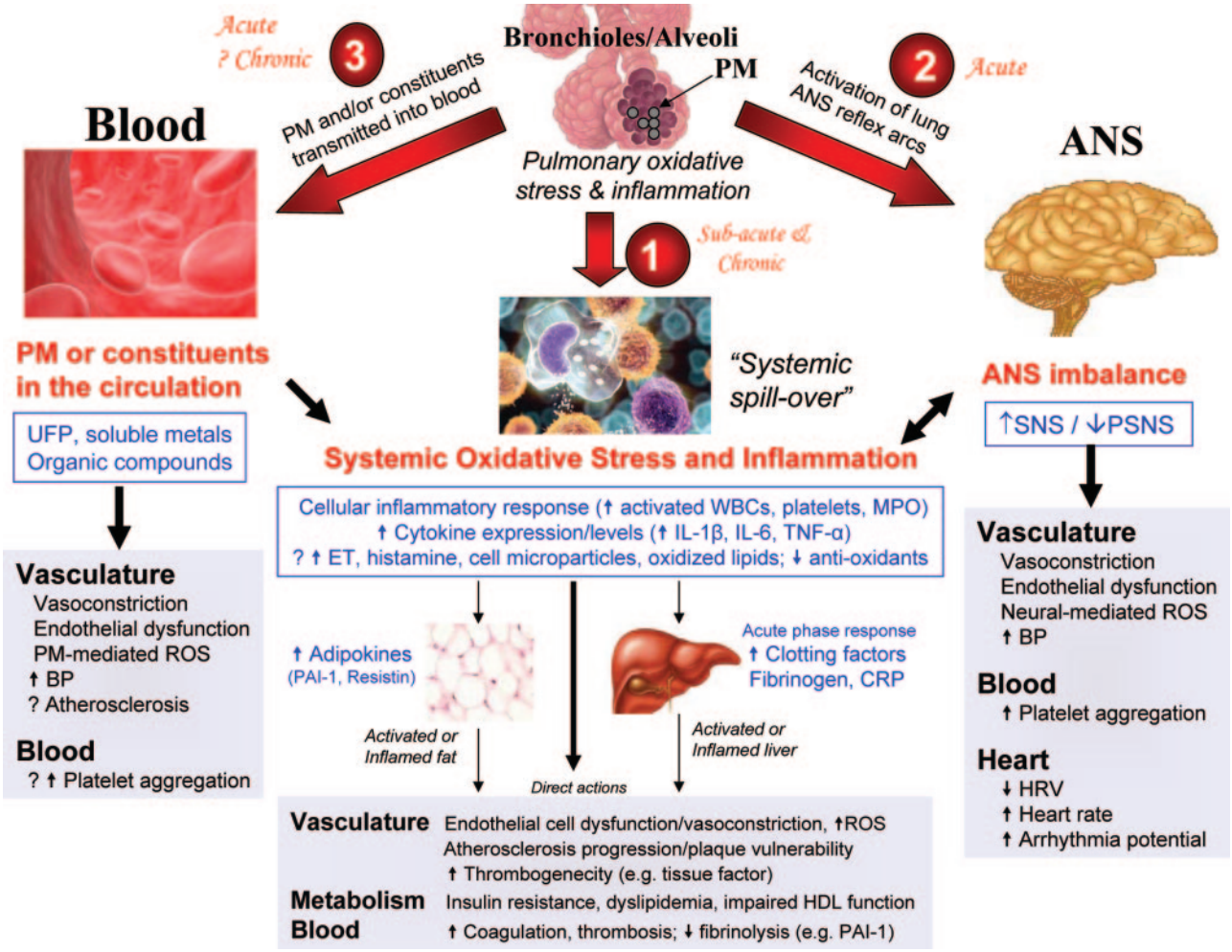

Figure 3. Biological pathways linking PM exposure with CVDs. The 3 generalized intermediary pathways and the subsequent specific biological responses that could be capable of instigating cardiovascular events are shown. MPO indicates myeloperoxidase; PAI, plasminogen activator inhibitor; PSNS, parasympathetic nervous system; SNS, sympathetic nervous system; and WBCs, white blood cells. A question mark (?) indicates a pathway/mechanism with weak or mixed evidence or a mechanism of likely yet primarily theoretical existence based on the literature.

cardiovascular effects. Studies before 2004 were reviewed previously,[1] and only some are again discussed here for contextual background. A number of new experiments have demonstrated very rapid effects of air pollution, such as vascular dysfunction, which argues for the existence of pathways that convey signals systemically within hours of PM inhalation. On the other hand, there is also support for chronic biological effects, such as the promotion of atherosclerosis. At the molecular level, persuasive evidence supports an integral role for ROS-dependent pathways at multiple stages, such as in the instigation of pulmonary oxidative stress, systemic proinflammatory responses, vascular dysfunction, and atherosclerosis. In sum, new studies continue to support the idea that inhalation of PM can instigate extrapulmonary effects on the cardiovascular system by 3 general "intermediary" pathways. These include pathway 1, the release of proinflammatory mediators (eg, cytokines, activated immune cells, or platelets) or vasculoactive molecules (eg, ET, possibly histamine, or microparticles) from lung-based cells; pathway 2, perturbation of systemic ANS balance or heart rhythm by particle interactions with lung receptors or nerves; and pathway 3, potentially the translocation of PM (ie, UFPs) or particle constituents (organic compounds, metals) into the systemic circulation (Figure 3).

Exposure Considerations

Animal and human exposure studies are discussed separately and apart from the effect of ambient PM because their methodologies and clinical relevancies vary widely. Controlled exposure studies involve exposing a subject to various size fractions of PM within a chamber connected to ambient air (concentrated or nonconcentrated) or a source of aerosolized particles. Virtual impactor systems that deliver concentrated ambient particles (CAPs) from "real-world" ambient air are a commonly used approach for mimicking exposures to higher levels of ambient particles without requiring invasive methods or the generation of artificial particles.[3] Both a strength and limitation, however, is that CAPs can vary considerably from day to day in composition. Additionally, only certain particle size ranges are typically concentrated (eg, PM from 0.1 to 2.5 μm in the fine-CAP system), whereas

ambient air contains a mixture of particle sizes, volatile organics, and gases that are not concentrated (and can be lowered). Potential interactions between PM and gaseous copollutants on health end points are therefore excluded, unless the latter are reintroduced in an artificial fashion. Other methods of controlled-inhalation exposures include diesel engine exhaust (diluted and aged mixtures of high numbers of fresh combustion UFPs with vapor-phase components), roadside aerosols, and wood-burning sources. Regarding animal exposures, intratracheal instillation methods may sometimes be required because of the limited availability of inhalation exposure systems. Unfortunately, particle size and surface characteristics—mostly retained in inhalation systems with fresh sources of pollution and which may be important in influencing biological effects—are likely significantly altered in instillation systems or by methods that use previously collected particulate. However, the use of carefully modeled exposures (eg, deposition calculation) and the recognition that areas of "hot spots" containing markedly higher PM levels within the lung may occur even during normal inhalation make the results of these experiments potentially relevant.[2] Further detailed discussions of exposure considerations are reviewed elsewhere.[290]

The protocol details vary considerably among the studies. Many aspects of exposure, including the duration, concentration, PM size ranges and composition, and gaseous copollutants, are important to consider. A wide variety of outcomes may be anticipated depending on the biological pathways evoked by differing exposures. Moreover, there are multiple determinants of the subsequent physiological responses, including the time frames of investigation, preexisting susceptibility, animal models, and the details of the outcomes investigated. All of these factors may explain some of the heterogeneity in the reported study results and must be taken into consideration when interpreting the findings.

Animal Exposure and Toxicological Studies

Studies that investigate the effects of exposure on susceptible animals (eg, those with preexisting cardiovascular or metabolic abnormalities) may be preferable in many circumstances because of the increasing recognition that the pathways underlying the biological effects of PM overlap (ie, modify and/or enhance) those of conventional cardiovascular risk factors. Such factors (eg, hypertension or atherosclerosis) may also be necessary or at least responsible for the evocation of a more readily observable or robust response. For example, in the context of systemic oxidative stress or inflammation, the cellular machinery for the generation of excess ROS and proinflammatory responses (eg, adhesion molecule and cytokine expression) is already primed or operational in susceptible animals.

Pulmonary Oxidative Stress and Inflammation

The molecular events responsible for triggering pulmonary oxidative stress and inflammation, along with the interactions between lung and immune cells, the inhaled PM, and the protective secretions (eg, surfactant, proteins, and antioxidants), are highly complex,[4–6] as reviewed in detail elsewhere.[290a,290b,414] In brief, size, charge, solubility, aggregation, ROS-producing potential, and chemistry play roles in determining the responses. These include the particle fate (eg, lung clearance versus retention rates), the nature of the PM-cell interactions (eg, immune versus lung cell uptake, host cell responses, and intracellular sequestration/location), and the dose (likely typically a small percentage of inhaled PM) and pathways of potential systemic transmission of PM or its constituents, such as in the circulation [free, intracellular within circulating cells, (lipo)protein-bound] or via lymphatic spread.[4,5,290a,290b] Because of their nano-scale size, UFPs may directly enter multiple lung cell types via nonphagocytic pathways and adversely affect organelles, such as mitochondria.[6,290a,290b] Larger unopsonized fine particles are more typically taken up by phagocytes through interactions with innate immunity receptors such as MARCO (macrophage receptor with collagenous structure) or other scavenger receptors.[5,290a,290b] This may in fact be a protective mechanism that sometimes prevents harmful lung inflammation. Certain particle compounds may directly generate ROS in vivo because of their surface chemistry (eg, metals, organic compounds, and semiquinones) or after bioactivation by cytochrome P450 systems (eg, polycyclic aromatic hydrocarbon conversion to quinones).[6,290a,290b] A particle surface or anions present on otherwise more inert particles may disrupt iron homeostasis in the lung and thereby also generate ROS via Fenton reactions.[291] Other PM constituents may do so indirectly by the upregulation of endogenous cellular sources (eg, nicotinamide adenine dinucleotide phosphate [NADPH]) oxidase)[292,293] or by perturbing organelle function (eg, mitochondria) by taken-up PM components.[261] Particle stimulation of irritant and afferent ANS fibers may also play a role in local and systemic oxidative stress formation.[294] Given the rich antioxidant defenses in the lung fluid, secondarily generated oxidization products of endogenous molecules (eg, oxidized phospholipids, proteins) or a reduction in endogenous antioxidants per se may be responsible at least in part for the state of oxidative stress in the lungs (along with instigating the subsequent cellular responses) rather than ROS derived directly from PM and its constituents.

Subsequent to oxidative stress, antioxidant and phase II defenses may be activated (eg, inducible nitric oxide synthase, glutathione) via transcription factor Nrf2-dependent pathways.[261] When inadequate, pathological oxidative stress can initiate a variety of pulmonary inflammatory responses. For example, ROS in the lungs has been shown to augment the signal transduction of membrane ligand (eg, epidermal growth factor by disrupting phosphatases) or pattern-recognition receptors (eg, toll-like receptors [TLR])[295–299] and/or stimulate intracellular pathways (eg, mitogen-activated protein kinases) that lead to the activation of proinflammatory transcription factors (eg, nuclear factor-$\kappa\beta$) that upregulate expression of a variety of cytokines and chemokines.[261] Alteration in lung cell redox status may itself stimulate nuclear factor-$\kappa\beta$. Biological components within coarse PM could also directly trigger inflammation (eg, nuclear factor-$\kappa\beta$ pathways) by binding to TLR2 or TLR4 receptors or other innate immune pattern-recognition receptors.[297] It is also possible that other components of metal-rich

PM could instigate inflammatory pathways via TLR activation directly or via the oxidation of endogenous biological compounds that then serve as TLR ligands.[300] Finally, there is some evidence that PM can activate inflammatory mitogen-activated protein kinase signaling by angiotensin II receptor–dependent pathways.[295] These inflammatory responses can also exacerbate the initial oxidative stress [eg, via upregulation of cellular NAD(P)H oxidase] and thus initiate a positive-feedback cycle.

Available studies support important contributions to pulmonary inflammation from innate immune cells such as neutrophils and macrophages (TNF-α, IL-6), as well as from the adaptive immune system, such as T cells (IL-1, IL-4, IL-6, and IL-10). Although the dominant source of cytokines likely represents the alveolar macrophages and lung epithelial cells, the role of other innate and adaptive immune cells cannot be ruled out.[299,301,302] Recently, myeloperoxidase activity was shown to increase after PM exposure in the same time course of appearance of cellular inflammation (primarily neutrophils) in the lung.[303] Gaseous components such as ozone may also amplify the toxicity of PM.[304]

Systemic Inflammation

In the context of examining the cardiovascular effects of air pollution, it is important to consider the inflammatory mediators that are released from lung cells after contact with PM, because some could conceivably spill over to the general circulation or increase liver production of acute-phase proteins (eg, CRP, fibrinogen). An increase in circulating proinflammatory mediators (eg, activated immune cells, cytokines) could thus serve as a pathway to instigate adverse effects on the heart and vasculature. Numerous experiments have demonstrated increased cellular and inflammatory cytokine content, such as IL-6, IL-1β, TNF-α, interferon-γ, and IL-8, of bronchial fluid and sometimes in circulating blood after acute exposure to a variety of pollutants.[292,305–311]

Critical roles for the elevations in systemic and pulmonary levels of IL-6 and TNF-α have been observed after PM exposure, typically coincident with pulmonary inflammation.[292,302,306,309,311–314] There is at least some evidence that the degree of pulmonary inflammation and systemic inflammation (IL-6) correlates with the elevation of systemic cytokines and systemic vascular dysfunction.[314] In a 4-week inhalation exposure to freshly generated diesel exhaust, IL-6 knockout mice did not demonstrate increased cellular inflammation or TNF-α in bronchial fluid, which implies a role for IL-6.[315] Consistent with these findings, acute intratracheal exposure to PM_{10} resulted in an increase in IL-6, TNF-α, and interferon-γ in the bronchial fluid.[316] However, in this study, IL-6$^{-/-}$ mice showed roughly the same levels of TNF-α in bronchial fluid as wild-type mice, although interferon-γ was decreased to control values.[316] The results also suggested that lung macrophages play an important role, because depletion of these cells abolished the increases in some of the cytokines and systemic cardiovascular responses. Although our understanding of the source of IL-6 and TNF-α and their involvement in the systemic inflammatory response after PM exposure remains incomplete, these and other experiments appear to suggest that at least with PM_{10} particles, alveolar macrophages play a dominant role.[309,314,316]

Among remaining uncertainties, the upstream signaling pathway responsible for the recognition of PM components that in turn produce the systemic inflammation has not been fully elucidated[317]; however, there is some evidence with other particulates and experimental models of lung injury that ROS generated by NADPH oxidase or pattern-recognition receptors may modulate some of these responses.[292,299,318] NADPH-oxidase knockout mice demonstrated significantly lower IL-6 and macrophage inflammatory protein-2 responses to collected PM than wild-type mice.[292] Extrapulmonary sources may also be involved in promulgating the systemic inflammation. $PM_{2.5}$ exposure in a model of diet-induced obesity in C57Bl/6 mice for a duration of 24 weeks resulted in elevations in TNF-α and IL-6. In addition, there were increases in circulating adipokines, such as resistin and plasminogen activator inhibitor-1.[319] The elevation in cytokines, thought to be derived from adipose sources, in addition to findings of adipose inflammation in that study, raises the possibility of additional systemic nonpulmonary sources of such cytokines.

Systemic Oxidative Stress

Numerous in vitro studies have demonstrated activation of ROS-generating pathways by PM incubation, such as NADPH oxidases, mitochondrial sources, cytochrome P450 enzymes, and endothelial nitric oxide synthase in cultured cells or in pulmonary and vascular tissue.[293,311,320–329] Similar to inflammation, the oxidative stress after PM inhalation may not always stay confined within the lungs.[330] The sources of excess ROS within cardiovascular tissue may include circulating immune cells or cytokines, depletion of defense mechanisms (eg, impaired high-density lipoprotein function), oxidation of lipoproteins or other plasma constituents,[331] activation of ANS pathways,[294] or circulating PM constituents (eg, soluble metals, organic compounds) reaching the vasculature.[261] Activation of ROS-dependent pathways modulates diverse responses with far-reaching consequences, including vascular inflammation/activation, atherosclerosis, impaired basal vasomotor balance, enhanced coagulation/thrombosis, and platelet activation.[290b]

Recent experiments have indeed confirmed the existence of footprints or markers of oxidative stress within the cardiovascular system in the in vivo context. Acute-exposure studies[332] have shown a relationship between the vascular dysfunction in spinotrapezius microvessels and the release of myeloperoxidase from leukocytes into the vasculature within only hours after the pulmonary instillation of PM.[332] Interestingly, an insoluble particle (TiO_2) induced very similar effects. More long-term studies[333] have demonstrated that 10 weeks of exposure to $PM_{2.5}$ increased superoxide production in response to angiotensin II and resulted in upregulation of NAD(P)H oxidase subunits and depletion of tetrahydrobiopterin in the vasculature. These effects had functional consequences in terms of increases in systemic vascular resistance and BP. In another investigation that involved apolipoprotein E–deficient (ApoE$^{-/-}$) fed a high-fat diet, chronic exposure

to $PM_{2.5}$ exacerbated vascular oxidant stress and promoted atherosclerosis progression.[334] The proatherogenic effects of ambient UFPs[331] versus $PM_{2.5}$ in genetically susceptible $ApoE^{-/-}$ mice in a mobile facility close to a Los Angeles freeway have also been compared. Exposure to UFPs resulted in an inhibition of the antiinflammatory capacity of plasma high-density lipoprotein and greater systemic oxidative stress, as evidenced by increased hepatic malondialdehyde and upregulation of Nrf2-regulated antioxidant genes.[331]

Other experiments[294] have suggested that ANS imbalance may play an important role in PM-induced cardiac oxidative stress. Pharmacological inhibition of the ANS could significantly reduce chemiluminescence in the heart after exposure.[303] More recently, an upstream modulator, the transient receptor potential vanilloid receptor-1, within the lung was identified as central to the inhaled CAP-mediated induction of cardiac chemiluminescence.[335] In these studies, capsazepine was able to abrogate ECG alterations in rats during the 5-hour exposure, which suggests that neural ANS pathways are crucial.

Thrombosis and Coagulation

Earlier studies using intratracheal instillation of high concentrations of diesel exhaust particles demonstrated the induction of lung inflammation, platelet activation, and increased peripheral vascular thrombosis in both arteries and veins after photochemical injury.[336,337] Thrombosis susceptibility was ascribed to direct passage of the instilled UFPs in the blood, because large polystyrene particles unlikely to cross the lung-blood barrier did not increase peripheral thrombosis. In a subsequent study, a persistent increase in thrombosis susceptibility to diesel exhaust particles was shown after 24 hours, an effect that was mitigated by pretreatment with sodium cromoglycate, which indicates that this response was secondary to histamine release from basophil degranulation.[338] These same effects, however, were mimicked by 400-nm polystyrene particles with a low likelihood of transgressing the pulmonary barrier, which implicates pulmonary release of histamine as a mediator of thrombosis at the later time point. Because histamine was increased in the plasma at 6 and 24 hours after exposure, and diphenhydramine mitigated diesel PM–induced thrombosis at later time points but not at 1 hour, it was hypothesized that additional direct effects of PM constituents reaching the circulation may be responsible for the earliest prothrombotic effects.[339] No increase in circulating von Willebrand factor was observed after instillation of both particles. Finally, pulmonary instillation of carbon nanotubes produced neutrophil lung influx 24 hours later. Circulating platelet-leukocyte conjugates were elevated 6 hours after exposure, whereas procoagulant microvesicular tissue factor activity and peripheral thrombotic potential were increased 24 hours later. Inhibition of P-selectin abrogated these responses, which demonstrates that rapid activation of circulating platelets by the pulmonary deposition of PM plays a vital role.[340] This series of studies suggests that release of lung cell–derived mediators (eg, histamine) after several hours along with the more rapid activation of circulating platelets by lung inflammation via P-selectin–dependent processes may mediate distant system prothrombotic effects without necessarily inducing systemic endothelial damage.

In a study using C57BL/6J mice, intratracheal PM_{10} particles rich in transition metals decreased bleeding, prothrombin, and activated partial thromboplastin times and enhanced the levels of several coagulation factors as well as thrombosis times in response to experimental $FeCl_3$ injury.[316] This prothrombotic effect was mitigated in IL-$6^{-/-}$ and macrophage-depleted mice, which suggests that IL-6, lung macrophages, and pulmonary inflammation are necessary initial steps. It is possible, however, that coarse-particle components (eg, endotoxin) could have been important mechanistically via TLR activation. The effect of fine PM or UFPs per se requires more investigation. Chronic ambient exposure to $PM_{2.5}$ has also been shown to increase tissue factor expression in macrophages and smooth muscle cells in atherosclerotic lesions. Complementary in vitro studies with cultured human smooth muscle cells and monocytes demonstrate dose-dependent increases in tissue factor in response to collected ambient particles.[341] Other findings also support potential procoagulant and thrombotic effects of PM.[342,343] These collective studies suggest that both short- and long-term PM inhalation can enhance thrombotic and coagulation tendencies, potentially via increases in circulating histamine and inflammatory cytokines and/or activated white cells and platelets. The plausibility of these pathways is supported by the well-recognized cross talk between inflammation and thrombosis.[344] Potential additional roles for UFPs or soluble constituents that reach the circulation and directly enhance platelet aggregation or systemic oxidative stress (thus activating the endothelium and blunting platelet-derived nitric oxide) require more investigation.

Systemic and Pulmonary Hypertension

Early animal studies suggested small or inconsistent effects of PM on BP,[345–347] sometimes dependent on the season[348] of exposures. A potential explanation may be variations in experimental protocols, including differences in the delivery, duration, and composition of exposure and the methods used to measure BP. Moreover, PM by itself may represent a relatively weak stimulus but may act more robustly in concert with other predisposing factors to affect BP. Sun et al[333] recently demonstrated a significant interactive effect of fine-CAP exposure with the vasoconstrictor angiotensin II in rats. Preexposure to $PM_{2.5}$ for a 10-week period resulted in enhancement of its prohypertensive response measured continuously by intra-arterial radiotelemetry. The exaggerated BP elevation was accompanied by endothelial dysfunction, including blunted endothelium-dependent vasodilation and enhanced vasoconstrictor reactivity, along with upregulation of NAPDH oxidase and Rho-kinase–signaling pathways. In vitro exposure to UFPs and $PM_{2.5}$ was also associated with an increase in Rho-kinase activity, phosphorylation of myosin light chain, and myosin phosphatase target subunit. Pretreatment with the nonspecific antioxidant *N*-acetylcysteine and Rho-kinase inhibitors prevented these responses, which suggests an ROS-mediated mechanism for particle-mediated effects on vascular smooth muscle constriction. Further

studies corroborated the role of exaggerated Rho-kinase pathway activity in potentiating the hypertensive response to angiotensin II in mice exposed to $PM_{2.5}$.[349] Moreover, particle exposure augmented angiotensin-mediated cardiac hypertrophy and collagen deposition. Blockade of Rho-kinase abolished these effects. These responses suggest that chronic $PM_{2.5}$ exposure disrupts normal vascular homeostasis and vasoactive mediator balance through ROS-dependent mechanisms in a manner that sensitizes the vessel toward vasoconstrictors. Activation of RhoA/Rho-kinase signaling pathways appears to play an important mechanistic role.

In conscious canines with implanted BP catheters, systemic arterial BP increased and baroreceptor sensitivity was rapidly altered over a few hours during CAP exposure.[350] Interestingly, α-adrenergic antagonism abrogated the responses. The findings support a mechanistic role for acute activation of the sympathetic nervous system by inhaled particles. In a study with Wistar-Kyoto male rats, CAP exposure for 4 days upregulated ET-A receptor expression in the heart. This alteration was also weakly correlated with an increase in BP, which suggests a role for enhance ET activity.[351] PM has also been demonstrated to alter the release of ET-1 and ET-3 from the lungs.[352] Elevation in pulmonary vascular resistance and pulmonary arterial pressure, which suggests constriction of the pulmonary vessels, has also been demonstrated in response to respirable carbon black particles.[353] Recently, ultrafine carbon particles were shown to increase BP in spontaneously hypertensive rats 1 to 3 days after a 24-hour exposure.[354] This response occurred concomitant with increased ET-1 messenger ribonucleic acid levels in lung tissue and small elevations in plasma renin concentration and angiotensin I and II in the systemic circulation. These findings further support the idea that ET may play a role in cardiovascular responses to PM exposure and suggest that activation of the renin-angiotensin system may also be involved. It is not clear whether the elevated circulating ET levels reflect increased release from the lungs and whether this mediates a systemic vasoconstrictor response. Alternatively, the increase may be more indicative of enhanced vascular tissue activity of these systems. Longer-term exposures of carbon black for 4 weeks in Sprague-Dawley rats has also been shown to significantly increase systolic BP concomitant with increases in serum levels of IL-6 and CRP.[355]

Finally, in vitro exposure to soluble and insoluble components of UFPs induces constriction in isolated pulmonary arterial rings and activates intracellular signaling pathways such as phosphorylation of extracellular signal–regulated kinase-1/2 and p38 mitogen-activated protein kinase in pulmonary endothelial cells. These effects were antagonized by losartan, and several metal components (copper and zinc) could replicate the responses.[295] This suggests a possible role for activation of angiotensin II receptor pathways relevant for the maintenance of vasomotor tone and smooth muscle constriction after inhalation of metal constituents within PM.

In sum, the studies demonstrate that long-term PM exposures over a period of weeks are capable of enhancing vasoconstrictive responsiveness of the vasculature (eg, increased Rho-kinase activity and reduced nitric oxide bioavailability) by inflammatory and ROS-dependent cell-signaling pathways. Shorter-term exposures over several hours to days may lead to vasoconstriction and increased pulmonary and systemic BP by pathways dependent on enhanced ET or angiotensin II signaling. Lung cells may release ET into the systemic circulation and thus increase its systemic activity, or the vascular ET system may be relatively upregulated because of increased ROS or reduced nitric oxide. Activation of the renin-angiotensin system may also occur because of systemic oxidative stress or inflammation or as a consequence of ANS imbalance. The very acute increase in BP that occurs concomitant with the inhalation of particles or within only minutes to hours after exposure appears to be mediated by autonomic imbalance that favors a relative activation of the sympathetic nervous system. No study has evaluated the effect of air pollution on renal sodium handling or long-term pressure natriuresis mechanisms, which are fundamental to the generation of chronic hypertension.

Vascular Dysfunction and Atherosclerosis

Many early experiments demonstrated the capacity of PM constituents to blunt nitric oxide–dependent dilation and enhance vasoconstrictor tone in ex vivo vascular studies because of excess ROS formation.[1] The first in vivo experiment demonstrated the proatherosclerotic actions of intratracheal PM_{10} instillation.[356] More recently, the pulmonary instillation of several different PM types was shown to rapidly impair microvascular endothelium-dependent vasodilation within days, likely by proinflammatory or ROS-dependent mechanisms (eg, myeloperoxidase).[332] Several animal studies have now demonstrated that long-term exposure to ambient $PM_{2.5}$, by use of ambient-exposure facilities without direct pulmonary instillation, not only causes endothelial dysfunction but also accelerates the progression of atherosclerosis. Sun et al[334] demonstrated that exposure of atherosclerosis-prone $ApoE^{-/-}$ mice to environmentally relevant levels of CAP, derived from regional northeastern $PM_{2.5}$, for 6 months in conjunction with a high-fat chow diet potentiated plaque development and heightened vascular inflammation (CD68+ macrophage infiltration and inducible nitric oxide synthase expression) and oxidant stress. The atherosclerotic plaque progression was also accompanied by alterations in vasomotor tone, including decreased endothelium-dependent vasodilation and heightened vasoconstriction to adrenergic stimuli. Importantly, the normalized average $PM_{2.5}$ concentration over the entire period was 15.2 $\mu g/m^3$, which approximates the annual NAAQS. Similar findings were reported in other chronic CAP exposures that involved an $ApoE^{-/-}$ model.[357] However, exposures to a double-knockout model of ApoE-deficient and low-density lipoprotein receptor–deficient mice increased plaque cellularity, reflective of inflammation, but did not enhance plaque burden. It is possible that the atherosclerotic severity of this phenotype precluded the observation of more subtle effects of CAP exposures.

Intratracheal instillation of UFP can acutely impair aortic endothelium-dependent vasodilation.[358] Moreover, repeated 10-week-long endotracheal dispersion of UFP carbon black increased atherosclerosis in low-density lipoprotein receptor–

knockout mice.[359] This occurred without evidence of systemic translocation of particles into the cardiovascular tissues. UFP inhalation by use of exposure facilities has also recently been shown to augment atherosclerosis, perhaps to a greater degree than $PM_{2.5}$. When investigating the effects of different PM size fractions, Araujo et al[331] compared the proatherogenic potential of exposure over 40 days to ambient particles <0.18 μm versus $PM_{2.5}$ in ApoE$^{-/-}$ mice. UFPs caused more adverse cardiovascular responses (eg, systemic oxidative stress, impaired high-density lipoprotein function) and greater potency in accelerating atherosclerotic lesion formation, although $PM_{2.5}$ did demonstrate qualitatively similar effects. Recent studies have also demonstrated that PM exposure likely promulgates systemic atherosclerosis by mechanisms that overlap those of other conventional cardiovascular risk factors.[360] Intratracheal instillation of PM_{10} particles caused a rapid impairment in endothelium-dependent vasodilation, stimulation of bone marrow–derived cells, and increased migration of monocytes into atherosclerotic plaques.[361,362] Systemic inflammation (IL-6) was also related to the degree of endothelial dysfunction.[314] Finally, the most compelling evidence for rapid impairment in nitric oxide bioavailability being directly involved in the origin of PM-induced endothelial dysfunction was demonstrated recently. Both fine-PM and UFM inhalation for only a few hours in normal rats blunted agonist-stimulated nitric oxide production within the microvasculature, measured by direct electrochemical sensors, concomitant with an observed impairment in vasomotor relaxation. Inhibition of myeloperoxidase or NADP(H) oxidase partially restored normal nitric oxide bioavailability and endothelial function, which suggests a role of activation of these endogenous radical-generating enzymes in this biological response.[363]

Potentially relevant adverse vascular effects of nonparticulate PM components should not be discounted. There may also exist some synergy between vapor phase, gas, and particle constituents in relation to instigation of cardiovascular responses. Recently,[364] it was demonstrated in apoE$^{-/-}$ mice that whole gasoline engine exhaust over 1 or 7 days increased vascular messenger ribonucleic acid expression of matrix metalloproteinase (MMP)-2 and MMP-9. Levels of ET-1 and ROS were similarly increased. The vascular ROS and MMP-2 elevations were attenuated by tempol. Endothelial receptor antagonism ameliorated the vascular expression of MMP-2, MMP-9, and ROS. In separate experiments, diesel exhaust exposure to rats for 5 hours augmented ET-induced vasoconstriction, potentially via a blunting of ET-B–induced nitric oxide release.[365] The findings suggest that exposure to a fresh mixture of PM, gases, and vapors may play a role in rapidly triggering atherosclerotic plaque vulnerability via ROS and ET-dependent upregulation of MMP levels.

Some studies suggest that predisposed animals may be more susceptible to air pollution–mediated vascular dysfunction. Diesel exhaust particles delivered by intraperitoneal injection impaired nitric oxide–dependent vasodilation only in apoE$^{-/-}$ mice with atherosclerosis and not in healthy control animals.[366] Aortas from prediabetic rats were found to be more susceptible to repeated exposures to oil combustion particles in causing noradrenergic-mediated constriction and impaired endothelium-dependent vasodilation.[367]

Taken together, the available studies suggest that short- and long-term particle exposures (including PM_{10}, $PM_{2.5}$, and UFP) can impair conduit and resistance arterial endothelium-dependent vasodilation. Chronic exposures have been shown to be capable of promoting atherosclerosis progression and enhancing plaque vulnerability. The underlying mechanisms likely involve vascular sequelae of systemic inflammation (due to interactions with innate immune cells and cytokines) or exaggerated oxidative stress pathways. Excess vascular ROS and inflammation will impair endogenous vasodilator bioavailability (eg, nitric oxide), enhance vasoconstrictor tone (eg, ET), and chronically activate multiple intracellular pathways that promote atherosclerosis.[368–370]

Heart Rate Variability

Some of the earliest indications of systemic effects of PM came from ECG studies in rats.[371] In general, reductions in several measures of HRV have been shown.[372–376] Most of the recent research has focused on exploring the roles of susceptibility and exposure characteristics. Decreases in heart rate and HRV indices have been reported to be pronounced in senescent mice, which indicates that aging may be a susceptibility factor.[353] Using an anesthetized model of postinfarction myocardium sensitivity, Wellenius and colleagues[377] did not demonstrate an effect of 1 hour of CAP exposure on heart rate or spontaneous ventricular arrhythmias. In contrast, in a post-MI heart failure model in Sprague-Dawley rats, diesel exhaust emissions reduced HRV in both healthy and heart failure groups and increased the incidence of premature ventricular contractions. Studies in mice have also indicated a potential role for transition metals and nickel in HRV alterations[376] and provide initial clues on the PM components that could influence autonomic tone.[48]

Some beginning insight into the neural pathways involved has been reported recently. PM-induced ECG changes in rats were shown to be prevented by inhibiting the transient receptor potential vanilloid receptor in the lungs. This suggests that the relevant neural mechanism that leads to alterations in HRV or heart rhythm may be induced by activation of receptor-mediated autonomic reflexes in the lung.[335] Circulating particle constituents or inflammatory mediators interacting with myocardial ion channels or electrophysiology did not appear to be a pertinent mechanism, at least in these studies.[335] However, it is unknown whether similar mechanisms can account for the HRV changes observed in humans, and a more detailed understanding of the anatomic pathways involved is required. Finally, it remains unclear whether the changes in cardiac HRV are actually caused by or merely illustrate an underlying alteration in ANS balance. Experiments that clearly define the direct contribution of sympathetic and parasympathetic nervous system activities (eg, microneurography, norepinephrine spillover rates, or autonomic receptor or ganglionic blockade) are needed.

MI and Arrhythmia

PM exposure can increase experimental infarct size and potentiate myocardial ischemia and arrhythmias in experi-

mental MI models. Relatively high concentrations of intratracheal UFP instillation induced pulmonary inflammation and doubled MI size in mice.[358] Conscious dogs exposed to fine CAP for several days experienced greater ST-segment changes during transient coronary artery occlusion.[378] These studies suggested that particulate-related changes in myocardial blood flow may be responsible, a hypothesis recently supported by experiments in chronically instrumented dogs exposed to fine CAP before transient occlusion of the left anterior descending artery. PM exposure was associated with a small but significant decrease in total myocardial flow, especially in the ischemic zone, and increases in coronary vascular resistance without an alteration in rate-pressure product.[379] The abnormalities were inversely related to PM mass, particle number, and black carbon concentration.

Exposure to residual oil fly ash increases arrhythmia frequency in rats with preexisting premature ventricular complexes, which suggests that PM sensitizes ischemic myocardium to abnormal automaticity[372]; however, CAP had no effect in rats.[380] Nevertheless, the data suggest that PM exposure may potentially be capable increasing the sensitivity of the myocardium to ischemia, likely by impairing myocardial blood flow and perfusion. In theory, this could play a role in enhancing the propensity for ventricular arrhythmias.

Insulin Resistance

Recently, Sun et al[319] exposed C57BL/6 mice fed high-fat chow to fine CAP or filtered air for 24 weeks. Mice exposed to $PM_{2.5}$ exhibited marked worsening of whole-body insulin resistance, systemic inflammation (increased IL-6 and TNF-α), and higher levels of adipokines, such as resistin and plasminogen activator inhibitor-1. $PM_{2.5}$ increased visceral adiposity and inflammation ($F4/80^+$ cells), with stromal vascular cells expressing higher TNF-α and IL-6 and lower IL-10 levels. Exposure also induced insulin-signaling abnormalities and reduced phosphorylation of Akt and endothelial nitric oxide synthase in aortic tissue, accompanied by abnormalities in vascular relaxation to insulin. Additionally, there was evidence that $PM_{2.5}$ exaggerated adhesion of monocytes in mesenteric microvessels, culminating in accumulation in visceral adipose. These intriguing findings suggest that longer-term exposure to PM air pollution may promote the chronic development of insulin resistance, obesity, and the metabolic syndrome.

Controlled-Exposure Studies in Humans

Several new human exposure studies have been published, a few of which have even included patients with CVD or risk factors. Similar to the animal studies, large variations among the exposure protocols, measured outcomes, and subject susceptibilities likely explain much of the differences among findings and must be considered when interpreting the results.

Systemic Inflammation

Controlled human exposure studies have measured the effects on circulating inflammatory markers such as CRP, IL-6, and TNF-α. In many of these single-episode short-term exposures, no overt changes in plasma cytokine levels were observed after CAP[381–383] or diesel exhaust.[345,384–386] Similarly, CRP levels have not consistently been found to increase in the time frame and context of most of these studies.[313,384–386]

However, there have also been some positive findings. Increases in IL-6[313] and TNF-α 24 hours after exposure to diesel exhaust in healthy adults have been reported. High levels of ambient particles can stimulate the bone marrow to enhance the release of neutrophils, band cells, and monocytes into the circulation, which causes a cellular inflammatory response.[387,388] Some controlled-exposure studies corroborate the existence of a cellular proinflammatory response that manifests as increases in circulating white blood cell or immune cell counts. In 1 study, increased peripheral basophils in healthy older adults were noted 4 hours after a 2-hour exposure to fine CAP.[389] In a similar study, increased white blood cell counts were observed in healthy young adults 12 hours after exposure.[381] Recently, investigators observed an increased in total white blood cell and neutrophil levels immediately after a 2-hour exposure to CAP in downtown Toronto, Ontario, Canada.[390] Conversely, decreases in blood monocytes, basophils, eosinophils, and CD54 and CD18 adhesion molecule expression on monocytes after exposure to ultrafine carbon (10 to 50 $\mu g/m^3$) among exercising asthmatic individuals and healthy adults have also been reported.[391] The authors suggested in the latter study that these results may represent the sequestration of these cells in tissue compartments such as the lung or vasculature, where there may be selective expression of the corresponding receptors for these ligands.[362] However, other recent human clinical studies have found no association between peripheral blood cell counts and exposure to fine PM or UFPs such as zinc oxide,[392] ultrafine carbon,[393] or diesel exhaust.[313,384,385]

More subtle, yet physiologically relevant or functional proinflammatory changes may be overlooked by the measurement of circulating cytokines or cell counts alone in human studies. Peretz et al[394] recently evaluated gene expression using an expression array in monocytes after 2 hours of exposure to diesel exhaust. Although initially a small study, 10 genes involved in the inflammatory response were modulated in response to exposure (8 upregulated, 2 downregulated). These findings will need to be reproduced in larger studies and raise the possibility that functional changes in inflammatory cells may occur without discernible changes in their levels in the peripheral circulation.[394]

In sum, the findings from controlled human exposures do not demonstrate a robust inflammatory response; however, they have been limited by the fact that they are, by necessity, of short duration and relatively low concentration. Additionally, the results do not preclude an effect of higher exposures, the presence of more subtle responses, or alterations in other cellular inflammatory pathways not measurable by circulating markers.

Systemic Oxidative Stress

The demonstration of systemic oxidative stress is difficult in human studies. Nonetheless, a few studies have reported positive findings. These include an increase in urinary excre-

tion of free 8-iso-prostaglandin-2α among healthy adults after a 4-hour exposure to concentrated wood smoke[395] and an increase in plasma antioxidant capacity 24 hours after a 1-hour exposure to diesel exhaust in a group of healthy volunteers.[313] The investigators speculated that systemic oxidative stress after exposure may have been responsible for this upregulation in antioxidant defense.[313] Other investigators[394] have observed significant differences in expression of genes involved in oxidative stress pathways due to diesel exhaust exposure. Bräuner et al[167] recently investigated the effect of ultrafine traffic particles on oxidative stress–induced damage to DNA in healthy young adults exposed to low concentrations of ambient urban particles ($PM_{2.5}$ and $PM_{10-2.5}$ mass of 9.7 and 12.6 $\mu g/m^3$, respectively) in an exposure chamber above a busy road with high traffic density. The authors observed increased levels of DNA strand breaks and formamidopyrimidine-DNA glycosylase sites in monocytes after exposure to PM but no changes in the DNA repair enzyme 7,8-dihydro-8-oxoguanine-DNA glycosylase. Similar to their previous findings with ambient levels,[168] the results suggest that short-term exposure to UFPs may result in damage to DNA. This may occur through oxidative stress pathways, although there was no increase in messenger ribonucleic acid levels in heme oxygenase-1, a gene known to be regulated by Nrf2, a transcription factor regulated by oxidative stress.[396] Moreover, more recent observations by the same investigators failed to demonstrate significant biomarker signals for lipid or protein oxidative damage after similar near-roadway exposures.[178] Although not entirely consistent, the available studies demonstrate that acute exposure to PM, perhaps even at ambient levels, may be capable of inducing acute systemic oxidative stress in human subjects under certain circumstances. The assays used to assess the footprint of systemic "oxidative stress" or damage may also play a significant role in the results.

Thrombosis and Coagulation

Several new studies of controlled human exposure have evaluated the effects of PM on hemostatic markers (eg, factor VII, fibrinogen, platelet count, D-dimer, and von Willebrand factor). Although some of these studies have not observed changes after acute exposures,[392] others have reported increases in fibrinogen levels at 8 to 24 hours after exposure to CAP.[381,397] Mills and colleagues[384,385] recently demonstrated a significant effect of diesel exhaust on fibrinolytic function in response to intermittent exercise both in healthy men and in men with coronary heart disease. In both groups of volunteers, bradykinin-induced release of tissue plasminogen activator was observed to decrease compared with filtered air at 6 hours after exposure to diesel exhaust. These perturbations in tissue plasminogen activator release did not persist 24 hours after exposure.[313] In a randomized, controlled crossover study involving "at-risk" metabolic syndrome patients, no changes in plasminogen activator inhibitor-1 were noted over a 24-hour duration; paradoxically, a decrease in von Willebrand factor was noted in this study.[398] In a similar experiment conducted in healthy adults, diesel exhaust had no effect on D-dimer, von Willebrand factor, CRP, or platelet counts compared with filtered air up to 22 hours after exposure.[386] Other investigators[395] recently evaluated the effect of wood smoke on markers of coagulation, inflammation, and lipid peroxidation in young healthy subjects. Serum amyloid A and the ratio of factor VIII to von Willebrand factor, an indicator of an increased risk of venous thromboembolism, were increased at 4 hours after exposure.[395] Samet et al[383] reported an association between various coagulation markers and exposure to ultrafine, fine, and thoracic coarse CAP among healthy young adults. Although exposure to coarse CAP did not result in significant changes in hemostatic variables, the overall trend suggested a prothrombotic effect. Exposure to UFPs increased D-dimer levels, whereas fine-CAP effects tended to increase fibrinogen, similar to previously reported findings.[381]

The measurement of blood levels of coagulation factors or biomarkers of thrombosis could potentially miss a relevant biological effect at the vascular wall. Recently, ex vivo thrombus formation was assessed by use of the Badimon chamber after controlled exposures to dilute diesel exhaust in healthy volunteers.[399] This protocol measures thrombus formation in native (nonanticoagulated) whole blood triggered by exposure to a physiologically relevant substrate, under flow conditions that mimic those found in diseased coronary arteries. It may therefore provide a superior estimate of actual in vivo conditions related to thrombosis potential. Interestingly, dilute diesel exhaust exposure increased thrombus formation within 2 hours, in association with increased platelet activation (ie, increased circulating platelet-monocyte aggregates and soluble CD40 ligand). Taken together, these new studies have provided additional evidence that short-term exposure to PM at near-ambient levels may have small yet potentially significant effects on hemostasis in humans. Whether direct interactions of circulating PM constituents with platelets, activation of platelets due to lung inflammation or secondary to elevated systemic cytokine levels, or an increase in procoagulant factors (eg, fibrinogen) as an acute-phase response to inflammation (or a combination of these pathways) is responsible warrants attention in future studies.

Arterial BP

Although several studies have evaluated the BP response to acute exposures, many inconsistencies in results have been reported.[400] This must be considered in the context that BP was not the primary outcome of interest in most studies, nor was it typically assessed with adequate sophistication. In one of the earliest studies, $PM_{2.5}$ increased systolic BP in healthy subjects but decreased it in asthmatic individuals.[401] Three other controlled studies did not report changes among healthy adults.[345,402,403] However, in a more detailed reanalysis of the changes in BP during the actual period of exposure to CAP plus ozone, Urch et al[404] found a significant increase in diastolic BP of 6 mm Hg. The magnitude of response was associated with the concentration of organic carbon within $PM_{2.5}$.[405] Recent follow-up studies redemonstrated an acute prohypertensive response during the inhalation of CAP in 2 separate cities.[390] The $PM_{2.5}$ mass during exposure and decreases in several HRV metrics were associated with the

magnitude of the short-lived diastolic BP elevation. This suggested that the most plausible mechanism for this acute response was CAP-induced ANS imbalance that favored sympathetic over parasympathetic cardiovascular tone. Whether this reaction occurred because of a generalized stress response, as a consequence of specific soluble PM constituents directly altering central nervous system activity, or via altered ANS reflex arcs due to the interaction of inhaled particles with lung receptors/nerve endings remains to be elucidated.

The effect of inhaled particulates on BP has also been investigated in several other recent controlled human exposure studies. Two new studies assessed BP changes after a 1-hour exposure to diesel exhaust. Mills et al[384] found a 6-mm Hg increase in diastolic BP 2 hours after exposure, which was of marginal statistical significance ($P=0.08$); however, this trend did not persist for 24 hours,[384] nor was it found among patients with coronary artery disease.[385] The available data to date suggest that short-term exposure to $PM_{2.5}$ or diesel exhaust is capable in certain circumstances of rapidly raising BP. The most consistent and largest effects were seen concomitant with the inhalation of particles. Thus far, the most likely mechanism for such rapid hemodynamic responses appears to be ANS imbalance. However, it is possible that reductions in nitric oxide bioavailability that modulate basal arterial tone toward vasoconstriction or increases in ET among other hemodynamically active molecules (eg, angiotensin II) also play a role in some circumstances.

Vascular Dysfunction

The first controlled human exposure study related to vascular function reported that CAP plus ozone exposure caused acute conduit arterial vasoconstriction in healthy adults.[1] Endothelium-dependent and -independent vasodilation remained intact. Recent follow-up experiments determined that $PM_{2.5}$, not ozone, was responsible for the adverse vascular effects. However, in these subsequent and larger experiments, fine-CAP exposure did prove capable of diminishing conduit artery endothelium-dependent vasodilation 24 hours (but not immediately) after exposure.[390] Postexposure $PM_{2.5}$ mass and TNF-α level were both associated with the degree of endothelial dysfunction, which suggests that systemic inflammation induced by higher levels of particles was likely responsible. Finally, the CAP-induced endothelial dysfunction occurred during exposures in Toronto, Canada, but not Ann Arbor, Mich, which suggests that the composition of the particles is probably an important determinant of the vascular responses.

An acute alteration in vascular function/tone after short-term controlled PM air pollution exposure was corroborated recently.[406] In 27 adults (10 healthy adults and 17 with the metabolic syndrome), a 2-hour exposure to dilute diesel exhaust caused a dose-dependent constriction of the brachial artery and elevation in plasma ET level without impairing endothelium-dependent vasodilation. Contrary to the hypothesis that metabolic syndrome patients would show greater effects, vasoconstriction was greater in magnitude among the healthy participants. In an additional study, 2-hour exposure to UFPs composed of elemental carbon impaired peak forearm blood flow response to ischemia 3.5 hours later. There were no other vascular changes or alterations at other time points. BP was also not affected.[407]

Several recent studies have also shown that dilute diesel exhaust can impair peripheral resistance vessel responses to acetylcholine, bradykinin, and nitroprusside 6 hours after exposure.[384] The blunted responses to acetylcholine persisted for 24 hours in healthy adults.[313] In contrast, bradykinin and sodium nitroprusside–mediated vasodilation and bradykinin-induced acute plasma tissue plasminogen activator release were not altered 24 hours later. In subsequent studies, patients with stable coronary artery disease exposed to dilute diesel exhaust for 1 hour during intermittent exercise demonstrated reduced bradykinin-mediated tissue plasminogen activator release; however, microvascular endothelial function was not impaired.[385] This may be related to some degree of preexisting endothelial dysfunction in these patients. However, exercise-induced ST-segment depression and ischemic burden were significantly greater during diesel compared with filtered air exposure. These important findings experimentally highlight that PM air pollution exposure can trigger, or augment existing, myocardial ischemia extremely rapidly (in fact, concomitant with exposure). Reduced coronary flow reserve (that was not observed or resolved at the time of the postexposure brachial artery studies) due to rapid alterations in coronary microvascular function may have contributed to the acute myocardial ischemia. Alternatively, acute ANS imbalance induced by diesel exhaust inhalation may have acutely altered coronary tone and impaired myocardial perfusion.

In a study that exposed healthy young adults to 100 $\mu g/m^3$ of diesel exhaust for 2 hours,[364] it was recently demonstrated that this air pollution mixture acutely raised plasma ET-1 and MMP-9 expression and activity within 30 minutes. These results corroborate the animal data that even short-term exposures can rapidly alter factors, such as MMP activity, that are mechanistically linked with causing atherosclerotic plaque disruption (and thus acute MI). The increase in ET levels also corroborates previous studies[406] that showed that diesel exhaust can acutely affect important endogenous regulators of vasomotor tone.

Controlled air pollution exposures have not always been shown to impair endothelial function or vasomotor tone. Despite an increase in exhaled 8-isoprostane concentrations that suggested pulmonary oxidative stress, fine CAP did not affect brachial flow–mediated dilation or basal diameter in northern Scotland exposures.[382] However, the $PM_{2.5}$ consisted of relatively inert ambient sea-salt particles and was extremely low in combustion-derived sources. This is in contrast to the particle chemistry in the investigators' previous diesel exposure studies that showed positive findings.[408,409] Moreover, 24-hour exposure to ambient pollution shunted into a chamber next to a busy street did not impair microvascular endothelial function in 29 healthy subjects, as assessed by digital tonometry.[178] This exposure to near-roadway ambient air, which consisted of ambient UFP and $PM_{2.5}$, did not alter biomarkers of inflammation, hemostasis,

or protein and lipid oxidation. The authors speculated that the relatively low concentrations of UFP numbers and PM mass or the young, healthy status of the subjects could explain the null findings. Taken together, these studies suggest that brief PM exposure can trigger conduit arterial vasoconstriction, possibly in relation to increased ET activity or augmented sympathetic ANS tone. Under certain circumstances, conduit and resistance arteriole endothelium-dependent vasodilation can also be impaired within a few hours. This abnormality is more likely due to reduced nitric oxide bioavailability as a consequence of systemic proinflammatory and oxidative responses; however, alternative mechanisms and endogenous vasoactive pathways have not been fully explored. It is also apparent that the composition, source, and concentration of pollution, along with the susceptibility of the human subjects, play important roles in determining the vascular effects of acute air pollution exposure.

Heart Rate Variability

The results of several new controlled human exposure studies provide limited evidence to suggest that acute exposure to near-ambient levels of PM may be associated with small changes in HRV. There are at least 4 studies to support this. In the first study, healthy elderly individuals experienced significant decreases in HRV immediately after exposure.[233] Some of these changes persisted for at least 24 hours. Gong et al[410] studied healthy and asthmatic adults exposed to coarse CAPs with intermittent exercise. HRV was not affected immediately after the exposure but decreased in both groups at 4 and 22 hours after the end of the exposure; greater responses were observed in nonasthmatic individuals.[410] In another study, healthy elderly subjects and patients with chronic obstructive pulmonary disease were exposed to approximately 200 $\mu g/m^3$ CAP and filtered air for 2 hours with intermittent mild exercise. HRV over multihour intervals was lower after CAP than after filtered air in healthy elderly subjects but not in subjects with lung disease. A significant negative effect of CAP on ectopic heartbeats during or after CAP exposure relative to filtered air was noted in the healthy subjects, whereas the group with pulmonary disease experienced an improvement during or after CAP relative to filtered air.[389] Other investigators recently compared the effects of 2-hour exposures with intermittent exercise to ultrafine (average concentration 47 $\mu g/m^3$), fine (average concentration 120 $\mu g/m^3$), and coarse (average concentration 89 $\mu g/m^3$) CAP among healthy subjects.[383] In both the ultrafine and coarse studies, a crossover design was used in which each subject was exposed to both PM and filtered air. In the case of the fine-PM study, subjects did not serve as their own control but were exposed to either PM or filtered air. Thoracic coarse fraction CAP produced a statistically significant decrease in the standard deviation of normal-to-normal heart rate 20 hours after exposure compared with filtered air. No statistically significant effects on HRV were observed after exposure to UFPs as measured during controlled 5-minute intervals. However, the authors did observe a significant decrease in the standard deviation of normal-to-normal heart rate after exposure to UFPs based on an analysis of the 24-hour measurements. No differences were reported in HRV with fine-PM exposures. Although some controlled-exposure studies have reported either no acute changes[390] or, on occasion, increases in HRV metrics in subsets of individuals,[208,393,401] these studies generally demonstrate that acute PM exposure is capable of reducing HRV. More consistent reductions have been found among older adults (compared with younger subjects or those with lung diseases, who show mixed responses) and perhaps with exposures to larger particles.[233,389] Whether pulmonary ANS reflex arcs are activated by the deposition of PM within the lung or whether other pathways are responsible for these physiological changes in human exposure studies requires more investigation.

Table 7. Summary of Level of Evidence Supporting Global Biological Pathways and Specific Mechanisms Whereby $PM_{2.5}$, Traffic-Related, or Combustion-Related Air Pollution Exposure Can Affect the Cardiovascular System

	Animal Studies	Human Studies
General "intermediary" pathways whereby PM inhalation can instigate extrapulmonary effects on the cardiovascular system		
Pathway 1: Instigation of systemic proinflammatory responses	↑ ↑ ↑	↑ ↑ ↑
Pathway 2: Alterations in systemic ANS balance/activity	↑	↑ ↑
Pathway 3: PM and/or associated constituents directly reaching the systemic circulation	↑	↑
Specific biological mechanisms directly responsible for triggering cardiovascular events		
Vascular dysfunction or vasoconstriction	↑ ↑ ↑	↑ ↑
Enhanced thrombosis or coagulation potential	↑ ↑	↑ ↑
Elevated arterial BP	↑ ↑	↑ ↑
Enhanced atherosclerosis or plaque vulnerability	↑ ↑	↑
Arrhythmias	↑	↑

The arrows are not indicators of the relative size of the association but represent a qualitative assessment based on the consensus of the writing group of the strength of the mechanistic evidence based on the number and/or quality, as well as the consistency, of the relevant studies.

↑ ↑ ↑ Indicates strong overall mechanistic evidence.

↑ ↑ Indicates moderate overall mechanistic evidence.

↑ Indicates some but limited or weak available mechanistic evidence.

Blank indicates lack of evidence.

Evidence Summary and Contextual Framework for Biological Mechanisms

Table 7 provides an outline of the level of evidence supporting the generalized intermediary pathways and specific mechanisms whereby PM exposures can be capable of eliciting

cardiovascular events. At the molecular level, oxidative stress as a critically important cause and consequence of PM-mediated cardiovascular effects has a sound experimental basis.[261,290b,294,319,333,334,345–349,351,361–364,411] At the integrated physiological level, the collective body of evidence continues to support the existence of 3 general pathways (Figure 3). Some of these responses, such as systemic inflammation (via pathway 1), likely require antecedent pulmonary oxidative stress or inflammation in order to be initiated. Others, including ANS imbalance (via pathway 2) and PM or its constituents reaching the systemic circulation (via pathway 3), may not. Although PM-associated metals[412] and certain UFPs[261,413–415] might be capable of translocating into the blood stream, some studies have been negative in this regard.[355,416] Many issues related to this pathway are controversial and require resolution.[416] These include the relevance of the dosages delivered to cardiovascular organs, the consequences of particle constituent modifications after interactions with lung tissue/fluids and plasma components, the means of transport within the circulation (eg, protein bound or within cells),[417] and the time course and ultimate sites of PM sequestration. It is also possible that increases in some vasoactive mediators or molecules with adverse effects on cardiovascular tissue, such as ET-1,[351–354] may occur in the lung and systemic circulation without the need for antecedent lung inflammation. Moreover, the 3 general pathways represent a simplification of complicated biological processes. They may not be mutually exclusive, may overlap temporally, and likely exhibit synergies in causing manifest cardiovascular disease events. Many of the biological pathways are also known to exhibit mutual interactions (eg, inflammation with thrombosis/coagulation and with autonomic function). The pathways are also likely to be principally active at differing time points (eg, more rapid cardiovascular effects of autonomic imbalance than systemic inflammation) and likely vary in importance in relation to different durations of exposure and in causing different cardiovascular sequelae. The chemical characteristics and sizes of inhaled PM may also determine the pathways activated. As opposed to UFPs or some particulate components or chemicals, larger fine and coarse PM are not likely transported into the circulation to any large degree and therefore are more apt to require intermediary pathways to cause extrapulmonary effects. It may also be that surface-bound components may be delivered into the circulation, whereas larger particles themselves serve as a means to deliver the responsible constituent into the pulmonary tree.

The hyperacute physiological responses that occur minutes to hours after PM inhalation are likely mediated principally via pathways 2 and 3. These include ANS-mediated changes (eg, elevated BP, arrhythmias, and vasoconstriction), along with direct effects of circulating PM constituents on platelets (eg, procoagulant and thrombotic changes) and the endothelium (eg, oxidative stress and vasoconstriction). These responses are liable to be the dominant mechanisms responsible for the actual triggering of acute cardiovascular events. Clinically meaningful effects undoubtedly become manifest only in the context of a susceptible patient, typified by the individual with "vulnerable plaque" in the case of acute coronary syndromes or strokes, "vulnerable myocardium" in the context of arrhythmias, or the "vulnerable circulation" in the context of a heart failure patient at risk for circulatory overload. On the other hand, the biological consequences of systemic inflammation, such as activated white cells and elevated cytokines (via pathway 1), typically require longer periods. Their penultimate effect is the induction of a chronic underlying vulnerable milieu that leads to atherosclerotic plaque vulnerability, enhanced coagulation/thrombotic and arrhythmia potential, and impaired basal vasomotor balance. These actions thereby predispose individuals for future cardiovascular events, particularly when they occur in conjunction with traditional risk factors or prompt susceptibility to the acute biological actions (via pathways 2 and 3) of later air pollution exposures.

This hypothetical segregation of the biological effects of PM exposure as acute or chronic and into the broad pathways is artificial. It is useful in the broad context of understanding potential pathways; however, there is no doubt a large degree of overlap among the mechanisms and the timing of physiological responses. This is most aptly conveyed as the influence of "acute on chronic" actions of exposure. For example, the activation of circulating platelets by the pulmonary deposition of particles or lung inflammation (eg, by P-selectin–dependent pathways, histamine, or IL-6) could occur within hours and more rapidly than typical of the other consequences of inflammation (eg, progression of atherosclerosis). In the presence of a vulnerable or eroded coronary plaque due to long-term air pollution exposure, this sudden prothrombotic tendency could instigate an acute ischemic event (alone or in conjunction with other effects of short-term PM exposure via pathways 2 and 3). Furthermore, the epidemiological cohort studies demonstrate a larger relative risk for increased cardiovascular-related mortality than for morbidity.[72,73,227,274] If this is a true biological response and not simply a statistical phenomenon or a shortcoming of the available data, it not only suggests that exposures are capable of triggering acute cardiovascular events but that PM air pollution may also exaggerate their severity even if they would have otherwise occurred for reasons unrelated to air pollution. Therefore, exposure to PM could also be responsible for promoting fatal over nonfatal events.

Conclusions and Recommendations

A wide array of new studies that range from epidemiology to molecular and toxicological experiments have provided additional persuasive evidence that present-day levels of air pollutants contribute to cardiovascular morbidity and mortality. Although not unexpected given the numerous and heterogeneous nature of the published studies, all findings related to every single cardiovascular end point have not been consistent. However, the overall weight of scientific evidence now supports several new conclusions since the 2004 statement. These consensus points are given below by the AHA writing group after considering the strength, consistency, and coherence of the epidemiological findings, as well as in the context of evaluating the extent of the studies that provided related mechanistic support.

- The preponderance of findings indicate that short-term exposure to $PM_{2.5}$ over a period of a few hours to weeks can trigger CVD-related mortality and nonfatal events, including myocardial ischemia and MIs, heart failure, arrhythmias, and strokes.
- The increase in risk for acute $PM_{2.5}$-associated cardiovascular morbidity and mortality is principally among susceptible, but not necessarily critically ill, individuals. Several studies suggest that susceptible individuals at greater risk may include the elderly, patients with preexisting coronary artery disease, and perhaps those with diabetes. Recent data suggest that women and obese individuals might also be at higher risk.
- Most studies support the idea that longer-term $PM_{2.5}$ exposures increase the risk for cardiovascular mortality to an even greater extent than short-term exposures. Because most studies have focused on mortality data, the effect of long-term exposures on nonfatal cardiovascular events is less consistent and requires more investigation.
- The $PM_{2.5}$ concentration–cardiovascular risk relationships for both short- and long-term exposures appear to be monotonic, extending below 15 $\mu g/m^3$ (the 2006 annual NAAQS level) without a discernable "safe" threshold.
- Long-term exposure to elevated concentrations of ambient $PM_{2.5}$ at levels encountered in the present-day environment (ie, any increase by 10 $\mu g/m^3$) reduces life expectancy within a population probably by several months to a few years. Given that $PM_{2.5}$ is most strongly associated with cardiovascular deaths in the cohort studies, the reduced life expectancy is most likely predominantly due to excess cardiovascular mortality.
- The available studies are suggestive that reductions in PM levels decrease cardiovascular mortality within a time frame as short as a few years.
- Many potential biological mechanisms exist whereby PM exposure could exacerbate existing CVDs and trigger acute cardiovascular events (over the short term) and instigate or accelerate chronic CVDs (over the long run). Experimental support is increasingly strong for several mechanisms, which lends biological plausibility for the epidemiological findings.
- The existing evidence suggests that PM air pollution is capable of augmenting the development and progression of atherosclerosis. There is some support for a potential effect on several other chronic CVDs, including hypertension, heart failure, and diabetes.
- Most recent studies support the conclusion that the overall absolute risk for mortality due to PM exposure is greater for cardiovascular than pulmonary diseases after both short- and long-term exposures.

There are several additional areas worthy of highlighting in which the study results are reasonably consistent but in which the writing group believed further research was required to formulate firm conclusions.

- Although there is only limited epidemiological evidence directly linking UFPs with cardiovascular health problems,[262] the toxicological and experimental exposure evidence is suggestive that this size fraction may pose a particularly high risk to the cardiovascular system. The likelihood of health effects and the causal pathways mediated specifically by UFP exposure have been debated among experts recently.[418] Future research may help to more fully elucidate whether particles within the ultrafine size range (0.001 to 0.1 μm) and/or their constituents are more harmful to the cardiovascular system or pose a relatively greater cardiovascular risk than particles between 0.1 and 2.5 μm in diameter.
- Similarly, many studies have found a strong association between metrics of traffic-related air pollution exposure and elevated cardiovascular risk. Whether this represents the harmful effects of UFPs or diesel exhaust particulates, major components of the traffic mixture, or other pollution components is unclear. Diesel and UFPs possess toxic properties that instigate harmful biological responses in experimental models. However, the particle size fraction(s) and roles played by other copollutants (gases, VOCs, SVOCs) within the traffic-related mixture have not been fully elucidated. Nevertheless, traffic-related pollution as a whole appears to be a specific source associated with cardiovascular risk. It likely poses a major public health burden, regardless of a putative higher toxicity, because of the commonness of exposure in modern society (eg, accounting for ≈60% of daily UFP exposure; http://www.catf.us/projects/diesel/).
- The importance of other specific sources, regional differences in pollution composition, and other specific constituents remains less clear. However, toxicological studies have identified several transition metals (eg, iron, vanadium, nickel, copper, and zinc), organic carbon species, semiquinones, and endotoxin as specific PM-related components capable of prompting oxidative stress and inflammation and thus likely imparting biological harm. Some source-apportionment studies also demonstrate that attention should be given to these compounds as being among the most likely mediators of clinical CVD. More studies are required in this regard to clarify this issue and to better define these and other potentially responsible constituents and sources.
- Although the focus of the present statement is on PM, we recognize that other air pollutants may also pose cardiovascular risk alone or in conjunction with fine-particle exposure. In this context, we believe additional research is necessary to make firm conclusions regarding the independent cardiovascular risks posed by several gaseous pollutants (eg, ozone and NO_2). Although ozone has been linked to increased cardiopulmonary mortality,[50] strokes,[126] and MIs[419] in some short-term studies, long-term exposure was not associated with cardiovascular mortality after accounting for PM in a recent analysis.[87] The recent finding that small changes in low levels of ambient carbon monoxide concentrations are related to cardiovascular hospitalizations also merits further exploration.[420]
- Several secondary aerosols (eg, nitrate and sulfate) are often associated with cardiovascular mortality; however, whether these compounds are directly harmful or are surrogate markers of toxic sources of exposure requires

more investigation. Similarly, the current literature regarding the independent cardiovascular risks posed by coarse particles is mixed, with most recent findings not supporting an association after accounting for the effects of $PM_{2.5}$.[43,72,104]

- Several recent cohort studies and intermediate end-point experiments suggest that obese individuals (and/or those with the metabolic syndrome) may be a susceptible population at greater risk for cardiovascular events due to $PM_{2.5}$ exposure. This is a tremendously important public health issue to corroborate because of the enormous and growing prevalence of obesity worldwide.

This updated review by the AHA writing group corroborates and strengthens the conclusions of the initial scientific statement. In this context, we agree with the concept and continue to support measures based on scientific evidence, such as the US EPA NAAQS, that seek to control PM levels to protect the public health. Because the evidence reviewed supports that there is no safe threshold, it appears that public health benefits would accrue from lowering $PM_{2.5}$ concentrations even below present-day annual (15 $\mu g/m^3$) and 24-hour (35 $\mu g/m^3$) NAAQS, if feasible, to optimally protect the most susceptible populations. Evaluations of the effectiveness of such efforts would be warranted as well. Within the framework of attempting to establish causality between associated variables in epidemiological studies, there are several generally accepted "aspects" that have been evaluated (the following phrases in italics per the Bradford Hill criteria)[421]: With regard to cardiovascular mortality and $PM_{2.5}$ exposure, there is a *consistent association* that satisfies both a *temporal and exposure-response relationship*. There is *coherence of findings* among several fields of science, including toxicology, human and animal exposures, and different types of epidemiological studies and time frames of exposure. Rigorous experiments demonstrate multiple *plausible biological mechanisms*. Finally, natural experiments have confirmed that a change (ie, reduction) in exposure produces a change (ie, decrease) in cardiovascular mortality. In this case, *specificity of outcomes* and *strength of the observation* are less pertinent, because PM exposure could be capable of causing multiple different types of events (eg, MIs, arrhythmias, and heart failure exacerbations), and the overall cardiovascular mortality relative risk posed for any single individual is expected to be small. Nevertheless, given the ubiquity of exposure, the overall public health consequences can be substantial and observable in population- or large cohort–based studies.

It is the opinion of the writing group that the overall evidence is consistent with a causal relationship between $PM_{2.5}$ exposure and cardiovascular morbidity and mortality. This body of evidence has grown and has been strengthened substantially since publication of the first AHA scientific statement.[1] At present, no credible alternative explanation exists. These conclusions of our independent review are broadly similar to those found in the EPA's Integrated Science Assessment for Particulate Matter final report (http://cfpub.epa.gov/ncea/cfm/recordisplay.cfm?deid=216546). In summary, the AHA writing group deems that $PM_{2.5}$ exposure is a "modifiable factor contributing to cardiovascular morbidity and mortality."

Clinical Recommendations

Several precautionary recommendations can be made for healthcare providers who interact with individuals who are at risk for CVDs. Although they have not been clinically tested or proven to reduce mortality, they are practical and feasible measures that may help to reduce exposures to air pollution and therefore potentially lower the associated cardiovascular risk. Moreover, a recent observational study found that patient awareness of air quality indices and media alerts along with health professional advice can significantly affect reported changes in outdoor activity to avoid exposure to air pollution.[422]

- Evidence-based appropriate treatment of the traditional cardiovascular risk factors should be emphasized. This may also lessen the susceptibility of patients to air pollution exposures.
- All patients with CVD should be educated about the cardiovascular risks posed by air pollution.
- Consideration should also be given to educating patients without CVD but who are at high risk (eg, the elderly, individuals with the metabolic syndrome or multiple risk factors, and those with diabetes).
- Part of patient education should include the provision of information regarding the available sources (local and national newspapers [*USA Today*], EPA World Wide Web site [http://airnow.gov/], and The Weather Channel and its World Wide Web site [http://www.weather.com/]) that provide a daily EPA Air Quality Index.
- On the basis of the forecast Air Quality Index, prudent recommendations for reducing exposure and limiting activity should be provided based on the patient's level of risk. A list of such recommendations is provided on the EPA World Wide Web site (http://airnow.gov/). For example, when the Air Quality Index for PM is "unhealthy" (151 to 200), then the recommendations are as follows: "People with heart or lung disease, older adults, and children should avoid prolonged or heavy exertion. Everyone else should reduce prolonged or heavy exertion." The action recommendations are as follows: "You can reduce your exposure to particles by 1) planning strenuous activity when particle levels are forecast to be lower, 2) reducing the amount of time spent at vigorous activity, or 3) choosing a less strenuous activity (eg, going for a walk instead of a jog). When particle levels are high outdoors, they also can be high indoors. Certain filters and room air cleaners are available that can help reduce particles indoors."
- Practical recommendations to reduce air pollution exposure should be given to at-risk patients. Although unproven to reduce cardiovascular events, there are a number of prudent and feasible measures, including reducing optional or unnecessary exposures. Additional measures could include eliminating or reducing nonmandatory travel to highly polluted regions and avoiding exposures or outdoor activities (eg, exercising, commut-

ing) during highly polluted times (eg, rush hours) or in proximity to major sources of pollution (eg, roadways, industrial sources). Choosing to exercise indoors with windows closed and using efficient air conditioning and filtering systems may be prudent for certain high-risk patients, particularly during peak pollution periods. Indeed, not only can central air conditioners reduce the indoor exposure level to PM from outdoor sources, there is some evidence that they might reduce the risk for cardiovascular hospitalizations associated with higher ambient pollution levels.[423] If travel/commutes cannot be avoided, maintaining optimal car filter systems, driving with windows closed, and recycling inside vehicle air may help reduce PM exposures (http://www.catf.us/projects/diesel/).[424,425]

However, at present, no specific recommendations regarding the appropriateness of undertaking more aggressive measures, even those shown to provide some benefits in a few studies (eg, wearing facemasks, installing PM filters in households), can be made based on the limited evidence. Similarly, although measures that decrease long-term PM exposures may produce even greater cardiovascular health benefits than the provided recommendations that focus on reducing short-term exposures, no specific recommendations (eg, moving to less polluted regions) can be prudently made at this time given the limited evidence. We acknowledge that occupational and indoor sources along with secondhand tobacco smoke are additional significant sources of personal PM exposures that should be avoided or reduced as much as possible. Finally, in developing nations, reducing exposure to indoor cooking sources of PM and air pollution from biomass combustion is a major issue of concern.[426] Additional suggestions are available on the EPA World Wide Web site.

Finally, although the existing evidence supports a causal relationship between $PM_{2.5}$ and cardiovascular mortality, we acknowledge the importance of continued research in areas of controversy and uncertainty to further understand the full nature of this issue. Although numerous insights have greatly enhanced our understanding of the PM-cardiovascular relationship since the first AHA statement was published,[1] the following list represents broad strategic avenues for future investigation:

Mechanistic Studies

- Better describe the physiological relevance in humans and the fundamental details of the mechanisms underlying the intermediate general mediating pathways (ie, PM or constituent transport into the circulation versus effects of inflammatory cytokines or activated immune cells versus ANS imbalance or other pathways) through which PM inhalation might mediate cardiovascular effects remote from the site of pulmonary deposition.
- Understand the clinical significance and relative importance of the observed biological responses (eg, vascular dysfunction, thrombosis, arrhythmia, ANS imbalance) in relation to the various causes of PM-mediated cardiovascular morbidity and mortality.
- Examine the efficacy of preventive measures (eg, patient education) and treatment modalities (eg, statins, antioxidants, fish oil, treatment of traditional risk factors, and reducing exposures by engineering controls, including filtration, personal protection via facemasks, or behavior modification) on cardiovascular health outcomes.
- Investigate the interaction between preexisting traditional cardiovascular risk factors (eg, diabetes, hypertension) and PM exposure, as well as the potential of air pollutants to exacerbate or worsen these risk factors. Determine the extent to which treatment of such factors (eg, with statins, aspirin, or angiotensin-converting enzyme inhibitors), especially among patients with known CVD, may modify the risk associated with PM exposure.
- Describe the biological effects of acute on top of chronic exposures (eg, synergistic effects versus reduced susceptibility to acute exposures due to augmented protective mechanisms).
- Determine the ability of long-term exposure to precipitate the development of chronic diseases, including clinically relevant atherosclerosis, hypertension, diabetes, and other vascular, metabolic, renal, or neurological diseases.

Epidemiological and Exposure Studies

- Expand our knowledge related to the "responsible" PM pollution constituents (eg, metals, organic compounds, semiquinones, endotoxin, and VOC and SVOC compounds), size fractions (eg, UFPs), sources (eg, traffic, power generation, and biomass combustion), and mixtures of pollutants.
- Investigate the cardiovascular health implications and importance of regional and intracity differences in composition and combinations of pollutants.
- Better understand the effects of mixtures of ambient pollutants (ie, potential synergism between PM and gaseous or vapor-phase pollutants such as ozone).
- Investigate the feasibility and utility of quantifying risk coefficients (concentration-response functions) according to PM source or relevant indices of pollutant mixtures, as a function of susceptibility (eg, age, preexisting disease), for reliable application in integrated, multipollutant risk assessments.
- Investigate the relative importance of various time frames of exposure in relation to PM causing cardiovascular events, including the relevance of epochs not well described, such as ultra-acute peak PM excursions (eg, 1 to 2 hours) and exposures of intermediate duration (eg, 1 to 12 months).
- Better document the time course and specific cardiovascular health benefits induced by reductions in PM.
- Better define susceptible individuals or vulnerable populations.
- Determine whether any "safe" PM threshold concentration exists that eliminates both acute and chronic cardiovascular effects in healthy and susceptible individuals and at a population level.

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Ana V. Diez-Roux	University of Michigan	EPA†; 1st EPA STAR grant to study the effects of long-term PM exposures on subclinical atherosclerosis and inflammatory markers in MESA; #2 is a subcontract to the University of Washington to participate in a long-term study of air pollution and progression of atherosclerosis, also in MESA	None	None	None	None	None	None
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Yuling Hong	American Heart Association‡	None	None	None	None	None	None	None
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Sanjay Rajagopalan	Ohio State University	None	None	Takeda*	None	None	None	None
David Siscovick	University of Washington	MESA AIR (ancillary study to MESA) funded by EPA†; NIEHS Discovery Center Study focused on air pollution and CVD†; NIH†	None	None	None	None	None	None
Sidney C. Smith, Jr	University of North Carolina	None	None	None	None	None	None	None
Laurie Whitsel	American Heart Association	None	None	None	None	None	None	None

This table represents the relationships of writing group members that may be perceived as actual or reasonably perceived conflicts of interest as reported on the Disclosure Questionnaire, which all members of the writing group are required to complete and submit. A relationship is considered to be "significant" if (1) the person receives $10 000 or more during any 12-month period, or 5% or more of the person's gross income; or (2) the person owns 5% or more of the voting stock or share of the entity, or owns $10 000 or more of the fair market value of the entity. A relationship is considered to be "modest" if it is less than "significant" under the preceding definition.

*Modest.

†Significant.

‡Dr Hong is currently with the Centers for Disease Control and Prevention, Atlanta, Ga.

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Reviewer	Employment	Research Grant	Other Research Support	Speakers' Bureau/Honoraria	Expert Witness	Ownership Interest	Consultant/Advisory Board	Other
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Doug Dockery	Harvard University	National Institute of Environmental Health Sciences†; Health Effects Institute†	None	None	None	None	Science Advisory Board to MESA Air Study, University of Washington*	None
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*Modest.

†Significant.

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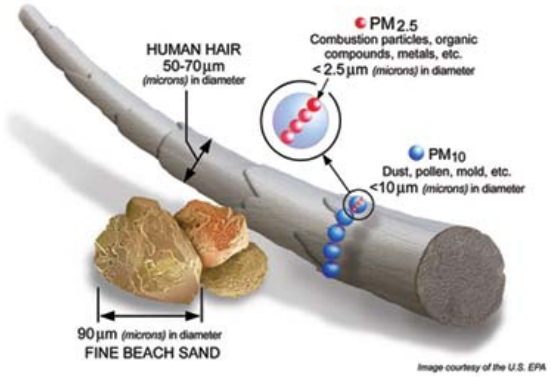
APPENDIX T

Particulate Matter

Particulate Matter (PM) Research



See a larger version of the image here

What is Particulate Matter?

Particulate matter (PM) is an air pollution term for a mixture of solid particles and liquid droplets found in the air. The pollutant comes in a variety of sizes and can be composed of many types of materials and chemicals. Particles that are small enough to be inhaled have the potential to cause health effects. Of particular concern is a class of particles known as fine particulate matter or PM2.5 that gets deep into the lung.

There are many sources of PM. The air pollutant can originate from natural processes, like forest fires and wind erosion, and from human activities, like agricultural practices, smokestacks, car emissions, and construction. Examples include dust, dirt, soot, soil, and smoke.

EPA Research on PM

EPA accelerated its investigations of fine particulate matter (PM2.5) in 1998 to improve understanding of the potential health effects of the small particles in the outside air and to find ways to reduce risks from the air pollutant. Studies at that time had provided compelling evidence that air pollution particles were responsible for thousands of deaths and hospitalizations, as well as substantial loss of work and school days.

Research has since confirmed the links between exposure to PM2.5 and increases in respiratory health problems, hospitalizations and premature death. EPA's PM research also has affirmed the need for air quality standards to reduce PM in the air to protect human health. However, many questions remain about particles and why they are associated with such significant health effects.

Research is being conducted to better understand:

- Which attributes of particles may be causing these health effects?
- Who may be most susceptible to their effects?
- How are people exposed to PM air pollution?
- How are particles formed in the atmosphere?
- How should particles be measured so data is relevant to health protection?
- What are the contributions from various sources in the different regions of the country?

Areas of research include:

Atmospheric Science

To learn how PM forms, interacts and where it comes from and travels

Exposure Science

To learn how and where people become exposed to PM

Health Effects Research

To understanding the impacts PM has on human health, particularly with those more susceptible

Sources Research

To describe and profile the many different sources of PM

Types of PM

Although it can be categorized in a number of ways, PM has traditionally been classified by size. In general, the smaller the particle, the stronger its potential impact on human health because it can be more easily inhaled. For this reason, EPA monitors and regulates particles in two size categories depending on their predicted penetration into the lung. These categories are:

Coarse particles (PM10)

Inhalable particles less than 10 micrometers (μm) in diameter used as a nominal surrogate for particles between 2.5 and 10μm in diameter; found near roadways and dusty industries

Fine particles (PM2.5)

Inhalable particles less than 2.5μm in diameter; generally found in smoke and haze, emitted from natural sources like forest fires and industrial combustion sources, or formed when gases react in the air. Ultrafine particles (PM0.1) are a subset of inhalable PM2.5 particles less than 0.1μm in diameter. They are not specifically regulated but have a strong link to combustion and therefore are garnering special attention.

PM can also be classified by its **source**:

- Primary particles: directly emitted from a natural or human source
- Secondary particles: produced when chemicals from natural and human sources react in the atmosphere often energized by sunlight

Human Health Effects

Inhalable particles, particularly fine particles, have the greatest demonstrated impact on human health. Their small size allows them to get deep into the lungs and from there they can reach or trigger inflammation in the lung, blood vessels or the heart, and perhaps other organs. Studies have linked PM exposure to health problems such as:

- Irritation of the airways, coughing, and difficulty breathing
- Reduced lung function
- Aggravated asthma
- Chronic bronchitis
- Irregular heartbeat
- Nonfatal heart attacks
- Some cancers

Research has found that certain populations are more vulnerable to these health effects, such as people with pre-existing heart or lung diseases, children, and older adults.

Environmental Effects

Effects of PM on the environment include:

- Reduced visibility (haze)
- Increased acidity of lakes and streams
- Nutrient balance changes in coastal waters and river basins
- Reduced levels of nutrients in soil
- Damage to forests and crops
- Reduced diversity in ecosystems
- Damage to stone and other materials

History of PM

When fossil fuels empowered new industries in the late 1800s, most people saw smoky skies and haze as signs of a city's prosperity, and there was little concern about any health effects they might have. That perception started to change in the mid-20th century as severe episodes of air pollution (e.g., Meuse Valley, Belgium; Donora, PA; London, UK) were linked to spikes in death rates and hospitalization.

Soon groups began measuring soot levels and noticing actual correlations between urban fogs and death rates. In response, local areas began monitoring and regulating emissions of these particles, but regulatory efforts remained uneven and localized until national awareness about the environment rose in the 1960s.

In 1970, the Clean Air Act established the first set of National Ambient Air Quality Standards (NAAQS) and created EPA as a federal Agency to protect the public health and welfare.

At that time, Congress designated seven (now six) criteria air pollutants to be of particular concern. What little research existed had shown these to be ubiquitous urban pollutants with potential negative health effects. The concern was at a nationwide level, not just localized in a particular region, city or industrial area. EPA currently regulates six criteria pollutants (PM, ozone, sulfur oxides, nitrogen oxides, carbon monoxide, and lead) to protect public health and the environment.

In the 1980s, as many smoke stacks cleared their emissions, the air looked clean and standard health assessments generally concurred with these improvements. The irritating haze of urban smog and ozone became the next focus of interest as towns and cities with associated vehicular traffic began to grow and expand. However, new studies (some supported by EPA) in the 1990s, using novel methods, began to reveal the insidious nature of forgotten PM, particularly a smaller class of inhalable particles, PM2.5, that was associated with respiratory health problems, heart and lung disease, and premature death at levels once thought to be safe.

In 1997, in response to these findings, EPA established a new standard for allowable levels of PM2.5 in the ambient environment to augment the existing regulation for larger PM particles or PM10.

In 1998, Congress augmented EPA's budget and mandate for enhanced emphasis on PM research and specifically called for EPA to fund up to five PM Research Centers to address the PM issue over the next five years. In 2005, these grants were re-awarded for a second five-year cycle.

Controlling PM Emissions

Efforts by EPA, other organizations, and the general public have successfully reduced ambient levels of PM in the United States. Between 2000, when monitoring began, and 2007, average national levels of PM2.5 declined by 11%. Similarly, national levels of PM10 declined by 28% between 1990 and 2007. However, these declines were regionally uneven; some areas experienced bigger declines while others actually experienced increases in PM levels during this period.

Revisions to the Clean Air Act in 1990 required each state to develop a State Implementation Plan (SIP) describing how it will reach and maintain the national standards. These SIPs vary by state, but generally include local monitoring of PM levels, strategies to reduce PM emissions, and steps to evaluate these strategies. Individual actions that can also make a difference include recycling, using energy-efficient products and appliances, planting deciduous trees, and driving less.

Related Links:

- What are the Six Common Air Pollutants?
- Air Trends
- The Green Book Nonattainment Areas for Criteria Pollutants

Selected References:

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APPENDIX U

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Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution

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Abstract

Context—Associations have been found between day-to-day particulate air pollution and increased risk of various adverse health outcomes, including cardiopulmonary mortality. However, studies of health effects of long-term particulate air pollution have been less conclusive.

Objective—To assess the relationship between long-term exposure to fine particulate air pollution and all-cause, lung cancer, and cardiopulmonary mortality.

Design, Setting, and Participants—Vital status and cause of death data were collected by the American Cancer Society as part of the Cancer Prevention II study, an ongoing prospective mortality study, which enrolled approximately 1.2 million adults in 1982. Participants completed a questionnaire detailing individual risk factor data (age, sex, race, weight, height, smoking history, education, marital status, diet, alcohol consumption, and occupational exposures). The risk factor data for approximately 500000 adults were linked with air pollution data for metropolitan areas throughout the United States and combined with vital status and cause of death data through December 31, 1998.

Main Outcome Measure—All-cause, lung cancer, and cardiopulmonary mortality.

Results—Fine particulate and sulfur oxide–related pollution were associated with all-cause, lung cancer, and cardiopulmonary mortality. Each 10-μg/m^3 elevation in fine particulate air pollution was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

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Author Contributions: *Study concept and design:* Pope, Burnett, Krewski, Thurston.
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Analysis and interpretation of data: Pope, Burnett, Krewski, Thurston.
Drafting of the manuscript: Pope, Burnett, Ito, Thurston.
Critical revision of the manuscript for important intellectual content: Pope, Thun, Calle, Krewski, Thurston.
Statistical expertise: Pope, Burnett, Krewski. *Obtained funding:* Pope, Thun, Thurston. *Administrative, technical, or material support:* Pope, Calle, Krewski, Ito, Thurston. *Study supervision:* Pope, Krewski.

Conclusion—Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

Based on several severe air pollution events,[1-3] a temporal correlation between extremely high concentrations of particulate and sulfur oxide air pollution and acute increases in mortality was well established by the 1970s. Subsequently, epidemiological studies published between 1989 and 1996 reported health effects at unexpectedly low concentrations of particulate air pollution.[4] The convergence of data from these studies, while controversial,[5] prompted serious reconsideration of standards and health guidelines[6-10] and led to a long-term research program designed to analyze health-related effects due to particulate pollution.[11-13] In 1997, the Environmental Protection Agency adopted new ambient air quality standards that would impose regulatory limits on fine particles measuring less than 2.5 μm in diameter ($PM_{2.5}$). These new standards were challenged by industry groups, blocked by a federal appeals court, but ultimately upheld by the US Supreme Court.[14]

Although most of the recent epidemiological research has focused on effects of short-term exposures, several studies suggest that long-term exposure may be more important in terms of overall public health.[4] The new standards for long-term exposure to $PM_{2.5}$ were originally based primarily on 2 prospective cohort studies,[15,16] which evaluated the effects of long-term pollution exposure on mortality. Both of these studies have been subjected to much scrutiny,[5] including an extensive independent audit and reanalysis of the original data.[17] The larger of these 2 studies linked individual risk factor and vital status data with national ambient air pollution data.[16] Our analysis uses data from the larger study and (1) doubles the follow-up time to more than 16 years and triples the number of deaths; (2) substantially expands exposure data, including gaseous copollutant data and new $PM_{2.5}$ data, which have been collected since the promulgation of the new air quality standards; (3) improves control of occupational exposures; (4) incorporates dietary variables that account for total fat consumption, and consumption of vegetables, citrus, and high-fiber grains; and (5) uses recent advances in statistical modeling, including the incorporation of random effects and nonpara-metric spatial smoothing components in the Cox proportional hazards model.

METHODS

Study Population

The analysis is based on data collected by the American Cancer Society (ACS) as part of the Cancer Prevention Study II (CPS-II), an ongoing prospective mortality study of approximately 1.2 million adults.[18,19] Individual participants were enrolled by ACS volunteers in the fall of 1982. Participants resided in all 50 states, the District of Columbia, and Puerto Rico, and were generally friends, neighbors, or acquaintances of ACS volunteers. Enrollment was restricted to persons who were aged 30 years or older and who were members of households with at least 1 individual aged 45 years or older. Participants completed a confidential questionnaire, which included questions about age, sex, weight, height, smoking history, alcohol use, occupational exposures, diet, education, marital status, and other characteristics.

Vital status of study participants was ascertained by ACS volunteers in September of the following years: 1984, 1986, and 1988. Reported deaths were verified with death certificates. Subsequently, through December 31, 1998, vital status was ascertained through automated linkage of the CPS-II study population with the National Death Index.[19] Ascertainment of deaths was more than 98% complete for the period of 1982-1988 and 93% complete after 1988.[19] Death certificates or codes for cause of death were obtained for more than 98% of all known deaths. Cause of death was coded according to the International Classification of Diseases, Ninth Revision (ICD-9). Although the CPS-II cohort included approximately 1.2 million participants with adequate questionnaire and cause-of-death data, our analysis was restricted to those participants who resided in US metropolitan areas with available pollution data. The actual size of the analytic cohort varied depending on the number of metropolitan areas for which pollution data were available. **TABLE 1** provides the number of metropolitan areas and participants available for each source of pollution data.

Air Pollution Exposure Estimates

Each participant was assigned a metropolitan area of residence based on address at time of enrollment and 3-digit ZIP code area.[20] Mean (SD) concentrations of air pollution for the metropolitan areas were compiled from various primary data sources (Table 1). Many of the particulate pollution indices, including $PM_{2.5}$, were available from data from the Inhalable Particle Monitoring Network for 1979-1983 and data from the National Aerometric Database for 1980-1981, periods just prior to or at the beginning of the follow-up period. An additional data source was the Environmental Protection Agency Aerometric Information Retrieval System (AIRS). The mean concentration of each pollutant from all available monitoring sites was calculated for each metropolitan area during the 1 to 2 years prior to enrollment.[17]

Additional information on ambient pollution during the follow-up period was extracted from the AIRS database as quarterly mean values for each routinely monitored pollutant for 1982 through 1998. All quarterly averages met summary criteria imposed by the Environmental Protection Agency and were based on observations made on at least 50% of the scheduled sampling days at each site. The quarterly mean values for all stations in each metro politan area were calculated across the study years using daily average values for each pollutant except ozone. For ozone, daily 1-hour maximums were used and were calculated for the full year and for the third quarter only (ie, July, August, September). While gaseous pollutants generally had recorded data throughout the entire follow-up period of interest, the particulate matter monitoring protocol changed in the late 1980s from total suspended particles to particles measuring less than 10 μm in diameter (PM_{10}), resulting in the majority of total suspended particle data being available in the early to mid-1980s and PM_{10} data being mostly available in the early to mid-1990s.

As a consequence of the new $PM_{2.5}$ standard, a large number of sites began collecting $PM_{2.5}$ data in 1999. Daily $PM_{2.5}$ data were extracted from the AIRS database for 1999 and the first 3 quarters of 2000. For each site, quarterly averages for each of the 2 years were computed. The 4 quarters were averaged when at least 1 of the 2 corresponding quarters for each year

had at least 50% of the sixth-day samples and at least 45 total sampling days available. Measurements were averaged first by site and then by metropolitan area. Although no network of $PM_{2.5}$ monitoring existed in the United States between the early 1980s and the late 1990s, the integrated average of $PM_{2.5}$ concentrations during the period was estimated by averaging the $PM_{2.5}$ concentration for early and later periods.

Mean sulfate concentrations for 1980-1981 were available for many cities based on data from the Inhalable Particle Monitoring Network and the National Aerometric Database. Recognizing that sulfate was artifactually overestimated due to glass fiber filters used at that time, season and region-specific adjustments were made.[17] Since few states analyzed particulate samples for sulfates after the early 1980s, individual states were directly contacted for data regarding filter use. Ion chromatography was used to analyze PM_{10} filters and this data could be obtained from metropolitan areas across the United States. Filters were collected for a single reference year (1990) in the middle of the 1982-1998 study period. The use of quartz filters virtually eliminated the historical overestimation of sulfate. Mean sulfate concentrations for 1990 were estimated using sulfate from AIRS, data reported directly from individual states, and analysis of archived filters.

Statistical Analysis

The basic statistical approach used in this analysis is an extension of the standard Cox proportional hazards survival model,[21] which has been used for risk estimates of pollution-related mortality in previous longitudinal cohort studies.[15,16] The standard Cox model implicitly assumes that observations are statistically independent after controlling for available risk factors, resulting in 2 concerns with regard to risk estimates of pollution-related mortality.[22] First, if the assumption of statistical independence is not valid, the uncertainty in the risk estimates of pollution-related mortality may be misstated. Second, even after controlling for available risk factors, survival times of par ticipants living in communities closer together may be more similar than participants living in communities farther apart, which results in spatial autocorrelation. If this spatial autocorrelation is due to missing or systematically mismeasured risk factors that are spatially correlated with air pollution, then the risk estimates of pollution-related mortality may be biased due to inadequate control of these factors. Therefore, in this analysis, the Cox proportional hazards model was extended by incorporating a spatial random-effects component, which provided accurate estimates of the uncertainty of effect estimates. The model also evaluated spatial autocorrelation and incorporated a nonparametric spatial smooth component (to account for unexplained spatial structure). A more detailed description of this modeling approach is provided elsewhere.[22]

The baseline analysis in this study estimated adjusted relative risk (RR) ratios for mortality by using a Cox proportional hazards model with inclusion of a metropolitan-based random-effects component. Model fitting involved a 2-stage process. In the first stage, survival data were modeled using the standard Cox proportional hazards model, including individual level covariates and indicator variables for each metropolitan area (without pollution variables). Output from stage 1 provided estimates of the metropolitan-specific logarithm of the RRs of mortality (relative to an arbitrary reference community), which were adjusted for individual

risk factors. The correlation between these values, which was induced by using the same reference community, was then removed.[23] In the second stage, the estimates of adjusted metropolitan-specific health responses were related to fine particulate air pollution using a linear random-effects regression model.[24] The time variable used in the models was survival time from the date of enrollment. Survival times of participants who did not die were censored at the end of the study period. To control for age, sex, and race, all of the models were stratified by 1-year age categories, sex, and race (white vs other), which allowed each category to have its own baseline hazard. Models were estimated for all-cause mortality and for 3 separate mortality categories: cardiopulmonary (*ICD-9* 401-440 and 460-519), lung cancer (*ICD-9* 162), and all others.

Models were estimated separately for each of the 3 fine particle variables, $PM_{2.5}$ (1979-1983), $PM_{2.5}$ (1999-2000), and $PM_{2.5}$ (average). Individual level covariates were included in the models to adjust for various important individual risk factors. All of these variables were classified as either indicator (ie, yes/no, binary, dummy) variables or continuous variables. Variables used to control for tobacco smoke, for example, included both indicator and continuous variables. The smoking indicator variables included: current cigarette smoker, former cigarette smoker, and a pipe or cigar smoker only (all vs never smoking) along with indicator variables for starting smoking before or after age 18 years. The continuous smoking variables included: current smoker's years of smoking, current smoker's years of smoking squared, current smoker's cigarettes per day, current smoker's cigarettes per day squared, former smoker's years of smoking, former smoker's years of smoking squared, former smoker's cigarettes per day, former smoker's cigarettes per day squared, and the number of hours per day exposed to passive cigarette smoke.

To control for education, 2 indicator variables, which indicated completion of high school or education beyond high school, were included. Marital status variables included indicator variables for single and other vs married. Both body mass index (BMI) values and BMI values squared were included as continuous variables. Indicator variables for beer, liquor, and wine drinkers and nonresponders vs non-drinkers were included to adjust for alcohol consumption. Occupational exposure was controlled for using various indicator variables: regular occupational exposure to asbestos, chemicals/ acids/solvents, coal or stone dusts, coal tar/pitch/asphalt, diesel engine exhaust, or formaldehyde, and additional indicator variables that indicated 9 different rankings of an occupational dirtiness index that has been developed and described elsewhere.[17,25] Two diet indices that accounted for fat consumption and consumption of vegetables, citrus, and high-fiber grains were derived based on information given in the enrollment questionnaire.[18] Quintile indicator variables for each of these diet indices were also included in the models.[18]

In addition to the baseline analysis, several additional sets of analysis were conducted. First, to more fully evaluate the shape of the concentration-response function, a robust locally weighted regression smoother[26] (within the generalized additive model framework[27]) was used to estimate the relationship between particulate air pollution and mortality in the second stage of model fitting. Second, the sensitivity of the fine particle mortality risk estimates compared with alternative modeling approaches and assumptions was evaluated. Standard Cox proportional hazards models were fit to the data including particulate air

pollution as a predictor of mortality and sequentially adding (in a controlled forward stepwise process) groups of variables to control for smoking, education, marital status, BMI, alcohol consumption, occupational exposures, and diet.

In addition, to evaluate the sensitivity of the estimated pollution effect while more aggressively controlling for spatial differences in mortality, a 2-dimensional term to account for spatial trends was added to the models and was estimated using a locally weighted regression smoother. The "span" parameter, which controls the complexity of the surface smooth, was set at 3 different settings to allow for increasingly aggressive fitting of the spatial structure. These included a default span of 50%, the span that resulted in the lowest unexplained variance in mortality rate between metropolitan areas, and the span that resulted in the strongest evidence (highest *P* value) to suggest no residual spatial structure. The risk estimates and SEs (and thus the confidence intervals) were estimated using generalized additive modeling[27] with S-Plus statistical software,[28] which provides unbiased effect estimates, but may underestimate SEs if there is significant spatial autocorrelation and significant correlations between air pollution and the smoothed surface of mortality. Therefore, evidence of spatial autocorrelation was carefully evaluated and tested using the Bartlett test.[29] The correlations of residual mortality with distance between metropolitan areas were graphically examined.

Analyses were also conducted of effect modification by age, sex, smoking status, occupational exposure, and education. Finally, models were fit using a variety of alternative pollution indices, including gaseous pollutants. Specifically, models were estimated separately for each of the pollution variables listed in Table 1, while also including all of the other risk factor variables.

RESULTS

Fine particulate air pollution generally declined in the United States during the follow-up period of this study. **FIGURE 1** plots mean $PM_{2.5}$ concentrations for 1999-2000 over mean $PM_{2.5}$ concentrations for 1979-1983 for the 51 cities in which paired data were available. The concentrations of $PM_{2.5}$ were lower in 1999-2000 than in 1979-1983 for most cities, with the largest reduction observed in the cities with the highest concentrations of pollution during 1979-1983. Mean $PM_{2.5}$ levels in the 2 periods were highly correlated (r=0.78). The rank ordering of cities by relative pollution levels remained nearly the same. Therefore, the relative levels of fine particle concentrations were similar whether based on measurements at the beginning of the study period, shortly following the study period, or an average of the 2.

As reported in **TABLE 2**, all 3 indices of fine particulate air pollution were associated with all-cause, cardiopulmo-nary, and lung cancer mortality, but not mortality from all other causes combined. **FIGURE 2** presents the nonpara-metric smoothed exposure response relationships between cause-specific mortality and $PM_{2.5}$ (average). The log RRs for all-cause, cardiopulmonary, and lung cancer mortality increased across the gradient of fine particulate matter. Goodness-of-fit tests indicated that the associations were not significantly different from linear associations (P>.20).

The fine particle mortality RR ratios from various alternative modeling approaches and assumptions are presented in **FIGURE 3**. After controlling for smoking, education, and marital status, the controlled forward stepwise inclusion of additional covariates had little influence on the estimated associations with fine particulate air pollution on cardiopulmonary and lung cancer mortality. As expected, cigarette smoking was highly significantly associated with el evated risk of all-cause, cardiopulmo-nary, and lung cancer mortality ($P<.001$). Estimated RRs for an average current smoker (men and women combined, 22 cigarettes/day for 33.5 years, with initiation before age 18 years) were equal to 2.58, 2.89, and 14.80 for all-cause, cardiopulmonary, and lung cancer mortality, respectively. Statistically significant, but substantially smaller and less robust associations, were also observed for education, marital status, BMI, alcohol consumption, occupational exposure, and diet variables. Although many of these covariates were also statistically associated with mortality, the risk estimates of pollution-related mortality were not highly sensitive to the inclusion of these additional covariates.

Figure 3 also demonstrates that the introduction of the random-effects component to the model resulted in larger SEs of the estimates and, therefore, somewhat wider 95% confidence intervals. There was no evidence of statistically significant spatial autocorrelation in the survival data based on the Bartlett test (P[H11022].20) after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, graphical examination of the correlations of the residual mortality with distance between metropolitan areas did not reveal significant spatial autocorrelation (results not shown). Nevertheless, the incorporation of spatial smoothing was included to further investigate the robustness of the estimated particulate pollution effect. Effect estimates were not highly sensitive to the incorporation of spatial smoothing to account for regional clustering or other spatial patterns in the data.

FIGURE 4 presents fine particle air pollution–related mortality RR ratios after stratifying by age, sex, education, and smoking status, and adjusting for all other risk factors. The differences across age and sex strata were not generally consistent or statistically significant. However, a consistent pattern emerged from this stratified analysis: the association with particulate pollution was stronger for both cardiopulmonary and lung cancer mortality for participants with less education. Also, for both cardiopulmonary and lung cancer mortality, the RR estimates were higher for nonsmokers.

FIGURE 5 summarizes the associations between mortality risk and air pollutant concentrations listed in Table 1. Statistically significant and relatively consistent mortality associations existed for all measures of fine particulate exposure, including $PM_{2.5}$ and sulfate particles. Weaker less consistent mortality associations were observed with PM_{10} and PM_{15}. Measures of the coarse particle fraction ($PM_{15\text{-}2.5}$) and total suspended particles were not consistently associated with mortality. Of the gaseous pollutants, only sulfur dioxide was associated with elevated mortality risk. Interestingly, measures of $PM_{2.5}$ were associated with all-cause cardiopulmonary, and lung cancer mortality, but not with all other mortality. However, sulfur oxide pollution (as measured by sulfate particles and/or sulfur dioxide) was significantly associated with mortality from all other causes in addition to all-cause, cardiopulmo-nary, and lung cancer mortality.

COMMENT

Thisstudydemonstratedassociationsbetween ambient fine particulate air pollution and elevated risks of both cardio-pulmonary and lung cancer mortality. Each 10-μg/m^3 elevation in long-term average $PM_{2.5}$ ambient concentrations was associated with approximately a 4%, 6%, and 8% increased risk of all-cause, cardiopulmonary, and lung cancer mortality, respectively, although the magnitude of the effect somewhat depended on the time frame of pollution monitoring. In addition, this analysis addresses many of the important questions concerning the earlier, more limited analysis of the large CPS-II cohort, including the following issues.

First, does the apparent association between pollution and mortality persist with longer follow-up and as the cohort ages and dies? The present analysis more than doubled the follow-up time to more than 16 years, resulting in approximately triple the number of deaths, yet the associations between pollution and mortality persisted.

Second, can the association between fine particulate air pollution and increased cardiopulmonary and lung cancer mortality be due to inadequate control of important individual risk factors? After aggressively controlling for smoking, the estimated fine particulate pollution effect on mortality was remarkably robust. When the analysis was stratified by smoking status, the estimated pollution effect on both cardio-pulmonary and lung cancer mortality was strongest for never smokers vs former or current smokers. This analysis also controlled for education, marital status, BMI, and alcohol consumption. This analysis used improved variables to control for occupational exposures and incorporated diet variables that accounted for total fat consumption, as well as for consumption of vegetables, citrus, and high-fiber grains. The mortality associations with fine particulate air pollution were largely unaffected by the inclusion of these indi vidualriskfactorsinthemodels.Thedata on smoking and other individual risk factors, however, were obtained directly by questionnaire at time of enrollment and do not reflect changes that may have occurred following enrollment. The lack of risk factor follow-up data results in some misclassificationofexposure,reducesthe precision of control for risk factors, and constrains our ability to differentiate time dependency.

Third, are the associations between fine particulate air pollution and mortality due to regional or other spatial differences that are not adequately controlled for in the analysis? If there are unmeasured or inadequately modeled risk factors that are different across locations, then spatial clustering will occur. If this clustering is independent or random across metropolitan areas, then the spatial clustering can be modeled by adding a random-effects component to the Cox proportional hazards model as was done in our analysis. The clustering may not be independent or random across metropolitan areas due to inadequately measured or modeled risk factors (either individual or ecological). If these inadequately measured or modeled risk factors are also spatially correlated with air pollution, then biased pollution effects estimates may occur due to confounding. However, in this analysis, significant spatial autocorrelation was not observed after controlling for fine particulate air pollution and the various individual risk factors. Furthermore, to minimize any potential confounding bias, sensitivity analyses, which directly modeled spatial trends using

nonparametric smoothing techniques, were conducted. A contribution of this analysis is that it included the incorporation of both random effects and nonparametric spatial smoothing components to the Cox proportional hazards model. Even after accounting for random effects across metropolitan areas and aggressively modeling a spatial structure that accounts for regional differences, the association between fine particulate air pollution and cardiopulmonary and lung cancer mortality persists.

Fourth, is mortality associated primarily with fine particulate air pollution or is mortality also associated with other measures of particulate air pollution, such as PM_{10}, total suspended particles, or with various gaseous pollutants? Elevated mortality risks were associated primarily with measures of fine particulate and sulfur oxide pollution. Coarse particles and gaseous pollutants, except for sulfur dioxide, were generally not significantly associated with elevated mortality risk.

Fifth, what is the shape of the concentration-responsefunction?Withinthe range of pollution observed in this analysis, the concentration-response function appears to be monotonic and nearly linear. However, this does not preclude a leveling off (or even steepening) at much higher levels of air pollution.

Sixth, how large is the estimated mortality effect of exposure to fine particulate air pollution relative to other risk factors? A detailed description and interpretationofthemanyindividualrisk factors that are controlled for in the analysis goes well beyond the scope of this report. However, the mortality risk associated with cigarette smoking has been well documented using the CPS-II cohort.[16] The risk imposed by exposure to fine particulate air pollution is obviously much smaller than the risk of cigarette smoking. Another risk factor that has been well documented using the CPS-II cohort data is body mass as measured by BMI.[30] The Word Health Organization has categorized BMI values between 18.5-24.9 kg/m^2 as normal; 25- 29.9 kg/m^2, grade 1 overweight; 30- 39.9 kg/m^2, grade 2 overweight; and 40 kg/m^2 or higher, grade 3 overweight.[31] In the present analysis, BMI values and BMI values squared were included in the proportional hazards models. Consistent with previous ACS analysis,[30] BMI was significantly associated with mortality, optimal BMI was between approximately 23.5 and 24.9 kg/m^2, and the RR of mortality for different BMI values relative to the optimal were dependent on sex and smoking status. For example, the RRs associated with BMI values between 30.0 and 31.9 kg/m^2 (vs optimal) would be up to approxi mately 1.33 for never smokers. Based on these calculations, mortality risks associated with fine particulate air pollution at levels found in more polluted US metropolitan areas are less than those associated with substantial obesity (grade 3 overweight), but comparable with the estimated effect of being moderately overweight (grade 1 to 2).

In conclusion, the findings of this study provide the strongest evidence to date that long-term exposure to fine particulate air pollution common to many metropolitan areas is an important risk factor for cardiopulmonary mortality. In addition, the large cohort and extended follow-up have provided an unprecedented opportunity to evaluate associations between air pollution and lung cancer mortality. Elevated fine particulate air pollution exposures were associated with significant increases in lung cancer mortality. Although

potential effects of other unaccounted for factors cannot be excluded with certainty, the associations between fine particulate air pollution and lung cancer mortality, as well as cardiopulmonary mortality, are observed even after controlling for cigarette smoking, BMI, diet, occupational exposure, other individual risk factors, and after controlling for regional and other spatial differences.

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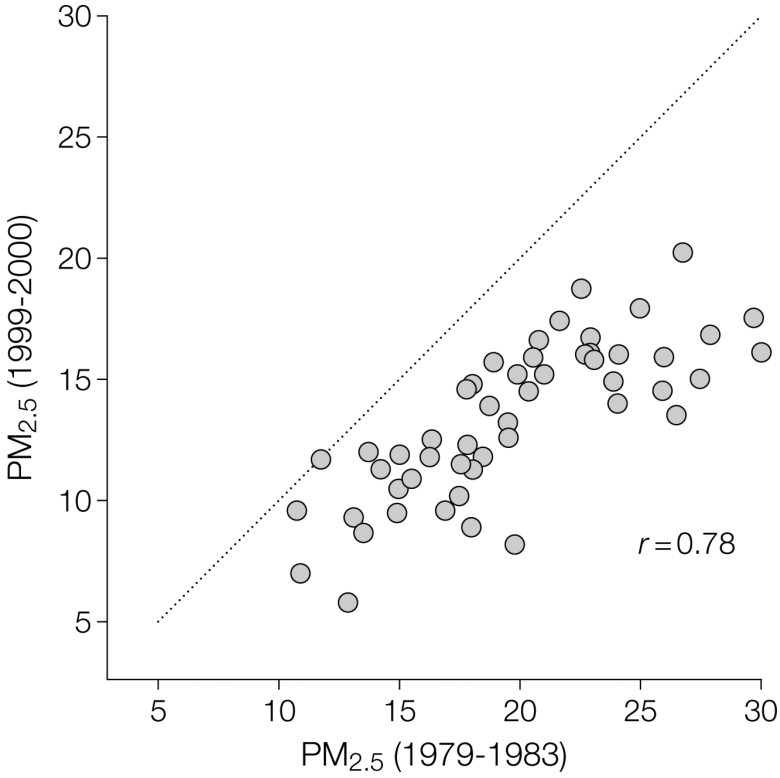

Figure 1.
Mean Fine Particles Measuring Less Than 2.5 μm in Diameter ($PM_{2.5}$)

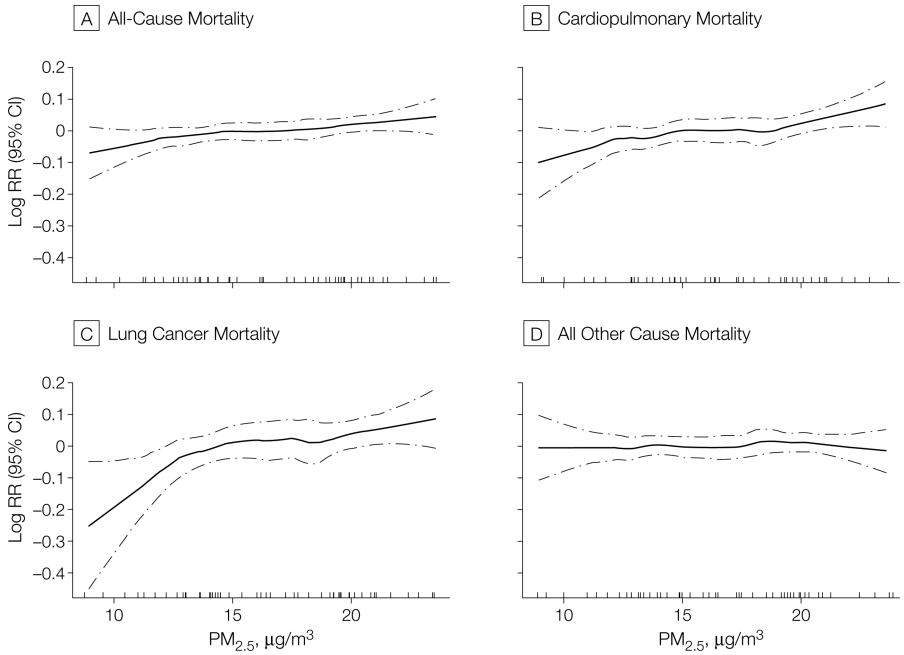

Figure 2.
Nonparametric Smoothed Exposure Response Relationship

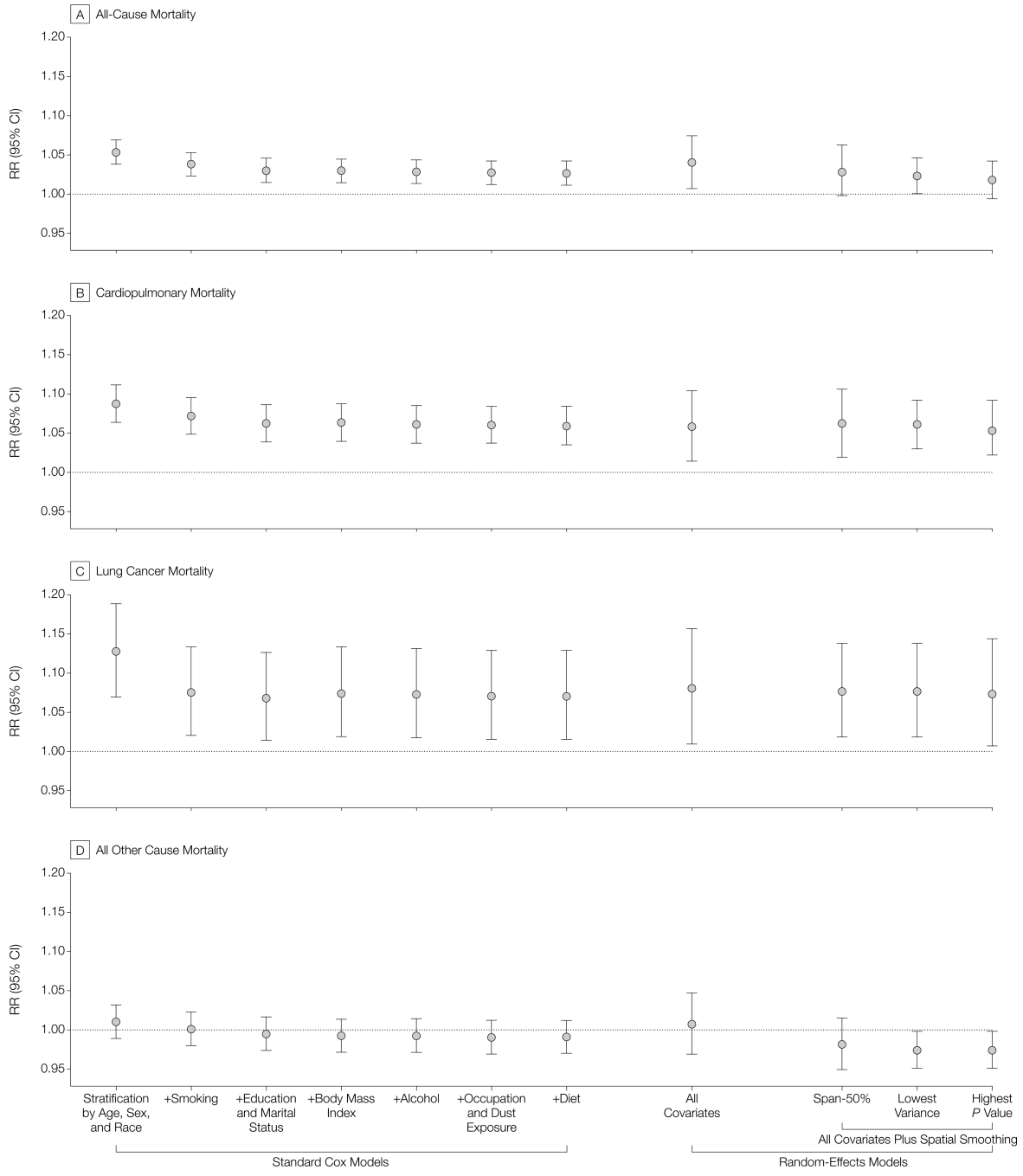

Figure 3.

Mortality Relative Risk (RR) Ratio Associated With 10-μg/m^3 Differences of $PM_{2.5}$ Concentrations

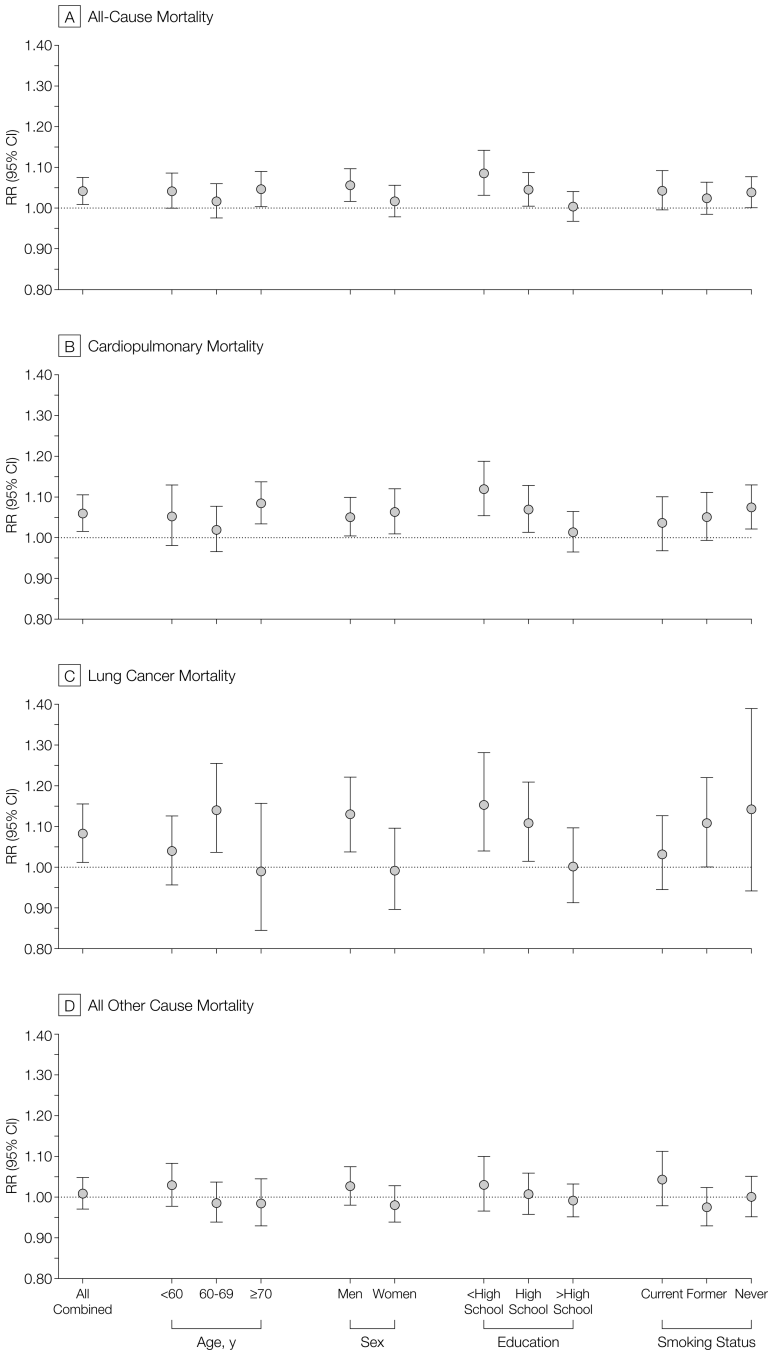

Figure 4.

Adjusted Mortality Relative Risk (RR) Ratio Associated With 10-μg/m^3 Differences of $PM_{2.5}$ Concentrations

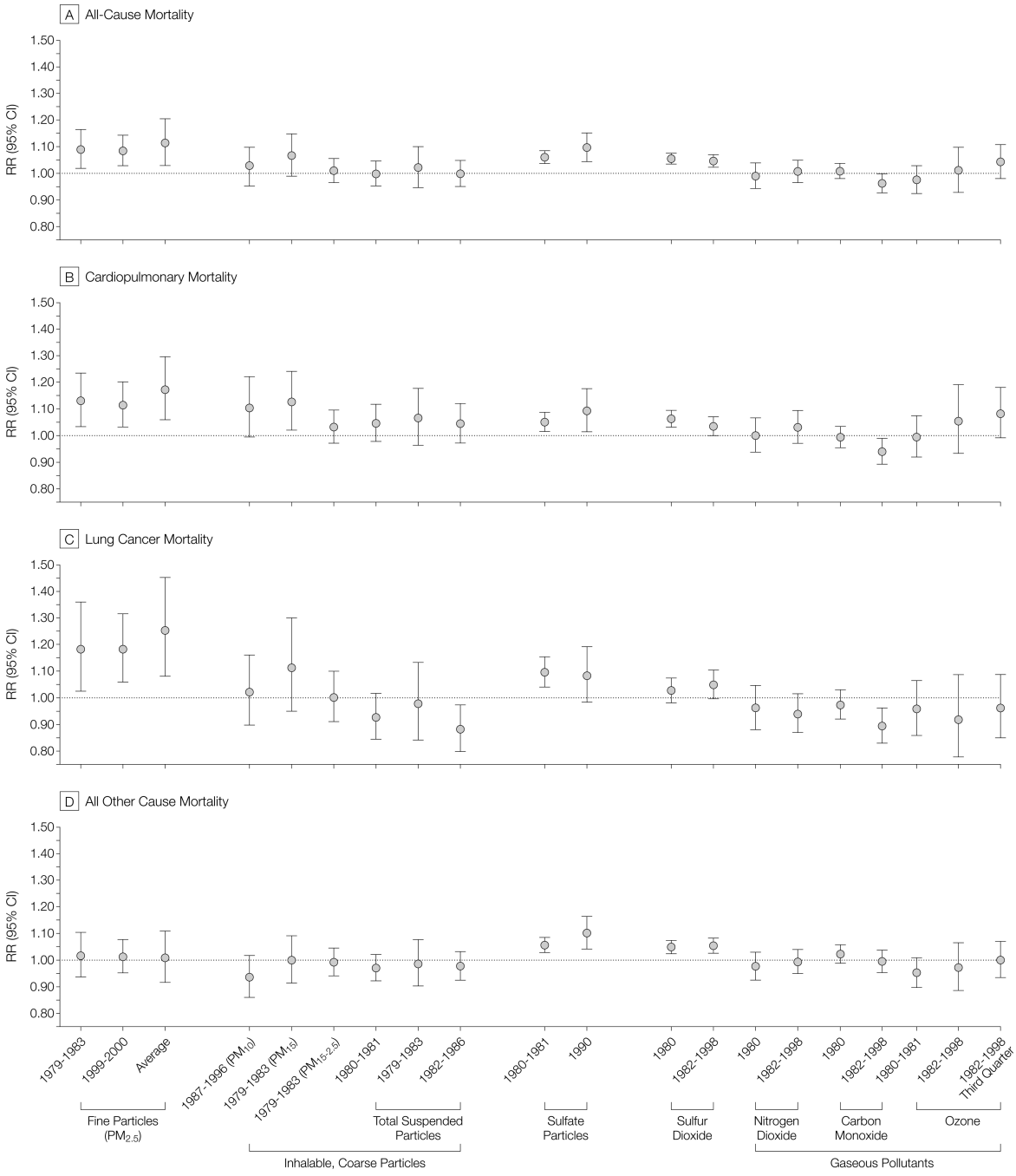

Figure 5.

Adjusted Mortality Relative Risk (RR) Ratio Evaluated at Subject-Weighted Mean Concentrations

Table 1

Summary of Alternative Pollution Indices*

Pollutant (Years of Data Collection)	Units	Source of Data	Data Compilation Team†	No. of Metropolitan Areas	No. of Participants, in Thousands	Mean (SD)
$PM_{2.5}$	$\mu g/m^3$					
1979-1983		IPMN	HEI	61	359	21.1 (4.6)
1999-2000		AIRS	NYU	116	500	14.0 (3.0)
Average				51	319	17.7 (3.7)
PM_{10}	$\mu g/m^3$					
1982-1998		AIRS	NYU	102	415	28.8 (5.9)
PM_{15}	$\mu g/m^3$					
1979-1983		IPMN	HEI	63	359	40.3 (7.7)
$PM_{15-2.5}$	$\mu g/m^3$					
1979-1983		IPMN	HEI	63	359	19.2 (6.1)
Total suspended particles	$\mu g/m^3$					
1980-1981		NAD	HEI	156	590	68.0 (16.7)
1979-1983		IPMN	HEI	58	351	73.7 (14.3)
1982-1998		AIRS	NYU	150	573	56.7 (13.1)
Sulfate	$\mu g/m^3$					
1980-1981		IPMN and NAD, artifact adjusted	HEI	149	572	6.5 (2.8)
1990		Compilation and analysis of PM_{10} filters	NYU	53	269	6.2 (2.0)
Sulfur dioxide	ppb	AIRS				
1980			HEI	118	520	9.7 (4.9)
1982-1998			NYU	126	539	6.7 (3.0)
Nitrogen dioxide	ppb	AIRS				
1980			HEI	78	409	27.9 (9.2)
1982-1998			NYU	101	493	21.4 (7.1)
Carbon monoxide	ppm	AIRS				
1980			HEI	113	519	1.7 (0.7)
1982-1998			NYU	122	536	1.1 (0.4)
Ozone	ppb	AIRS				
1980			HEI	134	569	47.9 (11.0)
1982-1998			NYU	119	525	45.5 (7.3)
1982-1998‡			NYU	134	557	59.7 (12.8)

*$PM_{2.5}$ indicates particles measuring less than 2.5 μm in diameter; PM_{10}, particles measuring less than 10 μm in diameter; PM_{15}, particles measuring less than 15 μm in diameter; $PM_{15-2.5}$, particles measuring between 2.5 and 15 μm in diameter; $\mu g/m^3$, micrograms per cubic meter; ppb, parts per billion; ppm, parts per million; IPMN, Inhalable Particle Monitoring Network; AIRS, Aerometric Information Retrieval System [Environmental Protection Agency]; and NAD, National Aerometric Database.

† HEI indicates data were compiled by the Health Effects Institute reanalysis team, which was previously published.[17] NYU indicates data were compiled at the New York University School of Medicine, Nelson Institute of Environmental Medicine (K.I. and G.D.T.).

‡ Daily 1-hour maximums were used. Values were calculated only for the third quarter (ie, July, August, September).

Table 2

Adjusted Mortality Relative Risk (RR) Associated With a 10-μg/m^3 Change in Fine Particles Measuring Less Than 2.5 μm in Diameter

	Adjusted RR (95% CI)*		
Cause of Mortality	**1979-1983**	**1999-2000**	**Average**
All-cause	1.04 (1.01-1.08)	1.06 (1.02-1.10)	1.06 (1.02-1.11)
Cardiopulmonary	1.06 (1.02-1.10)	1.08 (1.02-1.14)	1.09 (1.03-1.16)
Lung cancer	1.08 (1.01-1.16)	1.13 (1.04-1.22)	1.14 (1.04-1.23)
All other cause	1.01 (0.97-1.05)	1.01 (0.97-1.06)	1.01 (0.95-1.06)

* Estimated and adjusted based on the baseline random-effects Cox proportional hazards model, controlling for age, sex, race, smoking, education, marital status, body mass, alcohol consumption, occupational exposure, and diet. CI indicates confidence interval.

APPENDIX V

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Significance of biomass open burning on the levels of polychlorinated dibenzo-*p*-dioxins and dibenzofurans in the ambient air

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Abstract

In southern Taiwan, two areas (L- and Y-) with/without biomass open burning were selected to compare the PCDD/F concentrations and their congener profiles in the ambient air. The results of this study indicate that biomass (rice straw) open burning exhibited a significant impact on the PCDD/F concentration level in the ambient air. During the biomass burning season, the total PCDD/F I-TEQ concentrations in the ambient air of L- and Y-areas were approximately 4 and 17 times higher than those without biomass open burning, respectively. When 10% mass fraction of rice straw was burned, the contribution fraction of biomass burning on annual total PCDD/F I-TEQ emission was 3.28 and 8.11% for KC County and for Taiwan, respectively; however, when the calculation was on a weekly basis, the contribution fraction of biomass burning on weekly total PCDD/F I-TEQ emission was 30.6 and 53.4% for KC County and for Taiwan, respectively. The results of this study imply that during the week of biomass burning, it appears to be the most significant source of total I-TEQ PCDD emission. The results of this research can be applied to the study of other agricultural areas.

Keywords: Significance; Biomass open burning; PCDD/Fs; Ambient air

1. Introduction

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) have been found in the stack gas and fly ash of municipal solid waste incinerators [1], and have been extensively studied owing to their toxicity and associated adverse health effects [2–4]. In the USA, municipal waste incineration was the largest PCDD/F emission source (40.5%, 1393.5 rel. to 3444 g TEQ total) in the year 1995, but it contributed only 5.89% (83.8/1422) of the total in 2000. However, for the same year in the USA, the backyard barrel burning of refuse (one type of biomass burning) was the top PCDD/F emission source (35.1%, 498.5/1422) [5]. With regard to the sources of PCDD/F emission, biomass burning has become more and more important.

In open burning, due to its less than ideal combustion conditions, the air pollutant emission is greater than from well-controlled combustion sources on a mass pollutant per mass fuel (emission factor) basis. The emissions are also not spread evenly throughout the year; rather, they are typically episodic in time or season and localized/regionalized. Agricultural activities employ open burning as a rapid method for disposing of crop residue, releasing nutrients for the next growing cycle, and clearing land. Meanwhile, biomass open burning is also a large source of emission on a global scale in comparison to other broad classes of sources (e.g. mobile and industrial sources) [6].

The open burning of biomass during agricultural debris and forest fires, wildfires and land-clearing operations has been found to release significant amounts of polycyclic aromatic hydrocarbons (PAHs) [7,8]. Additionally, Hays et al. [9] reported that the combustion-derived PM (particulate matter) emission from wheat is enriched in potassium, K (31%, (w/w)) and chlorine, Cl (36%, w/w), whereas the PM emission from rice is largely carbonaceous (84%, w/w). PCDD/PCDFs can be formed

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from any combustion process where organic carbon, oxygen and chlorine are present [10,11].

Due to its incomplete combustion, except PAHs and PCDD/Fs, the biomass open burning typically produces soot and particulate matter (PM) that are visible as a smoke plume, carbon monoxide (CO), methane (CH_4) and volatile organic compound (VOCs) such as benzene. Depending on the sources, varying amounts of metals such as lead (Pb) or mercury (Hg) may be emitted [6].

Rice is a popular crop globally, thus, research concerning PCDD/F emissions from burning of its residues (rice straw) has drawn considerable attention recently. Laboratory pyrolytic experiments (2 L/min air, 700 °C) with rice straw showed PCDD/F emissions at 6 and 22 ng I-TEQ/kg of raw biomass [12]. Gullett and Touati [11] generated an initial PCDD/F emission factor of 0.5 ng toxic equivalency (TEQ)/kg from the combustion of wheat and rice. Lin et al. [13] reported that emissions from open burning of rice straw were 4200 and 158,800 tonnes/year for the KC area and Taiwan, respectively. Furthermore, Kao et al. [4] measured the concentrations of PCDD/Fs in ambient air which were impacted by the open burning of rice straw residue. They reported that the mean PCDD/F concentration (0.409 pg I-TEQ/Nm^3) in ambient air at a rice straw field was 4.6 times higher than that (0.089 pg I-TEQ/Nm^3) before open burning.

When industrial sources lower their emissions in response to environmental regulations, non-industrial sources such as open burning began to dominate the emission inventory [6]. Thus, the study of PCDD/F emission during rice straw burning has practical significance. Even though Gullett and Touati [11] concluded that wheat and rice straw burning is an apparently minor source of PCDD/Fs in the USA, the impact of biomass (rice) open burning on the levels of PCDD/Fs in the ambient air should not be set aside, especially on a short-term basis.

In Taiwan, the current study selected two areas (L- and Y-) with/without rice straw burning for comparing the PCDD/F concentrations and their congener profiles in the ambient air. The effect of biomass open burning on the ambient air quality of PCDD/F emission was presented and discussed.

2. Experimental

The current study selected two areas, Y- and L-, situated in southern Taiwan. Most people who live in these two areas earn a living by planting agricultural crops, with rice the most common. Five and six sampling sites were chosen (LA, LB, LC, LD and LE for L-area and YA, YB, YC, YD, YE and YF for Y-area), and each sampling site was located close to a field. August and December were the seasons the present study selected to do the sampling campaigns. In August, there was no biomass burning in the fields, as the crops were growing in these two areas. Meanwhile, in December, a very significant amount of agricultural waste (rice straw biomass) was burned to remove mosquitoes and other pests in the fields.

Each ambient air sample was collected using a PS-1 sampler (Graseby Andersen, GA) according to the revised EPA Reference Method T09A. The sampling flow rate was specified at ~0.225 m^3/min. Each sample was collected continuously on three consecutive days. The PS-1 sampler was equipped with a quartz-fiber filter for sampling particle-phase PCDD/Fs and followed by a glass cartridge for sampling gas-phase PCDD/Fs. Prior to sampling, XAD-2 resin was spiked with PCDD/Fs surrogate standards. To ensure the collected samples were contamination-free, one trip blank and one field blank were also taken when the field sampling was conducted [14].

Analyses of ambient air samples followed the US EPA Reference Method T09A. All chemical analyses were performed in the Super Micro Mass Research and Technology Centre of Cheng Shiu University. This centre is the first lab certified by the Taiwan EPA to analyze PCDD/Fs in Taiwan and has passed the international inter-calibration on PCDD/Fs in fly ash, sediment, mother's milk, human blood and cod liver. The sample analysis was performed according to the standard procedures [3,13–15]. Each collected sample was spiked with a known amount of the internal standard. After being extracted for 24 h, the extract was concentrated, treated with concentrated sulfuric acid, and this was then followed by a series of sample cleanup and fractionation procedures. The eluent was concentrated to ~1 ml, then transferred to a vial, and then further concentrated to near dryness by using a nitrogen stream. Prior to PCDD/Fs analysis, the standard solution was added to the sample to ensure recovery during the analysis process.

A high-resolution gas chromatograph (HRGC), coupled with a high-resolution mass spectrometer (HRMS), was used for the PCDD/Fs measurements. The HRGC was a Hewlett Packard 6970 series gas chromatograph, equipped with a DB-5 (J&W Scientific, CA, USA) fused silica capillary column (60 m, 0.25 mm i.d., 0.25 μm film thickness), and splitless injection. The initial oven temperature was 150 °C, and the temperature was programmed as follows: 150 °C, held for 1 min, increased by 30 °C/min to 220 °C, held for 12 min, increased at 1.5 °C/min to 240 °C, held for 20 min. Helium was used as the carrier gas. The HRMS was a Micromass Autospec Ultima (UK) mass spectrometer with a positive electron impact (EI+) source. The analyzer mode was selected ion monitoring (SIM) with a resolving power of 10,000. The electron energy was set at 35 eV, and the source temperature was set at 250 °C. An CTC A200S autosampler (CTC Analytics AG, GCPAL, Switzerland) was equipped with a pull-up speed of 55 μL/s and injection speed of 55 μL/s. Syringes for analyses were washed with two kinds of solvents: *n*-hexane and dichloromethane. The injection volume was 2 μL. The temperature of the injector and the interface was 300 °C.

3. Results and discussion

3.1. PCDD/Fs in the ambient air without biomass open burning

Tables 1 and 2 summarize the PCDD/F concentrations in the ambient air of L- and Y-areas, respectively, for both areas without biomass open burning during the sampling period. As can be seen from these two tables, the higher the total PCDD/F concentrations, the higher the total PCDD/F I-TEQ concentrations are at most sampling sites. Japan has an ambient air quality standard (JAQS) of 0.6 pg I-TEQ/Nm^3 [16], and those of L- and Y-areas

Table 1
PCDD/F concentrations in the ambient air of L-area without biomass open burning

PCDD/PCDFs (pg/Nm^3)	Location					
	LA	LB	LC	LD	LE	Mean
2,3,7,8-TeCDD	0.00498	0.00473	0.00295	0.00373	0.00117	0.00351
1,2,3,7,8-PeCDD	0.0117	0.0116	0.0102	0.0118	0.00313	0.00969
1,2,3,4,7,8-HxCDD	0.00907	0.00983	0.00848	0.00822	0.00320	0.00776
1,2,3,6,7,8-HxCDD	0.0198	0.0180	0.0160	0.0174	0.00627	0.0155
1,2,3,7,8,9-HxCDD	0.0158	0.0183	0.0131	0.0133	0.00440	0.0130
1,2,3,4,6,7,8-HpCDD	0.113	0.132	0.111	0.110	0.0429	0.102
OCDD	0.231	0.281	0.299	0.285	0.150	0.249
2,3,7,8-TeCDF	0.0459	0.0561	0.0366	0.0448	0.0152	0.0397
1,2,3,7,8-PeCDF	0.0543	0.0592	0.0422	0.0533	0.0155	0.0449
2,3,4,7,8-PeCDF	0.0625	0.0694	0.0505	0.0602	0.0221	0.0529
1,2,3,4,7,8-HxCDF	0.0657	0.0704	0.0541	0.0674	0.0192	0.0554
1,2,3,6,7,8-HxCDF	0.0634	0.0718	0.0520	0.0640	0.0178	0.0538
1,2,3,7,8,9-HxCDF	0.00405	0.00355	0.00347	0.00278	0.00149	0.00307
2,3,4,6,7,8-HxCDF	0.0588	0.0675	0.0493	0.0607	0.0186	0.0510
1,2,3,4,6,7,8-HpCDF	0.179	0.169	0.164	0.184	0.0508	0.149
1,2,3,4,7,8,9-HpCDF	0.0213	0.0268	0.0248	0.0262	0.0119	0.0222
OCDF	0.164	0.156	0.203	0.220	0.0430	0.157
PCDDs	0.405	0.475	0.461	0.450	0.211	0.400
PCDFs	0.718	0.749	0.679	0.783	0.216	0.629
PCDDs/PCDFs ratio	0.564	0.634	0.679	0.574	0.980	0.686
Total PCDD/Fs	1.12	1.22	1.14	1.23	0.427	1.03
PCDDs pg I-TEQ/Nm^3	0.0167	0.0167	0.0132	0.0149	0.00470	0.0132
PCDFs pg I-TEQ/Nm^3	0.0599	0.0667	0.0490	0.0590	0.0197	0.0509
PCDDs/PCDFs (TEQ) ratio	0.278	0.251	0.269	0.253	0.238	0.258
Total pg I-TEQ/Nm^3	0.0766	0.0835	0.0622	0.0739	0.0244	0.0641

Table 2
PCDD/F concentrations in the ambient air of Y-area without biomass open burning

PCDD/PCDFs (pg/Nm^3)	Location						
	YA	YB	YC	YD	YE	YF	Mean
2,3,7,8-TeCDD	0.00219	0.00269	0.00154	0.00361	0.00142	0.00257	0.00234
1,2,3,7,8-PeCDD	0.00437	0.00416	0.00256	0.00475	0.00161	0.00514	0.00377
1,2,3,4,7,8-HxCDD	0.00398	0.00275	0.00122	0.00241	0.00116	0.00341	0.00249
1,2,3,6,7,8-HxCDD	0.0133	0.00557	0.00211	0.00469	0.00194	0.0116	0.00654
1,2,3,7,8,9-HxCDD	0.00611	0.00352	0.00205	0.00253	0.00142	0.00585	0.00358
1,2,3,4,6,7,8-HpCDD	0.0395	0.0305	0.0159	0.0287	0.0123	0.0358	0.0271
OCDD	0.133	0.106	0.0774	0.125	0.0655	0.123	0.105
2,3,7,8-TeCDF	0.0167	0.0165	0.00921	0.0222	0.00626	0.0246	0.0159
1,2,3,7,8-PeCDF	0.0133	0.0116	0.00621	0.0151	0.00490	0.0163	0.0112
2,3,4,7,8-PeCDF	0.0192	0.0152	0.00806	0.0175	0.00529	0.0197	0.0142
1,2,3,4,7,8-HxCDF	0.0146	0.0108	0.00614	0.0123	0.00574	0.0144	0.0107
1,2,3,6,7,8-HxCDF	0.0150	0.0117	0.00570	0.0130	0.00484	0.0149	0.0109
1,2,3,7,8,9-HxCDF	0.00296	0.00167	0.00083	0.00158	0.000903	0.00187	0.00164
2,3,4,6,7,8-HxCDF	0.0165	0.0143	0.00646	0.0127	0.00497	0.0175	0.0121
1,2,3,4,6,7,8-HpCDF	0.0490	0.0330	0.0177	0.0296	0.0170	0.0384	0.0308
1,2,3,4,7,8,9-HpCDF	0.00456	0.00352	0.00237	0.00469	0.00258	0.00463	0.00373
OCDF	0.0270	0.0170	0.0194	0.0210	0.0185	0.0262	0.0215
PCDDs	0.202	0.155	0.103	0.172	0.0854	0.187	0.151
PCDFs	0.179	0.135	0.0821	0.150	0.0710	0.178	0.133
PCDDs/PCDFs ratio	1.13	1.15	1.25	1.15	1.20	1.05	1.16
Total PCDD/Fs	0.381	0.290	0.185	0.321	0.156	0.366	0.283
PCDDs pg I-TEQ/Nm^3	0.00724	0.00637	0.00359	0.00736	0.00287	0.00772	0.00586
PCDFs pg I-TEQ/Nm^3	0.0174	0.0140	0.00740	0.0160	0.00538	0.0184	0.0131
PCDDs/PCDFs (TEQ) ratio	0.420	0.450	0.490	0.460	0.530	0.420	0.462
Total pg I-TEQ/Nm^3	0.0246	0.0204	0.0110	0.0234	0.00824	0.0262	0.0190

were approximately 11% (=0.0641/0.6) and 3.2% (=0.0190/0.6), respectively, of JAQS. The above information revealed that without biomass burning, total PCDD/F I-TEQ concentration in both L- and Y-areas was at a low level.

It is worth mentioning that, for L-area, concentrations of PCDDs were lower than those of PCDFs at all sampling sites (locations LA–LE); In other words, the ratios of PCDDs/PCDFs were all less than unity (ranging from 0.564 to 0.980) accordingly. Meanwhile, the ratios of PCDDs/PCDFs (I-TEQ) at all sampling sites were also less than unity (ranging from 0.238 to 0.278), meaning that PCDFs were the primary distributors of toxicity for PCDD/Fs in L-area.

However, for Y-area, concentrations of PCDDs were higher than those of PCDFs at all sampling sites (locations YA–YF), and the ratios of PCDDs/PCDFs were more than unity (ranging from 1.05 to 1.25) instead. Meanwhile, the ratios of PCDDs/PCDFs (I-TEQ) at all sampling sites were also less than unity (ranging from 0.420 to 0.530). This means that PCDFs were still the primary distributors of toxicity for PCDD/Fs in Y-area. Although there were different kinds of PCDDs/PCDFs ratios between L- and Y-areas without biomass open burning, these two areas exhibited the same kinds of PCDDs/PCDFs (I-TEQ) ratios. The probable reason for this difference is that they were influenced by different pollution sources. In fact, there were some emission sources (included an electric arc furnace and a secondary aluminum smelter) situated about one kilometer upstream from location LD of L-area. During the sampling, they emitted PCDD/Fs more or less. Then, through dispersion, some of these PCDD/Fs were transported to the ambient air of location LD, and thus, changed the level of PCDDs/PCDFs ratio between L- and Y-areas.

Comparing the PCDD/F I-TEQ concentration in the ambient air of L- and Y-areas with four areas in Taiwan [15], we found that the level of L-area (0.0641 pg I-TEQ/Nm^3) was very close to suburban area (Pingtung, 0.0695 pg I-TEQ/Nm^3) and was much higher than that of remote area (Kenting, 0.0119 pg I-TEQ/Nm^3). While the level of Y-area (0.0190 pg I-TEQ/Nm^3) was quite close to that of remote area, which has the lowest level among these four areas in Taiwan.

Comparing the PCDD/F I-TEQ concentration in the ambient air of L- and Y-areas with other countries [15] (including Germany, Japan and Spain), we found that the levels of L- and Y-areas all fell into the range of rural areas (0.018–0.070 pg I-TEQ/Nm^3), which has the lowest PCDD/F I-TEQ concentration among various areas of these countries.

There are 75 PCDDs and 135 PCDFs differentiated from each other by the number and location of chlorine atom addition. The mixture of PCDD/Fs can be translated into profiles (mass fraction), which represent the distribution of individual PCDD/Fs. The comparison of a homologue pattern is a useful method to trace the source of contamination. Different sources of PCDD/Fs can usually be characterized by their different congener patterns [2,17]. The 17 congener profiles (mean of each location) of PCDD/Fs in the ambient air of L- and Y-areas are illustrated in Figs. 1 and 2, respectively; the y coordinate was the concentration of each congener divided by the sum concentration of the seventeen 2,3,7,8 chlorine substituted PCDD/Fs.

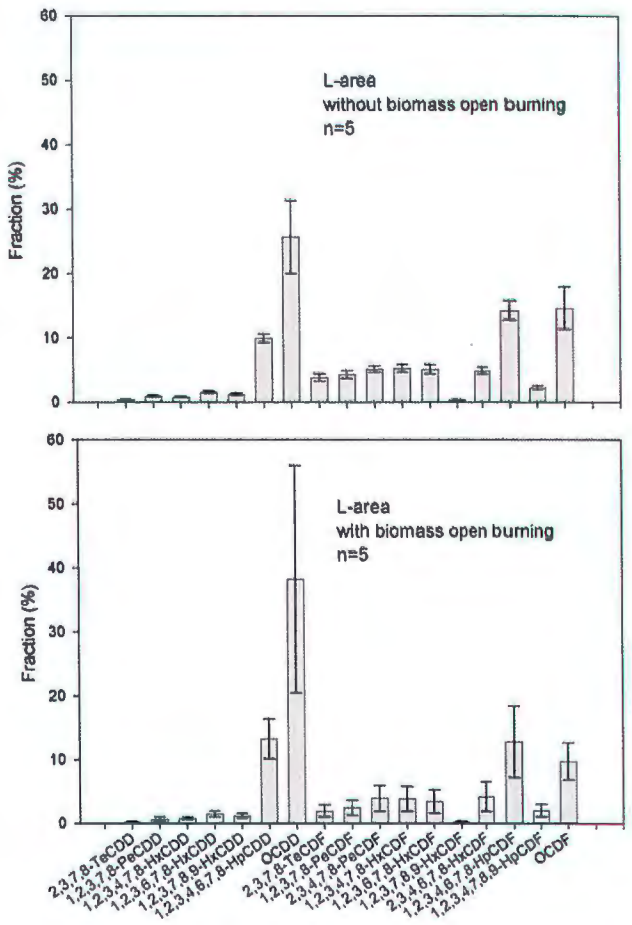

Fig. 1. Congener profiles of PCDD/Fs in the ambient air of L-area with/without biomass open burning.

As shown in the upper parts (without biomass open burning) of these two figures, the congener profiles did not exhibit significant differences for the L- and Y-areas. OCDD, OCDF, 1,2,3,4,6,7,8-HpCDD and 1,2,3,4,6,7,8-HpCDF were the four predominant congeners. This is the same case in the ambient air before rice straw open burning in the study of Kao et al. [4]. Nevertheless, the most dominant congeners were different. They were OCDD for both L- and Y-areas in the present study, and OCDF for both rice straw field and air quality monitor station (AQMS) in the study of Kao et al. [4].

3.2. PCDD/Fs in the ambient air with biomass open burning

Tables 3 and 4 summarize the PCDD/F concentrations in the ambient air of L- and Y-areas with rice straw burning, respectively. The mean of total PCDD/F concentration of L-area was around two times (9.53/4.64) higher than that of Y-area. Nevertheless, the mean of total PCDD/F I-TEQ concentration of the L-area was lower than that of the Y-area. Obviously, the high concentration level of OCDD (21.8 pg/Nm^3, Table 3) at location LD in L-area was responsible for the elevated mean of total PCDD/F concentration.

Table 3
PCDD/F concentrations in the ambient air of L-area with biomass open burning

PCDD/PCDFs (pg/Nm^3)	Location					
	LA	LB	LC	LD	LE	Mean
2,3,7,8-TeCDD	0.00405	0.00364	0.00625	0.00933	0.0148	0.00761
1,2,3,7,8-PeCDD	0.0173	0.0134	0.0219	0.0670	0.0737	0.0387
1,2,3,4,7,8-HxCDD	0.0227	0.0157	0.0209	0.144	0.0721	0.0551
1,2,3,6,7,8-HxCDD	0.0415	0.0268	0.0389	0.347	0.162	0.123
1,2,3,7,8,9-HxCDD	0.0278	0.0229	0.0310	0.262	0.129	0.0945
1,2,3,4,6,7,8-HpCDD	0.284	0.269	0.369	6.05	0.942	1.58
OCDD	0.791	0.840	1.13	21.8	1.45	5.20
2,3,7,8-TeCDF	0.0701	0.0455	0.0763	0.0637	0.162	0.0835
1,2,3,7,8-PeCDF	0.0888	0.0653	0.0851	0.100	0.201	0.108
2,3,4,7,8-PeCDF	0.1312	0.0985	0.122	0.169	0.426	0.189
1,2,3,4,7,8-HxCDF	0.129	0.0799	0.118	0.262	0.442	0.206
1,2,3,6,7,8-HxCDF	0.114	0.0680	0.105	0.214	0.415	0.183
1,2,3,7,8,9-HxCDF	0.00648	0.00559	0.00663	0.0133	0.0249	0.0114
2,3,4,6,7,8-HxCDF	0.140	0.0840	0.127	0.227	0.515	0.219
1,2,3,4,6,7,8-HpCDF	0.450	0.316	0.402	1.04	1.22	0.686
1,2,3,4,7,8,9-HpCDF	0.0700	0.0433	0.0580	0.147	0.228	0.109
OCDF	0.268	0.282	0.316	1.55	0.812	0.646
PCDDs	1.19	1.19	1.62	28.7	2.84	7.11
PCDFs	1.47	1.09	1.42	3.78	4.44	2.44
PCDDs/PCDFs ratio	0.810	1.09	1.14	7.59	0.638	2.25
Total PCDD/Fs	2.66	2.28	3.04	32.4	7.28	9.53
PCDDs pg I-TEQ/Nm^3	0.0255	0.0204	0.0311	0.200	0.0988	0.0752
PCDFs pg I-TEQ/Nm^3	0.122	0.0847	0.114	0.181	0.394	0.179
PCDDs/PCDFs (TEQ) ratio	0.210	0.241	0.274	1.11	0.251	0.417
Total pg I-TEQ/Nm^3	0.147	0.105	0.145	0.381	0.493	0.254

Table 4
PCDD/F concentrations in the ambient air of Y-area with biomass open burning

PCDD/PCDFs (pg/Nm^3)	Location						
	YA	YB	YC	YD	YE	YF	Mean
2,3,7,8-TeCDD	0.00607	0.00766	0.0312	0.00843	0.0149	0.00864	0.0128
1,2,3,7,8-PeCDD	0.0248	0.0299	0.131	0.0306	0.0591	0.0383	0.0523
1,2,3,4,7,8-HxCDD	0.0240	0.0363	0.0905	0.0317	0.0500	0.0439	0.0461
1,2,3,6,7,8-HxCDD	0.0533	0.0713	0.193	0.0649	0.104	0.0919	0.0964
1,2,3,7,8,9-HxCDD	0.0368	0.0779	0.167	0.0671	0.0694	0.0835	0.0836
1,2,3,4,6,7,8-HpCDD	0.313	0.430	0.851	0.414	0.555	0.598	0.527
OCDD	0.538	0.577	0.919	0.601	0.758	0.709	0.684
2,3,7,8-TeCDF	0.0511	0.0737	0.263	0.0823	0.151	0.0815	0.117
1,2,3,7,8-PeCDF	0.0757	0.122	0.417	0.119	0.185	0.113	0.172
2,3,4,7,8-PeCDF	0.127	0.190	0.664	0.202	0.285	0.197	0.278
1,2,3,4,7,8-HxCDF	0.145	0.245	0.570	0.209	0.285	0.192	0.274
1,2,3,6,7,8-HxCDF	0.131	0.224	0.535	0.191	0.259	0.200	0.257
1,2,3,7,8,9-HxCDF	0.0273	0.0459	0.0798	0.0357	0.0311	0.0361	0.0427
2,3,4,6,7,8-HxCDF	0.201	0.293	0.567	0.270	0.288	0.284	0.317
1,2,3,4,6,7,8-HpCDF	0.647	0.941	1.50	0.831	1.06	0.938	0.986
1,2,3,4,7,8,9-HpCDF	0.0756	0.133	0.180	0.0974	0.135	0.115	0.123
OCDF	0.465	0.576	0.735	0.512	0.542	0.582	0.569
PCDDs	0.996	1.23	2.38	1.22	1.61	1.57	1.50
PCDFs	1.95	2.84	5.51	2.55	3.22	2.74	3.14
PCDDs/PCDFs ratio	0.510	0.430	0.430	0.480	0.500	0.570	0.487
Total PCDD/Fs	2.94	4.07	7.89	3.77	4.83	4.31	4.64
PCDDs pg I-TEQ/Nm^3	0.0335	0.046	0.151	0.0449	0.0731	0.0564	0.0675
PCDFs pg I-TEQ/Nm^3	0.130	0.200	0.572	0.195	0.266	0.195	0.260
PCDDs/PCDFs (TEQ) ratio	0.260	0.230	0.260	0.230	0.280	0.290	0.258
Total pg I-TEQ/Nm^3	0.164	0.246	0.723	0.240	0.339	0.251	0.327

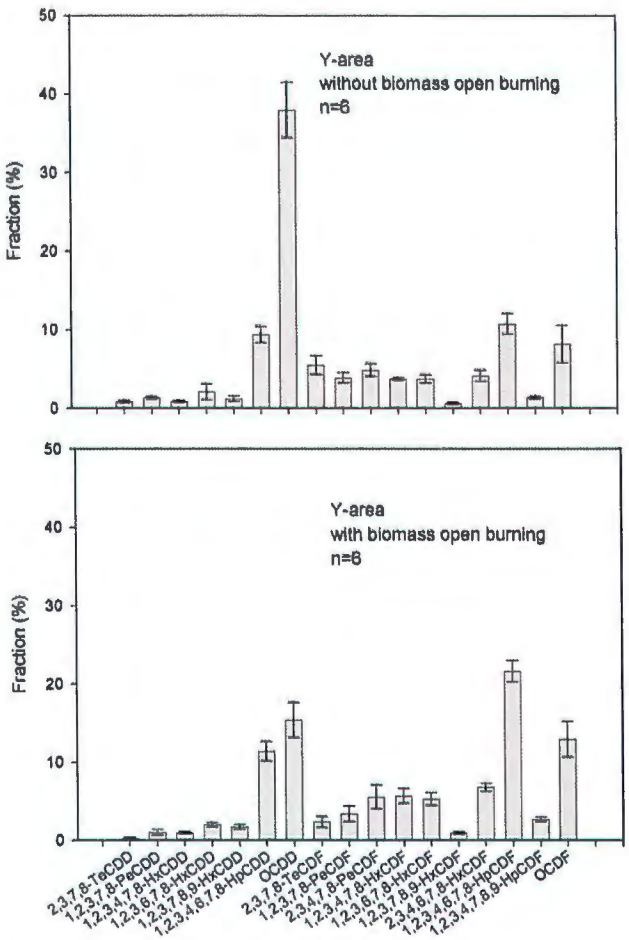

Fig. 2. Congener profiles of PCDD/Fs in the ambient air of Y-area with/without biomass open burning.

As mentioned in the previous section, there are a few emission sources (including an electric arc furnace and a secondary aluminum smelter) situated about one kilometer upstream from location LD. During the sampling, they emitted significant amounts of PCDD/Fs. Via dispersion, parts of these PCDD/Fs were then transported to the ambient air of location LD, and thus, increased the levels of OCDD and the corresponding total PCDD/F concentration. For example, the distance from emission sources to location LA, LB or LC is around 1.8–2.0 km, which is two times higher than that to location LD or LE (1 km). As shown in Table 3, the average total PCDD/F I-TEQ concentration of location LD and LE was 0.437 pg I-TEQ/Nm^3, which is 3.3 times higher than that of location LA, LB and LC (0.132 pg I-TEQ/Nm^3). On the other hand, OCDDs having the lowest toxic equivalent factor (TEF) were responsible for the lower total PCDD/F I-TEQ concentration of L-area [15]. Notably, if the results of LD location were excluded, the mean of total PCDD/F concentration of the L-area then was 3.82 pg/Nm^3, which was quite close to that of the Y-area (4.64 pg/Nm^3). Based on our comprehensive survey, there was no additional emission source besides rice straw burning, electric arc furnace and secondary aluminum smelters during sampling. These results revealed that after dismissing the influence of certain sources, the effects of biomass open burning on the level of PCDD/Fs could be found.

There are three mechanisms best known to establish the PCDD/F emission from combustion processes, in which PCDD/Fs are produced by de novo synthesis that is in the low temperature post-combustion zone. Usually, open burning occurs at low temperature of 250–450 °C [18,19] and uncontrolled conditions, which are favorable for forming incomplete products, such as PCDD/Fs. In addition to the low temperature condition, chlorine (Cl) content has been known to play a major role on forming PCDD/Fs during biomass burning.

The lower parts of Figs. 1 and 2 show the congener profiles of PCDD/Fs (mean of each location) in the ambient air of L- and Y-areas with biomass open burning. Similar to the trends in without biomass opening burning, the top four predominant species were still 1,2,3,4,6,7,8-HpCDD, OCDD, 1,2,3,4,6,7,8-HpCDF and OCDF for both L- and Y-area. The top predominant congener is OCDD for L-area, which result is the same with that of without biomass open burning. However, for Y-area, it is shifted from OCDD to 1,2,3,4,6,7,8-HpCDF.

When comparing with the case in the ambient air during rice straw open burning in the study of Kao et al. [4], OCDD, OCDF, 1,2,3,4,6,7,8-HpCDD and 1,2,3,4,6,7,8-HpCDF were the four predominant congeners for all circumstances. The top predominant congeners were OCDD and 1,2,3,4,6,7,8-HpCDF for L- and Y-areas, respectively. For rice straw field and AQMS, they were OCDD and OCDF, respectively.

3.3. Comparisons of PCDD/F levels in the ambient air with/without biomass open burning

As mentioned above, in L- and Y-areas (Tables 1–4), the PCDD/F concentrations in the ambient air with biomass open burning were significantly higher than those without biomass open burning. The means of total PCDD/F concentration with biomass open burning were about 9.0 (=9.53/1.03) and 16 (=4.64/0.283) times higher than those without biomass open burning, respectively. With respect to toxicity, the means of total PCDD/F I-TEQ concentrations were about 4.0 (=0.254/0.0641) and 17 (=0.327/0.0190) times higher, respectively. Similar results were also reported in the studies of Kao et al. [4] and Wevers et al. [20]. Apparently, biomass open burning (particularly rice straw), a common occurrence during November and December in Taiwan, was responsible for these elevated concentrations. In Taiwan, for advancing crop rotation and controlling insects (mosquitoes or other pests) and diseases, agricultural fire is an inexpensive and effective way to dispose of crop waste. In addition, the residual ash after combustion has a high fraction of organic components and can be used as fertilizers or manure. Thus, such waste is burned by the farmers in a very short period (e.g. within 2 weeks) once crops have been harvested.

During this short time, the impact of biomass burning on the PCDD/F concentration level in the ambient air quality is undoubtedly very significant. Table 5 lists the contribution of PCDD/F emission from rice straw burning at different mass fractions burned for KC County (one county in Taiwan) and Taiwan in general, respectively. The top four dominant emis-

Table 5
Contribution of PCDD/F emission from rice straw burned at different mass fractions

Mass fraction of rice straw burned (%)	Annual release (g I-TEQ/year)		Contribution (yearly) (%)		Contribution (weekly) (%)	
	KC County	Taiwan	KC County	Taiwan	KC County	Taiwan
0	0.00	0.00	0.00	0.00	0.00	0.00
10	0.200	7.70	3.28	8.11	30.6	53.4
20	0.400	15.4	6.56	16.2	46.8	71.6
30	0.600	23.1	9.84	24.3	56.9	80.7
40	0.800	30.8	13.1	32.4	63.8	86.2
50	1.00	38.5	16.4	40.5	68.8	89.9
60	1.20	46.2	19.7	48.6	72.6	92.5
70	1.40	53.9	23.0	56.7	75.5	94.5
80	1.60	61.6	26.2	64.8	77.9	96.0
90	1.80	69.3	29.5	72.9	79.9	97.2
100	2.00	77.0	32.8	81.1	81.5	98.2

sion sources of PCDD/Fs in KC County are coal-fired power plants, secondary aluminum smelting, electric arc furnaces, and open burning of rice straw, which contributed 56, 17, 13 and 3.3% to the total annual release (to the air), respectively. However, in Taiwan, they are sinter plants, coal-fired power plant, electric arc furnaces, and open burning of rice straw, which contributed 32, 28, 23 and 8.1%, respectively [13]. In 2004, the harvested areas of KC County and Taiwan in general were 6259 and 237,015 hectares (ha) [21]. Based on the field survey, the rice straw production per hectare was 6.7 tonnes and the fraction of rice straw burned was approximately 10%. The annual release can, thus, be calculated as follows: for KC County, 6259 ha × 6.7 tonnes/ha × 10%/year × 48.6 μg I-TEQ/tonnes × $1/10^6$ g/μg = 0.2 g I-TEQ/year; for Taiwan, 237015 ha × 6.7 tonnes/ha × 10%/year × 48.6 μg I-TEQ/tonnes × $1/10^6$ g/μg = 7.7 g I-TEQ/year. The 48.6 μg I-TEQ/tonnes for the emission factor of biomass burning was cited from Lin et al. [13]. Based on these estimated results along with the total annual release to air from major sources [13], the contribution of PCDD/F emission from rice straw burning at different mass fractions burned can be calculated, and is shown in Table 5.

As can be seen in the Table 5, when 10% mass fraction of rice straw was burned, the contribution fractions of biomass burning on annual total PCDD/F I-TEQ emission were 3.28 and 8.11% for KC County and for Taiwan, respectively. However, when the calculation was performed for a week with biomass (rice straw) burning and 10% mass fraction of rice straw was burned, the contribution fraction of biomass burning 1-week total PCDD/F I-TEQ emission were 30.6 and 53.4% for KC County and for Taiwan, respectively. Biomass burning is currently illegal in Taiwan, and Taiwan's central and local governments have tried to stop the biomass burning after the rice harvest seasons. However, approximately 10% of biomass is still being burned. If in some countries, the governments did not regulate or strongly prohibit biomass burning, the fraction of biomass burning would be higher than 50%. If 50% biomass was burned and the calculation of PCDD/F emission was in a week that such burning took place (Table 5), the contribution fractions of biomass burning on total PCDD/F I-TEQ emission were 68.8 and 89.9%, respectively, for KC County and the whole of Taiwan (Table 5). The above results imply that during the week of biomass burning and 50% biomass being burned, the open burning is the most significant source of total I-TEQ PCDD emission in both KC County and the whole of Taiwan.

Table 6 lists the comparisons of PCDD/F I-TEQ concentrations in the ambient air and emission factors of various kinds of biomasses burned. The PCDD/F I-TEQ concentrations in the ambient air of this study (0.254 and 0.327 pg I-TEQ/Nm^3) were close to those of Kao et al. [4] (0.409 and 0.458 pg I-TEQ/Nm^3), but much higher than that of Krauthacker et al. [22] (0.09 pg I-TEQ/Nm^3). The probable reason was the crop biomass burned of this study was similar to that of Kao et al. [4], and was different from that of Krauthacker et al. [22].

Comparing the current results with those of Gullett and Touati [11], some agreements and differences between Taiwan and the USA have been found. Firstly, they concluded that wheat and rice field burning are only minor contributors of PCDD/F to the US emission inventory. This was also the case in the study of Lin et al. [13], where they reported that in Taiwan open burning process (mainly rice straw) only has a minor contribution (8.2%) in comparison to ferrous and non-ferrous metal production (the major emission sources, 57%) and the total releases to the air. Secondly, the emission factor, 48.6 μg/tonnes, cited from the study of Lin et al. [13] was around 100 times higher than that Gullett and Touati [11] reported, 0.5 ng/kg or 0.5 μg/tonnes. Many factors were responsible for such a different emission factor, for instance, sampling train, the characteristics or ingredients of the biomass (rice or wheat), and extremely limited testing. Thirdly, "short period" field burning, previously mentioned in the present study, is a common activity in Taiwan. Nevertheless, the information regarding such burning behavior by the farmers in the USA was not so available.

Dioxin, formed in any combustion process where carbon, oxygen and chlorine are present. Among these three elements, chlorine obviously will be the limiting factor in forming dioxins. The most important reason probable was that the chlorine content of the rice straw in Taiwan was much higher than that in USA.

However, no matter what the differences are between Taiwan and the USA, biomass open burning could have a significant impact on the PCDD/F emission on a short-term basis. When

Table 6
Comparisons of PCDD/F I-TEQ concentrations in the ambient air and emission factors of various kinds of biomass burning

Fuel type (biomass burned)	PCDD/Fs (pg I-TEQ/Nm^3)	Emission factor (ng I-TEQ/kg of biomass)	Reference
Rice straw	0.254 (L-area)	–	This study
Rice straw	0.327 (Y-area)	–	This study
Rice straw	0.409	–	[4]
Wax apple stubble	0.458	–	[4]
Garden waste	0.09	–	[22]
Rice straw	–	6 and 22	[12]
Rice straw	–	48.6	[13]
Wax apple stubble	–	2.69	[13]
Ritual paper	–	1.36	[13]
Wheat straw	–	0.337–0.602	[23]
Rice straw	–	0.537	[23]
Rice and wheat straw	–	0.5	[11]

the impact on the ambient air quality is considered, the emission characteristics of the sources must be also taken into account. During the week of biomass burning, particularly rice straw burning, it appears to be the most important source of total I-TEQ PCDD emission. The ideas behind this research can be applied to the study of global agriculture.

4. Conclusions

(1) Biomass (rice straw) open burning exhibited a significant impact on the PCDD/F concentration level in the ambient air of L- and Y-areas, their means of total PCDD/F I-TEQ concentrations in the ambient air were about four and seventeen times higher than those without biomass open burning, respectively.
(2) When 10% mass fraction of rice straw was burned, the contribution fraction of biomass burning on annual total PCDD/F I-TEQ emission were 3.28 and 8.11% for KC County and for the whole of Taiwan, respectively. However, when the calculation was on the week that biomass (rice straw) burning took place and 10% mass fraction of rice straw was burned, the contribution fractions of biomass burning on that week's total PCDD/F I-TEQ emission were 30.6 and 53.4% for KC County and for all Taiwan, respectively.
(3) If 50% biomass was burned and the calculation of PCDD/F emission was in the week that biomass was burning, the contribution fractions of biomass burning on total PCDD/F I-TEQ emission were 68.8 and 89.9%, respectively for KC County and the whole of Taiwan.
(4) The results of this study imply that during the week of biomass burning, such burning is the most significant source of total I-TEQ PCDD emission. The ideas behind this research can be applied to the study of global agriculture.

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APPENDIX W

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Polychlorinated dibenzo-*p*-dioxins and dibenzofurans emissions from open burning of crop residues in China between 1997 and 2004

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Researches on emissions of PCDD/Fs from crop residue field burning are of great importance to establish a complete inventory for China.

Abstract

Annual emissions of polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) from open burning of crop residues in each province of China mainland between 1997 and 2004 were estimated to be ranged from 1.38×10^3 to 1.52×10^3 g I-TEQ/yr, with the average of $1.50 \pm 0.08 \times 10^3$ g I-TEQ/yr, which contributed to approximately 10% ~ 20% of the total emissions in China. The PCDD/F emissions mainly occurred in the largest crop-producing provinces, especially in those of higher economic levels. The major sources of PCDD/F emissions from open burning in China were found to be cereal residues (i.e. rice, wheat, and corn), which accounted about 70% of the total emissions. Moreover, the first-order one-variable grey differential equation model (GM (1,1) model) for annual emissions of PCDD/Fs was established based on grey system theory. The GM (1,1) model was proved to be robust to predict the annual PCDD/F emissions from crop residue field burning in forthcoming years.

Keywords: PCDD/Fs; Open burning; Crop residues; Grey system model; Emission

1. Introduction

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) are ubiquitous and some congeners (such as 2,3,7,8-PCDD/Fs) are highly toxic (deBruyn et al., 2004; Kubota et al., 2004). They are considered as typical persistent organic pollutants (POPs) and endocrine disrupting chemicals with great potential risk to human health (Niu et al., 2003, 2004; Costopoulou et al., 2006). Human exposure to PCDD/Fs has been a concern because of their wide occurrence and their adverse impacts on ecosystem and human health (Gomara et al., 2005). In particular, surprisingly high levels of PCDD/Fs were even observed in Polar Regions (Hung et al., 2002).

Efforts have been made to reduce PCDD/F emissions by many countries, and international agreements were adopted to reduce future environmental burdens. One example is the 1998 Aarhus Protocol on Persistent Organic Pollutants under the 1979 Geneva Convention on Long-range Transboundary Air Pollution (UNECE, 1998). The overall and long-term goal of the Aarhus Protocol on POPs is to eliminate any discharges, emissions, and losses of POPs to the environment. A recent international agreement is the Stockholm Convention on Persistent Organic Pollutants, which focuses on eliminating or at least controlling twelve POPs (including PCDD/Fs). This Convention has received 151 signatures, among which 136 parties have ratified it from 23 May 2001 until December 2006 (UNEP, 2006). According to the Article 5 of the Stockholm Convention, China has to take measures to reduce the

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release of unintentionally produced POPs, especially for the release of PCDD/Fs. As the premise to develop and implement a scientific action plan for PCDD/Fs reduction, the PCDD/F emissions from various sources should be determined or estimated in order to get the comprehensive national or regional inventory. Recently, some countries have developed national inventories of PCDD/F emissions, such as the United States, European Union, and Japan (Berdowski et al., 1997; US EPA, 1998; UNEP, 1999, 2001; Vestreng and Klein, 2002; Breivik et al., 2004; Quass et al., 2004).

In 2004, Jun et al. (2004) compiled the first inventory on PCDD/Fs release from potential PCDD/F emission sources in China. They suggested that the total emissions of PCDD/Fs from these sources ranged from about 7144 to 13,575 g I-TEQ/yr. However, the PCDD/F emissions from uncontrolled combustion activities including biomass burning have not been estimated in this inventory. In some previous studies, uncontrolled combustions including those from forest fires, open burning of agricultural residues, house fires, and backyard household waste combustion have been identified as a large potential source of PCDD/Fs but has not been regulated (Wasson et al., 2005; Gullett et al., 2006; Hedman et al., 2006; Pandelova et al., 2006).

Agricultural activities use open burning as a rapid method for disposing of crop residues, releasing nutrients for the next growing cycle, and clearing land (Gullett et al., 2006). Annually, a large amount of PCDD/Fs is released to the environment from open burning of crop residues in many countries (Gullett and Touati, 2002). As one of the largest crop production countries in the world, open burning of crop residues in the field could be an important source of PCDD/Fs in China. In fact, such burning has been a very popular practice of removing the material and fertilizing soil in countryside of many provinces in China. Therefore, estimation of PCDD/F emissions from open burning of crop residues is of great importance for China to complement the data gap of the existing inventory.

This study is the first effort to estimate the emission inventory of PCDD/Fs from the open burning of crop residues in the mainland of China, basically following the methodology of UNEP Dioxins Inventory Toolkit (UNEP, 2005). With the data of annual agricultural production, temporal variation of the emissions from 1997 to 2004 was addressed. Also geographical information system (GIS) was used to present the spatial distribution feature of the emissions at the provincial level. Moreover, the first-order one-variable grey differential equation model (abbreviated as GM (1,1) model) from grey system theory was developed based on discrete data and equal spacing in time, which can be used to predict future emissions of PCDD/Fs.

2. Materials and methods

2.1. Methodology of estimation

The annual output data of major produce (i.e. grain, cotton, oil-bearing crops, fiber crops and sugar crops) for the period of 1997–2004 in China mainland were collected from officially published yearbooks (NBSC, 1998–2005). Detailed information of individual provinces, autonomous districts, and municipalities on an annual basis was gathered, as shown in Table 1. These data were used for conducting study on spatially and temporally resolved emissions. It should be noted that the data in Table 1 are the only data available for the source quantification at up to date.

The annual output of crop residues was estimated by multiplying the output of each farm produce with the ratio of residue to crop. The ratios of residue to crop were available from the data in China Association of Rural Energy Industry (CAREI, 2000). The mass of crop residues of open burning in each province was then calculated by the burning percentage according to the characteristics of each province.

In this study, emissions of PCDD/Fs are given in TEQ using the International Toxic Equivalent system (NATO/CCMS, 1988). The PCDD/F emissions were developed according to an approach used by most of the agencies, including United States Environmental Protection Agency (US EPA).

$$\text{PCDD/F emissions per annum} = \text{Emission factor (EF)} \times \text{Activity rate} \quad (1)$$

The PCDD/F emissions per annum are given in grams I-TEQ per annum. According to Eq. (1), the annual flux is calculated by multiplying the EF with the open burning amount of crop residue produced (i.e. the activity rate).

2.2. Open burning ratios and emission factors

High variation (1% ~ 50%) of crop residues was expected for open burning in different provinces, autonomous districts, and municipalities according to limited literatures (Cao et al., 2005, 2006). These differences could be affected by many factors, such as the living standards, climate zones, vegetation covering situation, and the production of different crops, especially the income levels and the utilized cost of crop residues in the rural areas. Generally, the income level in the rural areas is in compliance with the percentage of crop residues burning. The ratios of open burning (ratio of crop residue field burning mass to the total crop residue production) in the developed areas are higher than those in the undeveloped areas and the ratio is proportional to the income level in the rural areas. In order to make a comparatively accurate estimation of the amount of crop residue burning in different provinces, the ratios of the open burning of crop residues were divided into five categories based on the living standards, the climate zones, vegetation covering situation, production sites, especially the average income per annum in the rural areas of each province according to the reports by Cao et al. (2006). The five categories are: >40%, 30% ~ 40%, 20% ~ 30%, 10% ~ 20%, <10%. The detailed information of the five regions was shown in Table 2 (Cao et al., 2005, 2006). For instance, with regard to the rural areas in Beijing and Shanghai, the ratios of the crop residue of open burning were >40% because of both the developed economy and low percentage of utilization of crop residues in these rural areas.

The EF units of PCDD/Fs from crop residue field burning were difficult to be determined (Guyton et al., 1986; Gullett and Touati, 2002). In the UNEP Dioxin Inventory Toolkit, all three versions (i.e. the 2001 draft version, the 2003 first edition and the 2005 second edition) present PCDD/F emission factors of 0.5 μg I-TEQ/t to air and 10 μg I-TEQ/t to land, respectively, for agricultural residue field burning. In the 2001 and 2003 Toolkits, these EF values were based on studies on wood combustion in household stoves and fireplaces that were conducted in Netherlands and Germany more than a decade ago. In the 2005 Toolkit, the same air EF value was based on the study by Ikeguchi and Tanaka (1999) in which leaves were burned in a furnace under the conditions simulating open burning. The EF values used in this study were based on the data presented in the UNEP Dioxin Inventory Toolkit (UNEP, 2003, 2005).

2.3. Grey system prediction theory

In 1982, grey system theory was firstly proposed by Deng (1982). Recently, it has become an effective method of resolving uncertainty problems under discrete data and incomplete information (Zhang et al., 2003; Trivedi and Singh, 2005). In grey system theory, accumulated generating operation

Table 1
PCDD/F emissions from crop residue field burning to the environment

Province	The percentage of open burning (%)	Emission of PCDD/Fs (g I-TEQ)							
		1997	1998	1999	2000	2001	2002	2003	2004
Beijing	46	18.9	19.3	15.6	11.2	8.7	7.1	5.0	6.3
Tianjin	36	11.0	11.6	9.5	7.4	10.0	9.5	8.8	9.1
Hebei	25	117.9	127.7	119.9	113.6	113.4	110.6	111.3	118.0
Shanxi	19	28.7	35.3	27.3	27.4	22.7	30.6	32.1	37.1
Inner Mongolia	20	49.7	56.8	52.0	44.5	47.0	53.0	52.2	56.0
Liaoning	24	47.8	72.8	64.0	40.8	54.3	57.8	59.4	69.3
Jilin	20	62.8	91.5	82.9	55.1	69.4	80.5	83.7	90.0
Heilongjiang	21	97.5	93.5	96.0	73.4	78.6	93.6	77.6	88.4
Shanghai	56	12.3	10.8	11.7	10.0	8.2	6.9	5.3	5.6
Jiangsu	36	147.6	134.5	148.7	132.7	129.7	125.0	108.0	125.1
Zhejiang	42	51.5	48.2	48.8	44.2	40.1	35.0	30.3	31.5
Anhui	19	67.0	58.1	67.1	62.0	67.1	71.7	58.4	71.4
Fujian	32	23.7	23.5	23.1	20.9	20.3	19.2	18.1	18.4
Jiangxi	21	31.4	27.0	29.3	28.2	28.0	26.9	25.1	27.5
Shandong	27	176.8	204.2	203.4	191.6	187.1	166.4	175.4	185.6
Henan	20	130.1	136.5	146.4	142.9	144.8	148.2	120.8	148.4
Hubei	23	68.2	65.3	63.9	62.4	59.4	54.7	53.8	60.2
Hunan	22	52.5	49.4	55.0	53.1	52.2	48.4	48.0	51.7
Guangdong	37	61.4	63.3	69.7	57.7	52.6	49.2	47.7	47.0
Guangxi	19	33.2	33.6	28.0	33.0	33.1	34.3	34.4	35.1
Hainan	22	4.4	4.4	3.7	4.3	4.1	4.1	4.4	4.2
Chongqing	19	22.9	22.0	21.8	21.9	20.1	21.5	21.9	23.4
Sichuan	19	75.9	78.0	77.7	74.8	64.6	70.2	69.3	71.8
Guizhou	14	17.9	19.7	22.7	21.2	19.5	19.4	19.5	20.9
Yunnan	15	26.3	28.0	27.4	30.3	30.6	30.0	29.0	30.4
Tibet	13	1.3	1.4	1.6	1.6	1.7	1.7	1.7	1.6
Shaanxi	14	22.8	29.3	24.9	24.6	22.9	23.7	23.2	24.4
Gansu	14	16.4	20.3	18.4	16.0	16.7	17.6	18.0	17.9
Qinghai	15	3.0	3.2	2.9	2.1	2.5	2.3	2.2	2.4
Ningxia	17	6.5	7.5	8.1	6.2	6.9	7.9	7.7	7.8
Xinjiang	16	28.8	30.4	29.6	29.8	30.1	32.1	31.4	32.9
Total		1.52×10^3	1.61×10^3	1.60×10^3	1.44×10^3	1.45×10^3	1.46×10^3	1.38×10^3	1.52×10^3

Statistical data of Hong Kong, Macao and Taiwan are not concluded in this study.

(AGO) and inverse accumulated generating operation (IAGO) are the main methods that provide a manageable approach to treat disorganized evidence. The series generated can be used to build a grey forecasting model. A grey system model is a kind of dynamic model described by a differential equation. As a prediction model, the grey model (GM) is the core of grey system theory, which collects available data to obtain the internal regularity without any assumptions. It avoids the inherent defects of conventional, large sample statistical methods, and only requires a limited number of discrete data to estimate the behavior of a system with incomplete information (Deng, 1989; Zhang et al., 2003).

The grey system prediction theory uses very few data (four or more) to create the model and the calculation is relatively simple. The grey model GM (1,1), i.e. first-order one-variable grey differential equation model, is the most important prediction model. A condition to establish a GM (1,1) is that the data should be taken at equal intervals.

Assume that $= \{x^{(0)}(1), x^{(0)}(2), \ldots, x^{(0)}(n)\}$ is an original series. The first-order AGO series obtained from $x^{(0)}$ is $x^{(1)} = \{x^{(1)}(1), x^{(1)}(2), \ldots, x^{(1)}(n)\}$, where

$$x^{(1)}(k) = \sum_{j=1}^{k} x^{(0)}(j), \quad k = 1, 2, \ldots, n. \tag{2}$$

If $n \geq 4$, $x^{(0)}$, $x^{(1)} \in R^+$, the grey dynamic prediction model GM (1,1) can be expressed by a one-variable, first-order differential equation.

$$\frac{dx^{(1)}}{dt} + ax^{(1)} = b \tag{3}$$

Using Laplace transform, Eq. (3) can be expressed in frequency domain as

$$sx^{(1)}(s) - u(0) + ax^{(1)}(s) = \frac{b}{s} \tag{4}$$

where $u(0)$ is the initial value of the system. $u(0) = x^{(0)}(1) = x^{(1)}(1)$.

$$x^{(1)}(s) = \frac{x^{(0)}(1) - \frac{b}{a}}{s + a} + \frac{\frac{b}{a}}{s} \tag{5}$$

The solution of continuous system form and discrete system form are then obtained by

$$\hat{x}^{(1)}(t+1) = \left(x^{(0)}(1) - \frac{b}{a}\right) e^{-at} + \frac{b}{a} \tag{6}$$

and

$$\hat{x}^{(1)}(i+1) = \left(x^{(0)}(1) - \frac{b}{a}\right) e^{-at} + \frac{b}{a} \tag{7}$$

where the coefficients a and b stand for the development and grey input coefficients, respectively.

By least-squares method, the coefficients a and b can be obtained as

$$\begin{bmatrix} a \\ b \end{bmatrix} = (A^T A)^{-1} A^T X_n \tag{8}$$

$$A = \begin{bmatrix} -z^{(1)}(1) & 1 \\ -z^{(1)}(2) & 1 \\ \vdots & \vdots \\ -z^{(1)}(n) & 1 \end{bmatrix} \tag{9}$$

Table 2
Open burning ratios of crop residues in different provinces of China mainland

Ratios of open burning	Provinces	Remarks
>40%	Beijing, Shanghai	Developed areas and low use ratio of crop residues in rural areas
30 ~ 40%	Tianjin, Jiangsu, Zhejiang, Fujian, Guangdong, Jilin and Heilongjiang	Developed areas, low use ratio of crop residues and low population density in rural areas
20 ~ 30%	Shanxi, Shaanxi, Liaoning, Henan, Shandong, Hebei, Hainan, Jiangxi, Hubei, Hunan and Anhui	Resources production regions, less-developed areas and relative high use ratio of crop residues in rural areas
10 ~ 20%	Ningxia, Gansu, Xinjiang, Guangxi, Sichuan, Chongqing, Yunnan and Guizhou	High use ratio of crop residues in rural areas
<10%	Inner Mongolia, Qinghai and Tibet	High use ratio of crop residues in rural areas

$$X_n = \begin{bmatrix} x^{(0)}(2) \\ x^{(0)}(3) \\ \vdots \\ x^{(0)}(n) \end{bmatrix} \tag{10}$$

Then, the predicted PCDD/F emission values can be estimated by Eq. (11)

$$\hat{x}^{(1)}(i+1) = \hat{x}^{(1)} - \hat{x}^{(1)}(i) = \left(x^{(0)}(1) - \frac{b}{a}\right)(1 - e^{a})\, e^{-ai} \tag{11}$$

The qualitative evaluation of the GM (1,1) model performance can be verified according to the following method. The zeroth order residue $q^{(0)}(k)$ can be expressed as

$$q^{(0)}(k) = x^{(0)}(k) - \hat{x}^{(0)}(k) \tag{12}$$

where $x^{(0)}(k)$ and $\hat{x}^{(0)}(k)$ are estimated values and predicted values, respectively.

The correlative value of posterior error (c) and micro-error probability (p) can be obtained as

$$c = \frac{S_1}{S_2} \tag{13}$$

$$p = p\{|q^{(0)}(k) - \overline{q}| < 0.6745 S_2\} \tag{14}$$

where $\overline{q}$ and $\overline{x}$ are the average zeroth order residues and the average estimated values, respectively. When c is lower than 0.35 and p is greater than 0.95, the GM (1,1) model is considered to be reliable and feasible.

It was suggested that the system of PCDD/F emissions can be viewed as a grey system. The annual release of PCDD/Fs between 1997 and 2004 can be viewed as an original series. In addition, grey dynamic model GM (1,1), which stands for the first order with one variable based on grey system theory as a prediction model can be used in the prediction problem of the PCDD/F emissions from crop residue field burning.

3. Results and discussion

3.1. Annual PCDD/F emissions

The annual emissions of PCDD/Fs from field burning of crop residue in China mainland between 1997 and 2004 were estimated based on the ratios of open burning and their EF values. The results of PCDD/F emissions from open burning of major farm produce in the individual provinces of China mainland are given in Table 1. The emission values presented in Table 1 were the sum of PCDD/F emissions to air and land. The annual release of PCDD/Fs from the open burning of crop residues ranged from 1.38×10^3 to 1.52×10^3 g I-TEQ between 1997 and 2004 with the average PCDD/F emissions of $(1.50 \pm 0.08) \times 10^3$ g I-TEQ. The results indicated that the mass of burning crop residues in the field was relatively stable from 1997 to 2004.

Recently, Jun et al. (2004) reported that the total PCDD/F emissions were approximately 7144–13,575 g I-TEQ/yr in China mainland. However, contributions of PCDD/Fs from uncontrolled combustion activities, such as wood combustion, fallen leaves burning, crop residue field burning, and so on were not included in their study. It can be concluded that the open burning of field crop residues contributed to approximately 10% ~ 20% of the total PCDD/F emissions in China mainland according to the reports by Jun et al. (2004). Compared with the PCDD/F emissions from open burning of crop residues in other countries, the emissions of China mainland were slightly higher than the 1163 g I-TEQ/yr emitted from Mexico, and much higher than the 124.2 g I-TEQ/yr emitted from Argentina (Costner, 2005). These differences were most possibly resulted from the high production of crops in China, sequentially resulted in a large amount of crop residue that was burned as waste annually. According to this large magnitude of emissions, open burning of crop residues should be considered as one of key sources for taking measures to reduce the emissions of PCDD/Fs.

3.2. Contributions of various crop residues

Individual contributions of different crops to PCDD/Fs in China mainland from 1997 to 2004 were illustrated in Fig. 1. The major sources of PCDD/F emissions in China were identified as the combustion of cereal residues (e.g. rice, wheat, and corn). In China, cereal production contributed to the largest percentage of crop production around 50% of the total planting areas. Consequently, the average mass of cereal residues accounts to about 78% of the total mass of crop residues and the emissions from cereal residue field burning account to about 70% of the total emissions of PCDD/Fs from open burning of crop residues between 1997 and 2004. Driven by the demands in both national and international markets, crop planting structure will be changed accordingly in the future. For instance, the production of wheat decreased from 1.23×10^{11} kg in 1997 to 9.20×10^{10} kg in 2004. However, cereal residues could still be the major source of PCDD/F emissions from open field burning. Thus, controlling the open burning of the cereal residue in the field will be the

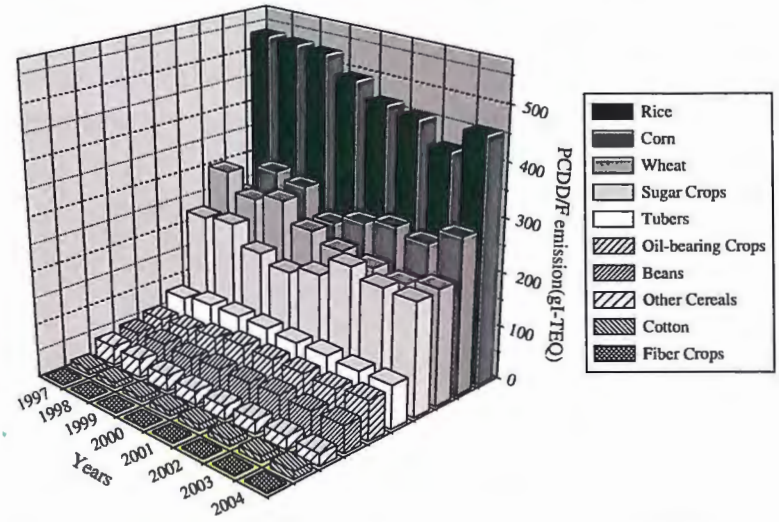

Fig. 1. PCDD/F emissions in China mainland from open burning of different crop residues from 1997 to 2004.

most effective measure to reduce the corresponding PCDD/F emissions.

3.3. Geographical distribution

Due to enormous differences in economic development, population density, size, geographical location, and climates zones, cereal productions were different among various provinces in China. The geographical distribution of annual PCDD/F emissions in individual provinces, autonomous districts, and municipalities directly under the jurisdiction of the central government was illustrated in Fig. 2. Clearly, the annual PCDD/F emissions at provincial level varied from 1.6 ± 0.1 g I-TEQ in Tibet to 187.3 ± 12.6 g I-TEQ in Shandong, and the source patterns also varied widely.

The results from this study suggested that the PCDD/F emissions from open burning of crop residues would be relatively high in the following regions: (1) Well-developed provinces. With the development of the peasant's living levels, the field burning of crop residues was replaced by commercial resource as a better choice of increasing the peasant's living qualities. For example, the ratios of commercialization in peasants' daily consumption of energy in Jiangsu and Zhejiang province were over 90%; therefore, about 40% of crop residues was abandoned or burned in the field. As shown in Table 1, the PCDD/F emissions from the rural areas of Jiangsu and Zhejiang were 131.4 ± 13.1 g I-TEQ and 41.2 ± 8.2 g I-TEQ, respectively. (2) Bread basket provinces. For example, Shandong was abundant with grains, which resulted in the largest amount of PCDD/F emissions in China mainland. Also in Northeast areas (e.g. Heilongjiang and Jilin), the larger quantities of crop residues from the agricultural activity caused correspondingly the more crop residue open burning in the field. As a result, the emissions of PCDD/Fs from the open burning of crop residues mainly occurred in the largest crop production provinces, especially in the provinces with rapid economic growth in recent decades, such as Jiangsu, Shandong, Hebei, Henan, etc. However, the PCDD/F emissions were quite low in the western areas with low agricultural production. Compared with the great share of PCDD/F emissions of wheat and corn in Northern and Eastern China, oil crops and fiber crops in Southern China provided less PCDD/F emissions.

3.4. GM (1,1) model

The GM (1,1) model from grey system theory was developed based on discrete data and equal spacing in time, i.e. the emissions of PCDD/Fs from 1997 to 2004. The accuracy of this model was increased by using residual error correction, which was helpful to effectively predict the emissions of PCDD/Fs from open burning crop residues. The results of the grey dynamic GM (1,1) prediction model were shown in Table 3. From the tested results calculated by some indexes, such as correlative value of posterior error ($c < 0.35$) and micro-error probability ($p > 0.95$), the model of grey system theory for the emissions of PCDD/Fs was examined and certified, which proved the model reliability and feasibility. Based on the GM (1,1) model, emissions of PCDD/Fs from 2005 to 2010 were predicted, respectively. The predicted emission values for PCDD/Fs were shown in Table 4. As a result from Table 4, the annual emission of PCDD/Fs from crop residue field burning will decrease slowly in the following few years. The predicated results suggested that emissions of PCDD/Fs will decrease from 1.52×10^3 g I-TEQ in 2005 to 1.24×10^3 g I-TEQ in 2010.

As shown in Table 4, the PCDD/F emission from open burning of crop residues seems to decrease gradually in the forthcoming years. However, the magnitude would be still quite large. In China, there is a change of crop planting structure in the past few years. Some crops that produce fewer residues were planted. For instance, from 1997 to 2004, there is an increasing trend for the production of sugar crops which

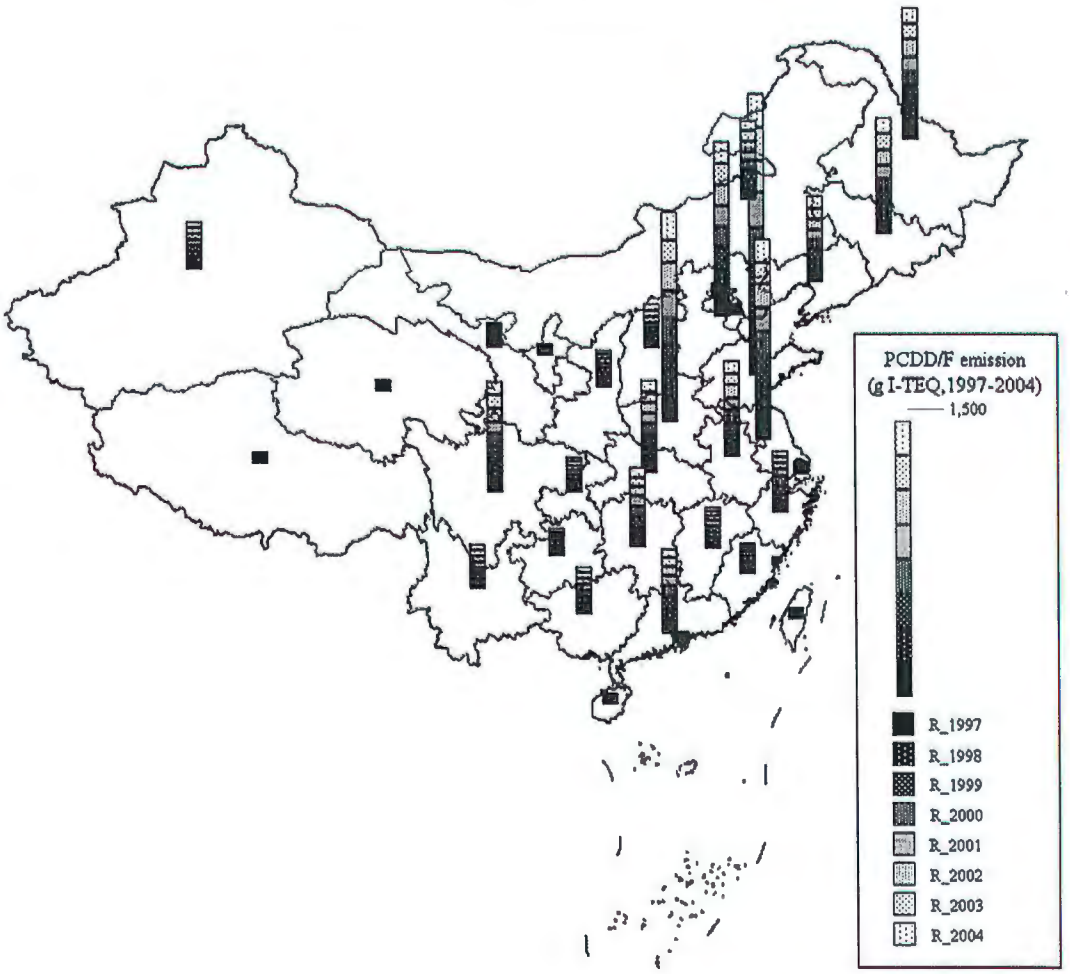

Fig. 2. PCDD/F emissions at the provincial level between 1997 and 2004.

has the low ratio of residue to crop. The crop planting structure adjustment may lead to the reduction of PCDD/F emissions to some extent. Although there is a trend in the rural areas of China to replace agricultural residues with coal or natural gas for heating and/or cooking, biomass burning is still a common practice in the rural areas of China because of the easy handling of the waste. Promisingly, the effort has been taken to reduce the emissions in the field. For example, the central government and local governments issued regulations in 1999 and 2000, respectively, to ban open-air burning of straw and to encourage environment-friendly usage of the material including biogas production in the rural areas and on-site burying. The regulations were performed successfully in Zhejiang province. Additionally, using the crop residues to produce the plant fiber tableware or to generate electricity is also an effective way to prohibit pollution from open burning of crop residues. This action has been taken in some provinces (e.g. Shandong, Hebei, Shaanxi, etc.). In these provinces, implementation of these measures will probably lead to a decline in PCDD/F emissions from biomass burning in the field. Hopefully, the total emissions of PCDD/Fs from open burning of crop residues in near future will be lower than the predicted values by the grey dynamic GM (1,1) model.

4. Conclusions

Annual emissions of PCDD/Fs from the open burning of crop residues in China mainland were estimated to be ranging from 1.38×10^3 to 1.52×10^3 g I-TEQ per annum between

Table 3
The results of GM (1,1) model for PCDD/F emissions from open burning of crop residues

	GM (1,1)	Modification by residual error		
		The first time	The second time	The third time
a	1.68×10^{-2}	2.14×10^{-1}	4.17×10^{-2}	-9.27×10^{-2}
b	1.61×10^{3}	2.06×10^{2}	6.10×10^{1}	2.62×10^{1}
c	8.25×10^{-1}	5.34×10^{-1}	3.81×10^{-1}	3.15×10^{-1}
p	4.29×10^{-1}	5.71×10^{-1}	9.99×10^{-1}	9.99×10^{-1}

Table 4
Predicted emission values of PCDD/Fs from crop residue field burning during 2006–2010

Years	2005	2006	2007	2008	2009	2010
Predicted values (g I-TEQ)	1.35 $\times 10^3$	1.32 $\times 10^3$	1.30 $\times 10^3$	1.28 $\times 10^3$	1.26 $\times 10^3$	1.24 $\times 10^3$

1997 and 2004, with the average value of $1.50 \pm 0.08 \times 10^3$ g I-TEQ, which contributed to approximately 10% ~ 20% of the total PCDD/F emissions in China mainland. The geographical distributions and contributions of various sources of the PCDD/F emissions were also discussed. Generally, the PCDD/F emissions from the open burning of crop residues mainly occurred in the largest crop-producing provinces, especially in those with rapid economic growth. The major sources of PCDD/F emission from crop residue field burning in China mainland were identified as the combustion of cereal residues, which accounted to about 70% of the total emissions of PCDD/Fs from crop residue field burning. Moreover, the dynamic GM (1,1) model for annual releases of PCDD/Fs from open burning of crop residues was established using the methodology of grey modeling prediction based on grey system theory. The tested results showed satisfactory performance of the established GM (1,1) model, with the correlative value of posterior error $c < 0.35$ and micro-error probability $p > 0.95$. Predictive results by the model showed a slightly decreasing trend of annual PCDD/F emission in the forthcoming years, with the magnitude greater than 1200 g I-TEQ/yr until 2010. This confirms the necessity to take measures to restrict the open burning of crop residues. As the priority, provinces with greater agricultural production as well as high economic level should be focused.

Acknowledgments

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Health Guidelines for Vegetation Fire Events, Lima, Peru, 6-9 October 1998.
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APPENDIX X

A REVIEW OF FACTORS AFFECTING THE HUMAN HEALTH IMPACTS OF AIR POLLUTANTS FROM FOREST FIRES

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SUMMARY

Although total emissions and adverse health effects have been documented in past studies, the overall toxicity of exposure to smoke or haze from forest fires has yet to be fully evaluated. A review of the literature identifies potential factors that influence forest fire emissions and allows for extrapolation of possible health effects. Fire dynamics involves fire, fuel, and climatological factors. The ecosystem's chemical and physical features combined with environmental parameters (humidity, temperature, and wind speed) and the type of ignition, affect the combustion factor and efficiency and, therefore, the amount of biomass consumed, the composition of smoke emissions, and the rate of release of emissions. Characteristics of fuel (e.g., arrangement, size distribution, moisture, and chemical composition) affect the phases of combustion, for which the quantities and rates of releases vary. Exposure to combustion products can have potentially detrimental short and long term effects on human health. These products, their known health effects, and the factors influencing their effects are described for (1) particulate matter; (2) polycyclic aromatic hydrocarbons; (3) carbon monoxide; (4) aldehydes; (5) organic acids; (6) semivolatile and volatile organic compounds; (7) free radicals; (8) ozone; (9) inorganic fraction of particles; (10) trace gases and other releases; and (11) radionuclides. The biological aspects of severe aerosol loading require further investigation.

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INTRODUCTION

Given the objective of protecting public health, a complete understanding of the spectrum of health effects from biomass fires requires knowledge of the full range and potential of factors that might affect health outcomes and related impacts. Although total emissions and adverse health effects have been documented in public health and medical literature, the overall toxicity of exposure to smoke or haze has yet to be fully evaluated. A review of the current literature, primarily from atmospheric chemistry, identifies several factors that potentially influence the impact of air pollutants from forest fires on the susceptibility of individuals and vulnerable groups. Knowledge of adverse effects on public health from direct and indirect linkages is lacking, and efforts to elucidate the biological mechanisms by which exposures to biomass smoke affect human health have yet to be described. However, an extrapolation of potential factors on health effects can be attempted, taking into consideration biological plausibility, linkage between cause and effect, and coherence of past and current studies.

FIRE DYNAMICS AND THE COMBUSTION PROCESS

Fire dynamics is a complex process involving fire, fuel and climatological factors including altitude and meteorology. Under ideal conditions (i.e., complete combustion), the burning of organic material is an oxidation process that primarily produces water vapor and carbon dioxide (CO_2) (1). In natural and anthropogenic fires, combustion is incomplete due to an insufficient supply of oxygen (O_2). As a result, incompletely oxidized compounds (e.g., carbon monoxide) or reduced compounds (e.g., methane, nonmethane hydrocarbons, ammonia) are formed (1). These compounds are found in smoke, which often consists of irritant respirable particles and gases, and in some cases may be carcinogenic. Smoke itself is a complex mixture with components that depend on fuel type, moisture content, fuel additives such as pesticides sprayed on foliages or trees, and combustion temperature (2).

The combustion process involves two key parameters: the amount of biomass material burned and the proportion of a compound released during combustion, or emission factor, which is measured by grams (g) of pollutant

per kilogram (kg) of fuel consumed. Combustion efficiency, defined as the ratio of carbon released by the fire as CO_2, is a fundamental parameter that integrates many of the variables affecting biomass volatilization and oxidation (3). The ecosystem's chemical and physical features combined with environmental parameters (humidity, temperature, wind speed) and the type of ignition, affect the combustion factor and combustion efficiency and, therefore, the amount of biomass consumed, the composition of smoke emissions, and the rate of release of emissions (4).

Combustion can consist of several phases relative to the time after ignition: (i) flaming phase, 0-20 minutes; (ii) initial smoldering phase, 20-80 minutes; and (iii) smouldering phase, 80-200 minutes. These processes are different phenomena involving different chemical reactions that result in diverse products (3). Characteristics of fuel (e.g., arrangement, size distribution, moisture, and chemical composition) affect the duration of each phase (3). The relative amount of biomass consumed through flaming and smoldering combustion can also vary due to these factors (4).

Exposure to combustion products can have potentially detrimental short and long term effects on human health. Although some of these products have been observed to occur in varying amounts after biomass fires (5, 6), little or no information exists about the intensity of human exposure and resulting health effects. The products, their known health effects, and the factors influencing their effects have been described for particulate matter (PM), polycyclic aromatic hydrocarbons (PAHs), carbon monoxide (CO), aldehydes, organic acids, semivolatile and volatile organic compounds (VOCs), free radicals, ozone, inorganic fraction of particles, trace gases and other releases, and radionuclides.

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COMBUSTION PRODUCTS OF BIOMASS FUELS

Particulate matter (PM)

Health effects

The solid component of smoke is PM, which at respirable size presents risks to human health. The PM mass is categorized into two modes: fine particle, with a mean-mass diameter of 0.3 micrometers (μm), and a coarse particle, with a mean-mass diameter greater than 10 μm. Even at low concentrations, fine particles have been observed to cause changes in lung function, leading to increases in respiratory and cardiovascular mortality and morbidity including asthma. Fine particles may reach the alveoli, and if not sufficiently cleared in the lungs and in great concentrations, may enter the bloodstream or remain in the lung, resulting in chronic lung disease such as emphysema. Airborne particulates may also contain toxic recondensed organic vapors, such as PAHs, which are indicated to be carcinogenic in animals (7).

Factors affecting health effects

The size of the fire event may influence resulting health effects. Particulate matter is produced abundantly, at least 0.6 tons per second after large fires, during forest fire combustion (8). 40 to 70 per cent of fine PM consists of organic carbon material, containing known carcinogens (9). 2-5 per cent is graphitic carbon; the remainder inorganic ash. Particulate matter may carry absorbed and condensed toxicants, and possibly, free radicals.

Polycyclic aromatic hydrocarbons (PAHs)

Health effects

PAHs comprise a group of organic compounds with two or more benzene rings, such as methyl anthracene, pyrene, chrysene, benzo(a)anthracene, flurorantheneene, and methylchrysene. Benzo(a)pyrene is considered the most carcinogenic. Exposure to PAHs has been linked to lung cancer in railway workers exposed to diesel exhaust fumes and occupational lung cancer in coke oven and coal gas workers (7).

Factors affecting health effects

Factors related to combustion may affect the production and quantity of PAHs released into the atmosphere. Although little is known about which combustion conditions yield highest amounts of PAHs, some combustion conditions produce PAHs more abundantly than others. Low intensity backing fires (i.e., a line of fire moving into new fuel in an upwind direction) are found to produce larger amounts of benzo(a)pyrene than heading fires, which move into new fuel in the same direction as the wind movement (10). In one study, the amount of benzo(a)pyrene ranged from 98 μg/g to 274 μg/g of PM for low intensity backing fires and 2 μg/g to 3μg/g for heading fires (11).

Fuel characteristics also affect the production of PAHs during combustion. Emission of benzo(a)pyrene increased as the density of live vegetation covering the prescribed fire units thickened (5). Under natural conditions of a tropical medium such as savanna fires, PAHs were observed to be abundantly produced, mainly in gaseous form. The total flux of PAH emitted in tropical Africa during the biomass burning season is estimated to be 605±275 tons per year for gaseous PAHs and 17±8 tons per year for particulate PAHs. Although savanna fires occur during only a few months of the year, their contribution to the global burden of atmospheric PAH is significant compared to anthropogenic sources (12).

In wind tunnel simulations of open burning, emission factors for 19 PAHs were measured for agricultural and forest biomass fuels, including cereal grasses, agricultural tree prunings, and fir and pine wood. Yields of total PAH varied from 5 milligrams (mg)/kg to 685 mg/kg depending on burning conditions and fuel type; barley straw and wheat straw emitted PAHs including benzo(a)pyrene at much higher levels than other cereals and wood fuel types. Total PAH emission rates increased with increasing particulate matter emission rates and with declining combustion efficiency (13).

Studies from wood stoves indicate that higher burn rates lead to fewer total organic emissions but the proportion of PAHs increases (5). Emission rates for PAHs were observed to be highest for temperatures in the range 500-800°C (14), and were consistent with results from a study of PAHs released in low intensity backing fires (9,11). The emission and mutagenic activity of PAHs from small wood stoves were observed to be greatly influenced by the quality of wood, with high levels (10-30 mg PAH) detected per kilogram of virgin wood (15).

Carbon monoxide (CO)

Health effects

Carbon monoxide gas causes tissue hypoxia by preventing the blood from carrying sufficient oxygen (16). At low to moderate concentrations, health effects include impaired thinking and perception, headaches, slow reflexes, reduced manual dexterity, decreased exercise capacity, and drowsiness. At higher concentrations, death may result. Persons at higher risk include those with preexisting cardiovascular and respiratory disease, infants, the elderly, and pregnant women. Unborn children are particularly susceptible because CO has a longer duration for clearance in the foetal circulatory system and foetuses cannot compensate for a reduction in oxyhemoglobin without a sustained increase in cardiac output (7).

Factors affecting health effects

Together with CO_2, CO accounts for 90 per cent to 95 per cent of the carbon produced during the combustion of biomass (17). CO release correlates highly with the release of other compounds in smoke, including PM and formaldehyde. CO emission factors range from 60 g/kg to more than 300 g/kg of fuel consumed. Studies indicate that CO emission factors in Brazil ranged from 167 g/kg to 209 g/kg, which is generally higher than similar measurements for logging slash fires in the western United States where the average emission factor is 171 g/kg. The differences are thought to result from variations in vegetation or moisture content.

Although one study indicates a significant increase in blood carboxyhemoglobin levels in non-smoking people who used biomass fuels for cooking (18), the factors that affect health effects have yet to be fully evaluated. According to the United States Occupational Safety and Health Administration (OSHA) regulations, the time-weighted average exposure limit is 50 parts per million (ppm) CO for an 8-hour work shift; the National Institute for Occupational Safety and Health limit is 35 ppm for 8 hours of exposure and 200 ppm for no defined time (5). Systematic studies of the effects of CO on human health should be performed, with carboxyhaemoglobin levels checked soon after exposure.

Aldehydes

Health effects

Aldehydes are primarily mucous membrane irritants. Some, such as formaldehyde, may be carcinogenic and in combination with other irritants may lead to an increase in the carcinogenicity of other compounds, such as PAHs (9). Formaldehyde and acrolein are the main aldehydes released during biomass burning. Formaldehyde, which is probably the most abundantly produced compound of this class, causes eye, nose, and throat irritation during smoke exposure (5). Low molecular weight aldehydes such as acrolein are thought to cause pulmonary lesions in rabbits (5).

Factors affecting health effects

These compounds have been poorly quantified as byproducts of the forest fuel combustion in the open environment. Sharkey (9) states that it is highly likely that acrolein is an irritant in smoke near firelines, with concentrations as high as 0.1 ppm to 10 ppm near fires.

Organic acids

Health effects

Organic acids, such as formic acid produced by the oxidation of formaldehyde, are known to form during the combustion of biomass fuels. Anticipated health effects include irritation of mucous membranes.

Factors affecting health effects

Organic acid production rates and combustion conditions, and the synergistic effects of some or all of these compounds, are unknown (9). It has been observed, however, that under equilibrium, high humidity could drive reactions to the production of organic acids. Aldehydes, for example, can produce acidic groups (e.g., formic acid, acetic acid) under conditions of high moisture.

Semivolatile and volatile organic compounds (VOCs)

Health effects

Some VOCs may cause skin and eye irritation, drowsiness, coughing and wheezing, while others (e.g., benzene and 1,3-butadiene) may be carcinogenic. Benzene and benzo(a)pyrene may be genetoxic carcinogens (7).

Factors affecting health effects

VOCs may have significant vapor pressures at ambient temperatures. Some compounds, such as benzene, naphthalene, and toluene, are partitioned between gaseous, liquid, or solid phases at ambient temperatures. Little work has been done to characterize VOCs in forest fires. To date, methane and CO gases are produced in proportion to semivolatile and VOCs and serve as indicators of their abundance (9).

Free radicals

Health effects

Free radicals are abundantly produced during the combustion of forest fuels. Free radicals may react with human tissues. They have been observed to persist up to 20 minutes following formation and pose a problem for firefighters exposed to fresh aerosols. Additional research is needed to determine the types and quantities of free radicals emitted during the combustion of biomass fuels, their persistence in the atmosphere, and subsequent health effects (9).

Factors affecting health effects

None noted at this time.

Ozone (O_3)

Health effects

O_3 is an extremely reactive oxidant. At high concentrations, it may impair lung function and reduce respiratory resistance to infectious diseases. People at risk include those with chronic respiratory illness. At low levels, human health may be affected when physical exercise is combined with several hours of exposure, i.e., tissue dose is enhanced by increased respiratory rate (7). At low concentrations, O_3 can cause symptoms such as coughing, choking, shortness of breath, excess sputum, throat tickle, raspy throat, nausea, and impaired lung function when exercising. Long-term health effects include decreased lung function and chronic obstructive pulmonary disease (7).

Factors affecting health effects

Concentrations of concern are not expected in areas close to fires. O_3 is formed photochemically near the top of smoke plumes under high sunlight. It is also formed when smoke is trapped in valleys or where there is a temperature inversion. Additionally, increased levels of O_3 may be encountered at high elevations.

OSHA has established standards for occupational exposure to O_3; however, these regulations have yet to be evaluated, along with other chemicals for biomass burning.

Inorganic fraction of particles

Health effects

Toxicologic effects of inorganic fractions from biomass fires have not been quantified. Health effects are dependent on the substance in question, such as lead, asbestos, and sulphur.

Factors affecting health effects

Inorganic materials, which are generally present in trace levels in smoke particles, are dependent on the chemistry of the fuels burned and the intensity at which the fire burns. Often, variability in the mineral content of fuels is enough to affect combustion. For example, in the United States,

particles in the Los Angeles Basin were found to have a higher lead content than particles from fires in the Pacific Northwest due to deposition of lead deposits in those areas (5). Similarly, asbestos fibers were carried with smoke from areas that naturally contained high deposits of asbestos (5). Also, organic soils of the southeast and fuels in the Yellowstone geyser basins would have emissions with sulphur-containing gases because they contain naturally occurring areas of high sulphur deposition (5).

Savannah fire aerosols are characterized by enrichments in elements such as potassium (K), chlorine (Cl), zinc (Zn), and bromine (Br), whereas forest fire emissions are enriched in silicon (Si) and calcium (Ca). Of the trace elements, K is found in relatively high concentrations in wood smoke. The combustion of hardwoods produces more ash and therefore higher concentrations of trace elements than does the combustion of softwoods (19).

Trace gases and other releases

Health effects

Trace gases, particularly polychlorinated dibenzo-p-dioxins (PCDDs), are extremely persistent and widely distributed in the environment. Very little human toxicity data related to PCDD are available. Data of health effects in occupational settings are based on exposure to chemicals contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD), which has a half-life of 7 to 11 years in humans. Dioxin exposures are associated with an increased risk of severe skin lesions (e.g., chloracne), and hyperpigmentation, altered liver function and lipid metabolism, general weakness associated with weight loss, changes in activities of various liver enzymes, immune system depression, and endocrine and nervous system abnormalities. TCDD also causes cancer of the liver and other organs in animals. Exposure to dioxin-contaminated chemicals has resulted in increased incidences of soft-tissue sarcoma and non-Hodgkin's lymphoma (20).

Methyl bromide and methyl chloride, which are sources of Br and Cl that destroy stratospheric ozone (21), may be carcinogenic (22). Health effects resulting from the emission of these compounds from biomass fires have yet to be determined.

Factors affecting health effects

Smoke emitted during the global annual combustion of about 2 to 3 billion metric tons of plant materials contains numerous toxic materials, some of which are dioxins. Forest and brush fires are major sources of PCDDs (23, 24). If flame temperatures exceed 1000 °C, essentially no dioxins are produced; however, this is rarely the case. In a study that involved burning various wood specimens in different stoves, total dioxins released were as much as 160 µg dioxin/kg wood. Soot collected and analyzed by well-designed and documented procedures indicated the presence of tetrachlorinated, hexachlorinated, heptachlorinated, and octachlorinated dioxins (23, 24).

Wood combustion products are spread around the world by winds. Consequently, PCDDs are found in soils in remote areas and tend to be bioconcentrated in the food chain (23).

Biomass fires emit a complex mixture of particulates and gases into the atmosphere. Globally, the diversity of combustion products results from wide ranges in fuel types and fire behaviour, which are induced by large variations in ecological types and weather phenomena. For instance, forest fires have lower combustion efficiency than grass fires, and therefore a larger fraction of smouldering compounds. During less efficient smouldering combustion, a large number of organic compounds are formed (5).

Radionuclides and herbicides

Health effects

Radionuclides, such as iodine-129 (^{129}I), cesium-137 (^{137}Cs), and chlorine-36 (^{36}Cl), can be released into the atmosphere, soil, and water, with immediate and long term consequences on health (25). They can cause cancer, depending on where in the body they are localized. For example, iodine is concentrated in the thyroid gland and the radioactive isotopes can cause thyroid cancer.

Fire occurring immediately after the application of herbicides may lead to adverse health effects in forest workers; however, in one study, no herbicide residues from an application containing the active ingredients imazapyr, triclopyr, hexazinone, and pioloram were detected in 140 smoke samples and 14 fires (26).

Factors affecting health effects

Fires can mobilize radionuclides from contaminated biomass through suspension of gases and particles in the atmosphere or solubilization and enrichment of the ash. Loss to the atmosphere increased with fire temperature, and during a typical field fire, 80 per cent to 90 per cent of the I and Cl, and up to 40 per cent to 70 per cent of the Cs was lost to the atmosphere (25).

In assessing exposure, factors such as fire conditions (high density smoke versus low density smoke) and personnel location should be taken into consideration when assessing exposure to airborne herbicide residues in smoke from prescribed fires (26).

RADIATIVE EFFECTS THROUGH GLOBAL COOLING

Smoke particles affect the global radiation balance by reflecting solar radiation directly, acting as cloud condensation nuclei, and increasing cloud reflectivity. The radiative effects of aerosols generated from biomass burning, dust storms, and forest fires could increase global cooling (i.e., or reduce the rate of global warming). Anthropogenic increases of smoke emissions may help weaken the net greenhouse warming from anthropogenic trace gases (27).

The effect is measured by Direct Radiative Forcing (DRF), the perturbation in the energy balance of the earth-atmosphere system; positive and negative values indicate warming and cooling, respectively, of the troposphere. For comparison, the net incoming solar radiation at the top of the atmosphere is 342 watts per square meter (W/m^2). Radiative forcing due to aerosols is comparable in magnitude to current anthropogenic greenhouse gas forcing but opposite in sign. The DRF due to long-lived greenhouse gases is 2.45 ± 0.37 W/m^2; the global average of DRF due to anthropogenic aerosols is -0.5 W/m^2, largely attributed to sulphate particles from fossil fuel combustion and smoke particles from biomass burning (28).

Severe aerosol loading results in immediate health effects, specifically respiratory disease from particle inhalation. It also may lead to a reduction in photosynthesis, which may affect the incidence of infectious and mosquito-borne diseases in the long-term. In regions of intensive biomass

burning, the photosynthetically active spectrum of sunlight (wavelengths of 400-700 nanometers (nm)) is reduced by 35 per cent to 40 per cent for two months (27). In one study, smoke from biomass burning caused significant aerosol optical thickness and up to an 81 per cent reduction in ultraviolet-B (UV-B) rays (29).

UV-B in natural sunlight kills airborne bacteria (27). The bactericidal effects of solar UV-B are well-known, and significantly reduced UV-B resulting from severe air pollution in regions where UV-B levels are ordinarily high might enhance the survivability of pathogenic organisms in air and water and on surfaces exposed to sunlight. In one study, exposing drinking water to normal intensities of UV-B has reduced diarrhea in children in Kenya by 33 per cent (30).

An increased incidence of respiratory, cardiopulmonary and other diseases are known to be associated with severe air pollution, but the biological mechanisms remain unknown.

The increased incidence of infectious and mosquito-transmitted disease has been raised as a possible consequence of severe aerosol loading. The larvae and pupae of some disease-transmitting mosquitoes are highly photophobic to ultraviolet-A (UV-A) rays and green wavelengths of sunlight. In 1995, in Brazil, smoke reduced sunlight in UV-A (340 nm range) as much as 45 per cent in an area peripheral to the region of maximum burning (29). Experiments with Culex pipiens, a vector for encephalitis, indicated that the females deposited eggs in the darkest nurseries and that their larvae avoided UV light (27).

The biological aspects of severe aerosol loading require further investigation. Estimates of emission factors of pollutants from forest fires and biological mechanisms leading to adverse health effects will improve global accounting of radiation-absorbing gases and particles that may be contributing to climate change and will provide strategic data for fire management.

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APPENDIX Y

http://www.epa.gov/agriculture/tburn.html
Last updated on 6/27/2012

Agriculture

Agricultural Burning

You will need Adobe Reader to view some of the files on this page. See EPA's PDF page to learn more about PDF, and for a link to the free Acrobat Reader.

Prescribed fire has long been a useful management tool for croplands, rangelands, and forests. As concern for air quality increases, however, it becomes more important to ensure that intentional or prescribed burning is used responsibly. EPA is working with the agricultural community to devise reasonable, science-based policies that define the role of agricultural burning in a way that allows efficient agricultural production as well as a healthy environment.

Related topics
Air

Related publications from the Ag Center
Agricultural Burning
Air

Related contacts
Air

More information from EPA
National Homeland Security Research Center - The National Homeland Security Research Center (NHSRC) develops and delivers reliable, responsive expertise and products based on scientific research and evaluations of technology. Our expertise and products are widely used to prevent, prepare for, and recover from public health and environmental emergencies arising from terrorist threats and incidents.

More information from USDA
CSREES National Air Quality Program
USDA Forest Service: BlueSky Framework - A Web-Based Information System to Help Manage Prescribed Burning, Wildland Fires and Agricultural Burning

More information from universities EXIT Disclaimer
Center for Ag Air Quality Engineering and Science

Background: Clean Air Act -- Title I

Pursuant to Title I of the CAA, EPA has established national ambient air quality standards (NAAQSs) to limit levels of "criteria pollutants," including carbon monoxide, lead, nitrogen dioxide, particulate matter, ozone, and sulfur dioxide.

EPA calls these pollutants "criteria air pollutants" because the agency has regulated them by first developing health-based criteria (science-based guidelines) as the basis for setting permissible levels. One set of limits (primary standard) protects health; another set of limits (secondary

standard) is intended to prevent environmental and property damage.

A geographic area that meets or does better than the primary standard is called an attainment area; areas that don't meet the primary standard are called nonattainment areas. A single geographic area may have acceptable levels of one criteria air pollutant but unacceptable levels of one or more other criteria air pollutants; thus, an area can be both attainment and nonattainment at the same time.

Under Section 110 of the CAA, each state must develop a State Implementation Plan (SIP) to identify sources of air pollution and to determine what reductions are required to meet federal air quality standards. A State Implementation Plan is a detailed description of the programs a state will use to carry out its responsibilities under the Clean Air Act.

Technical guidance and data sources

EPA maintains a Clearinghouse for Inventories and Emission Factors (CHIEF). The CHIEF web site provides access to tools for estimating emissions of air pollutants in various geographic domains (e.g. urban areas, regions, or the entire nation). It serves as EPA's central clearinghouse for the latest information on air emission inventories and emission factors. Emission estimation data bases, newsletters, announcements, and guidance on performing inventories are included in CHIEF.

The Factor Information Retrieval (FIRE) Data System (one of the tools offered by CHIEF) is a database management system containing EPA's recommended emission estimation factors for criteria and hazardous air pollutants. FIRE includes information about industries and their emitting processes, the chemicals emitted, and the emission factors themselves. The FIRE database is designed for use by local, state, and federal agencies, environmental consultants, and others who require emission factor information for estimating both criteria and toxic air emissions from stationary sources.

More information from EPA

Compilation of Air Pollutant Emission Factors -- AP-42, Fifth Edition, Volume I
EIIP Preferred and Alternative Methods for Estimating Air Emissions (PDF) (72 pp, 252K) -- Volume III, Chapter 16 - Open Burning
EIIP Preferred and Alternative Methods for Estimating Air Emissions -- Volume VIII, Chapter 11 -- Methods for estimating greenhouse gas emissions from burning of agricultural crop wastes (currently under revision)

Enforcement

The 1990 Clean Air Act gives important enforcement powers to EPA. It used to be very difficult for EPA to penalize a company for violating the Clean Air Act. The 1990 law enables EPA to fine violators. Other parts of the 1990 law increase penalties for violating the Act and bring the Clean Air Act's enforcement powers in line with other environmental laws.

Related topics

Clean Air Act

More information from EPA

Text of the Clean Air Act
Clean Air Act -- Plain English Guide
State Air Programs
Clean Air Act (CAA) Enforcement Policy and Guidance

Telephone assistance from EPA
EPA's Control Technology Center (919-541-0800) provides general assistance and information on CAA standards.

Air Emissions From Agricultural Practices

Under Section 110 of the CAA, each state must develop a State Implementation Plan (SIP) to identify the sources of air pollution and to determine what reductions are required to meet federal air quality standards.

State implementation plans are collections of the regulations used by a state to reduce air pollution. The Clean Air Act requires that EPA approve each state implementation plan. Members of the public are given opportunities to participate in review and approval of state implementation plans.

The degree to which ambient air emissions from farming practices -- such as prescribed burning -- are allowed are location-specific (specific to a geographic area) within each State Implementation Plan. Visibility standards may also apply through the State Implementation Plan. Locations that are in areas that have been classified as "nonattainment areas" under the National Ambient Air Quality Standards are subject to more restrictions.

The U.S. Department of Agriculture (USDA) has established the Agriculture Air Quality Task Force. EXIT Disclaimer EPA is an active participant in the Task Force. The Task Force has unanimously endorsed a listing of high priority research needs to improve the level of understanding of the impact of agriculture on air quality levels.

On February 25, 1998, the USDA and EPA announced a Memorandum of Understanding (MOU) to ensure that the two agencies work together to provide a healthy environment with clean air in harmony with a strong agriculturally productive nation. The MOU establishes a framework for the two agencies to share expertise and a process for involving the agricultural community in a cooperative effort to address agriculture-related air quality issues, including emissions from agricultural burning.

EPA will work with the task force to refine the distinction between wildland fires (which are covered by EPA's Interim Air Quality Policy on Wildland and Prescribed Fires) and agricultural burning.

Related environmental requirements
CAA Section 109 - 110
40 CFR Part 52 EXIT Disclaimer - contains approval and promulgation of each state's specific implementation plan (SIP)

Related information
Particulates

More information from EPA
National Ambient Air Quality Standards
Nonattainment Areas for Criteria Pollutants
Classifications of Particulate Matter Nonattainment Areas

Prescribed Burning

Prescribed burning is a land treatment, used under controlled conditions, to accomplish natural resource management objectives. It is one of several land treatments, used individually or in combination, including chemical and mechanical methods.

Prescribed fires are conducted within the limits of a fire plan and prescription that describes both the acceptable range of weather, moisture, fuel, and fire behavior parameters, and the ignition method to achieve the desired effects. Prescribed fire is a cost-effective and ecologically sound tool for forest, range, and wetland management. Its use reduces the potential for destructive wildfires and thus maintains long-term air quality. Also, the practice removes logging residues, controls insects and disease, improves wildlife habitat and forage production, increases water yield, maintains natural succession of plant communities, and reduces the need for pesticides and herbicides.

The major air pollutant of concern is the smoke produced. Smoke from prescribed fires is a complex mixture of carbon, tars, liquids, and different gases. This open combustion source produces particles of widely ranging size, depending to some extent on the rate of energy release of the fire. The major pollutants from wildland burning are particulate, carbon monoxide, and volatile organics. Nitrogen oxides are emitted at rates of from 1 to 4 g/kg burned, depending on combustion temperatures. Emissions of sulfur oxides are negligible.

Some pollution prevention practices that can be used during prescribed burning operations include the following:

- Carefully plan burning to adhere to weather, time of year, and fuel conditions that will help achieve the desired results and minimize impacts on water quality.
- Intense prescribed fire for site preparation should not be conducted in the streamside management areas.
- Avoid conditions requiring extensive blading of firelines by heavy equipment.
- Revegetate firelines with adapted herbaceous species.
- Avoid burning on steep slopes with high erosion hazard areas or highly erodible soils.
- Construct firelines in a manner that minimizes erosion and sedimentation and prevents runoff from directly entering watercourses.

Related publications from the Ag Center
Ag Sector Profiles (Sector Notebooks)

More information from EPA
Compilation of Air Pollutant Emission Factors: Wildfires and Prescribed Burning (Chapter 13.1) (PDF) (14 pp, 76K)
Interim EPA Air Quality Policy on Wildland and Prescribed Fires

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Particulates

Particulate matter is the term for solid or liquid particles found in the air. Some particles are large or dark enough to be seen, such as soot or smoke. Others are so small they can be detected only with an electron microscope.

Breathing particulate matter can cause serious health problems. Particulates also reduce visibility in many parts of the United States. They can also accelerate corrosion of metals and damage paints and building materials such as concrete and limestone.

Sources of particulates
"Coarse" particles are larger than 2.5 micrometers and generally come from sources such as vehicles traveling on unpaved roads, materials handling, crushing and grinding operations such as cement manufacturing, and combustion sources.

Particles less than 2.5 micrometers (0.0004 inch) in diameter are known as "fine" particles. Fine particles result from fuel combustion in motor vehicles, power plants and industrial facilities, residential fireplaces, woodstoves, wildfires, and prescribed forest burning. Fine particles can also be formed when combustion gases are chemically

transformed into particles.

Health effects of particulates
Particulate matter less than 10 micrometers in size, including fine particles less than 2.5 micrometers, can penetrate deep into the lungs. On a smoggy day, one can inhale millions of particles in a single breath. Tens of millions of Americans live in areas that exceed the national health standards for particulates.

In recent studies, exposure to particulate pollution -- either alone or with other air pollutants -- has been linked with premature death, difficult breathing, aggravated asthma, increased hospital admissions and emergency room visits, and increased respiratory symptoms in children. People most at risk from exposure to fine particulate matter are children, the elderly, and people with chronic respiratory problems.

Environmental effects of particulates
Fine particles scatter and absorb light, creating a haze that limits our ability to see distant objects. Particle plumes of smoke, dust, and/or colored gases that are released to the air can generally be traced to local sources such as industrial facilities or agricultural burning. Regional haze is produced by many widely dispersed sources, reducing visibility over large areas that may include several states.

The Clean Air Act established special goals for visibility in some national parks and wilderness areas. In 1994, EPA began developing a regional haze program that is intended to ensure that continued progress is made toward the national visibility goal of "no manmade impairment." Such control efforts would likely result in improved public health protection and visibility in areas outside national parks as well.

More information from EPA
Information on Particulate Matter
Grand Canyon Visibility Transport Commission Recommendations (PDF) (109 pp, 1.5MB) EXIT Disclaimer
Grand Canyon Visibility -- Draft Regulatory Language (PDF) (8 pp, 21K)
Annex to the Report of the Grand Canyon Visibility Transport Commission (PDF) (162 pp, 329K)

State Programs and Information EXIT Disclaimer

- **Arizona**
  Arizona State Smoke Management Program
  Prescott National Forest -- Fire Information: Prescribed Burn
- **California**
  Smoke Management Program
  Rice Straw Burning: Overview
  Rice Straw Burning: 2003, 2001, 1999, and 1997 Reports to Legislature
  Rice Straw Demonstration Project Fund
- **Florida**
  Prescribed Fire Information
- **Idaho**
  Fire Management
  Airshed Group
- **Kansas**
  Kansas Flint Hills Smoke Management
  Kansas Flint Hills Smoke Management Plan (PDF) (53 pp, 935K)
  Open Burning Regulations (PDF) (3 pp, 20K)
- **Louisiana**
  Sugarcane Burning (PDF) (4 pp, 149K)
  Smoke Management Guidelines for Sugarcane Harvesting (PDF) (14 pp, 177K)

- **Nebraska**
  University of Nebraska: Grassland Management with Prescribed Fire (PDF) (6 pp, 799K)
- **Oregon**
  Smoke Management Statute
  Oregon Department of Forestry
- **South Carolina**
  Prescribed Forest Burning BMPs
- **Washington**
  Fire Information
  Agricultural Burning Focus Sheet (PDF) (2 pp, 25K)
  Agriculture Burning Regulation
  -- Text (PDF) (19 pp, 72.1K)
  -- Summary

More information from the states EXIT Disclaimer
Air Pollution State Resource Locator - State and regional regulatory agencies and rules covering topics such as open burning, smoke and dust

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Federal Government Information

- **National Oceanic and Atmospheric Administration**
  Wildfire Smoke Forecasting at NOAA's Air Resources Laboratory
- **National Park Service**
  Use of a Deterministic Fire Growth Model To Test Fuel Treatments
- **U.S. Forest Service**
  Emissions From National Forests in Oregon and Washington
- **U.S. Geological Survey**
  Prescribed Burning Guidelines in the Northern Great Plains

Federal, Regional, and State Contacts

- EPA Office of Enforcement and Compliance Assurance -- Air Enforcement Division
- EPA Control Technology Center Hotline: 919-541-0800
- Air Pollution Contacts in EPA Regions
- EPA Regional and State Air Quality Contacts

APPENDIX Z

Smoke From Cane Fire Forces Intermittent Closures on Kuihelani

Updated 06:11 AM HST, June 12, 2013
Posted 06:17 PM HST, June 11, 2013

Kuihelani cane fire, 6/11/13. Photo courtesy, Susan Figg.

By Wendy Osher

A portion of the Kuihelani Highway on Maui was partially closed on two separate occasions on Tuesday, as smoke from a scheduled cane fire hampered vision along the roadway.

The first closure was reported at 5:45 am. on Tuesday, June 11, along the Kuihelani Highway between the Honoapi‘ilani Highway and Waiko Road.

At one point during the closure, police began diverting southbound traffic onto Waiko Road before completely opening the highway at 8:09 a.m.

The same stretch of highway was closed again at 1:25 p.m. due to poor visibility from heavy smoke in the same area, with a detour set up at Waiko Road for approaching south-bound traffic.

Police issued an updated advisory at 3:42 p.m. saying that all roads in Maui County were open.

According to a notification issued by Hawaiian Commercial and Sugar Company, there was a scheduled cane burn in the area that was planned between 4 and 6 a.m. along the Honoapi‘ilani Highway in Waikapu.

A separate notification was issued just before 11 a.m., indicating that cane burning is scheduled for the same time tomorrow, Wednesday, June 12, in the same area.

The company began their 141st harvest season in mid-March.

According to the material published by HC&S, company officials say schedules can change based on updated weather forecasts and operating conditions at the processing plant.

The public can access updated cane burning schedules via text, email, or online at HCSugar.com.

****Additional Information: Maui police have since confirmed that there were two accidents on Tuesday morning in the area of the road closure. Police say there were no life-threatening injuries. Authorities could not confirm the cause of accidents.*

UF UNIVERSITY of FLORIDA
IFAS Extension

The History of U.S. Sugar Protection [1]

Jose Alvarez and Leo C. Polopolus[2]

This is part of the Sugar Policy series, which discusses policy issues facing the U.S. sweetener industry in general and Florida's sweetener industry in particular. The objective of this article is to summarize the history of government protection and control in the U.S. sugar industry. Readers interested in a more detailed description are referred to Alvarez and Polopolus (1988).

Introduction

National sugar policy has been affected by statute since 1789 when the First Congress of the United States imposed a tariff upon foreign sugar. The purpose of this and subsequent tariffs was to provide revenue for the government. From 1789 to 1930, a total of 30 Acts dealing with sugar were passed, not including several modifications. In 1842, however, the purpose of the tariff was expanded to include a policy of protection by subjecting refined sugar to a higher tariff than raw sugar, thereby attempting the promotion of a domestic refining industry and the expansion of domestic raw sugar production. It was not until 1934 that a federal sugar program was enacted. Higher government intervention was motivated by the belief that sugar production and marketing was regulated in both producing and importing countries more than any other commodity. The absence of true competition between domestically produced sugar and sugar produced with cheap labor or under subsidy in other countries led to further controls started with the first Sugar Act.

Figure 1.

1. This is EDIS document SC 019, a publication of the Department of Food and Resource Economics, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida, Gainesville, FL. First published November 1990; revised June 2002 and May 2012. This publication is also part of the Florida Sugarcane Handbook, an electronic publication of the Department of Agronomy, University of Florida, Gainesville, FL. For more information, contact the editor of the Sugarcane Handbook, Ronald W. Rice (rwr@ufl.edu). Please visit the EDIS website at http://EDIS.ifas.ufl.edu.

2. Jose Alvarez, Professor, Department of Food and Resource Economics, Everglades Research and Education Center, Belle Glade, FL; and Leo C. Polopolus, Professor Emeritus, Department of Food and Resource Economics, Florida Cooperative Extension Service, Institute of Food and Agricultural Sciences, University of Florida, Gainesville, FL 32611.

The Sugar Acts, 1934-1974

The first Sugar Act, known as the Jones-Costigan Act, became law on May 9, 1934. Its main six features were as follows:

1. the determination each year of the quantity of sugar needed to supply the nation's requirements at prices reasonable to consumers and fair to producers;

2. the division of the U.S. sugar market among the domestic and foreign supplying areas by the use of quotas and subordinate limitations on offshore direct consumption sugar;

3. the allotment of these quotas among the various processors in each domestic area;

4. the adjustment of production in each domestic area to the established quota;

5. the levying of a tax on the processing of sugarcane and sugar beets, the proceeds of which were to be used to make payments to producers to compensate them for adjusting their production to marketing quotas to increase their income;

6. the equitable division of sugar returns among beet and cane processors, growers, and farm workers.

The U.S. Congress repealed the processing tax (declared unconstitutional by the Supreme Court in early 1936) and payment provisions. The President recommended new legislation that resulted in the enactment of the Sugar Act of 1937. The new Act contained an excise tax that was unrelated to government payments to growers, which would be made out of the U.S. Treasury from funds appropriated for that purpose. Quota allocations resulting from any market expansion were to be shared proportionally by both domestic and foreign areas. This Sugar Act was originally scheduled to expire in 1940 but was extended to 1941, then from 1941 to 1944, from 1944 to 1946, and again from 1946 through December 31, 1947.

The Sugar Act of 1948, which superseded the 1937 Act, only changed the method of establishing quotas, with Cuba receiving preferential treatment because of its response in increasing output during World War II to supply sugar to the U.S. at prices far below those that would have prevailed in a free market. The new Sugar Act was amended and extended several times, through December 31, 1960. During that year, sugar imports from Cuba were officially suspended and its quota transferred to other foreign countries on a temporary basis. Two short-term extensions in 1960 and 1961 provided for continuation of the Act until June 30, 1962. The Act was again amended in July 1962 and extended through December 31, 1966. Several amendments followed in subsequent years until December 31, 1974, when the Act expired.

The policy instruments of the previous Acts were many and varied. They included tariffs (with a minor role), quotas (the major role), fees (used for a relatively short period of time), taxes (for revenue generation), payments, allotments (not always in effect), and minimum wage provisions.

The reasons for the demise of the Sugar Act have been explained by Crawford (1978) in the following manner:

Beginning in the 1970s, the energy crisis, inflation, and global commodity shortages struck at the basic foundation of the program. The supply management system's assumption of a world surplus of sugar was challenged, as world consumption outstripped production in 4 of the 5 years from 1970 through 1974. The result was a dramatic swelling of sugar prices, which the Sugar Act's provisions were unable to stem (p. 17).

Federal Sugar Programs Since 1974

The expiration of the Sugar Act caused immediate repercussions in the U.S. domestic and foreign sugar policy areas. Corley (1975) listed the changes occurring in the policy instruments of the program as follows:

- The current duty rates for sugar were retained and a global quota of seven million tons was established by Presidential Proclamation.
- Import restrictions on refined sugar were eliminated.
- Direct payments to producers no longer existed after 1974.
- The Secretary of Agriculture lost the authority to allocate sugar quotas for the domestic areas.
- Acreage allotments were eliminated and production became unrestricted.
- The excise tax on domestic and on imported refined sugar was to be terminated on June 30, 1975.

The conditions that prevailed in 1974, when world sugar prices averaged a record 57 cents per pound raw value in November and 30 cents during that calendar year, did not persist very long. Just three years later, low sugar prices (which averaged 8.1 cents per pound in 1977 and 7.8 cents per pound in 1978) and increased costs of production, processing, and marketing prompted the government to initiate price support programs to ensure favorable returns to the industry.

An interim price support program was instituted in 1977 by the Secretary of Agriculture under the authority of the Agricultural Act of 1949. Processors received the difference between the price objective of 13.5 cents a pound, raw sugar basis, and the average market price, which was defined as the price deemed necessary to support efficient producers. In return, processors were required to pay producers of sugar beets and sugarcane at least $22.84 and $17.48 (U.S. average) per ton of average-quality sugar beets and sugarcane, respectively.

The Food and Agriculture Act of 1977 mandated a loan or purchase program for the 1977 and 1978 crops. The payment program ended on November 7, 1977. Loan rates were established at 13.5 and 14.73 cents per pound, raw value, for the 1977 and 1978 crops, respectively. Processors agreed to pay producers at least those prices if they met USDA minimum wages for field workers. Processors who obtained the loan used sugar as a collateral, and interest was charged only if the loan was redeemed. They could default on the loan and forfeit the sugar to the Commodity Credit Corporation (CCC) if the market price was not high enough. In order to minimize the risk of forfeiture, a "market price objective" was maintained through import duties and fees on imported sugar. Forfeitures, however, occurred in both 1977 and 1978.

A similar program was adopted for the 1979 crop. No program was in effect for the 1980 and 1981 sugar crops since, during those years, the industry was again enjoying relatively high market prices.

The Agriculture and Food Act of 1981 mandated a price support program for sugar for the 1982 through 1985 crop years. Loan rates for cane sugar were set at 17 cents a pound for the 1982 crop, 17.5 cents for 1983, 17.75 for 1984, and 18 cents for 1985. Beet sugar was to be supported at the levels that the Secretary of Agriculture thought to be fair and reasonable in relation to the support level for cane sugar. A "market stabilization price" (MSP) was established for raw cane sugar above the purchase or loan rate to discourage sale or forfeiture of any sugar to the CCC. This MSP minus the purchase or loan rate covers freight and related marketing expenses, the interest required to redeem the loan, and an incentive factor to encourage processors to sell sugar in the marketplace rather than to the CCC. Import duties were increased and a system of country-by-country import quotas was established.

The Food Security Act of 1985, which left almost intact the major provisions of the previous Act, was to apply to the 1986 through the 1990 crop years with a loan rate of no less than 18 cents a pound. Special emphasis was given to the operation of the program at no cost to the federal government by restricting supplies. This Act was replaced by the 1990 Farm Bill drafted by Congress.

The sugar program in the 1990 Farm Bill, also known as the Food, Agriculture, Conservation, and Trade Act of 1990, covered the 1991 through 1996 sugar crops. Although most of the provisions in previous legislation remained unchanged, some new features were included. The loan rate remained at 18 cents per pound despite efforts to reduce it. The Market Stabilization Price of 21.95 cents per pound established by the USDA for the 1989-90 crop remained unchanged. A minimum import quota of 1.25 million short tons, raw value, with a two-tiered tariff scheme, was established, as well as marketing controls on domestic sugar if imports were projected to fall below 1.25 million tons. As a result, a new re-export program had to be designed. A program service fee was imposed. Finally, marketing controls could be imposed if imports were projected to fall below 1.25 million tons.

The 1996 Farm Bill, also known as the Federal Agricultural Improvement and Reform (FAIR) Act, became effective for the 1996 through 2002 crops. The loan rate remained at 18 cents per pound. Loans are recourse when the tariff rate (TRQ) quota on sugar imports is at 1.5 million tons or below, and nonrecourse when the TRQ exceeds that level. Cane processors must pay a penalty of one cent per pound of sugar forfeited to the government, and beet processors must pay a 1.07 cents per pound penalty. Interest rates established by the CCC were set at one percent higher than in the past. Finally, the import quota system remained the same, while marketing controls on domestic sugar were eliminated.

President Bush has now signed the 2002 Farm Bill. No major changes in the sugar program were contained in the legislation. This farm bill is discussed in another article in this series (SC056, Sugar and the 2002 U.S. Farm Bill).

References

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Corley, J.R. (1975). Effects of the sugar act's expiration. Foreign Trade Newsletter, U.S. Department of Agriculture Extension Service, Washington, D.C. (February).

Crawford, H. (1978). Sugar: Many questions face the Congress. *Food Update* 9: 16-19.

Sugar Policy series

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The History of U.S. Sugar Protection

The Sugar Program: Description and Debate

The Florida Sugar Industry

Archived, available in University of Florida Digital Collections

Domestic and International Competition in Sugar Markets

Sugar and the General Agreement on Tariffs and Trade

Sugar and the North American Free Trade Agreement

Sugar and the World Trade Organization (WTO): Major Issues

Sugar and the World Trade Organization (WTO): Progress To-Date

APPENDIX CC

United States Department of Agriculture

Economic Research Service

Policy

Related Amber Waves Articles

Complex Array of Factors Influence World Sugar Prices

Indian Sugar Market More Volatile

U.S. Sugar Program at a Crossroads

***Note**: This topic page may contain material that has not yet been updated to reflect the new Farm Act, signed into law on February 7. ERS has published highlights and some implications of the Act's new programs and provisions. Sign up for the ERS Farm Bill e-newsletter to receive notices of topic page updates and other new Farm Bill-related materials on the ERS website.*

Domestic Price Support

Flexible Marketing Allotments

Disposition of Sugar Owned by the CCC

Sugar Tariff-Rate Quotas and Other Trade Measures

Re-Export Programs

Dominican Republic-Central American Free Trade Agreement

The U.S. sugar program uses price supports, domestic marketing allotments, and tariff-rate quotas (TRQs) to influence the amount of sugar available to the U.S. market. The program supports U.S. sugar prices above comparable levels in the world market. The origin of the program can be traced to legislation in the Agriculture and Food Act of 1981 (1981 Farm Act). The program has been reauthorized with some modifications in succeeding Farm Acts. An important aspect of the program is that it operates, to the maximum extent possible, at no cost to the Federal Government by avoiding loan forfeitures to USDA's Commodity Credit Corporation (CCC).

A new measure introduced in the Food, Conservation, and Energy Act of 2008 (2008 Farm Act) to help avoid loan forfeitures is the Feedstock Flexibility Program (FFP). The FFP will divert sugar in excess of domestic food consumption requirements to ethanol production. The main challenge to the program comes from sugar imports from Mexico that now enter duty-free under the terms of the North American Free Trade Agreement (NAFTA).

Domestic Price Support

The 2008 Farm Act provides for USDA to make loans available to processors of domestically grown sugarcane and to domestic processors of sugar beets at set loan rate levels for fiscal years (FY) 2009-13. Loans are taken for a maximum term of 9 months and must be liquidated along with interest charges by the end of the fiscal year in which the loan was made. Unlike most other commodity programs, the sugar program makes loans to processors and not directly to producers. The reason is that sugarcane and sugar beets, being bulky and very perishable, must be processed into sugar before they can be traded and stored. To qualify for loans, processors must agree to provide payments to producers that are proportional to the value of the loan received by the processor for sugar beets and sugarcane delivered by producers. USDA has the authority to establish minimum producer payment amounts.

The loans are nonrecourse. When a loan matures, USDA must accept sugar pledged as collateral as payment in full, in lieu of cash repayment of the loan, at the discretion of the processor. "In-process" sugar and syrups must be converted into raw cane or refined beet sugar at no cost to the CCC before being eligible for forfeiture. The processor is not required to notify USDA of the intention to forfeit the sugar under loan. The loan rates for raw cane and beet sugar are set in the 2008 Farm Act.

The loan rate for raw cane sugar is:

18 cents per pound in FY 2009,

18.25 cents per pound in FY 2010,

18.50 cents per pound in FY 2011, and

18.75 cents per pound in FY 2012-13.

The loan rate for refined beet sugar is:

22.9 cents per pound in FY 2009 and

128.5 percent of the loan rate for raw cane sugar in FY 2010-13.

The 2008 Farm Act allows processors to obtain loans for in-process sugar and syrups at 80 percent of the loan rate.

Flexible Marketing Allotments

Sugar sold in the United States for domestic human consumption by domestic sugar beet and sugarcane processors is subject to marketing allotments, as a way to guarantee the sugar loan program operates at no cost to the Federal Government. The overall allotment quantity (OAQ) is determined subject to two conditions: 1) domestic sugar prices remain above forfeiture levels and 2) the OAQ is at least 85 percent of estimated deliveries for domestic human consumption for the marketing year (October to September). Allotments are in effect the entire year; there are no criteria for suspension. During the course of the marketing year, USDA is required to adjust allotment quantities to avoid the forfeiture of sugar to CCC.

OAQ allocations are divided between refined beet sugar at 54.35 percent of the overall quantity and raw cane sugar at 45.65 percent of the overall quantity. For cane sugar, Hawaii is allotted 325,000 short tons, raw value (STRV). The allocations for the mainland cane sugar-producing States (Florida, Louisiana, and

Texas) are assigned based on the States' and processors' production in crop years 1999-2003. Beet sugar processors are assigned allotments based on their sugar production in crop years 1998-2000. The 2008 Farm Act sets out allocation conditions for new entrants and for the effect of sale of factories between processors.

The 2008 Farm Act provides for a number of contingencies that could require reassignment of allotments during the crop year. If a cane processor that has been allocated an OAQ share cannot market the share, it is reassigned to the other processors within the same State, taking into account their ability to make up the deficit and also the interests of producers served by the processors. If the deficit cannot be eliminated by this step, then the remainder is allocated to the other cane-producing States, and then to the processors in those States. If the deficit still is not eliminated, it is assigned to the CCC for sale from CCC inventories. If CCC inventories are insufficient to cover the deficit, then the deficit is assigned to imports. The procedure for a beet sugar processor deficit is similar, except there is no reassignment based on States where processing takes place. There is no provision for cane sugar OAQ deficits to be reassigned to beet sugar processors, or for beet sugar OAQ deficits to be reassigned to cane sugar processors.

The 2008 Farm Act explicitly states that sugar forfeited to the CCC counts against marketing allotments made in the year in which the loan to the processor was made. This clarification reinforces that sugar in excess of a processor's allotment at the end of the marketing year cannot be forfeited. Other marketings counting against allotments include a sale of sugar under the FFP; export of sugar from the U.S. Customs Territory eligible to receive credits under re-export programs for refined sugar or sugar-containing products administered by USDA's Foreign Agricultural Service (FAS); sale of sugar eligible to receive credit for the production of polyhydric alcohol under the FAS-administered Polyhydric Alcohol Program; and for any integrated processor and refiner, the movement of raw cane sugar into the refining process.

Feedstock Flexibility Program

The Feedstock Flexibility Program operates to avoid sugar loan forfeitures to the CCC by requiring the diversion of sugar from food use to ethanol production. On September 1 (1 month before the end of the marketing year), the Secretary of Agriculture announces the amount of sugar (if any) for the CCC to purchase and to be made available for sale to ethanol producers. Raw, refined, and in-process sugars are eligible for purchase. Such sugar can be purchased from any marketer located in the United States. As mentioned previously, sugar purchased from a sugarcane or sugar beet processor is counted against that processor's marketing allotment.

Disposition of Sugar Owned by the CCC

The 2008 Farm Act provides for specific ways to dispose of sugar owned by the CCC without increasing future forfeiture risk. Like the Farm Security Act of 2002 (2002 Farm Act), the 2008 Farm Act includes the payment-in-kind (PIK) authority to transfer ownership of CCC sugar to processors in exchange for reductions in production through reduced sugar crop planting. For area already planted, the processor cannot commercially market the crop other than as a bioenergy feedstock.

The 2008 Farm Act explicitly authorizes the sale of CCC sugar for the production of ethanol and for the buyback of certificates of quota entry (also referred to as certificates for quota eligibility, or CQEs) to reduce tariff-rate quota imports. To comply with the goal of preventing sugar forfeitures, the 2008 Farm Act prohibits the sale of CCC sugar for domestic human consumption. (Such sales would seem to be permissible if they resulted from a reassignment of OAQ from a sugar processor to the CCC, as provided for under the 2002 and 2008 Farm Acts. In this instance, the likelihood of sugar forfeiture would seem to be minimal.)

Sugar Tariff-Rate Quotas and Other Trade Measures

The United States establishes separate tariff-rate quotas (TRQs) for imports of raw cane sugar and refined sugar (also called "certain other sugars, syrups, and molasses"). Prior to the start of the fiscal year (October 1-September 30), the Secretary of Agriculture announces the quantity of sugar that may be imported at the preferential in-quota tariff rate during that fiscal year. There is no limit to the quantity

that may be imported at the higher over-quota tariff rate.

Under the Uruguay Round Agreement on Agriculture (AoA), the United States agreed to make available for import a minimum quantity of raw and refined sugar each marketing year. This amount is equal to 1.139 million metric tons, raw value (MTRV), or 1.256 million STRV. Included in this amount is a commitment to import at least 22,000 MTRV, or 24,251 STRV, of refined sugar. The United States administers additional TRQs on imports of various sugar-containing products that originally had been subject to absolute quotas under Section 22 of the Agricultural Adjustment Act of 1933. There are four of these additional TRQs, none of which apply to Mexico under NAFTA.

According to the Harmonized Tariff Schedule of the United States (Ch.17, Additional U.S. Note 5 (a) (ii)), whenever the Secretary of Agriculture believes that domestic supplies of sugars may be inadequate to meet domestic demand at reasonable prices, the Secretary may modify any quantitative limitations that have previously been established, but not below the minimum quantities under the AoA.

The raw cane sugar TRQ is currently allocated by Office of the U.S. Trade Representative (USTR) to 40 countries based on a representative period (1975-81) when trade was relatively unrestricted. The refined sugar tariff rate quota is currently allocated to Canada and Mexico, and there is a quantity of refined sugar that is available to all countries on a first-come, first-served basis. Likewise, there is an allocation for specialty sugars, which is also on a first-come, first-served basis.

The in-quota tariff for sugar is equal to 0.625 cents per pound. Most countries have the low-tier tariff waived under either the Generalized System of Preferences (see Agricultural Trade Preferences and the Developing Countries, page 3, for more information), the Caribbean Basin Initiative, or under U.S. free trade agreements. The over-quota tariff is 15.36 cents per pound for raw sugar and 16.21 cents per pound for refined sugar. In addition to the over-quota tariffs, there are safeguard duties based on the value or quantity of the imported sugar. Currently, these duties are based on value.

Re-Export Programs

The United States also operates two re-export programs, as well as a sugar-for-polyhydric alcohol import program, to help U.S. sugar refiners and manufacturers of sugar-containing products compete in world markets. The Refined Sugar Re-Export Program establishes a license against which a company can import sugar at world prices for refining and sale to replace sugar in the market that has been exported as refined sugar or as sugar in sugar-containing products. The Sugar-Containing Products Re-Export Program allows U.S. participants to buy sugar at world prices for use in products that will be exported onto the world market. Raw cane sugar imports under these programs are not subject to the sugar TRQs. All refined sugars derived from either sugar beets or sugarcane are substitutable under these programs.

Dominican Republic-Central American Free Trade Agreement

Under the Dominican Republic-Central American Free Trade Agreement (DR-CAFTA), there are specific provisions for trade in sugar. The United States establishes country-specific TRQs for the DR-CAFTA countries, starting at a total of 107,000 metric tons in 2006 (year 1) and growing to 151,140 metric tons in year 15, thereafter growing by 2,640 metric tons per year, into perpetuity. A 2,000-metric-ton TRQ, with no growth, is established for Costa Rica for specialty sugar. Each country's duty-free access will be the lesser of its trade surplus or its TRQ for that year. Provisions have been agreed to allow alternative forms of compensation to be established to facilitate sugar stock management by the United States.

2014 Farm Bill

UNITED STATES DEPARTMENT OF AGRICULTURE
FARM SERVICE AGENCY

FACT SHEET

March 2014

Sugar Loan Program, Sugar Marketing Allotments and Feedstock Flexibility Program

OVERVIEW

The Sugar Loan Program provides nonrecourse loans to processors of domestically grown sugarcane and sugar beets. Generally, loan programs provide producers interim financing at harvest time to meet cash flow needs without having to sell their commodities when market prices are typically at harvest-time lows. Allowing producers to store production at harvest facilitates more orderly marketing of commodities throughout the year. The Agricultural Act of 2014 (2014 Farm Bill) provides the Farm Service Agency (FSA) the authority to administer nonrecourse loans for the 2011 through 2018 crops on behalf of the Commodity Credit Corporation (CCC).

LOAN ELIGIBILITY

Sugar and in-process sugar loans are available beginning October 1 each fiscal year and mature at the earlier of:

(1) The end of the nine-month period beginning on the first day of the first month after the month in which the loan is made, or
(2) The end of the fiscal year in which the loan is made.

In the case of a loan made in the last three months of a fiscal year (July, August and September), the processor may re-pledge the sugar as collateral for a second loan (referred to as a supplemental loan) in the subsequent fiscal year. The supplemental loan is made at the loan rate in effect at the time the initial loan was made, and matures in nine months less the quantity of time that the initial loan was in effect.

To be eligible, processors must:

- Process sugar from domestically grown sugar beets or sugarcane from producers who are in compliance with both highly erodible and wetlands regulations;
- Agree to all terms and conditions in the loan application;
- Execute a note, a security agreement and a storage agreement with CCC.

LOAN RATE

The 2014 Farm Bill requires the Secretary of Agriculture to provide nonrecourse loans to processors of domestically grown:

(1) Sugarcane at a rate equal to 18.75 cents per pound for raw cane sugar for each of the 2011 through 2018 crop years, and
(2) Sugar beets at a rate equal to 24.09 cents per pound for refined beet sugar for each of the 2011 through 2018 crop years.

Loan rates are adjusted to reflect the processing location of the sugar pledged as collateral. (See the tables under National Average Loan Rates by Crop Year).

The in-process sugar loan rate equals 80 percent of the loan rate applicable to raw cane sugar or refined beet sugar, as determined by the Secretary on the basis of the source material for the in-process sugar and syrups. In-process sugars and syrups do not include raw sugar, liquid sugar, invert sugar or syrup, or other finished products otherwise eligible for sugar loans.

- In-process sugar forfeiture: The law authorizes CCC to accept forfeiture of in-process sugar and syrup loan collateral as full loan repayment if the processor converts them within one month after loan maturity into raw cane sugar or refined beet sugar of acceptable grade and quality for sugar eligible for the loans. If forfeited in-process sugars are not converted into raw cane sugar or refined beet sugar of suitable quality and transferred to CCC within one month, CCC may charge liquidated damages.

- In-process sugar crystallization: If the processor does not forfeit the collateral, but instead

further processes the in-process sugar into raw cane sugar or refined beet sugar and repays the loan, the processor may obtain a loan at the higher rate for the raw cane sugar or refined beet sugar. The term of a loan made for an in-process sugar, when combined with the term of a loan made for raw cane sugar or refined beet sugar derived from in-process sugars, may not exceed nine months.

National Average Loan Rates by Crop Year

Crop Year	2013
(Cents Per Pound)	
Raw Cane Sugar	18.75
Refined Beet Sugar	24.09

Regional 2013-crop Loan Rates of Raw Cane Sugar

Crop Year	2013
(Cents Per Pound)	
Florida	18.19
Hawaii	17.95
Louisiana	19.61
Texas	18.36
Sugar processed in Hawaii but placed under loan on the United States mainland	18.75

Regional 2013-crop Loan Rates of Refined Beet Sugar

Crop Year	2013
(Cents Per Pound)	
Mich. & Ohio	25.47
Minn. & E ½ N.D.	23.82
NE ¼ Colo., Neb., SE ¼ Wyo.	24.40
Mont. & NW ¼ Wyo., & W ½ N.D.	24.17
Idaho, Oregon, & Washington	23.58
California	24.89

MINIMUM PRICE SUPPORT FOR SUGARCANE

As a condition to receive CCC sugar loans, sugarcane processors are required to pay their growers at least the minimum payments as specified in the annual CCC loan rate news release and available in their FSA county office.

SUGAR BEET MINIMUM PAYMENT

Sugar beet grower minimum payments are the amount specified in the grower/processor contract.

MARKETING ALLOTMENTS

At the beginning of each fiscal year, CCC will estimate the domestic human consumption of sugar and establish marketing allotments for sugar sold for domestic human consumption that has been processed from sugarcane, sugar beets or in-process beet sugar. The Secretary will strive to establish an overall allotment quantity that results in no forfeitures of sugar to CCC under the sugar loan program and assigns domestic producers at least 85 percent of the market share of domestic human consumption for the crop year. The Secretary shall make estimates of sugar consumption, stocks, production and imports for a crop year as necessary, but not later than the beginning of each of the second through fourth quarters of the crop year.

- Beet Sugar: Beet sugar's allotment is derived by multiplying the overall allotment quantity for the crop year by 54.35 percent. This allotment may only be filled with sugar domestically processed from sugar beets or in-process beet sugar.

- Cane Sugar: Cane sugar's allotment is derived by multiplying the overall allotment by 45.65 percent. Offshore states receive an allocation of 325,000 short tons, raw value, of cane sugar. Remaining cane sugar is allotted to individual mainland cane sugar states. Cane sugar's allotment may only be filled with sugar processed from domestically grown sugarcane.

CCC establishes cane state allotments and sugarcane processor allocations as mandated by regulation. A state cane sugar allotment may be filled only with sugar processed from sugarcane grown in the state covered by the allotment.

If a sugarcane processor is unable to market its allocation, CCC will reassign the estimated quantity of the deficit to other processors within that state. If after reassignment, the deficit cannot be eliminated, CCC will reassign the deficit proportionately to

allotments for other cane sugar states. If this deficit cannot be eliminated, CCC will reassign the deficit to CCC for sale of CCC sugar inventory unless such sales would have a significant effect on the price of sugar. If the deficit still has not been eliminated, CCC will reassign the remainder to raw cane sugar imports. Likewise, if a sugar beet processor is unable to market its allocation, CCC will reassign the deficit to other sugar beet processors, then to CCC, and then to raw cane sugar imports.

During any crop year or portion thereof for which marketing allotments have been established, no sugar beet or sugarcane processor shall market a quantity of sugar for human consumption in excess of the allocation established for such processor, except to enable another processor to fulfill an allocation or for export. Processors knowingly violating their allocation shall be liable to CCC for a civil penalty in an amount equal to three times the U.S. market value at the time of the commission of the violation of that quantity of sugar involved in the violation.

FEEDSTOCK FLEXIBILITY PROGRAM

The 2014 Farm Bill also provides authority for CCC to purchase surplus sugar and sell it to bioenergy producers in order to forestall loan forfeitures.

If forfeitures do occur, the Secretary can dispose of the inventory through sale to bioenergy producers, operate a payment-in-kind program, or purchase certificates of quota eligibility, or any use permitted for CCC sugar before the 2014 Farm Bill was enacted; however, unless there is an emergency shortage of sugar for human consumption, the sugar cannot be sold in a manner that increases the supply of sugar available for human consumption.

Other requirements apply. Check with a local FSA county office or online at www.fsa.usda.gov for details.

APPENDIX EE

Table 3b--World raw sugar price, ICE Contract 11 nearby futures price, monthly, quarterly, and by calendar and fiscal year 1/

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	1st Q.	2nd Q.	3rd Q.	4th Q.	Calendar	Fiscal
									Cents per pound									
1989	9.96	10.67	11.82	12.30	12.02	12.62	13.86	13.80	14.10	13.99	14.78	13.47	10.82	12.31	13.92	14.08	12.78	NA
1990	14.45	14.69	15.46	15.37	14.67	12.92	11.83	10.86	10.89	9.70	9.93	9.70	14.87	14.32	11.19	9.78	12.54	13.61
1991	8.99	8.66	9.15	8.56	7.90	9.44	9.13	8.80	9.11	8.76	8.59	8.87	8.93	8.63	9.02	8.74	8.83	9.09
1992	8.44	8.10	8.33	9.58	9.60	10.50	9.69	9.36	9.01	8.78	8.68	8.30	8.29	9.89	9.35	8.59	9.03	9.07
1993	8.45	8.75	11.03	11.52	12.10	10.44	9.84	9.48	9.48	10.58	10.34	10.65	9.41	11.35	9.60	10.52	10.22	9.74
1994	10.54	11.00	12.03	11.18	11.92	12.09	11.73	11.92	12.48	12.60	13.75	14.75	11.19	11.73	12.04	13.70	12.17	11.37
1995	14.88	14.44	14.27	13.32	11.62	11.93	10.27	11.01	11.02	10.58	10.80	11.42	14.53	12.29	10.77	10.93	12.13	12.82
1996	11.75	12.41	12.01	11.33	10.95	11.76	11.66	11.70	11.61	10.71	10.51	10.61	12.06	11.35	11.66	10.61	11.42	11.50
1997	10.55	10.82	10.87	11.21	11.00	11.29	11.31	11.65	11.27	11.87	12.25	12.28	10.75	11.17	11.41	12.13	11.36	10.98
1998	11.43	10.57	9.72	9.30	8.84	7.98	8.60	8.40	7.16	7.62	8.17	7.96	10.57	8.71	8.05	7.92	8.81	9.87
1999	7.92	6.74	5.76	5.15	4.77	5.57	5.72	6.13	6.86	6.83	6.53	5.95	6.81	5.16	6.23	6.44	6.16	6.53
2000	5.56	5.25	5.28	6.10	7.00	8.46	9.74	10.65	10.06	10.41	9.51	9.72	5.37	7.19	10.15	9.88	8.15	7.28
2001	10.11	9.68	8.75	8.57	8.98	8.89	8.55	7.95	7.36	6.60	7.28	7.41	9.51	8.81	7.95	7.09	8.34	9.04
2002	7.43	6.25	6.06	5.77	5.64	5.40	5.79	5.86	6.73	7.28	7.52	7.56	6.58	5.60	6.13	7.45	6.44	6.35
2003	7.89	8.79	7.86	7.51	7.03	6.53	6.73	6.71	6.02	5.70	5.57	4.67	8.18	7.02	6.49	5.31	6.75	7.29
2004	5.83	5.63	6.50	6.56	6.62	7.05	8.17	7.88	7.91	8.96	8.67	8.79	5.99	6.74	7.98	8.81	7.38	6.51
2005	8.92	8.92	8.90	8.42	8.51	8.92	9.60	9.88	10.44	11.61	11.81	13.93	8.91	8.62	9.97	12.45	9.99	9.08
2006	16.19	17.94	17.08	17.21	16.90	15.27	15.86	12.98	11.41	11.51	11.73	11.70	17.07	16.46	13.42	11.65	14.65	14.85
2007	10.90	10.72	10.34	9.62	9.09	8.86	9.90	9.61	9.52	9.99	9.90	10.45	10.65	9.19	9.68	10.11	9.91	10.29
2008	11.66	13.13	12.88	11.85	10.93	10.80	13.21	13.68	12.29	11.70	11.83	11.32	12.55	11.20	13.06	11.62	12.11	11.73
2009	12.24	13.01	12.93	13.13	15.47	15.54	17.82	21.72	22.25	23.16	22.77	24.90	12.73	14.71	20.60	23.61	17.91	14.91
2010	28.38	26.60	19.26	16.12	14.60	15.81	17.62	19.22	23.72	28.58	28.90	31.09	24.75	15.51	20.19	29.52	22.49	21.01
2011	32.09	31.77	28.15	25.43	21.85	26.07	30.51	28.87	27.71	26.30	24.52	23.42	30.67	24.45	29.03	24.75	27.22	28.42
2012	24.05	25.81	24.73	22.98	20.25	20.44	22.76	20.53	19.47	20.39	19.31	19.50	24.86	21.22	20.92	19.73	21.69	22.94
2013	18.37	18.28	18.33	17.71	17.08	16.79	16.38	16.44	17.33	18.81	17.58	16.41	18.33	17.19	16.72	17.60	17.46	17.99
2014	15.42	16.28	17.58	17.01	17.50	17.22	17.18	15.89	14.60	16.48	15.89	14.99	16.43	17.24	15.89	15.79	16.34	16.79
2015	15.06	14.52	12.84										14.14					

1/ Contract No. 11 nearby

Source: New York Board of Trade (https://www.theice.com/marketdata/reportcenter/reports.htm?reportId=10)

Last updated: 4/1/2015

APPENDIX FF

USDA United States Department of Agriculture

Farm Service Agency
1400 Independence Ave, SW
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www.fsa.usda.gov

News Release

Release 0152.14

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USDA Announces 2014-Crop Sugar Loan Rates and FY 2015 Sugar Program Provisions

WASHINGTON, Sept. 26, 2014 – The U.S. Department of Agriculture's (USDA) Commodity Credit Corporation today announced loan rates for 2014-crop sugar as required by the 2014 Farm Bill. The 2014-crop national average loan rate is 18.75 cents per pound for raw cane sugar and 24.09 cents per pound for refined beet sugar, the same as last year. These national loan rates are adjusted regionally to reflect marketing cost differentials.

The Commodity Credit Corporation also announced sugar program provisions of the fiscal year (FY) 2015 domestic sugar program.

USDA's Sugar Loan Program provides price support loans to processors of sugar beets and domestically grown sugarcane. Price support loans are nonrecourse, meaning producers have the option of delivering the pledged sugar collateral to the Commodity Credit Corporation as full payment for the loan at maturity. USDA's Farm Service Agency is administering sugar nonrecourse loans on behalf of the Commodity Credit Corporation for the 2014 crop. Sugar and in-process sugar loans are available beginning Oct. 1, 2014, and mature at the earlier of (1) the end of the nine month period beginning the first day of the first month after the month in which the loan is made, or (2) the end of the fiscal year in which the loan is made.

Loan Rates for Refined Beet Sugar

The refined beet sugar processing regions and applicable 2014-crop (FY 2015) loan rates in cents per pound of refined beet sugar are:

- Michigan and Ohio – 25.32
- Minnesota and the eastern half of North Dakota – 23.72
- Northeastern quarter of Colorado, Nebraska and the southeastern quarter of Wyoming – 24.36
- Montana, northwestern quarter of Wyoming and the western half of North Dakota – 23.81
- Idaho, Oregon and Washington – 23.98
- California – 25.03

Loan Rates for Raw Cane Sugar

The 2014-crop (FY 2015) raw cane sugar loan rates in cents per pound of cane sugar, raw value are:

- Florida – 18.11
- Hawaii - 17.45 (18.75 cents per pound if stored on the mainland)
- Louisiana - 19.58
- Texas – 18.49

Sugar beet and sugarcane processors receiving Commodity Credit Corporation loans in FY 2015 must make minimum grower payments for all sugar beets and sugarcane received from growers. Processors failing to meet the required minimum grower payment will be ineligible for loans. Sugar beet grower minimum payments are the amount specified in the grower/processor contract.

Sugarcane processors must, at minimum, pay growers for their share of production from molasses and sugar per ton of cane as specified here:

States minimum payments are:

- Florida – $28.51 per net ton
- Hawaii – $29.62 per net ton
- Louisiana – $28.71 per gross ton
- Texas – $25.73 per gross ton

Commodity Credit Corporation has not modified the FY 2015 raw sugar loan schedule of premiums and discounts because the raw cane sugar loan rate has not changed. These schedules can be found in the Farm Service Agency handbook 10-SU, available at http://go.usa.gov/dTZ9, or at local Farm Service Agency state and county offices.

Initial FY 2015 Sugar Marketing Allocations

The Commodity Credit Corporation is announcing the initial FY 2015 overall sugar marketing allotment, which is established at 9,987,500 short tons, raw value. The overall sugar marketing allotment is equal to 85 percent of the estimated human consumption for the crop year of 11,750,000 short tons, raw value as forecast in the September 2014 World Agricultural Supply and Demand Estimates report. Statute requires that a fixed portion of the overall sugar marketing allotment be assigned to the beet sector and the cane sector. The Commodity Credit Corporation distributed the FY 2015 beet sugar allotment of 5,428,206 short tons, raw value (54.35 percent of the overall sugar marketing allotment) among the sugar beet processors and the cane sugar allotment of 4,559,294 short tons, raw value (45.65 percent of the overall sugar marketing allotment) among the sugarcane states and processors.

The Commodity Credit Corporation determined that:

- In 2004, Puerto Rican processors permanently terminated operations because no sugar had been processed for two complete years. The Puerto Rico allocation of 6,356 short tons, raw value is reassigned to Hawaii and then further reassigned to the mainland sugarcane-producing states, because Hawaii is not expected to use all of its cane sugar allotment.
- A Hawaiian cane processor, Gay and Robinson, Inc., permanently terminated operations because it had not processed sugarcane for two consecutive crop years. The Gay and Robinson, Inc. allocation of 73,145 short tons, raw value is reassigned to Hawaii and then further reassigned to the mainland sugarcane-producing states, because Hawaii is not expected to use all of its cane sugar allotment.
- Farm level proportionate shares were not necessary in Louisiana in FY 2015, the only state eligible for proportionate shares, because the cane sugar sector was not expected to fill its allotment.

USDA will closely monitor stocks, consumption, imports and all sugar market and program variables on an ongoing basis. USDA will continue to administer the sugar program as transparently as possible using the latest available data, and make adjustments as necessary to ensure adequate supplies of both raw and refined sugar in the domestic market.

The initial FY 2015 sugar marketing state allotments and processor allocations are as follows:

FY 2015 OVERALL BEET/CANE ALLOTMENTS AND ALLOCATIONS	
Distribution	**Initial FY 2015 Allocations**
Beet Sugar	5,428,206
Cane Sugar	4,559,294

TOTAL OAQ	9,987,500
BEET PROCESSORS' MARKETING ALLOCATIONS:	
Amalgamated Sugar Co.	1,162,220
American Crystal Sugar Co.	1,996,116
Michigan Sugar Co.	560,601
Minn-Dak Farmers Co-op.	376,983
So. Minn Beet Sugar Co-op.	732,635
Western Sugar Co.	554,200
Wyoming Sugar Growers, LLC	45,451
TOTAL BEET SUGAR	5,428,206
STATE CANE SUGAR ALLOTMENTS:	
Florida	2,318,566
Louisiana	1,793,672
Texas	201,557
Hawaii	245,499
TOTAL CANE SUGAR	4,559,294
CANE PROCESSORS' MARKETING ALLOCATIONS:	
Florida	
Florida Crystals	954,615
Growers Co-op. of FL	417,076
U.S. Sugar Corp.	946,876
TOTAL	2,318,566
Louisiana	
Louisiana Sugar Cane Products, Inc.	1,245,224
M.A. Patout & Sons	548,448
TOTAL	1,793,672
Texas	
Rio Grande Valley	201,557
Hawaii	
Hawaiian Commercial & Sugar Company	245,499

For more information, contact your local USDA Service Center or Barbara Fecso at (202) 720-4146, barbara.fecso@usda.gov.

USDA is an equal opportunity provider and employer. To file a complaint of discrimination, write: USDA, Office of the Assistant Secretary for Civil Rights, Office of Adjudication, 1400 Independence Ave., SW, Washington, DC 20250-9410 or call (866) 632-9992 (Toll-free Customer Service), (800) 877-8339 (Local or Federal relay), (866) 377-8642 (Relay voice users).

APPENDIX GG

United States Department of Agriculture
Foreign Agricultural Service

Sugar Import Program

Imports of sugar into the United States are governed by tariff-rate quotas (TRQs), which allow a certain quantity of sugar to enter the country under a low tariff. TRQs apply to imports of raw cane sugar, refined sugar, sugar syrups, specialty sugars and sugar-containing products. Import restrictions are intended to meet U.S. commitments under the North American Free Trade Agreement (NAFTA) and the Uruguay Round Agreement on Agriculture (which resulted in the creation of the World Trade Organization).

USDA establishes the annual quota volumes for each federal fiscal year (beginning October 1) and the U.S. Trade Representative allocates the TRQs among countries. Sugar and related products paying a higher, over-quota tariff may enter the country in unlimited quantities.

More information about U.S. trade in sugar and sweeteners is available from USDA's Economic Research Service <http://www.ers.usda.gov/topics/crops/sugar-sweeteners/trade.aspx>.

Regulations

15 CFR Part 2011 <http://www.ecfr.gov/cgi-bin/text-idx?sid=62ee7a6324325d70f6ebe1b86af57331&node=15:3.2.1.7.10&rgn=div5>

USDA also administers three re-export programs involving sugar.

- **The Refined Sugar Re-Export Program** is designed to facilitate use of domestic refining capacity to export refined sugar into the world market. The program establishes a license against which a refiner can: export domestically produced refined sugar and later import low-duty raw cane sugar; import low-duty raw cane sugar for refining and distribution to licensed U.S. manufacturers of sugar-containing products and/or licensed producers of polyhydric alcohol for non-food purposes; or import raw sugar, refine it and export it into the world market.
- **The Sugar-Containing Products Re-Export Program** is designed to put U.S. manufacturers of sugar-containing products on a level playing field in the world market. U.S. participants in the Sugar-Containing Products Re-Export Program may buy world-priced sugar from any licensed refiners for use in products to be exported to the world market.
- **The Sugar for the Production of Polyhydric Alcohol Program** is established to provide world-priced sugar to licensed U.S. manufacturers of polyhydric alcohols. Participating U.S. manufacturers purchase world-priced sugar from licensed refiners for use in the production of polyhydric alcohols, except polyhydric alcohols used as a substitute for sugar in human food consumption.

Regulations

7 CFR Part 1530 - The Refined Sugar Re-Export Program, the Sugar Containing Products Re-Export Program, and the Polyhydric Alcohol Program <http://www.ecfr.gov/cgi-bin/retrieveecfr?gp=1&sid=d18597eebea5031c0925ee4b22e26438&ty=html&h=l&n=7y10.1.3.4.2&r=part>

Applying

Specialty Sugar Certificate Application

- Global Specialty Sugar Certificate <http://www.fas.usda.gov/programs/sugar-import-program/applying-specialty-sugar-certificate>
- Panama Specialty Sugar Certificate <http://www.fas.usda.gov/programs/sugar-import-program/applying-panama-specialty-sugar-certificate>

U.S. Sugar Re-Export Administration, Application and Reporting

Re-Export Program Online Reporting System <http://www.fas.usda.gov/sugars/fassugarshome.aspx>

APPENDIX HH

Table 4--U.S. raw sugar price, duty fee paid, New York, monthly, quarterly, and by calendar and fiscal year 1/

Year	Jan.	Feb.	Mar.	Apr.	May	June	July	Aug.	Sep.	Oct.	Nov.	Dec.	1st Q.	2nd Q.	3rd Q.	4th Q.	Calendar	Fiscal
									Cents per pound									
1960	5.89	6.00	6.11	6.17	6.09	6.25	6.48	6.47	6.59	6.52	6.53	6.46	6.00	6.17	6.51	6.50	6.30	NA
1961	6.39	6.32	6.25	6.25	6.46	6.48	6.39	6.06	6.06	6.19	6.29	6.40	6.32	6.40	6.17	6.29	6.30	6.35
1962	6.45	6.37	6.43	6.43	6.43	6.45	6.39	6.54	6.43	6.52	6.44	6.54	6.42	6.44	6.45	6.50	6.45	6.40
1963	6.70	6.80	7.04	8.26	11.08	8.70	7.95	6.65	7.45	9.42	9.34	8.78	6.85	9.35	7.35	9.18	8.18	7.51
1964	9.29	8.02	7.33	7.43	6.65	6.45	6.25	6.18	6.20	6.27	6.17	6.55	8.21	6.84	6.21	6.33	6.90	7.61
1965	6.85	6.79	6.61	6.59	6.73	6.72	6.73	6.77	6.82	6.82	6.80	6.75	6.75	6.68	6.77	6.79	6.75	6.63
1966	6.88	6.92	6.84	6.89	6.90	6.92	7.00	7.05	7.11	7.15	7.12	7.14	6.88	6.90	7.05	7.14	6.99	6.91
1967	7.13	7.21	7.18	7.22	7.25	7.32	7.30	7.33	7.34	7.37	7.38	7.30	7.17	7.26	7.32	7.35	7.28	7.22
1968	7.41	7.38	7.35	7.42	7.48	7.53	7.59	7.59	7.62	7.66	7.58	7.62	7.38	7.48	7.60	7.62	7.52	7.45
1969	7.67	7.69	7.76	7.80	7.82	7.74	7.50	7.75	7.83	7.89	7.79	7.73	7.71	7.79	7.69	7.80	7.75	7.70
1970	8.11	7.96	7.90	7.90	8.16	8.22	8.16	8.19	8.16	8.14	7.96	8.02	7.99	8.09	8.17	8.04	8.07	8.01
1971	8.35	8.44	8.37	8.29	8.46	8.54	8.58	8.66	8.57	8.52	8.63	8.84	8.39	8.43	8.60	8.66	8.52	8.37
1972	9.10	9.02	9.16	8.89	8.76	8.77	9.17	9.33	9.39	9.32	9.03	9.19	9.09	8.81	9.30	9.18	9.09	8.97
1973	9.38	9.14	9.45	9.65	10.06	10.25	10.25	10.75	10.97	11.15	11.10	11.34	9.32	9.99	10.66	11.20	10.29	9.79
1974	12.63	17.09	18.11	19.25	23.05	26.30	28.35	32.60	33.71	38.83	57.30	46.74	15.94	22.87	31.55	47.62	29.50	20.39
1975	40.15	36.07	28.52	26.07	19.27	15.96	19.89	21.11	17.36	15.45	15.03	14.80	34.91	20.43	19.45	15.09	22.47	30.61
1976	15.42	15.04	16.27	15.58	15.97	14.40	14.59	11.32	9.80	10.65	10.46	10.22	15.58	15.32	11.90	10.44	13.31	14.47
1977	10.95	11.06	11.67	12.57	11.34	10.28	10.15	11.21	10.41	10.23	10.42	11.75	11.23	11.40	10.59	10.80	11.00	10.91
1978	13.28	14.75	14.00	13.93	13.62	13.57	12.63	13.29	14.41	15.17	14.24	14.25	14.01	13.71	13.44	14.55	13.93	12.99
1979	14.63	15.31	15.53	14.29	14.33	14.61	15.59	15.92	15.98	15.91	16.29	18.30	15.16	14.41	15.83	16.83	15.56	14.99
1980	19.66	24.69	21.18	22.67	31.89	32.10	28.75	33.13	36.03	41.69	39.28	30.29	21.84	28.89	32.64	37.09	30.11	25.05
1981	29.61	26.07	23.81	19.91	17.43	18.95	19.09	17.42	15.49	15.66	16.28	17.07	26.50	18.76	17.33	16.34	19.73	24.92
1982	18.16	17.77	17.13	17.89	19.57	21.03	22.15	22.45	20.88	20.44	20.79	20.83	17.69	19.50	21.83	20.69	19.92	18.84
1983	21.23	21.76	21.86	22.43	22.59	22.54	22.09	22.55	22.20	21.94	21.83	21.47	21.62	22.52	22.28	21.75	22.04	21.78
1984	21.51	21.90	22.00	22.03	22.01	22.06	21.89	21.72	21.70	21.56	21.40	21.10	21.80	22.03	21.77	21.35	21.74	21.84
1985	20.72	20.38	20.91	20.97	21.09	21.27	21.23	20.59	19.51	18.68	18.89	19.89	20.67	21.11	20.44	19.15	20.34	20.89
1986	20.67	21.01	20.95	20.85	20.88	20.99	20.97	20.87	20.87	21.08	21.17	21.12	20.88	20.91	20.90	21.12	20.95	20.46
1987	21.50	21.76	21.76	21.81	22.01	22.06	22.07	21.88	21.88	21.69	21.75	21.76	21.67	21.96	21.94	21.73	21.83	21.68
1988	21.83	22.11	22.16	22.16	22.13	22.54	23.43	21.90	21.77	21.74	21.70	21.99	22.03	22.28	22.37	21.81	22.12	22.10
1989	21.88	22.07	22.12	22.30	22.45	22.99	23.56	23.57	23.50	23.14	23.24	22.84	22.02	22.58	23.54	23.07	22.81	22.49
1990	23.11	22.93	23.58	23.81	23.58	23.33	23.42	23.27	23.23	23.29	23.15	22.47	23.21	23.57	23.31	22.97	23.26	23.29
1991	21.86	21.42	21.46	21.23	21.29	21.42	21.25	21.83	22.06	21.76	21.75	21.50	21.58	21.31	21.71	21.67	21.57	21.89
1992	21.38	21.56	21.36	21.38	21.04	20.92	21.10	21.34	21.55	21.61	21.39	21.11	21.43	21.11	21.33	21.37	21.31	21.39
1993	20.76	21.16	21.56	21.76	21.36	21.42	21.89	21.85	21.97	21.80	21.87	22.00	21.16	21.51	21.90	21.89	21.62	21.49
1994	22.00	21.95	21.95	22.08	22.18	22.44	22.72	21.84	21.78	21.58	21.57	22.35	21.97	22.23	22.11	21.83	22.04	22.05
1995	22.65	22.69	22.46	22.76	23.10	23.09	24.47	23.18	23.21	22.67	22.60	22.63	22.60	22.98	23.62	22.63	22.96	22.76
1996	22.39	22.68	22.57	22.71	22.62	22.48	21.80	22.51	22.38	22.37	22.12	22.14	22.55	22.60	22.23	22.21	22.40	22.50
1997	21.88	22.07	21.81	21.79	21.70	21.62	22.04	22.21	22.30	22.27	21.90	21.93	21.92	21.70	22.18	22.03	21.96	22.00
1998	21.85	21.79	21.74	22.14	22.31	22.42	22.66	22.19	21.92	21.67	21.83	22.19	21.79	22.29	22.26	21.90	22.06	22.09
1999	22.41	22.38	22.55	22.57	22.65	22.61	22.61	21.24	20.10	19.50	17.45	17.87	22.45	22.61	21.32	18.27	21.16	22.07
2000	17.70	17.24	18.46	19.43	19.12	19.31	17.64	18.12	18.97	21.15	21.39	20.56	17.80	19.29	18.24	21.03	19.09	18.40
2001	20.81	21.18	21.40	21.51	21.19	21.04	20.64	21.10	20.87	20.90	21.19	21.43	21.13	21.25	20.87	21.17	21.11	21.07
2002	21.03	20.69	19.92	19.73	19.52	19.93	20.86	20.91	21.65	21.94	22.22	22.03	20.55	19.73	21.14	22.06	20.87	20.65
2003	21.62	21.91	22.14	21.87	21.80	21.62	21.32	21.26	21.34	20.92	20.91	20.37	21.89	21.76	21.31	20.73	21.42	21.76
2004	20.54	20.57	20.86	20.88	20.69	20.03	20.14	20.10	20.47	20.31	20.40	20.55	20.66	20.53	20.24	20.42	20.46	20.54
2005	20.57	20.36	20.54	21.21	21.96	21.89	21.94	20.49	21.10	21.71	21.83	21.74	20.49	21.69	21.18	21.76	21.28	20.94
2006	23.61	24.05	23.10	23.56	23.48	23.32	22.44	21.38	21.27	20.22	19.66	19.59	23.59	23.45	21.70	19.82	22.14	22.62
2007	20.03	20.59	20.85	20.91	21.27	21.33	22.72	21.80	21.42	20.56	20.25	20.12	20.49	21.17	21.98	20.31	20.99	20.87
2008	20.24	20.21	20.65	20.54	20.83	21.80	23.76	23.15	23.10	21.46	19.83	20.00	20.37	21.06	23.34	20.43	21.30	21.27
2009	20.15	19.83	19.75	21.58	21.64	22.47	23.02	26.18	28.91	30.48	31.86	33.30	19.91	21.90	26.04	31.88	24.93	22.07
2010	39.36	40.13	35.11	30.86	30.89	32.73	33.66	34.24	38.17	39.30	38.84	38.35	38.20	31.49	35.36	38.83	35.97	34.23
2011	38.46	39.69	39.65	38.32	35.04	35.65	37.93	40.16	40.15	38.19	37.92	36.32	39.27	36.34	39.41	37.48	38.12	38.46
2012	34.69	33.57	34.94	31.87	30.20	28.89	28.68	28.84	26.27	23.89	22.52	22.41	34.40	30.32	27.93	22.94	28.90	32.53
2013	21.20	20.72	20.82	20.38	19.51	19.31	19.22	20.97	21.05	21.82	20.61	19.95	20.91	19.73	20.41	20.79	20.46	21.00
2014	20.27	21.65	22.03	24.33	24.66	25.65	24.78	25.64	25.36	26.41	24.26	24.81	21.32	24.88	25.26	25.16	24.15	23.06
2015	25.24	24.62	24.07										24.64					

1/ Contract No. 14/16, duty fee paid New York. Average of nearest futures month for which an entire month of prices will be available. For example, April 2001's price average of 21.51 cents is the average of closes for the July 2001 futures during the month of April since there was not a full month of May 2001 futures in

April (the May 2001 futures expired April 10th, July 2001 became the nearest futures, so July 2001 was used for the entire month of April).

Source: Intercontinental Exchange.

Last Updated: 4/1/2015

Florida Crop/Pest Profile: Sugarcane[1]

Mark Mossler[2]

Sugarcane Production Facts

- In 2006, Florida ranked first nationally in value of sugar produced from sugarcane -- approximately $425 million. That amount was half of the total U.S. value of sugar from sugarcane that year. In general, the total amount of sugar produced from sugarcane in Florida is more than 20 percent of total sugar (from sugar beet and sugarcane) produced in the United States annually (1,2).
- Florida, Louisiana, Texas and Hawaii are the only areas in the United States where sugarcane is grown commercially. Approximately 400,000 acres of sugarcane are harvested annually in Florida, producing approximately 1.5 million tons of sugar annually (1,2).
- Sugarcane is Florida's most valuable field crop, worth more economically than the combined value of all other field crops grown in Florida, including corn, soybean, tobacco, and peanuts. In terms of Florida's overall agricultural economy, sugarcane ranks third, behind the greenhouse/nursery industry, which is first in Florida, and the citrus industry, which is second in Florida.
- All Florida sugarcane is crushed at one of four mills in South Florida. Sugarcane milling companies grow about two-thirds of the cane; the remainder is produced by independent farmers. Sugar mills produce raw sugar, which is refined at two refineries in the region or shipped by barge to refineries on the East Coast. Some sugar is marketed in its raw state (2).

Figure 1. A stand of sugarcane in Immokalee, FL, 2006.
Credits: Josh Wickham, UF/IFAS

Regions of Sugarcane Production in Florida

Sugarcane, a tropical grass, is adapted to all portions of Florida. However, the commercial sugarcane industry is located in South Florida around Lake Okeechobee, especially in the Everglades Agricultural Area. The vast majority of sugarcane production in Florida (70 percent of the

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acreage and 75 percent of the tonnage) is produced in Palm Beach County. The remainder of commercial sugarcane production in Florida occurs in the adjacent counties of Hendry, Glades, and Martin (2). While most sugarcane is grown on muck organic soils -- which predominate near Lake Okeechobee -- approximately 20 percent of Florida's commercial sugarcane production is on sandy mineral soils (3).

Sugarcane Production Practices

Sugarcane is a multi-year crop and can be grown perennially in Florida. Sugarcane is propagated vegetatively. Following harvest of the first "plant cane" crop, the regrowth ("ratoon cane") is harvested about once each year until plant populations decline. Typically, an average of three annual crops will be harvested from a sugarcane field before the field is plowed under and replanted.

The majority of sugarcane is planted from September through December. Portions of a mature sugarcane field are reserved to grow "seed cane" -- whole, mature stalks cut directly from the field, either by machine or by laborers using machetes.

Stalks of seed cane are dropped in furrows, cut by hand into smaller pieces, and covered with soil 3 - 8 inches deep**.** If a sugarcane field is harvested early enough in the growing season (before January), the field will likely be replanted to sugarcane (termed successive planting). If the field is harvested at a later date, replanting may be delayed until the following season. During this fallow period, the field is often planted to another crop, such as rice or sweet corn (termed regular or fallow planting).

In 2005-06, 30 percent of the sugarcane crop in Florida was plant cane, and 70 percent was ratoon cane (2,4). During that same growing season, approximately one-third of the sugarcane was planted successively while the remainder was planted following a fallow period or rotational crop (2,4).

Sugarcane is harvested from late October through mid-April. Sugar yields are typically higher as the weather turns cooler. In order to complete the entire South Florida sugarcane harvest within the optimal time frame, some fields must be harvested before sugarcane plants have reached maximum yield potential. Consequently, research has examined which cultivars should be harvested in early-, mid- or late-season (5). Glyphosate is also used as a ripening agent for early harvest (6).

The fields are burned to remove field trash, and then mechanically harvested cane is loaded onto trucks or rail cars to be transported to the mill.

Worker activities. The only part of sugarcane cultivation that requires field workers is planting, which is 85 percent conducted manually. Approximately 120,000 acres are planted each year, and a single worker may plant up to five acres per day (4).

Insect/Mite Management in Florida Sugarcane Crops

Most sugarcane is resistant to insect pests that are common in semi-tropical environments, but some pests still infest sugarcane. These pests include the following: sugarcane borer, white grubs, wireworms, yellow sugarcane aphid, and lesser cornstalk borer. Weather and cultural practices play a role in the level of pest pressure present from one season to the next (7).

Insect and Mite Pests in Florida Sugarcane Crops

SUGARCANE BORER

The sugarcane borer, *Diatraea saccharalis*, is the larvae of a moth. Sugarcane borer is an important above-ground pest of sugarcane in Florida. All varieties of sugarcane currently grown in Florida are susceptible to sugarcane-borer infestation, but different sugarcane varieties exhibit variation in damage and yield losses. Although this moth's principal host is sugarcane, many other grasses have been reported as alternative hosts.

Damage results from the sugarcane borer larvae tunneling into the stalk, causing loss of stalk weight (tonnage/acre) and sucrose yield. Weakened stalks are also subject to breaking and lodging. Additionally, the borer's tunneling into the stalk allows points of entry for secondary invaders. Sugarcane borer infestation early in the sugarcane plant life and boring near the shoot meristem can also kill the shoot, resulting in "dead heart" (7).

Regular scouting is the foundation of an Integrated Pest Management Program (IPM) for sugarcane-borer control. Scouting is necessary to estimate the pest-infestation level and beneficial borer parasites. A regular scouting program will also increase the chances of detecting other pests that may be damaging the crop (7). Sugarcane fields should be scouted every two or three weeks from March through November. One Florida sugarcane company scouts each 40-acre field in at least four locations within the field. At

each location, five stalks are randomly sampled from each of five stools spaced 10 feet apart (five stalks/five stools/location).

Borers must be detected before they tunnel extensively into stalks so that, if necessary, control measures can be applied before any damage to stalks occurs. Characteristic signs that plants are infested are pinholes in leaves, tiny holes into midribs, holes into stalks, and frass (light-brown, fibrous waste material) at these holes. An infestation of borers cannot be positively identified until the sugarcane borers are actually observed. Scouts should examine leaves, the whorl, and behind leaf-sheaths, as well as splitting open stalks to detect borers tunneling inside stalks. Detecting two to three live larvae per 100 sampled stalks is generally thought to be enough to cause concern about economic damage (7).

One study of five commercial varieties showed that an average of one bored internode per stalk reduced sugar yield by an average of 5.6 pounds per ton of sugarcane. The range of loss was from 2.3 pounds per ton of sugarcane to 6.7 pounds per ton of sugarcane. Another investigation showed bored internodes produce 45 percent less sugar than undamaged internodes. Interestingly, certain localized regions of the Everglades Agricultural Area seem to be considerably more prone to borer infestations. Environmental explanations are presumed, but definite reasons are not clear (8).

WHITE GRUBS

Also found in Florida sugarcane fields are white grubs, the larvae of several beetle species of the genera *Ligyrus*, *Cyclocephala*, *Phyllophaga*, and *Anomala*. Of these grub pests, the species *Ligyrus subtropicus* is the most abundant, causes by far the most damage to sugarcane, and, as a result, imposes the greatest economic loss for sugarcane growers. These pests tend to infest sugarcane in muck soils, damaging sugarcane by feeding on roots and underground stems. The first symptom of infestation is a yellowing (chlorosis) of the leaves, a condition usually followed by stunted growth, dense browning, lodging, plant uprooting, and, in heavily infested areas, death of the plant. Symptoms may appear as early as September.

Fields of plant cane usually have little or no grub infestation (7,9). Instead, damage from an *L. subtropicus* infestation is usually more severe in ratoon crops and most evident around the edges of a field, the area where the infestation usually begins. From there, the infestation slowly spreads in an irregular pattern throughout the field. Sugarcane fields infested with *L. subtropicus* may need to be replanted because ratoon regrowth and productivity can be severely reduced by this pest. Heavily infested areas may not be worth harvesting.

Growers use cultural-control practices for sugarcane grubs. Discing infested fields kills many grubs and allows birds to kill many more. Additionally, planting rotational crops for a weed-managed fallow period and short-term flooding of the standing crop are common methods of grub control in Florida.

WIREWORMS

Wireworms, the larval stage of click beetles, may cause severe damage to numerous crops in Florida, including sugarcane. At least 12 species of wireworms have been found in South Florida, but only the corn wireworm, *Melanotus communis*, is considered to cause significant economic damage to sugarcane. Traditionally, *M. communis* has been a more important pest of sugarcane grown on muck soil than sugarcane grown on sandy soil (7,10).

Generally, wireworms are a pest of newly planted sugarcane and only rarely a pest in ratoon sugarcane. Wireworms feed on the buds and root primordia during germination of sugarcane seed pieces. After germination, wireworms feed on shoots and roots. Most of the injury to young shoots is near the point where the shoots join the seed piece or ratoon stubble.

Wireworm injury can be identified as relatively large, ragged holes cut into buds or young shoots. The death of buds and young shoots leads to stand reductions. Wireworm injury to sugarcane also facilitates the entrance of the fungus that causes sugarcane red rot disease (7,10).

YELLOW SUGARCANE APHID

Yellow sugarcane aphid (YSA), *Sipha flava*, is a small, yellow aphid with short legs, antennae and mouth parts. The aphid's body is covered in short, stiff hairs. Aphids are born live, not from eggs, and take 2 - 3 weeks to develop to reproductive adult stage, at which point the aphid can produce 3 - 5 nymphs per day for another 2 - 3 weeks. Winged forms of the aphid are produced under crowded conditions when the sugarcane plant is beginning to suffer significant damage.

Natural enemies of YSA include 10 species of ladybird beetles and several species of flower flies. These beetles and flies can greatly reduce YSA populations. Additionally, heavy summer rains usually reduce YSA populations to a low level. However, reduction in the size of a YSA

infestation by such means may not occur before the aphids have caused plant damage (7).

Yellow sugarcane aphids cause rust-like reddening and death of sugarcane leaves. Reduced growth and reduced tillering result when these aphids feed on very young plants. Longer, faster-growing leaves and internodes are some of the results of YSA feeding, as well as thinner, lighter stalks with shorter node lengths and narrow widths. Prolonged feeding by large populations of YSA can cause plant death.

After YSA has been brought under control, new sugarcane growth emanating from previously YSA-infested sugarcane will have node lengths and leaf dimensions similar to sugarcane plants that have not been infested with YSA (7). However, node diameters on this new growth will likely be smaller than if the crop had not been infested with YSA. As a result, thinner stalks can be expected from previously YSA-infested plants. These lighter-weight stalks will contain less accumulated sugar; thus, a decline in overall sugar yield can be expected from a crop even after YSA has been brought under control (7).

Aphid populations can build quickly to numbers too numerous to count for sampling purposes. Leaf damage is a good indicator of season-long effects on growth and yield. Leaves with less than 50 percent green tissue can be quickly counted and averaged over an area to compare long-term effects of YSA feeding with the relative size of the infestation. Resistance appears to be a viable control strategy since YSA shows a preference for certain cultivars (7).

LESSER CORNSTALK BORER

The larva of the lesser cornstalk borer moth, *Elasmopalpus lignosellus*, is a slender, brown worm with creamy-white to bluish-green bands, 1/2-inch to ¾-inch long when full grown. The lesser cornstalk borer larvae bore into young plants at or below the soil surface and usually cause a "dead-heart" similar to damage caused to sugarcane by the sugarcane borer or wireworms. Damage above the growing point appears as rows of holes in emerging leaves, evidence of lesser cornstalk borer feeding on developing leaf whorls at or below the growing point.

Larvae of the lesser cornstalk borer construct a silken tube in the soil; the tube extends outward from the cane plant. The larva pupates in this burrow and transforms into a small moth. The presence of these silk tubes and/or a small, circular entrance hole distinguishes dead-hearts caused by lesser cornstalk borers from those caused by wireworms.

The life cycle of the lesser cornstalk borer ranges from 38 - 65 days. Generations overlap considerably because of the mild South Florida winter, with no sharp seasonal breaks in populations. Most damage resulting from this pest occurs to young sugarcane plants during cool, dry periods. Ratoon cane usually recovers better from lesser cornstalk borer attack than does young plant cane. The protection this pest gets from its silk tube makes parasites of the lesser cornstalk borer inefficient as biological control agents and also makes chemical control measures difficult (11).

Cultural Management of Insects and Mites in Florida Sugarcane Crops

To manage sugarcane borer economically, use sugarcane varieties that exhibit resistance to infestation and damage. Varieties highly susceptible to the sugarcane borer are eliminated during the breeding program that develops new varieties for commercial release. Other important cultural-control tactics include destroying cane trash and stubble in infested fields and using seed pieces free of borer damage (8).

For white grubs, discing infested fields, crop rotation, and short-term flooding in the standing crop are effective methods of control. The best time to kill grubs by flooding is in August, when water temperatures are warm, rainfall is abundant, and feeding damage by the grubs is just starting (9).

For wireworms, flooding can be effective with a minimum of six weeks of continuous flooding during the summer. Longer flooding durations are needed during colder months. Flooding during late spring and summer will kill the wireworms and prevent egg-laying by the adult click beetles. Flooding fallow fields or growing rice as a rotation crop reduces the need to use a soil insecticide at sugarcane planting the following fall (10,12).

For yellow sugarcane aphids, several methods can assist sugarcane resistance to damage. These methods are variety-dependent and include tolerance and antibiotic effects on aphid development. Winged YSAs usually restrict their primary colonization to susceptible cultivars, including the following: 'CP 61-620', 'CP 72-1210', 'CP 72-2086', 'CP 80-1827' and 'CP 89-2143'.

Additionally, red imported fire ants, predatory earwigs and many species of ladybird beetles exert some control over these aphids. Heavy rains are also helpful to dislodge aphids, washing the pests from the sugarcane plants (11).

Biological Management of Insects and Mites in Florida Sugarcane Crops

Alabagrus stigmatera and *Cotesia flavipes* are important wasp parasitoids of the sugarcane borer larvae in Florida. *A. stigmatera* is a large, solitary (one-per-host) parasite and active all year. *C. flavipes* is a small, gregarious (many-per-host) parasite and usually most active after July. *C. flavipes* is the most important parasitoid. *C. flavipes*, an endoparasite (parasite that grows within the host), injects eggs directly into the borer larvae.

Whenever sugarcane borer infestation approaches the economic-injury threshold, sugarcane borer larvae from the infested field should be dissected to determine the level of parasitism. If 50 percent or more of the sugarcane borer's larvae are parasitized, insecticides are not recommended. Insecticide applications may harm the parasite population without gaining additional control of the sugarcane borer.

Cotesia flavites is an introduced parasitic wasp that has become established in Florida. *Cotesia* and the red imported fire ant are effective biological control agents for the sugarcane borer. Augmentative releases of *Cotesia* parasitoids have been highly effective for managing the sugarcane borer in sugarcane grown in Florida, Brazil, Costa Rica (8) and Jamaica.

Chemical Management of Insects and Mites in Florida Sugarcane Crops

Soil insecticides are used on about half of new plantings of sugarcane in Florida in any given year. Ethoprop and phorate are equally used at about 1 lb ai/A to control wireworms when infestation levels are high. Approximately 2,000 acres of sugarcane in Florida are treated yearly with carbofuran at 0.5 lb ai/A to control lesser cornstalk borer when damage levels are high. However, in 2008 the U.S. Environmental Protection Agency (EPA) announced plans to cancel registration for carbofuran. Other carbo insecticides registered for sugarcane in 2008 in Florida were azadirachtin, B.t., carbaryl, cyfluthrin, cyhalothrin, esfenvalerate, and pyrethrins +/- rotenone, spinosad, and tebufenozide. Methoprene is available for control of fire ants.

Disease Management in Florida Sugarcane Crops

Although there are many sugarcane diseases throughout the world, few such diseases have affected Florida sugarcane historically. For example, until 2008, no fungicides were used on Florida sugarcane to control rusts. Plant breeding programs designed to develop rust-resistant varieties have historically kept brown rust impacts under economic thresholds for any given growing season. However, orange rust was discovered in Florida in 2007. This disease may require growers of susceptible cultivars -- such as 'CP 80-1743' and 'CP 72-2086' -- to use fungicides to maintain acceptable yields.

Disease Pests in Florida Sugarcane Crops

BROWN RUST

Sugarcane production in Florida has been affected by brown rust (*Puccinia melanocephela*) since 1978. This fungal pathogen is now found almost everywhere sugarcane is grown throughout the world. The spread of brown rust has had considerable economic impact. As a result, the practice of screening new cultivars for resistance has become an integral part of Florida sugarcane breeding programs. However, due to genetic variability within the pathogen population, resistance to brown rust has not been stable. For example, 'CP70-1133', an important resistant variety grown for years without symptoms is now classified as moderately susceptible. Other important commercial clones have demonstrated increasing susceptibility to sugarcane rust over time (13).

Assessment of yield loss due to brown rust is difficult, but realistic estimates have been obtained. During 1988, rust was particularly severe on 'CP 78-1247' in Florida. A comparison of yields from 'CP 78-1247' that year with a variety of equal yield potential revealed that yield losses due to brown rust were nearly 40 percent (averaged over 13 different locations in Florida where the varieties were grown side-by-side).

Another study of sugarcane-yield loss -- conducted by establishing a nearly disease-free check using a fungicide as a means for comparison -- demonstrated yield losses of 20-25 percent on a moderately susceptible sugarcane variety, 'CP 72-1210'. This variety dominated the Florida sugarcane industry during the late 1980s, occupying more than 60 percent of Florida's sugarcane area. Economic losses on 'CP 72-1210' in 1987 - 88 were estimated as surpassing $40 million using a conservative estimate of 20 percent yield loss (13).

Brown rust is a leaf disease. The earliest symptoms are small, elongated, yellowish spots, visible on both the top and bottom leaf surfaces. The spots increase in size, turn brown to orange-brown or red-brown in color, and develop a slight, but definite chlorotic halo.

Lesions resulting from brown rust are seldom more than 1-3 mm in width. The lesions typically range from 2-10 mm in length, but occasionally reach 30 mm in length (14). The symptoms of brown-rust infection are usually most numerous toward the leaf tip, becoming less numerous toward the base. Pustules, which produce spores, develop on the lower leaf surface although certain sugarcane cultivars may develop pustules on the upper leaf surface. Pustules may remain active over a considerable period of time during cool seasons of the year with long dew periods.

Environmental factors most influential for rust development are leaf wetness and cool-to-moderate atmospheric temperatures. Several hours of free moisture on the leaf surface at a favorable temperature is necessary for successful spore germination, infection, and spread of the disease. While long dew periods and rainfall events both contribute to leaf wetness, rainfall events may not be as favorable for rust development. Heavy rains tend to remove spores from the atmosphere, and the spores are ineffective if they land on the soil, rather than on leaves. Increased soil moisture also favors rust infection by increasing humidity within the canopy and, thereby, lengthening the duration of leaf wetness.

In Florida, brown rust is most severe from February to May, when atmospheric temperatures are cool-to-moderate. Newly planted sugarcane plants, from 3 - 6 months in age, are more susceptible to brown rust than is ratoon cane (13). It appears, further, that plant cane is more susceptible to brown rust than is ratoon cane.

Eventually, lesions on sugarcane leaves affected with brown rust will darken, and the surrounding leaf tissues will become necrotic. On highly susceptible varieties, large numbers of pustules may occur on a leaf, coalescing to form large, irregular necrotic areas. High rust severity may result in premature death of even young leaves. Severe rust infections can cause reductions in both stalk mass and stalk numbers, thereby reducing cane yield (13).

ORANGE RUST

Previously, orange rust was known as a minor disease of sugarcane in Asia and Australia. The causal organism of orange rust (*Puccinia kuehnii*) was found in Florida sugarcane in July 2007 and was also detected in sugarcane fields in Costa Rica, Guatemala and Nicaragua (15).

Orange rust caused an estimated 10 percent loss on susceptible sugarcane varieties in Florida in the late summer of 2007. In the late spring of 2008, more orange rust was observed on susceptible Florida sugarcane varieties, and the disease appeared earlier in the season (16).

Plant cane and ratoon crops appear to be equally susceptible to orange rust. Mature cane is as susceptibe as young sugarcane plants, and the disease persists through hot summer months. The biology of orange rust is similar to that of brown rust. However, varietal resistance to brown rust does not simultaneously confer resistance to orange rust. For example, the variety CP80-1743 has fairly high resistance to brown rust, but not to orange rust.

One third of the overall area planted in sugarcane in Florida is planted with varieties that are susceptible to orange rust, including 'CP80-1743' and 'CP72-2086' (16). Researchers are currently determining whether orange rust is affecting 'CP89-2143' in two locations in Florida. This variety comprises about 20 percent of the acreage planted in sugarcane in Florida (16).

Non-Chemical Management of Disease Pests in Florida Sugarcane Crops

Planting resistant varieties of sugarcane is the best means of controlling rusts on sugarcane. However, resistance has not been stable or durable on certain varieties because of the development of rust variants. It is highly recommended that growers diversify their varietal holdings for this reason. Such diversification is an effective means of limiting risk of yield loss and reducing the likelihood of a new race of rust evolving.

Varietal diversification may also play an important role in holding down overall area-wide disease pressure, thereby reducing the natural-selection pressure for one particular rust variant. A general rule of epidemiology suggests that varietal diversification may assist in preserving the durability of host-plant resistance in currently resistant varieties (13).

Soil factors identified with high levels of rust infection on sugarcane include low soil pH and/or high fertility, so avoid growing susceptible varieties in areas with these soil conditions. Sugarcane grown in fields that have received recent applications of nutrient amendments is typically prone to rust. If possible, plant such fields with varieties that have durable rust resistance(13).

Chemical Management of Disease Pests in Florida Sugarcane Crops

Few effective disease-management tools are available for sugarcane. Propiconazole is available as a dip treatment for

plant-cane pieces, and phosphorous-based fungicides are registered for foliar application. However, neither of these treatments is greatly effective for a leaf disease, such as rust, that attacks sugarcane 3 - 6 months after planting.

Because of the limited treatments available to control disease in sugarcane, the Florida Department of Agriculture and Consumer Services (FDACS) has declared a crisis exemption under Section 18 of the Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) for the use of two fungicides (pyraclostrobin and metconazole) for control of orange rust. It is envisioned that the EPA will issue a three-year quarantine exemption for this use (16).

As of mid-July 2008, approximately 10,000 acres of sugarcane in Florida had been sprayed with pyraclostrobin. The applications cost approximately $30/acre ($25/acre for the fungicide and $5/acre application cost independent of whether it is applied by ground or air).

Weed Management in Florida Sugarcane Fields

Weed control is most critical early in the season, prior to sugarcane-canopy closure over the row middles. Heavy weed infestations can add unnecessary expense to harvesting sugarcane. Additionally, a weed that is allowed to mature and produce seed will multiply weed-control problems for years to come. The most important weed pests in Florida sugarcane production are fall panicum, napiergrass, yellow and purple nutsedge, and pigweeds (18-20).

Figure 2. Mechanical cultivation for weed control in sugarcane crops near Belle Glade, FL, 2004.
Credits: Thomas Wright, UF/IFAS

Weed Pests in Florida Sugarcane Fields

NUTSEDGE

Yellow nutsedge (*Cyperus esculentus*) and purple nutsedge (*C. rotundus*) are important weed pests of most crops in Florida. Both of these perennial sedges are found in disturbed habitats throughout Florida and the southeastern United States. Yellow nutsedge may produce some seed, but reproduces primarily by rhizomes and tubers. The first plant develops rhizomes, which end in tubers that produce new plants. Tuber production is favored by low nitrogen levels and high temperatures (80 - 91 F). Nutsedges are tolerant of high soil moisture and intolerant of shade.

Purple nutsedge can reproduce from tubers when conditions are harsh, making this weed especially difficult to control. Unlike the rhizomes of yellow nutsedge, purple nutsedge rhizomes growing off the first plant produce new plants and tubers in a series (tuber-chains). Purple nutsedge can reproduce by seed and can survive a wide range of environmental conditions, growing well in nearly all soil types and over a range of soil moisture, soil pH, and elevation. Purple nutsedge is also able to survive extremely high temperatures (20).

PIGWEED

Common species of pigweed in Florida include smooth pigweed (*Amaranthus hybridus*), spiny amaranth (*A. spinosus*) and livid amaranth (*A. lividus*). Pigweeds are summer annuals with taproots. These broadleaf plants reproduce by seed and can reach heights of 6 feet, creating a very competitive interaction with young sugarcane (20).

FALL PANICUM

Fall panicum (*Panicum dichotomiflorum*) is an annual that primarily emerges in spring and summer. Seed germination can occur almost year-round in Florida sugarcane fields. Fall panicum is sensitive to shading and is typically not found in sugarcane once canopy closure occurs. Fall panicum typically reaches a height of 1.5 - 4 feet and has been reported to reach more than 6 feet in height. The growth habit of this weed can range from erect to sprawling, and the plant can form large, loose tufts (18).

NAPIERGRASS

Napiergrass (*Pennisetum purpureum*) is an enormous, cane-like grass established throughout South Florida. In the sugarcane production areas of South Florida, napiergrass is commonly seen growing along roadsides, on the banks of canals and ditches, and in disturbed or cultivated areas.

Napiergrass, also known as elephantgrass, is of African origin and has been introduced to all tropical areas of the world because of the plant's ability to quickly produce large amounts of biomass. Napiergrass is widely used as a forage crop in Central America, South America, and Africa.

Napiergrass was introduced to South Florida and Texas as a forage crop in the early twentieth century. However, napiergrass is no longer widely used for this purpose in Florida and has become a considerable weed problem in Florida. The Florida Exotic Pest Plant Council has identified napiergrass as an invasive species in South Florida (19). Napiergrass, which has no natural diseases or insect pests in Florida, has been documented in almost 30 counties throughout the state.

Napiergrass produces many small, viable seeds, which are easily dispersed. Napiergrass also has a thick, aggressive rhizome structure.

Non-chemical Management of Weed Pests in Florida Sugarcane Fields

CROP ROTATION

Crop rotation patterns will affect weed management for a sugarcane crop. The need for weed management intensifies in successive planting operations due to cultivation, which contributes to germination of weed seeds. Traditionally, the fallow period between final ratoon harvest and replanting of sugarcane has effectively been used to manage troublesome weed populations by means of mechanical cultivation, crop rotation, and flooding.

Flooding fallow fields aids in weed control through the development of an anaerobic environment, where germination of weed seeds and seedling growth are impaired. In any given year in Florida, rice is grown as a rotational crop on approximately 10,000 acres (12,20). Typically, once the rice-growing season is over, these fields are replanted to sugarcane.

CROP COMPETITION

Crop competition for sunlight is one of the most effective means of weed control. A good stand of sugarcane that emerges rapidly and uniformly and forms a complete canopy (shading the row middles early in the season) is effective in reducing weed competition. Loss of sugarcane plants in ratoon crops -- whether due to rodent, insect, or harvest -- creates open spaces in the sugarcane canopy, under which weeds proliferate. A concentrated effort to maintain maximum cane population throughout all phases of production benefits weed-control efforts (20).

CULTIVATION

Cultivation can suppress weed growth. A height differential must be established between cane plants and weeds to ensure sugarcane plants get the early advantage in the competition for sunlight. Only when the cane plants are growing taller than competing weeds can mechanical cultivation be effective.

Cultivation when weeds are not present -- due to application of a herbicide or earlier cultivation -- is not recommended. Cultivation when weeds are not present can encourage germination of additional weed seeds and remove the layer of herbicide present when soil-applied herbicides are used. Mechanical cultivators must be able to cut through surface debris and thoroughly mix the soil in ratoon crops (20).

Chemical Management of Weed Pests in Florida Sugarcane Fields

Herbicides are routinely used in sugarcane production in Florida, as in commercial sugarcane production elsewhere in the world. The most commonly used materials include atrazine, 2,4-D, asulam, and ametryn. Herbicides used on less than 10 percent of the sugarcane acreage in Florida are metribuzin, halosulfuron, and pendimethalin (21,22). Other herbicides registered for use in Florida sugarcane as of 2008 are carfentrazone, dicamba, diuron +/- hexazinone, flumioxazin, glyphosate, paraquat, and trifloxysulfuron (20).

ATRAZINE

Atrazine is a restricted-use pesticide and the main herbicide used by sugarcane growers in Florida. Atrazine is applied to nearly all of the sugarcane grown in Florida as a preemergence treatment and sometimes as an early postemergent treatment. Atrazine controls most annual grass and broadleaf weeds. Atrazine is applied at a rate of 3 - 4 lb ai/A. The price of atrazine is approximately $3.50 per pound of active ingredient. The approximate cost of a maximum labeled application (4.0 lb ai/A) is $14/A (23,24). A special local-needs registration allows up to 10 lb of ai/A per crop.

2,4-D

The herbicide 2,4-D is selective against broadleaf weeds when sprayed on the foliage and is routinely used for the management of spiny amaranth, ragweed, morning glory, and many other weeds. This herbicide is applied by ground or air to approximately three-quarters of Florida's sugarcane acreage at a rate of 1.9 lb ai/A (21,23). Higher rates of application are used for large or difficult-to-control weeds,

such as alligatorweed. The price of 2,4-D is approximately $3 per pound of active ingredient, and the approximate cost of a maximum labeled application (2.0 lb ai/A) is $6/A (20,23).

AMETRYN

Ametryn is applied as a directional or semi-directional spray to annual grass and broadleaf-weed seedlings. Ametryn is applied to approximately 40 percent of Florida's sugarcane acreage. Ametryn is applied by ground or air at a rate of approximately 0.5 lb ai/A (22). The price of ametryn is approximately $7 per pound of active ingredient, and the approximate cost of a maximum labeled application (1.1 lb ai/A) is $8/A (20,23).

ASULAM

Asulam is a herbicide applied to foliage of immature seedling grasses. Application may be broadcast overall, directed, or semi-directed in sugarcane at least 14 inches tall. Asulam is applied only once per year. Asulam controls alexandergrass, broadleaf panicum, and other annual grasses. Asulam activity appears slow. Weed growth ceases at the time of application, and weed death may take 2 - 3 weeks.

Asulam is applied by ground or air to approximately 20 percent of Florida's sugarcane acreage at a rate of approximately 1 lb ai/A (22). The price of asulam is approximately $12 per pound of active ingredient, and the approximate cost of a maximum labeled application (3.3 lb ai/A) is $40/A (20,23).

METRIBUZIN

Metribuzin is applied by ground to a small number of sugarcane acres in Florida. Metribuzin is not registered for use in sugarcane grown on sand soils.

Metribuzin controls most annual grass and broadleaf weeds and is often mixed with pendimethalin. Metribuzin is applied at the time of planting or ratooning and prior to weed emergence or early post emergence.

Metribuzin is applied at a rate of 0.8 lb ai/A (22). The price of metribuzin is approximately $23 per pound of active ingredient. The approximate cost of a maximum labeled application (1.9 lb ai/A) is $44/A (20,23).

PENDIMETHALIN

Pendimethalin provides preemergent control of annual grasses. Additionally, pendimethalin is often mixed with metribuzin or atrazine for broadleaf weed control.

Pendimethalin is applied by ground or air to a small number of sugarcane acres in Florida. Pendimethalin controls most annual grasses and is often mixed with metribuzin. Pendimethalin is applied at the time of planting or ratooning, prior to weed emergence.

Rainfall or shallow cultivation within seven days of application to incorporate the herbicide into the soil is useful to achieve the best efficacy. Pendimethalin is applied at a rate of 3.3 lb ai/A on muck soils and 2.4 lb ai/A on sand soils (22). The price of pendimethalin is approximately $6 per pound of active ingredient. The approximate cost of a maximum labeled application (4 lb ai/A) is $24/A (20,23).

HALOSULFURON

Halosulfuron is applied by ground to a small number of sugarcane acres in Florida. Halosulfuron controls purple and yellow nutsedge, as well as some broadleaf weed species. Halosulfuron may be applied to any stage of sugarcane growth. Halosulfuron is applied at a rate of 0.05 lb ai/A (22). The price of halosulfuron is approximately $245 per pound of active ingredient, and the approximate cost of a maximum labeled application (0.06 lb ai/A) is $15/A (20,23).

Nematode Management in Florida Sugarcane Crops

Plant-parasitic nematodes are microscopic roundworms found in soil. Ectoparastic nematodes feed on sugarcane from the exterior of the root while endoparasitic nematodes enter the plant tissue to feed from within. General symptoms of nematode damage to sugarcane plants include stunting, premature wilting, leaf yellowing and related symptoms characteristic of nutrient deficiencies. Stunting and poor stand development tend to occur in patches throughout the field as a result of the irregular distribution of nematodes within the soil. Ratoon cane is generally most susceptible to damage from nematodes (25).

Most species of plant-parasitic nematodes favor sandy-soil conditions and are rarely a problem on muck soils. However, sugarcane grown in sandy areas has the potential for dramatic yield losses from nematodes (25).

Nematode Pests in Florida Sugarcane Crops

Sting nematode (*Belonolaimus longicaudatus*), an ectoparasite, is the most damaging nematode to sugarcane in Florida. Stubby-root (*Trichodorus* and *Paratrichodorus* spp.), ring (*Criconemoides* and related genera), and stunt

(*Tylenchorhynchus* and *Quinisulcius*) nematodes are other ectoparasites that may damage sugarcane and are common in Florida. Endoparasites that may damage sugarcane in Florida are lesion (*Pratylenchus zeae*), lance (*Hoplolaimus* spp.), and root-knot (*Meloidogyne* spp.) nematodes.

Non-chemical Management of Nematodes in Florida Sugarcane Crops

CROP ROTATION

Rotation with flooded rice crops can reduce populations of plant-parasitic nematodes. Many of the nematodes that feed on sugarcane can also feed on rice under dry conditions. However, because rice is normally grown in standing water, most nematodes are killed by the flooded conditions (25).

FLOODING

Flooding can be an effective management strategy for nematode control in sugarcane. The area should be flooded for a 4-week period, then drained and left dry for 2 weeks, and then flooded again for 4 weeks (25). This practice is difficult on sandy soils, where nematodes are most prevalent.

SOIL AMENDMENT

Sediment collected in the sugarcane juice clarification process is called filtercake, cachaza, or mill mud. Soil amendment with this substance has been shown to reduce populations of plant-parasitic nematodes on sugarcane. Filtercake can be added as an amendment to sandy areas to reduce nematode damage. The addition of organic matter, including sugarcane filtercake, to sandy soil can also improve plant tolerance and make nematode damage less severe (25).

Chemical Management of Nematodes in Florida Sugarcane Crops

In sandy soils, where nematodes are most prevalent in sugarcane crops, nematicides are infrequently used. Ethoprop, in addition to managing wireworms, may provide nematicidal benefit. The fumigant 1,3-dichloropropene also provides temporary nematode abatement (25).

Vertebrate Management in Florida Sugarcane Fields

Several species of rat feed on sugarcane plants and can in some cases impose measurable economic loss. A special local-need registration for the rodenticide diphacinone exists for sugarcane growers in Florida (24). Zinc phosphide is also registered to control vertebrates in sugarcane fields in Florida. Rodenticides are applied on the margins of sugarcane fields, but are used on less than one percent of the area planted to sugarcane in Florida.

Key Contact

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Pioneer Florida Museum and Village

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Click photo to view slides of cane grinding and cane syrup making

The Pioneer Florida Museum has a Cane Syrup Mill complete with hard working mule. Demonstratons of cane grinding and syrup making are presented during the year at special events.
Sugarcane was a Pioneer Florida cash crop and was harvested mostly by hand and sometimes mechanically. The museum has a mill grinder which could produce income for a pioneer family. Hand harvesting still accounts for more than half of the world's production. Harvesters use cane knives or machetes, cut the standing cane just above the ground.

Early development of cane grinding in Florida was done with a mule or horse. Later, mechanical harvesting, a sugarcane combine (or chopper harvester), a harvesting machine originally developed in Australia, was used. The Austoft 7000 series was the original design for the modern harvester and has now been copied by other companies including Cameco and John Deere. The machine cuts the cane at the base of the stalk, separates the cane from its leaves, and deposits the cane into a haulout transporter while blowing the thrash back onto the field. A modern machines can harvest 100 tons of cane each hour, but cane harvested using earlier machines had to be transported to the processing plant rapidly. Once cut, sugarcane begins to lose its sugar content, and damage inflicted on the cane during mechanical harvesting accelerates this decay.

APPENDIX KK

Sugar Cane & Sugar Beets

The world of money-in-politics can be sour, but sometimes, its sweet.

Sugar is the only industry in the entire agribusiness sector that has consistently supported Democrats during the past two decades. Sugar cane is grown in southern states such as Florida, while sugar beets are primarily grown in the upper Midwest and Great Plains and converted into a number of sugar products. *[Read more Background]*

Top Contributors, 2013-2014

Contributor	Amount
American Crystal Sugar	$2,219,849
Florida Crystals	$720,430
Michigan Sugar	$515,400
American Sugar Cane League	$393,750
Southern Minn Beet Sugar Co-Op	$309,500
Minn-Dak Farmers Co-Op	$283,000
US Sugar	$242,100
Western Sugar Cooperative	$209,500
Florida Sugar Cane League	$182,817
Snake River Sugar	$126,250
American Sugarbeet Growers Assn	$110,500
Sugar Cane Growers Co-op of Florida	$107,250
US Beet Sugar Assn	$57,000
Rio Grande Valley Sugar Growers	$39,500
St Mary Sugar Cooperative	$16,750
Amalgamated Sugar	$16,300
MK Enterprises	$8,500
Lula-Westfield LLC	$7,750
California Beet Growers Assn	$6,750

Contributions to Democrats Republicans Outside Spending Groups

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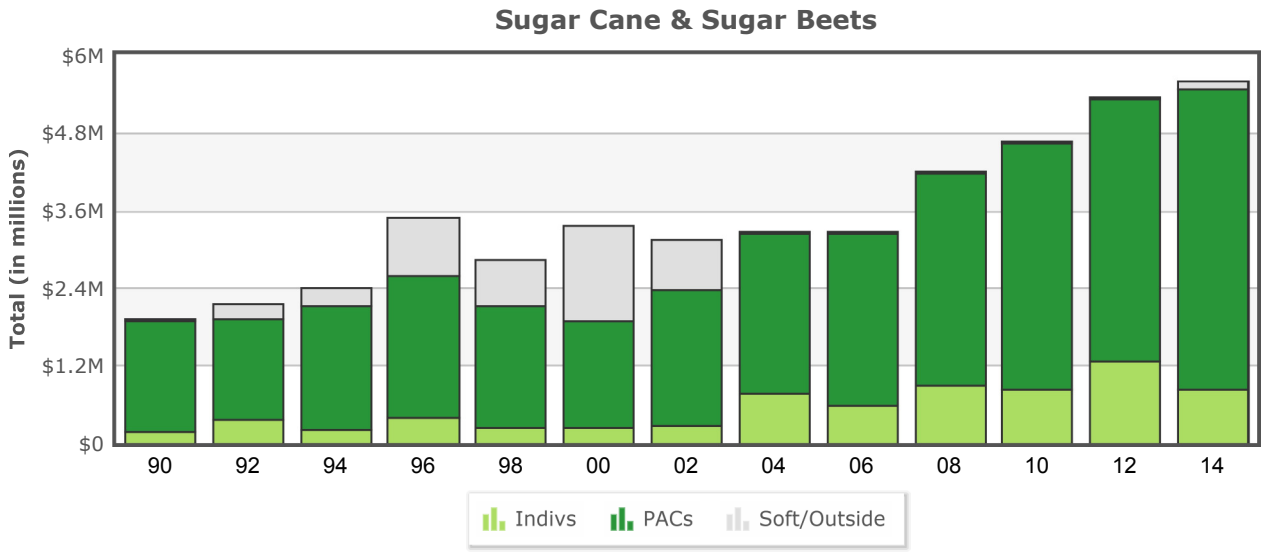
Contribution Trends, 1990-2014

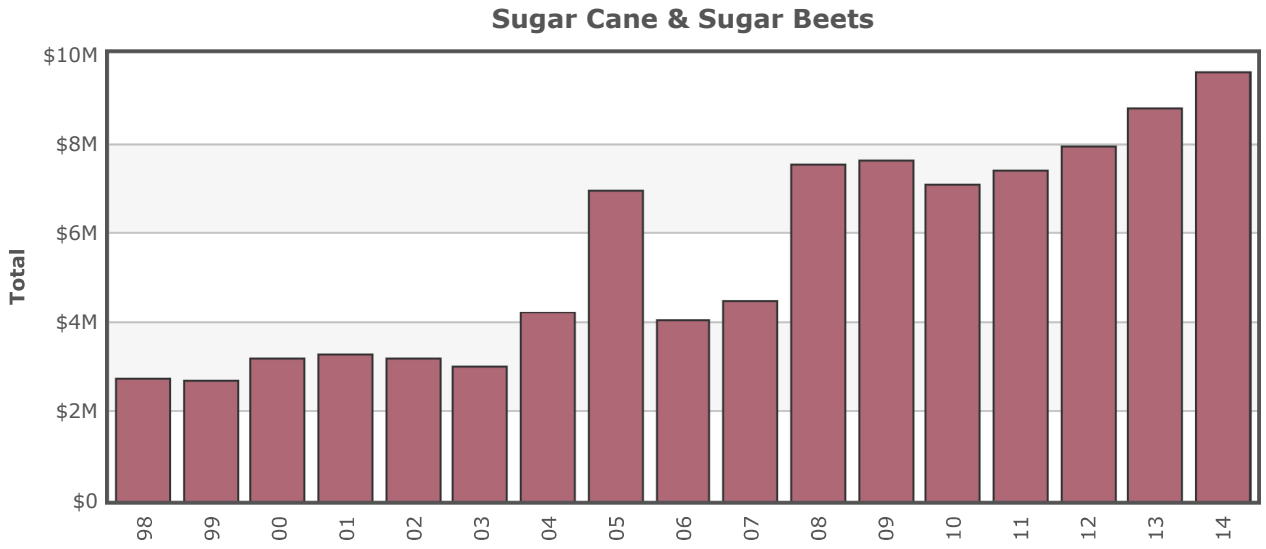


...view Totals

Top Lobbying Clients, 2014

Client/Parent	Total
American Sugar Alliance	$2,180,000
American Crystal Sugar	$2,039,484
US Beet Sugar Assn	$1,800,000
Fanjul Corp	$1,190,000
Florida Sugar Cane League	$1,000,000

Lobbying Totals, 1998-2014



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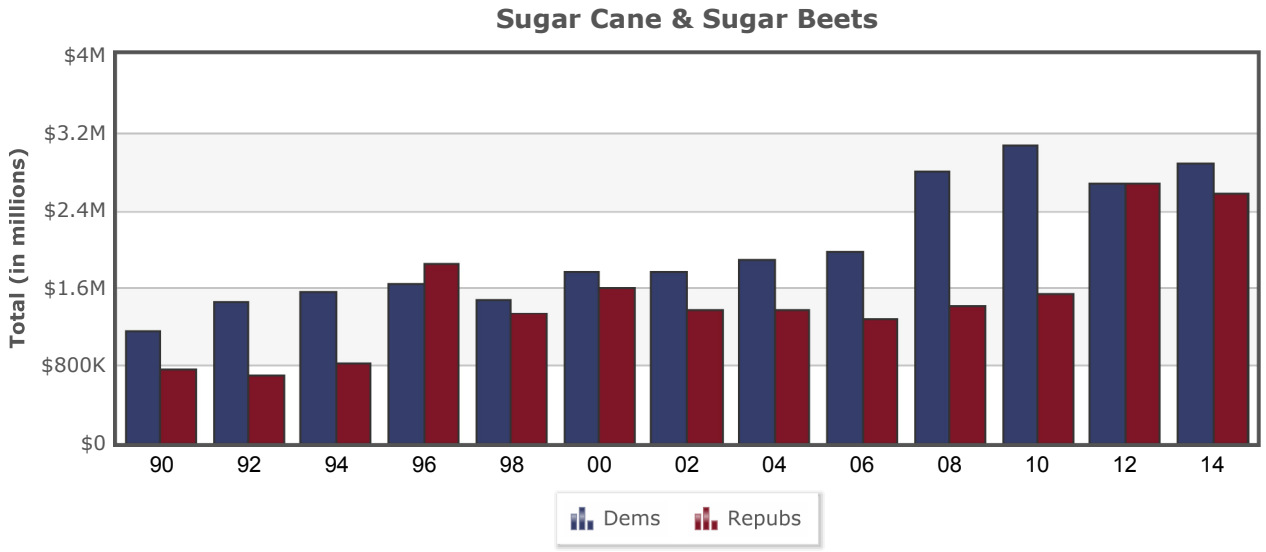
Party Split, 1990-2014



Top Recipients, 2013-2014

Candidate	Office	Amount
Cochran, Thad (R-MS)	Senate	$81,500
Peterson, Collin (D-MN)	House	$72,150
Schumer, Charles E (D-NY)	Senate	$54,400
Conaway, Mike (R-TX)	House	$54,000
Landrieu, Mary L (D-LA)	Senate	$49,750

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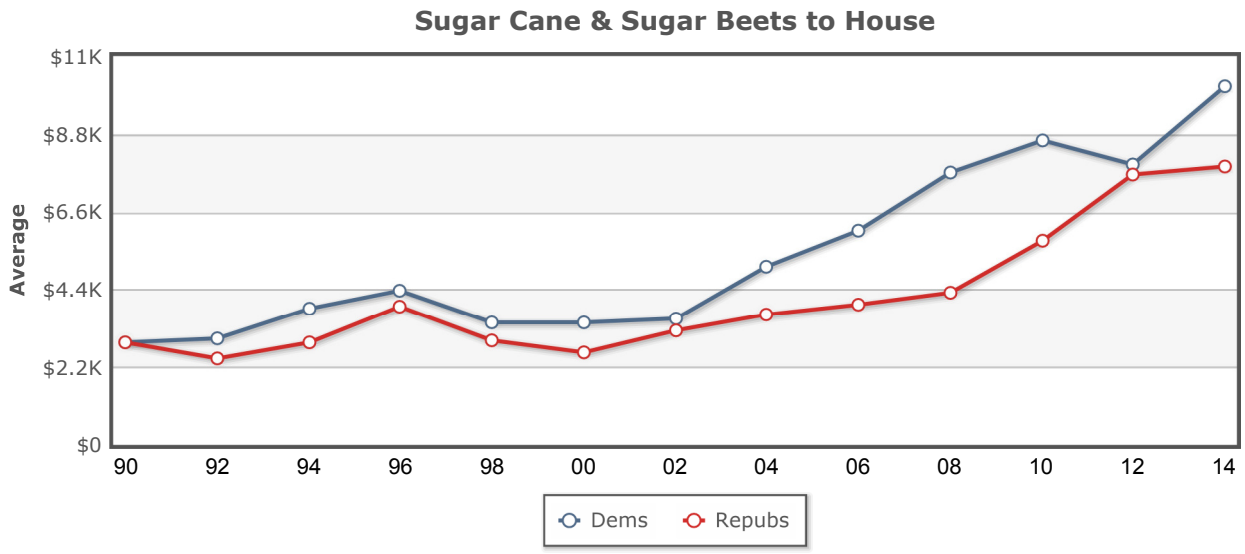
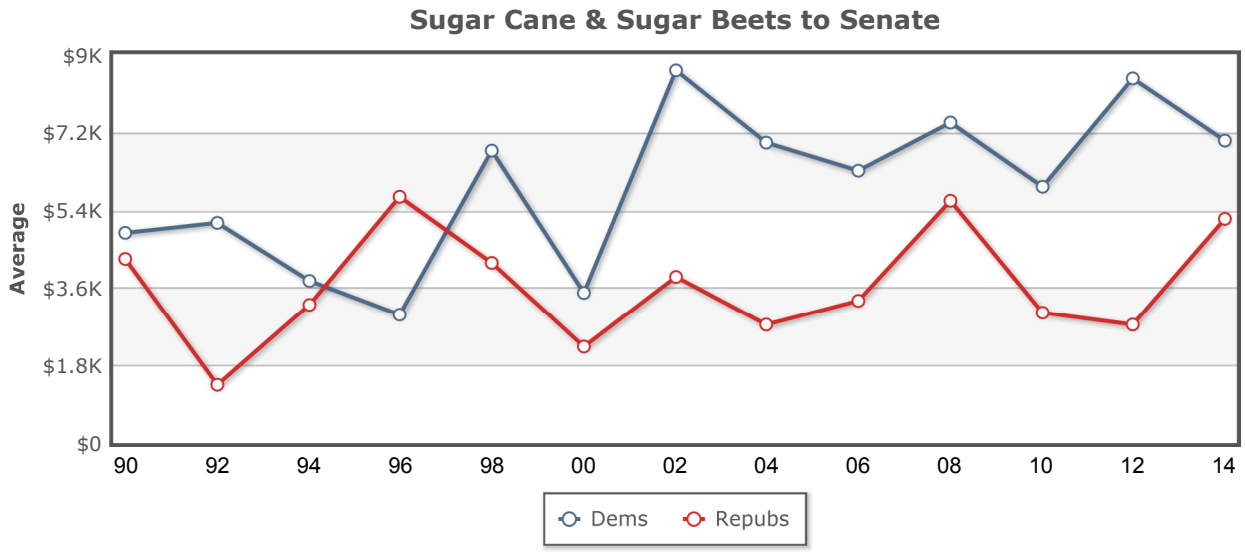
Average Contributions to Members of Congress, 1990-2014





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Sugar

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Data for the current election cycle were released by the Federal Election Commission on Monday, February 02, 2015

Sugar Cane & Sugar Beets

Sugar Cane & Sugar Beets: Background

The world of money-in-politics can be sour, but sometimes, its sweet.

Sugar is the only industry in the entire agribusiness sector that has consistently supported Democrats during the past two decades. Sugar cane is grown in southern states such as Florida, while sugar beets are primarily grown in the upper Midwest and Great Plains and converted into a number of sugar products.

Individuals and political action committees associated with the sugar industry have contributed more than $41.7 million since 1990, with about 58 percent going to Democrats. Over the years, the amount the industry contributes has increased and the industry's affection for Democrats has waned. During the 2008 campaign cycle, the industry contributed more than $4.2 million, with 65 percent going to Democrats. By the 2012 cycle, the industry accounted for more than $5.3 million in contributions, with the money evenly split 50-50 between Democrats and Republicans

The top contributor by far remains American Crystal Sugar, whose PAC contributed about $2 million to federal candidates during the 2012 cycle, with more than 57 percent going to Democrats.

On the lobbying front, the sugar industry spent $7.9 million in 2012, which is roughly the amount it has spent since 2008, when the industry's spending jumped up dramatically from just $4.5 million in 2007. The majority of the sugar industry's lobbying expenditures come from three groups -- American Crystal Sugar and two industry trade groups. Much of the increase in the industry's lobbying can be attributed to their spending habits.

The U.S. Beet Sugar Association -- a trade group of sugar beet farmers and producers - has spent between $1.8 million and $2 million a year on lobbying for for the last several years. The group's expenditures jumped from $560,000 in 2007 to $2 million in 2008.

American Crystal Sugar -- the largest sugar beet producer in the nation - made a similar dramatic jump in spending on lobbying. The company has spent more than $1 million on lobbying every year since 2008 but the company did not lobby at all in 2007, and prior to that had only lobbied once, spending a mere $60,000 in 2005.

The third major group, the American Sugar Alliance has been a force in Washington for some time, spending more than $1 million a year on lobbying every year since 2003. In 2012 it spent more than $1.7 million on lobbying.

The sugar industry is closely entwined with federal agricultural and trade policy, and the industry closely monitors and seeks to influence Washington on the Farm Bill and various trade agreements, like the Central American Free Trade Agreement (CAFTA), the North American Free Trade Agreement (NAFTA) and the Trans -Pacific PArtnership.

-- Russ Choma

Updated September 2013

Industries in this Sector:

Sugar

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OpenSecrets.org is your nonpartisan guide to money's influence on U.S. elections and public policy. Whether you're a voter, journalist, activist, student or interested citizen, use our free site to shine light on your government.

APPENDIX LL

2.5 Open Burning

2.5.1 General[1]

Open burning can be done in open drums or baskets, in fields and yards, and in large open dumps or pits. Materials commonly disposed of in this manner include municipal waste, auto body components, landscape refuse, agricultural field refuse, wood refuse, bulky industrial refuse, and leaves.

Current regulations prohibit open burning of hazardous waste. One exception is for open burning and detonation of explosives, particularly waste explosives that have the potential to detonate, and bulk military propellants which cannot safely be disposed of through other modes of treatment.

The following Source Classification Codes (SCCs) pertain to open burning:

Government

5-01-002-01	General Refuse
5-01-002-02	Vegetation Only

Commercial/Institutional

5-02-002-01	Wood
5-02-002-02	Refuse

Industrial

5-03-002-01	Wood/Vegetation/Leaves
5-03-002-02	Refuse
5-03-002-03	Auto Body Components
5-03-002-04	Coal Refuse Piles
5-03-002-05	Rocket Propellant

2.5.2 Emissions[1-22]

Ground-level open burning emissions are affected by many variables, including wind, ambient temperature, composition and moisture content of the debris burned, and compactness of the pile. In general, the relatively low temperatures associated with open burning increase emissions of particulate matter, carbon monoxide, and hydrocarbons and suppress emissions of nitrogen oxides. Emissions of sulfur oxides are a direct function of the sulfur content of the refuse.

2.5.2.1 Municipal Refuse -

Emission factors for the open burning of municipal refuse are presented in Table 2.5-1.

2.5.2.2 Automobile Components -

Emission factors for the open burning of automobile components including upholstery, belts, hoses, and tires are presented in Table 2.5-1.

Emission factors for the burning of scrap tires only are presented in Tables 2.5-2, 2.5-3, and 2.5-4. Although it is illegal in many states to dispose of tires using open burning, fires often occur at

Table 2.5-1 (Metric And English Units). EMISSION FACTORS FOR OPEN BURNING OF MUNICIPAL REFUSE

EMISSION FACTOR RATING: D

Source	Particulate	Sulfur Oxides	Carbon Monoxide	TOC[a]		Nitrogen Oxides
				Methane	Nonmethane	
Municipal Refuse[b]						
kg/Mg	8	0.5	42	6.5	15	3
lb/ton	16	1.0	85	13	30	6
Automobile Components[c]						
kg/Mg	50	Neg	62	5	16	2
lb/ton	100	Neg	125	10	32	4

[a] Data indicate that total organic compounds (TOC) emissions are approximately 25% methane, 8% other saturates, 18% olefins, 42% others (oxygenates, acetylene, aromatics, trace formaldehyde).
[b] References 2 and 7.
[c] Reference 2. Upholstery, belts, hoses, and tires burned together.

tire stockpiles and through illegal burning activities. If the emission factors presented here are used to estimate emissions from an accidental tire fire, it should be kept in mind that emissions from burning tires are generally dependent on the burn rate of the tire. A greater potential for emissions exists at lower burn rates, such as when a tire is smoldering, rather than burning out of control. In addition, the emission factors presented here for tire "chunks" are probably more appropriate than for "shredded" tires for estimating emissions from an accidental tire fire because there is likely to be more air-space between the tires in an actual fire. As discussed in Reference 21, it is difficult to estimate emissions from a large pile of tires based on these results, but emissions can be related to a mass burn rate. To use the information presented here, it may be helpful to use the following estimates: tires tested in Reference 21 weighed approximately 7 kilograms (kg) (15.4 pounds [lb]) and the volume of 1 tire is approximately 0.2 cubic meter (m^3) (7 cubic feet [ft^3]). Table 2.5-2 presents emission factors for particulate metals. Table 2.5-3 presents emission factors for polycyclic aromatic hydrocarbons (PAH), and Table 2.5-4 presents emissions for other volatile hydrocarbons. For more detailed information on this subject consult the reference cited at the end of this chapter.

2.5.2.3 Agricultural Waste -

Organic Agricultural Waste -

Organic refuse burning consists of burning field crops, wood, and leaves. Emissions from organic agricultural refuse burning are dependent mainly on the moisture content of the refuse and, in the case of the field crops, on whether the refuse is burned in a headfire or a backfire. Headfires are started at the upwind side of a field and allowed to progress in the direction of the wind, whereas backfires are started at the downwind edge and forced to progress in a direction opposing the wind.

Other variables such as fuel loading (how much refuse material is burned per unit of land area) and how the refuse is arranged (in piles, rows, or spread out) are also important in certain instances. Emission factors for open agricultural burning are presented in Table 2.5-5 as a function of refuse type and also, in certain instances, as a function of burning techniques and/or moisture content when these variables are known to significantly affect emissions. Table 2.5-5 also presents typical fuel loading values associated with each type of refuse. These values can be used, along with

Table 2.5-2 (Metric And English Units). PARTICULATE METALS EMISSION FACTORS FROM OPEN BURNING OF TIRES[a]

EMISSION FACTOR RATING: C

Tire Condition	Chunk[b]		Shredded[b]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
Aluminum	3.07	6.14	2.37	4.73
Antimony[c]	2.94	5.88	2.37	4.73
Arsenic[c]	0.05	0.10	0.20	0.40
Barium	1.46	2.92	1.18	2.35
Calcium	7.15	14.30	4.73	9.47
Chromium[c]	1.97	3.94	1.72	3.43
Copper	0.31	0.62	0.29	0.58
Iron	11.80	23.61	8.00	15.99
Lead[c]	0.34	0.67	0.10	0.20
Magnesium	1.04	2.07	0.75	1.49
Nickel[c]	2.37	4.74	1.08	2.15
Selenium[c]	0.06	0.13	0.20	0.40
Silicon	41.00	82.00	27.52	55.04
Sodium	7.68	15.36	5.82	11.63
Titanium	7.35	14.70	5.92	11.83
Vanadium	7.35	14.70	5.92	11.83
Zinc	44.96	89.92	24.75	49.51

[a] Reference 21.
[b] Values are weighted averages.
[c] Hazardous air pollutants listed in the *Clean Air Act.*

Table 2.5-3 (Metric And English). POLYCYCLIC AROMATIC HYDROCARBON EMISSION FACTORS FROM OPEN BURNING OF TIRES[a]

EMISSION FACTOR RATING: D

Tire Condition	Chunk[b,c]		Shredded[b,c]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
Acenaphthene	718.20	1436.40	2385.60	4771.20
Acenaphthylene	570.20	1140.40	568.08	1136.17
Anthracene	265.60	531.20	49.61	99.23
Benzo(A)pyrene	173.80	347.60	115.16	230.32
Benzo(B)fluoranthene	183.10	366.20	89.07	178.14
Benzo(G,H,I)perylene	36.20	72.40	160.84	321.68
Benzo(K)fluoranthene	281.80	563.60	100.24	200.48
Benz(A)anthracene	7.90	15.80	103.71	207.43
Chrysene	48.30	96.60	94.83	189.65
Dibenz(A,H)anthracene	54.50	109.00	0.00	0.00
Fluoranthene	42.30	84.60	463.35	926.69
Fluorene	43.40	86.80	189.49	378.98
Indeno(1,2,3-CD)pyrene	58.60	117.20	86.38	172.76
Naphthalene[d]	0.00	0.00	490.85	981.69
Phenanthrene	28.00	56.00	252.73	505.46
Pyrene	35.20	70.40	153.49	306.98

[a] Reference 21.
[b] 0.00 values indicate pollutant was not found.
[c] Values are weighted averages.
[d] Hazardous air pollutants listed in the *Clean Air Act*.

Table 2.5-4 (Metric And English Units). EMISSION FACTORS FOR ORGANIC COMPOUNDS FROM OPEN BURNING OF TIRES[a]

EMISSION FACTOR RATING: C

Tire Condition	Chunk[b,c]		Shredded[b,c]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
1,1'Biphenyl, methyl	12.71	25.42	0.00	0.00
1h Fluorene	191.27	382.54	315.18	630.37
1-Methyl naphthalene	299.20	598.39	227.87	455.73
2-Methyl naphthalene	321.47	642.93	437.06	874.12
Acenaphthalene	592.70	1185.39	549.32	1098.63
Benzaldehyde	223.34	446.68	322.05	644.10
Benzene[d]	1526.39	3052.79	1929.93	3859.86
Benzodiazine	13.12	26.23	17.43	34.87
Benzofuran	40.62	81.24	0.00	0.00
Benzothiophene	10.31	20.62	914.91	1829.82
Benzo(B)thiophene	50.37	100.74	0.00	0.00
Benzsisothiazole	0.00	0.00	151.66	303.33
Biphenyl[d]	190.08	380.16	329.65	659.29
Butadiene[d]	117.14	234.28	138.97	277.95
Cyanobenzene	203.81	407.62	509.34	1018.68
Cyclopentadiene	67.40	134.80	0.00	0.00

Table 2.5-4 (cont.).

Tire Condition	Chunk[b,c]		Shredded[b,c]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
Dihydroindene	9.82	19.64	30.77	61.53
Dimethyl benzene	323.58	647.16	940.91	1881.83
Dimethyl hexadiene	6.22	12.44	73.08	146.15
Dimethyl naphthalene	35.28	70.55	155.28	310.57
Dimethyldihydro indene	5.02	10.04	27.60	55.20
Ethenyl, dimethyl benzene	11.50	23.01	196.34	392.68
Ethenyl, methyl benzene	12.48	24.95	21.99	43.98
Ethenyl benzene[d]	539.72	1079.44	593.15	1186.31
Ethenyl cyclohexene	4.85	9.70	89.11	178.22
Ethenylmethyl benzene	103.13	206.26	234.59	469.19
Ethyenylmethyl benzene	0.00	0.00	42.04	84.07
Ethyl, methyl benzene	79.29	158.58	223.79	447.58
Ethyl benzene	138.94	277.87	335.12	670.24
Ethynyl, methyl benzene	459.31	918.62	345.25	690.50
Ethynyl benzene	259.82	519.64	193.49	386.98
Heptadiene	6.40	12.79	42.12	84.24
Hexahydro azepinone	64.35	128.69	764.03	1528.05

Table 2.5-4 (cont.).

Tire Condition	Chunk[b,c]		Shredded[b,c]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
Indene	472.74	945.48	346.23	692.47
Isocyano benzene	283.78	567.55	281.13	562.25
Isocyano naphthalene	10.75	21.51	0.00	0.00
Limonene	48.11	96.22	2309.57	4619.14
Methyl, ethenyl benzene	21.15	42.30	67.05	134.10
Methyl, methylethenyl benzene	35.57	71.13	393.78	787.56
Methyl, methylethyl benzene	109.69	219.39	1385.03	2770.07
Methyl benzaldehyde	0.00	0.00	75.49	150.98
Methyl benzene	1129.80	2259.60	1395.04	2790.08
Methyl cyclohexene	3.91	7.83	33.44	66.88
Methyl hexadiene	15.59	31.18	102.20	204.40
Methyl indene	50.04	100.07	286.68	573.36
Methyl, methylethyl benzene	11.76	23.52	114.33	228.66
Methyl naphthalene	144.78	289.56	122.68	245.37
Methyl, propyl benzene	0.00	0.00	30.14	60.28
Methyl thiophene	4.39	8.78	10.52	21.03
Methylene indene	30.37	60.75	58.91	117.82

Table 2.5-4 (cont.).

Tire Condition	Chunk[b,c]		Shredded[b,c]	
Pollutant	mg / kg tire	lb / 1000 tons tire	mg / kg tire	lb / 1000 tons tire
Methylethyl benzene	41.40	82.79	224.23	448.46
Phenol[d]	337.71	675.41	704.90	1409.80
Propenyl, methyl benzene	0.00	0.00	456.59	913.18
Propenyl naphthalene	26.80	53.59	0.00	0.00
Propyl benzene	19.43	38.87	215.13	430.26
Styrene[d]	618.77	1237.53	649.92	1299.84
Tetramethyl benzene	0.00	0.00	121.72	243.44
Thiophene	17.51	35.02	31.11	62.22
Trichlorofluoromethane	138.10	276.20	0.00	0.00
Trimethyl benzene	195.59	391.18	334.80	669.59
Trimethyl naphthalene	0.00	0.00	316.26	632.52

[a] Reference 21.
[b] 0.00 values indicate the pollutant was not found.
[c] Values are weight averages.
[d] Hazardous air pollutants listed in the *Clean Air Act*.

Table 2.5-5 (Metric And English Units). EMISSION FACTORS AND FUEL LOADING FACTORS FOR OPEN BURNING OF AGRICULTURAL MATERIALS[a]

EMISSION FACTOR RATING: D

Refuse Category	Particulate[b]		Carbon Monoxide		TOC[c]				Fuel Loading Factors (waste production)	
					Methane		Nonmethane			
	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	Mg/hectare	ton/acre
Field Crops[d]										
Unspecified	11	21	58	117	2.7	5.4	9	18	4.5	2
Burning techniques not significant[e]										
Asparagus[f]	20	40	75	150	10	20	33	66	3.4	1.5
Barley	11	22	78	157	2.2	4.5	7.5	15	3.8	1.7
Corn	7	14	54	108	2	4	6	12	9.4	4.2
Cotton	4	8	88	176	0.7	1.4	2.5	5	3.8	1.7
Grasses	8	16	50	101	2.2	4.5	7.5	15		
Pineapple[g]	4	8	56	112	1	2	3	6		
Rice[h]	4	9	41	83	1.2	2.4	4	8	6.7	3.0
Safflower	9	18	72	144	3	6	10	20	2.9	1.3
Sorghum	9	18	38	77	1	2	3.5	7	6.5	2.9
Sugar cane[i]	2.3-3.5	6-8.4	30-41	60-81	0.6-2	1.2-3.8	2-6	4-12	8-46	3-17
Headfire Burning[j]										
Alfalfa	23	45	53	106	4.2	8.5	14	28	1.8	0.8
Bean (red)	22	43	93	186	5.5	11	18	36	5.6	2.5
Hay (wild)	16	32	70	139	2.5	5	8.5	17	2.2	1.0
Oats	22	44	68	137	4	7.8	13	26	3.6	1.6
Pea	16	31	74	147	4.5	9	15	29	5.6	2.5
Wheat	11	22	64	128	2	4	6.5	13	4.3	1.9

Table 2.5-5 (cont.).

Refuse Category	Particulate[b]		Carbon Monoxide		TOC[c]				Fuel Loading Factors (waste production)	
					Methane		Nonmethane			
	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	Mg/hectare	ton/acre
Backfire Burning[k]										
Alfalfa	14	29	60	119	4.5	9	14	29	1.8	0.8
Bean (red)	7	14	72	148	3	6	10	19	5.6	2.5
Hay (wild)	8	17	75	150	2	4	6.5	13	2.2	1.0
Oats	11	21	68	136	2	4	7	14	3.6	1.6
Wheat	6	13	54	108	1.3	2.6	4.5	9	4.3	1.9
Vine Crops	3	5	26	51	0.8	1.7	3	5	5.6	2.5
Weeds										
Unspecified	8	15	42	85	1.5	3	4.5	9	7.2	3.2
Russian thistle (tumbleweed)	11	22	154	309	0.2	0.5	0.8	1.5	0.2	0.1
Tales (wild reeds)	3	5	17	34	3.2	6.5	10	21		
Orchard Crops[d,l,m]										
Unspecified	3	6	26	52	1.2	2.5	4	8	3.6	1.6
Almond	3	6	23	46	1	2	3	6	3.6	1.6
Apple	2	4	21	42	0.5	1	1.5	3	5.2	2.3
Apricot	3	6	24	49	1	2	3	6	4	1.8
Avocado	10	21	58	116	3.8	7.5	12	25	3.4	1.5
Cherry	4	8	22	44	1.2	2.5	4	8	2.2	1.0
Citrus (orange, lemon)	3	6	40	81	1.5	3	5	9	2.2	1.0
Date palm	5	10	28	56	0.8	1.7	3	5	2.2	1.0
Fig	4	7	28	57	1.2	2.5	4	8	4.9	2.2
Nectarine	2	4	16	33	0.5	1	1.5	3	4.5	2.0

Table 2.5-5 (cont.).

Refuse Category	Particulate[b]		Carbon Monoxide		TOC[c]				Fuel Loading Factors (waste production)	
					Methane		Nonmethane			
	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	Mg/hectare	ton/acre
Orchard Crops[d,l,m]										
Olive	6	12	57	114	2	4	7	14	2.7	1.2
Peach	3	6	21	42	0.6	1.2	2	4	5.6	2.5
Pear	4	9	28	57	1	2	3.5	7	5.8	2.6
Prune	2	3	24	47	1	2	3	6	2.7	1.2
Walnut	3	6	24	47	1	2	3	6	2.7	1.2
Forest Residues[n]										
Unspecified	8	17	70	140	2.8	5.7	9	19	157	70
Hemlock, Douglas fir, cedar[p]	2	4	45	90	0.6	1.2	2	4	ND	ND
Ponderosa pine[q]	6	12	98	195	1.7	3.3	5.5	11	ND	ND

[a] Expressed as weight of pollutant emitted per weight of refuse material burned. ND = no data.

[b] Reference 12. Particulate matter from most agricultural refuse burning has been found to be in the submicrometer size range.

[c] Data indicate that total organic compound (TOC) emissions average 22% methane, 7.5% other saturates, 17% olefins, 15% acetylene, 38.5% unidentified. Unidentified TOCs are expected to include aldehydes, ketones, aromatics, cycloparaffins.

[d] References 12-13 for emission factors, Reference 14 for fuel loading factors.

[e] For these refuse materials, no significant difference exists between emissions from headfiring and backfiring.

[f] Factors represent emissions under typical high moisture conditions. If ferns are dried to <15% moisture, particulate emissions will be reduced by 30%, CO emissions 23%, TOC emissions 74%.

[g] Reference 11. When pineapple is allowed to dry to <20% moisture, as it usually is, firing technique is not important. When headfired at 20% moisture, particulate emissions will increase to 11.5 kg/Mg (23 lb/ton) and TOCs will increase to 6.5 kg/Mg (13 lb/ton).

[h] Factors are for dry (15% moisture) rice straw. If rice straw is burned at higher moisture levels, particulate emissions will increase to 14.5 kg/Mg (29 lb/ton), CO emissions to 80.5 kg/Mg (181 lb/ton), and VOC emissions to 11.5 kg/Mg (23 lb/ton).

[i] Reference 20. See Section 8.12 for discussion of sugar cane burning. The following fuel loading factors are to be used in the corresponding states: Louisiana, 8 - 13.6 Mg/hectare (3 - 5 ton/acre); Florida, 11 - 19 Mg/hectare (4 - 7 ton/acre); Hawaii, 30 - 48 Mg/hectare (11 - 17 ton/acre). For other areas, values generally increase with length of growing season. Use larger end of the emission factor range for lower loading factors.

Table 2.5-5 (cont.).

[j] See text for definition of headfiring.

[k] See text for definition of backfiring. This category, for emission estimation purposes, includes another technique used occasionally to limit emissions, called into-the-wind striplighting, which is lighting fields in strips into the wind at 100 - 200 meter (300 - 600 feet) intervals.

[l] Orchard prunings are usually burned in piles. There are no significant differences in emissions between burning a "cold pile" and using a roll-on technique, where prunings are bulldozed onto the embers of a preceding fire.

[m] If orchard removal is the purpose of a burn, 66 Mg/hectare (30 ton/acre) of waste will be produced.

[n] Reference 10. NO_X emissions estimated at 2 kg/Mg (4 lb/ton).

[p] Reference 15.

[q] Reference 16.

the corresponding emission factors, to estimate emissions from certain categories of agricultural burning when the specific fuel loadings for a given area are not known.

Emissions from leaf burning are dependent upon the moisture content, density, and ignition location of the leaf piles. Increasing the moisture content of the leaves generally increases the amount of carbon monoxide, hydrocarbon, and particulate emissions. Carbon monoxide emissions decrease if moisture content is high but increase if moisture content is low. Increasing the density of the piles increases the amount of hydrocarbon and particulate emissions, but has a variable effect on carbon monoxide emissions.

The highest emissions from open burning of leaves occur when the base of the leaf pile is ignited. The lowest emissions generally arise from igniting a single spot on the top of the pile. Particulate, hydrocarbon, and carbon monoxide emissions from windrow ignition (piling the leaves into a long row and igniting one end, allowing it to burn toward the other end) are intermediate between top and bottom ignition. Emission factors for leaf burning are presented in Table 2.5-6. For more detailed information on this subject, the reader should consult the reference cited at the end of this section.

2.5.2.4 Agricultural Plastic Film -

Agricultural plastic film that has been used for ground moisture and weed control. Large quantities of plastic film are commonly disposed of when field crops are burned. The plastic film may also be gathered into large piles and burned separately or burned in an air curtain. Emissions from burning agricultural plastic are dependent on whether the film is new or has been exposed to exposed to vegetation and possibly pesticides. Table 2.5-7 presents emission factors for organic compounds emitted from burning new and used plastic film in piles or in piles where air has been forced through them to simulate combustion in an air curtain. Table 2.5-8 presents emission factors for PAHs emitted from open burning of inorganic plastic film.

Table 2.5-6 (Metric And English Units). EMISSION FACTORS FOR LEAF BURNING[a]

EMISSION FACTOR RATING: D

Leaf Species	Particulate[b]		Carbon Monoxide		TOC[c]			
					Methane		Nonmethane	
	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton	kg/Mg	lb/ton
Black Ash	18	36	63.5	127	5.5	11	13.5	27
Modesto Ash	16	32	81.5	163	5	10	12	24
White Ash	21.5	43	57	113	6.5	13	16	32
Catalpa	8.5	17	44.5	89	2.5	5	6.5	13
Horse Chestnut	27	54	73.5	147	8	17	20	40
Cottonwood	19	38	45	90	6	12	14	28
American Elm	13	26	59.5	119	4	8	9.5	19
Eucalyptus	18	36	45	90	5.5	11	13.5	27
Sweet Gum	16.5	33	70	140	5	10	12.5	25
Black Locust	35	70	65	130	11	22	26	52
Magnolia	6.5	13	27.5	55	2	4	5	10
Silver Maple	33	66	51	102	110	20	24.5	49
American Sycamore	7.5	15	57.5	115	2.5	5	5.5	11
California Sycamore	5	10	52	104	1.5	3	3.5	7
Tulip	10	20	38.5	77	3	6	7.5	15
Red Oak	46	92	68.5	137	14	28	34	69
Sugar Maple	26.5	53	54	108	8	16	20	40
Unspecified	19	38	56	112	6	12	14	28

[a] References 18-19. Factors are an arithmetic average of results obtained by burning high and low moisture content conical piles, ignited either at the top or around the periphery of the bottom. The windrow arrangement was only tested on Modesto Ash, Catalpa, American Elm, Sweet Gum, Silver Maple, and Tulip Poplar, and results are included in the averages for these species.

[b] The majority of particulate is submicrometer in size.

[c] Tests indicate that total organic compound (TOC) emissions average 29% methane, 11% other saturates, 33% olefins, 27% other (aromatics, acetylene, oxygenates).

Table 2.5-7 (Metric And English Units). EMISSION FACTORS FOR ORGANIC COMPOUNDS FROM BURNING PLASTIC FILM[a]

EMISSION FACTOR RATING: C

Pollutant	Units	Condition Of Plastic			
		Unused Plastic		Used Plastic	
		Pile[b]	Forced Air[c]	Pile[b]	Forced Air[c]
Benzene	mg/kg plastic	0.0478	0.0288	0.0123	0.0244
	lb/1000 tons plastic	0.0955	0.0575	0.0247	0.0488
Toluene	mg/kg plastic	0.0046	0.0081	0.0033	0.0124
	lb/1000 tons plastic	0.0092	0.0161	0.0066	0.0248
Ethyl benzene	mg/kg plastic	0.0006	0.0029	0.0012	0.0056
	lb/1000 tons plastic	0.0011	0.0058	0.0025	0.0111
1-Hexene	mg/kg plastic	0.0010	0.0148	0.0043	0.0220
	lb/1000 tons plastic	0.0020	0.0296	0.0086	0.0440

[a] Reference 22.
[b] Emission factors are for plastic gathered in a pile and burned.
[c] Emission factors are for plastic burned in a pile with a forced air current.

Table 2.5-8 (Metric And English Units). POLYCYCLIC AROMATIC HYDROCARBON EMISSION FACTORS FROM OPEN BURNING OF AGRICULTURAL PLASTIC FILM[a]

EMISSION FACTOR RATING: C

Pollutant	Units	Condition Of Plastic			
		Unused Plastic		Used Plastic	
		Pile[b]	Forced Air[c]	Pile[b]	Forced Air[c,d]
Anthracene	µg/kg plastic film	7.14	0.66	1.32	0.40
	lb/1000 tons plastic film	0.0143	0.0013	0.0026	0.0008
Benzo(A)pyrene	µg/kg plastic film	41.76	1.45	7.53	0.00
	lb/1000 tons plastic film	0.0835	0.0029	0.0151	0.0000
Benzo(B)fluoranthene	µg/kg plastic film	34.63	1.59	9.25	0.93
	lb/1000 tons plastic film	0.0693	0.0032	0.0185	0.0019
Benzo(e)pyrene	µg/kg plastic film	32.38	1.45	9.65	0.00
	lb/1000 tons plastic film	0.0648	0.0029	0.0193	0.0000
Benzo(G,H,I)perylene	µg/kg plastic film	49.43	2.11	14.93	0.00
	lb/1000 tons plastic film	0.0989	0.0042	0.0299	0.0000
Benzo(K)fluoranthene	µg/kg plastic film	13.74	0.66	2.51	0.00
	lb/1000 tons plastic film	0.0275	0.0013	0.0050	0.0000
Benz(A)anthracene	µg/kg plastic film	52.73	2.91	14.41	1.19
	lb/1000 tons plastic film	0.1055	0.0058	0.0288	0.0024
Chrysene	µg/kg plastic film	54.98	3.70	17.18	1.19
	lb/1000 tons plastic film	0.1100	0.0074	0.0344	0.0024

Table 2.5-8 (cont.).

Pollutant	Units	Condition Of Plastic			
		Unused Plastic		Used Plastic	
		Pile[b]	Forced Air[c]	Pile[b]	Forced Air[c,d]
Fluoranthene	µg/kg plastic film	313.08	53.39	107.05	39.12
	lb/1000 tons plastic film	0.6262	0.1068	0.2141	0.0782
Indeno(1,2,3-CD)pyrene	µg/kg plastic film	40.04	2.78	10.70	0.00
	lb/1000 tons plastic film	0.0801	0.0056	0.0214	0.0000
Phenanthrene	µg/kg plastic film	60.40	12.56	24.05	8.72
	lb/1000 tons plastic film	0.1208	0.0251	0.0481	0.0174
Pyrene	µg/kg plastic film	203.26	18.24	58.81	5.95
	lb/1000 tons plastic film	0.4065	0.0365	0.1176	0.0119
Retene	µg/kg plastic film	32.38	2.91	18.77	3.04
	lb/1000 tons plastic film	0.0648	0.0058	0.0375	0.0061

[a] Reference 22.
[b] Emission factors are for plastic gathered in a pile and burned.
[c] Emission factors are for plastic burned in a pile with a forced air current.
[d] 0.00 and 0.0000 values indicate pollutant was not found at that factor level.

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