CITY OF CARMEL-BY-THE-SEA
FOREST AND BEACH COMMISSION

MEETING AGENDA
Thursday, 10 July 2014
Regular Meeting – 3:30 p.m.

City Hall, Council Chambers
East side of Monte Verde St. between Ocean & 7th Avenues
Carmel, California

CALL TO ORDER AND ROLL CALL

COMMISSION MEMBERS: LISA BOARDMAN – CHAIR
DAVID REFUERZO
KAREN FERLITO
KATHY BANG
MICHAEL CARTER

ROLL CALL

PLEDGE OF ALLEGIANCE

APPEARANCES

Anyone wishing to address the Commission on matters within the jurisdiction of the City and not on the agenda may do so now. Matters not appearing on the Commission's agenda will not receive action at this meeting but may be referred to staff for a future meeting. Presentations will be limited to three (3) minutes, or as otherwise established by the Commission. **Persons are not required to give their names, but it is helpful for speakers to clearly state their full names in order that the Secretary may identify them in the minutes of the meeting.** Always speak into the microphone, as the meeting is recorded. The City Council Chambers is equipped with a portable microphone for anyone unable to come to the podium. Assisted listening devices are available upon request of the Secretary. If you need assistance, please advise the Secretary as to which item you would like to comment on and the microphone will be brought to you.

CONSENT AGENDA

These matters include routine administrative actions, which are usually approved by a single majority vote. Individual items may be removed from Consent by a member of the Council or the public for discussion and action.

1. Consideration of the minutes for the 12 June 2014 regular meeting and the 25 June 2014 special meeting. Page 1.

2. Adopt the revised 2014 regular meeting calendar of the Forest and Beach Commission. Page 11.

ORDERS OF BUSINESS

3. Receive update on beach fire management. Page 12.

4. Consideration of forming ad hoc committee of the Forest and Beach Commission regarding management of beach fires. No page.

5. Consideration of an Arbor Day event for 2014. Page 128.

6. Receive an update on oak tree diseases and tree care. Page 132.

REPORTS FROM STAFF AND COMMISSIONERS

7. Receive the July 2014 report from the Ad Hoc Beach Committee. No page.

8. Forester's report
   A. June tree data – distributed at the meeting. No page.
   B. Project updates.
   C. Parks activities.
   D. Beach activities.
   E. Friends of Carmel Forest program reminder.

9. Public Services Director comments. No page.
   A. Project Status.
   B. Update of Council Items.
   C. Future Agenda Items.

ADJOURNMENT

Any writings or documents provided to a majority of the Forest and Beach Commission regarding any item on this agenda will be made available for public inspection in the Planning and Building Department located at City Hall, on Monte Verde between Ocean and 7th Avenues during normal business hours.

If there is not a special meeting, the next regular meeting of the Forest and Beach Commission will be:
14 August 2014
Tour of Inspection – as required
3:30 p.m. - Regular Agenda

The City of Carmel-by-the-Sea does not discriminate against persons with disabilities. The City of Carmel-by-the-Sea Telecommunication's Device for the Deaf /Speech Impaired (TDD) number is 1-800-735-2929.

CITY OF CARMEL-BY-THE-SEA
FOREST AND BEACH COMMISSION

REGULAR MEETING MINUTES
Thursday, June 12, 2014
Tour of Inspection 3:00 p.m.
Regular Meeting 3:30 p.m.

I. CALL TO ORDER AND ROLL CALL FOR TOUR OF INSPECTION

PRESENT:	Commission Members Ferlito, Refuerzo, Bang, Carter
ABSENT:	Commission members: Boardman
STAFF PRESENT:	Mike Branson, City Forester

II. TOUR OF INSPECTION

The Commission toured the following site:

1. Mission St., 4 north of Vista Ave.

III. ROLL CALL FOR THE REGULAR MEETING

The meeting was called to order at 3:41 p.m.

PRESENT:	Karen Ferlito David Refuerzo Michael Carter Kathy Bang
ABSENT:	Lisa Boardman
STAFF PRESENT:	Mike Branson, City Forester Sharon Friedrichsen, Public Services Director

IV. PLEDGE OF ALLEGIANCE

Members of the audience joined the Commission in the Pledge of Allegiance.

V. APPEARANCES

Skip Lloyd, representing the Friends of Mission Trail Nature Preserve, thanked the Commission for moving items forward for funding in next year's budget.

Barbara Livingston commented on the budget meeting and asked about the duties of the new full-time Public Services position.

Nancy Porteus-Thomas, representing SAND, supported the city moving forward on the North Dunes restoration project. She supports a science and logic based approach for implementation and pledged support as the project moves ahead.

John Lambert asked when the North Dunes may be back on an agenda.

No other appearances.

VI. CONSENT AGENDA

1. Consideration of the minutes for the 3 April 2014 regular meeting.

It was moved by BANG to approve the minutes; seconded by CARTER.

The motion carried by the following vote:

AYES:	Ferlito, Refuerzo, Carter, Bang
NOES:	None
ABSENT:	Boardman
ABSTAIN:	None

VII. APPLICATIONS / PUBLIC HEARINGS

1. Consideration of an application to remove one Monterey pine (25" diameter) on public property due to damage to the applicant's driveway and foundation. The site is located on Mission St., 4 houses northeast of Vista Ave. The applicant is Jon Borden.

City Forester Branson presented his staff report on the tree removal application and answered questions from the Commission.

Commissioner Ferlito asked about the lack of a site plan.

City Forester Branson said a site plan was not necessary due to the tree being in the public right-of-way.

Following discussion on the tree, a motion was made by BANG to deny the application and recommend the applicant work with staff to address the driveway repairs; seconded by FERLITO.

The motion carried by the following vote:

AYES:	Bang, Ferlito, Refuerzo, Carter
NOES:	None
ABSENT:	Boardman
ABSTAIN:	None

VIII. ORDERS OF BUSINESS

1. Review of the landscape plan for the Carmel Event Center Project located at the southeast corner of Dolores St. and 7th Avenue.

This Item was deferred to later in the meeting.

4. Discussion on protocols and procedures regarding dissemination and discussion of materials that are relevant to matters on Forest and Beach Commission agendas.

Director Friedrichsen commented on why this item is on the agenda and introduced City Attorney Don Freeman.

City Attorney Freeman presented an overview of protocols and procedures on communications between Commissioners, staff, and the public that involve items that may appear or are on Commission agendas. He also answered several questions from the Commissioners regarding, ad hoc committees, liability, social media, document distribution, and communication with the public.

Public appearances opened at 4:21 p.m.

Maggie Eaton asked about social meetings where Commissioners or Council members are present and issues may be discussed.

Barbara Livingston asked about sending materials to the City Council members and if is better for the City Administrator to distribute materials.

Skip Lloyd commented that city support groups are covered by the city insurance when working on approved projects.

Barbara Livingston asked if she has a question for a body, should she send it to the City Administrator or the department head.

Public comments closed at 4:25 p.m.

City Attorney Freeman answered the questions from the public.

3. Consideration of forming an ad hoc committee to address improvements to the Scenic Rd. Beach Bluff Pathway.

City Forester Branson presented the staff report.

Commissioner Bang asked about an ad hoc committee with Commissioners and other experts to develop a plan and then disband and let the Commission decide on implementation.

Commissioner Ferlito asked if the Mayor should appoint a small task force.

Director Friedrichsen will ask for direction on this item and bring back more details on how to implement the review of the Pathway plan.

Public appearances opened at 4:36 p.m.

Barbara Livingston hoped that some original beach task force committee members could be included in any new task force. She also asked that any committee look at the past to see what went wrong..

Public appearances closed at 4:37 p.m.

This Item was continued to another meeting.

2. Receive update on beach fire management options and consideration of holding a special meeting on Carmel Beach to discuss various fire management options.

City Forester presented the update on signage, charcoal cleanup, and other fire related items.

Commissioner Bang commented on the Planning Commission review of the proposed beach signs.

Director Friedrichsen commented on the signage program and coordination with the Coastal Commission on new signs at the base of the bluffs. She also reported that funding was approved for new signage and the City will be soliciting the help of a signage consultant.

Staff answered additional question from Commissioners regarding a charcoal cleanup contract and training of beach workers.

Director Friedrichsen noted that the City web site will post cleanup activities that are planned for Carmel Beach and provided a handout of beach management activities.

Public appearances opened at 4:52 p.m.

Mary Liskin pointed out that the beach with white sands is the number one asset to the city. She is concerned about the dollars spent for cleanup and thought that prevention and containment were the best practices.

Scott McKenzie thanked the Commission for keeping a way of life in Carmel. He thought the sand is more grey than other years and thinks the city can do better. He also thought throwing wood over the banks is an issue, signage is not too helpful, and enforcement was helpful. He proposed the possibility of a commercial program to manage fires and would like to meet with staff to introduce his plan.

Margaret Eaton liked the idea of the Conservation Corps cleaning the beach but thought they should use the collection data sheets to see if their efforts and the extra enforcement are successful.

Skip Lloyd note that the charcoal is as bad as ever. He agreed that prevention is good and that cleanup should occur as soon as possible. He likes the idea of a special meeting on the beach and asked that other items like the Leverone basket could be demonstrated.

Barbara Livingston thought that performance measures are needed to see if additional enforcement is working. She also thought that rings were a good idea to limit the number of fires and hopes the beach workers get proper training.

Public appearances closed at 5:08 p.m.

After further discussion on beach fire issues and a date for a special meeting, Commissioner BANG made a motion to hold a special meeting on Carmel Beach at 3:30 p.m. on Wednesday, June 25, 2014 at the 13th Ave. beach access; seconded by REFUERZO.

The motion carried by the following vote:

AYES:	Ferlito, Refuerzo, Bang, Carter
NOES:	None
ABSENT:	Boardman
ABSTAIN:	None

1. Review of the landscape plan for the Carmel Event Center Project located at the southeast corner of Dolores St. and 7th Avenue.

City Forester Branson presented an overview of the proposed landscape plan and its compliance with the conditions approved by the Forest and Beach Commission.

Commissioner Ferlito asked about the Blue Point Cypress trees.

Public appearances opened and closed at 5:46 p.m.

City Forester Branson answered questions from the Commissioners regarding replacing the two public trees and the choice of Deodar cedars around the site.

After further discussion FERLITO made a motion to have staff work with the project to replace the two city trees on 7th Ave. but not cedar trees, to plant a non-cedar upper canopy tree on the eastern part of the property, to plant a new tree in the area near building #2, and to plant a small tree in the Dolores St. planter; seconded by REFUERZO.

The motion carried by the following vote:

AYES:	Ferlito, Refuerzo, Bang, Carter
NOES:	None
ABSENT:	Boardman
ABSTAIN:	None

IX. REPORTS FROM STAFF AND COMMISSIONERS

1. Receive the May 2014 and June 2014 reports from the Ad Hoc Beach Committee.

Commissioner Bang and Refuerzo presented their report on Carmel Beach issues.

Public appearances opened and closed at 6:14 p.m.

2. Forester's report

Forester Branson presented his reports and answered questions from the Commission.

Commissioner Ferlito asked about the title, duties, and hiring of the new staff position.

Director Friedrichsen answered Commissioner Ferlito's question.

Commissioner Ferlito noted that the Friends of Carmel Forest are holding a tree lecture and two tree walks on July 10th & 11th with Dr. Matt Ritter.

3. Public Services Director's comments.

Director Friedrichsen commented on the recent budget adoption by the city council and on department activities for moving the shoreline assessments, North Dunes restoration, and beach signage forward. She also noted the ASBS project was nearing completion, the new restrooms at Santa Lucia Ave. are under construction, and the Planning Commissions direction on commercial activities along the shoreline.

Commissioner Refuerzo commented on the August meeting date and a possible conflict with the Tour d' Elegance on that day. Perhaps a change to the 7th should be discussed at the next meeting.

X. ADJOURNMENT

There being no further business, Chairperson BOARDMAN adjourned the meeting at 6:50 p.m.

Approved,

_____

Lisa Boardman, Chairperson

Respectfully submitted,

_____

Mike Branson, City Forester

CITY OF CARMEL-BY-THE-SEA
FOREST AND BEACH COMMISSION

REGULAR MEETING MINUTES
Wednesday, June 25, 2014
Tour of Inspection 3:30 p.m.
Special Meeting 4:30 p.m.

I. CALL TO ORDER AND ROLL CALL FOR TOUR OF INSPECTION

PRESENT:	Commission Members Ferlito, Refuerzo, Bang, Carter
ABSENT:	Commission members: Boardman
STAFF PRESENT:	Mike Branson, City Forester
	Sharon Friedrichsen, Public Services Director

II. TOUR OF INSPECTION

The Commission toured the following site:

1. Carmel Beach at the 13th Ave. access point.

III. ROLL CALL FOR THE SPECIAL MEETING

The meeting was called to order at 4:30 p.m.

PRESENT:	Karen Ferlito
	David Refuerzo
	Michael Carter
	Kathy Bang
ABSENT:	Lisa Boardman, Chairperson
STAFF PRESENT:	Mike Branson, City Forester
	Sharon Friedrichsen, Public Services Director

IV. PLEDGE OF ALLEGIANCE

Members of the audience joined the Commission in the Pledge of Allegiance.

V. APPEARANCES

Barbara Livingston asked about new oak diseases and what homeowners can do to protect their trees. She also asked about a plan for removal of tree stumps and replacing them with new trees.

Neils Reimers thanked the Commission for taking on the beach issues.

Jason Wax urged the city to address oak tree issues and let citizens know what to do.

No other appearances.

VIII. ORDERS OF BUSINESS

1. Discuss the use of fire baskets or rings as an option for managing beach fires and provide recommendations related to the number and location of fires, fire baskets or rings that may be allowed at the Carmel Beach as part of a pilot program.

City Forester Branson presented a report on the items that were presented during the tour on Carmel Beach.

Public appearances opened at 4:40 p.m.

Barbara Livingston wanted to know what the Commissioners thought of the condition of the beach and if they looked from the bluffs to consider additional fencing to prevent cut-throughs.

Linda Anderson liked the ad hoc committee report and identifying that Carmel Beach has no limit on the number of beach fires. She thought that fire rings were a good idea and the Leverone baskets had implementation issues. She also noted the bluffs were taking a beating from firewood being thrown down them.

Jason Wax pointed out that a small minority causes much o the problems on the beach. He proposed banning fires since the cause litter and the fires are never cleaned up despite the City's efforts. If fires are allowed then perhaps gas fires should be used.

Mary Liskin agreed with baskets instead of fixed structures. She thought a program where the baskets are picked up at night would help. She also thought signage won't work and prevention is the best idea.

Kevin Urquart thought enforcement is needed and a small minority is the cause of most of the problems. He pointed out that there are many homes that also have chimneys along Scenic Road. He would like to continue to have fires but perhaps limiting the number.

Scott McKenzie identified himself as a systems engineer who has reviewed the beach fire issues. He presented a proposal for a commercial activity, managed by a non-profit, to address beach fires. He thought rings, signage, and fences are a blight. His proposal can resolve the fire issues except is currently not allowed on the beach. He urged further discussion of his idea by the City.

Margaret Eaton pointed out that smoke from fires is another problem. She favors a program where only smokeless or low smoke fuels are allowed. She also prefers the 100' spacing of fire rings and asked for demonstrations of alternative fuels in the future.

She also asked for a survey of other beaches that allow fires to see what they have done to address fire management.

Skip Lloyd thanked the Commission for going down on the beach. He pointed out that the white sands are an asset and should be the standard. He thought that unlimited fires lead to other problems and that beach use has increased and problems are multiplying. He pointed out that Scenic Road homes should be concerned about the smoke and blight of unlimited fires.

Richard Pepe also thinks that a20% of the people are causing the problems along the beach. He pointed out a recent poll by the Monterey Herald that 75% of respondents support less regulation of beach fires. He would like to see a mock up of the fire ring proposal.

Sandra Pepe pointed out there is a long history of beach fires. She also said that the beach gets lots of use but it is a fun place for planned and spontaneous family activities that have little cost.

Hugo Ferlito said that 10-15 years ago he would have resisted changes to the beach rules. He thinks the beach is being loved to death and that current levels of use are not sustainable. He favors limits on the number or areas for fires. He supports increasing cleanup, education, and enforcement.

Public appearances closed at 5:00 p.m.

Commissioner Bang reviewed her research on how other beaches manage fires. She proposed that no fires should be allowed directly on the sand and there should be a limit to the number of fires. She also thought that other ideas such as permits and smokeless fuel deserve exploration.

After further discussion on beach fire issues, Commissioner BANG made a motion to ask the City Council to approve the principle of no fires in the sand and to limit the number of fires, and to remand back to the Forest and Beach Commission and Planning Commission how to implement these two principles; seconded by FERLITO.

The motion carried by the following vote:

AYES:	Ferlito, Refuerzo, Bang, Carter
NOES:	None
ABSENT:	Boardman
ABSTAIN:	None

X. ADJOURNMENT

There being no further business, Vice-chairperson REFUERZO adjourned the meeting at 5:38 p.m.

Approved,

_____

Lisa Boardman, Chairperson

Respectfully submitted,

_____

Mike Branson, City Forester

MEMORANDUM

TO: Lisa Boardman, Chairperson
Members of the Forest and Beach Commission

FROM: Mike Branson, City Forester

DATE: 3 July 2014

SUBJECT: Revised Meeting Calendar for 2014

Staff has drafted a revised regular meeting calendar to reflect the change in meeting date to the second Thursday of each month.

2014 REVISED FOREST AND BEACH COMMISSION REGULAR MEETING CALENDAR:

January 2nd - recommend moving to the 9th **(APPROVED MOVE TO 9TH , 3-0)**
February 6th
March 6th
April 3rd
May 1st
June 5th
~~July 3rd~~ July 10th
~~August 7th~~ August 14th
~~September 4th~~ September 11th
~~October 2nd~~ October 9th
~~November 6th~~ November 13th
December 4th December 11th

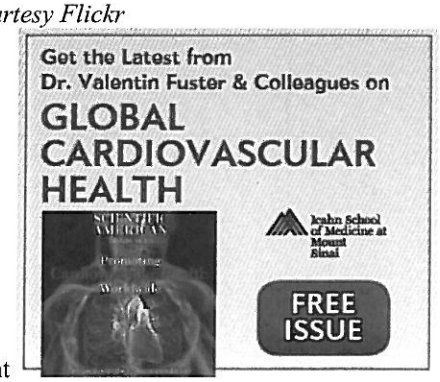
If you are unable to attend any of the scheduled meetings, please advise the City Forester well in advance of your absence.

ADVERTISEMENT

The Environmental Dangers of Backyard Fire Pits

These popular landscape features may have a global impact
Oct 3, 2008

Saffanna, courtesy Flickr



Advertisement

Dear EarthTalk: Backyard fire pits have become the latest must-have gardening feature. How bad are they on the environment?

-- Michael O'Laughlin, Tigard, OR

12

With Fall setting in and the mercury starting to drop, many of us want to extend our time outdoors, and sitting around a backyard fire pit has become one of the most popular means to do so. But even though it may be fun—s'mores anyone?—it is not good for the environment, especially during times when air quality is already poor.

It's hard to assess the larger impact of backyard fire pits on local or regional air quality, but no one questions the fact that breathing in wood smoke can be irritating if not downright harmful. According to the U.S. Environmental Protection Agency (EPA), so-called fine particles (also called particulate matter) are the most dangerous components of wood smoke from a health perspective, as they "can get into your eyes and respiratory system, where they can cause health problems such as burning eyes, runny nose and illnesses such as bronchitis."

Fine particles also aggravate chronic heart and lung diseases, and have been linked to premature deaths in those already suffering from such afflictions. As such, the EPA advises that anyone with congestive heart failure, angina, chronic obstructive pulmonary disease, emphysema or asthma should steer clear of wood smoke in general. Children's exposure to wood smoke should also be limited, as their respiratory systems are still developing and they breathe more air (and air pollution) per pound of body weight than adults.

Geography and topography play a role in how harmful wood smoke can be on a community-wide level. People living in deep, steep-walled valleys where air tends to stagnate should be careful not to light backyard fires during smog alerts or other times when air quality is already poor. Lingering smoke can be an issue even in wide-open areas, especially in winter when temperature inversions limit the flow of air.

The Washington State Department of Ecology reports that about 10 percent of the wintertime air pollution statewide can be attributed to fine particles from wood smoke coming out of wood burning stoves. While a wood stove may be a necessary evil as a source of interior heat, there is no excuse for lighting up a backyard fire pit during times when you could be creating health issues for your neighbors.

Another potential risk to using a backyard fire pit is sparking a forest fire. Some communities that are surrounded by forestland voluntarily institute seasonal burn bans so that residents won't inadvertently start a forest fire while they are out enjoying their backyard fire pits. If you live in one of these areas, you probably already know it and would be well advised to follow the rules.

If you must light that backyard fire pit, take some precautions to limit your friends' and family's exposure to wood smoke. The Maine Bureau of Air Quality recommends using only seasoned firewood and burning it in a way that promotes complete combustion—small, hot fires are better than large smoldering ones—to minimize the amount of harmful smoke. The moral of the story: If you need to burn, burn responsibly.

CONTACTS: U.S. Environmental Protection Agency (EPA), www.epa.gov; Washington State Department of Ecology, www.ecy.wa.gov; Maine Bureau of Air Quality, www.maine.gov/dep/air/.

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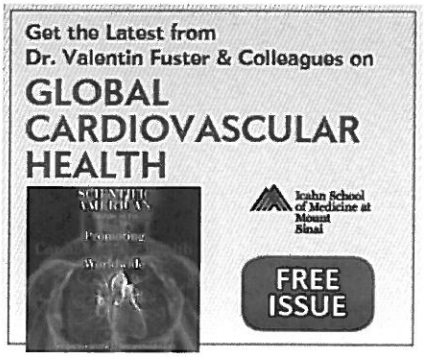

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Latest News

Mike Branson

From: kathleen bang [kathybang@mac.com]
Sent: Thursday, June 26, 2014 9:53 PM
To: Sharon Friedrichsen; Mike Branson
Subject: Fwd: laws against outdoor wood burning

For F and B

- Iowa's Supreme Court in 1998 declared that government bodies do not have the right to allow burning that results in smoke crossing property lines.
- The State of Washington has laws to address neighbors' wood smoke. According to the Puget Sound Clean Air Agency, "generating excessive smoke is not only unneighborly, it's illegal. Under state regulations, smoke from a person's chimney cannot exceed 20 percent opacity for six consecutive minutes. Greater smoke densities could result in fines from air pollution control officials. It is always illegal to smoke out your neighbor. Everyone has a right to breathe clean air. If smoke from your fire is affecting your neighbors, it is considered a nuisance and subject to enforcement action."
- Many states have restricted the use of wood burning in fireplaces and wood-burning stoves on certain high pollution days. Colorado, Utah, Albuquerque, New Mexico and many towns in California have set up pollution numbers to call to find out if you can burn wood.

(14)

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the dangers of campfire smoke

Friday, April 24, 2009

The Dangers of Campfire Smoke

It use to be we'd love to sit around a camp fire, in the great out doors. The bigger the fire, the better or so we thought.

But times have changed.

"Fresh air, and no stink" are you kidding me?

Yes, It's our dream to be in the great outdoors, breathing fresh air, and at night sitting out side with friends, and star gazing and probably enjoying a nice cold beer.

Have you really given much thought into what it is your breathing in, once you light that fire.

You try to not to breath it, but the wind shifts, and you get smoke in your eyes and throat. Cough ~ cough~ we grin, move our chairs yet again.. and have another drink, to clear our throats.

We've thrown in paper plates, napkins and Kleenex in there as well as news paper and boxes, etc. those are **not chemical free items**. and what goes in comes out into the air we breath and a good chance back into our lungs.

While trash will eventually disintegrate in the flame, they do release extremely **toxic vapors** when burned

The contents of that smoke are more dangerous than many people think.
According to Clean Air Revival Inc.'s Web site, the

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15

Environmental Protection Agency estimates that wood smoke is 12 times more **carcinogenic** than equal amounts of tobacco smoke — and that it stays active in the body up to 40 times longer than tobacco smoke.

Children appear to be at the greatest risk of health conditions such as acute bronchitis and respiratory infections, the **EPA** said.

Granted, it's your choice to fire smoke free..and risk it and your friends.. key word here is Choice..

Mind you If your a smoker, you probably don't care..

If your stranded somewhere, and need it for warmth you'll do it. mind you that's a slightly different situation.

But ,if your just camping, can you not do with out the fire !
Just sit with friends, enjoying the stars, and stories..
and of course your nice cold beer..

The stars will look brighter.. your lungs will thank you.
and as bonus your clothes won't reak of smoke..

If you find you just can't live without a campfire, just keep your camp fire small, and toss in NO garbage..

Remember the smaller the fire, the easier it is to control and the smaller amount of firewood you'll use..

~~*~~

The Dangers of wood smoke

Wood fires are popular, both for heating and for pure recreation. Many people thoroughly enjoy an evening around an old fashioned campfire. However, studies presented at

16

The Society of Toxicology's 1995 annual meeting held in Baltimore strongly suggested that exposure to the smoke associated with wood fires may be bad for one's health.

Wood smoke contains numerous toxic substances, including known carcinogens, such as polycyclic aromatic hydrocarbons, aldehydes, carbon monoxide, and tine organic particles. Based on epidemiological studies in children, wood smoke has implicated in increasing respiratory illnesses. Controlled studies on mice and rats have confirmed such associations. In one study, carried out by EPA (Enviroment Protection Agency scientists, a group of mice was exposed to wood smoke for six hours, a second group was exposed to the emissions from an oil furnace, and a third group (the control group) was not exposed to any type of smoke or emissions. All of the mice were then exposed to an air-borne bacteruim which causes respiratory infections. After sex weeks only 5% of the mice in the control group and in the group exposed to oil emissions had died of the infection, whereas 21% of the mice exposed to the wood smoke had died. Independent studies undertaken at New York University School of Medicine using rats exposed to wood smoke and respiratory pathogens (such as the bacterium staphylococcus aureus) showed similar results. Based on such data, the researchers are convinced of the potential health associated with breathing wood smoke.

From
Case studies in environmental science By Robert M. Schoch
you can view his site at :
http://books.google.ca/books?id=GcNVVA8od_UC&pg=PA123&lpg=PA123&dq=dangers+of+campfire+smoke&source=bl&ots=cyehT3LC6M&sig=dwQFcBD7JMiaOxbnAcA8sW1y_s8&hl=en&ei=MlnySaKmIpPoMJKLzMMP&sa=X&oi=book_result&ct=result&resnum=9#PPA123,M1

17

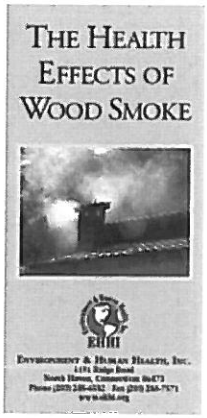
info@ehhi.org

Wood Smoke

The Health Effects of Wood Smoke

Health Issues | Exposure Issues | What Others are Doing | References

Health Issues



Download Brochure

- Although wood smoke conjures up fond memories of sitting by a cozy fire, it is important to know that the components of wood smoke and cigarette smoke are quite similar, and that many components of both are carcinogenic. Wood smoke contains fine particulate matter, carbon monoxide, formaldehyde, sulfur dioxide and various irritant gases such as nitrogen oxides that can scar the lungs. Wood smoke also contains chemicals known or suspected to be carcinogens, such as polycyclic aromatic hydrocarbons (PAHs) and dioxin.
- Wood smoke interferes with normal lung development in infants and children. It also increases children's risk of lower respiratory infections such as bronchitis and pneumonia.
- Wood smoke exposure can depress the immune system and damage the layer of cells in the lungs that protect and cleanse the airways.
- According to the Environmental Protection Agency (EPA), toxic air pollutants are components of wood smoke. Wood smoke can cause coughs, headaches, eye, and throat irritation in otherwise healthy people.
- For vulnerable populations, such as people with asthma, chronic respiratory disease and those with cardiovascular disease, wood smoke is particularly harmful— even short exposures can prove dangerous.
- The particles of wood smoke are extremely small and therefore are not filtered out by the nose or the upper respiratory system. Instead, these small particles end up deep in the lungs where they remain for months, causing structural damage and chemical changes. Wood smoke's carcinogenic chemicals adhere to these tiny particles, which enter deep into the lungs.
- Recent studies show that fine particles that go deep into the lungs increase the risk of heart attacks and strokes. EPA warns that for people with heart disease, short- term exposures have been

Wood Smoke

Wood Smoke Home

The Dangers to Health from Outdoor Wood Furnaces

Environment and Human Health, Inc.'s research shows outdoor wood furnaces are dangerous to people's health

Scientists and physicians say Outdoor Wood Furnaces are a serious health threat to those who live near them.

EHHI Testimony On An Act Adding Wood Smoke to the Public Health Nuisance Code and Concerning Outdoor Wood-burning Furnaces

The Health Effects of Wood Smoke

Outdoor Wood Smoke: Number of Complaints by Location

American Lung Association Position Paper on Wood Smoke

Connecticut Attorney General's position on Wood Smoke

EHHI asks the CT Legislature to help the many people in the state who are being made sick from breathing their neighbor's wood smoke

EHHI Op-ed on Wood Smoke

EHS Circular Letter # 2009 -32 - A letter from Connecticut Department of Public Health that

linked to heart attacks and arrhythmias. If you have heart disease, these tiny particles may cause you to experience chest pain, palpitations, shortness of breath, and fatigue.

discusses wood smoke as a public nuisance

Exposure Issues

- The particulate matter in wood smoke is so small that windows and doors cannot keep it out—even the newer energy-efficient weather-tight homes cannot keep out wood smoke.
- The EPA estimates that a single fireplace operating for an hour and burning 10 pounds of wood will generate 4,300 times more PAHs than 30 cigarettes. PAHs are carcinogenic.
- A study by the University of Washington in Seattle showed that 50 to 70 percent of the outdoor levels of wood smoke were entering homes that were not burning wood. EPA did a similar study in Boise, Idaho, with similar results.

What Others Are Doing

- Iowa's Supreme Court in 1998 declared that government bodies do not have the right to allow burning that results in smoke crossing property lines.
- The State of Washington has laws to address neighbors' wood smoke. According to the Puget Sound Clean Air Agency, "generating excessive smoke is not only unneighborly, it's illegal. Under state regulations, smoke from a person's chimney cannot exceed 20 percent opacity for six consecutive minutes. Greater smoke densities could result in fines from air pollution control officials. It is always illegal to smoke out your neighbor. Everyone has a right to breathe clean air. If smoke from your fire is affecting your neighbors, it is considered a nuisance and subject to enforcement action."
- Many states have restricted the use of wood burning in fireplaces and wood-burning stoves on certain high pollution days. Colorado, Utah, Albuquerque, New Mexico and many towns in California have set up pollution numbers to call to find out if you can burn wood.

What Needs to Be Done

There is much we can do to protect the public's health from wood smoke exposures. Fireplace and wood stove chimneys should be regulated so that they are high enough to protect neighbors from exposures. Individual towns should pass zoning regulations to protect public health. State legislatures and state departments of health should strengthen local health departments with specific wood smoke language so that they can deal on a case-by-case basis with situations in which people are made sick by their neighbors' smoke. As the State of Washington Clean Air Agency has stated: "It is always illegal to smoke out your neighbor."

(19)

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- San Francisco Bay Area Air Quality Management District | Santa Clara County Woodsmoke Rebate Program
- San Joaquin Valley Air Pollution Control District | Wood-burning Fireplace, Stove and Insert Rule | Today's Wood Burn Burn Status
- Yolo-Solano Air Quality Management District | Woodburning Appliances Rule

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20

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JAMA. 2002 Mar 6;287(9):1132-41.

Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution.

Pope CA 3rd[1], Burnett RT, Thun MJ, Calle EE, Krewski D, Ito K, Thurston GD.

Author information

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 500 000 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-microg/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,

(21)

respectively. Measures of coarse particle fraction and total suspended particles were not consistently associated with mortality.

CONCLUSION: Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

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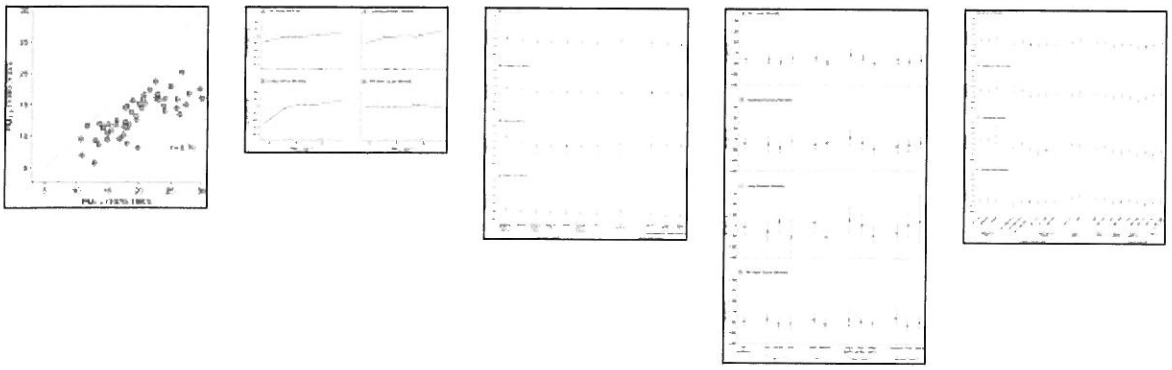

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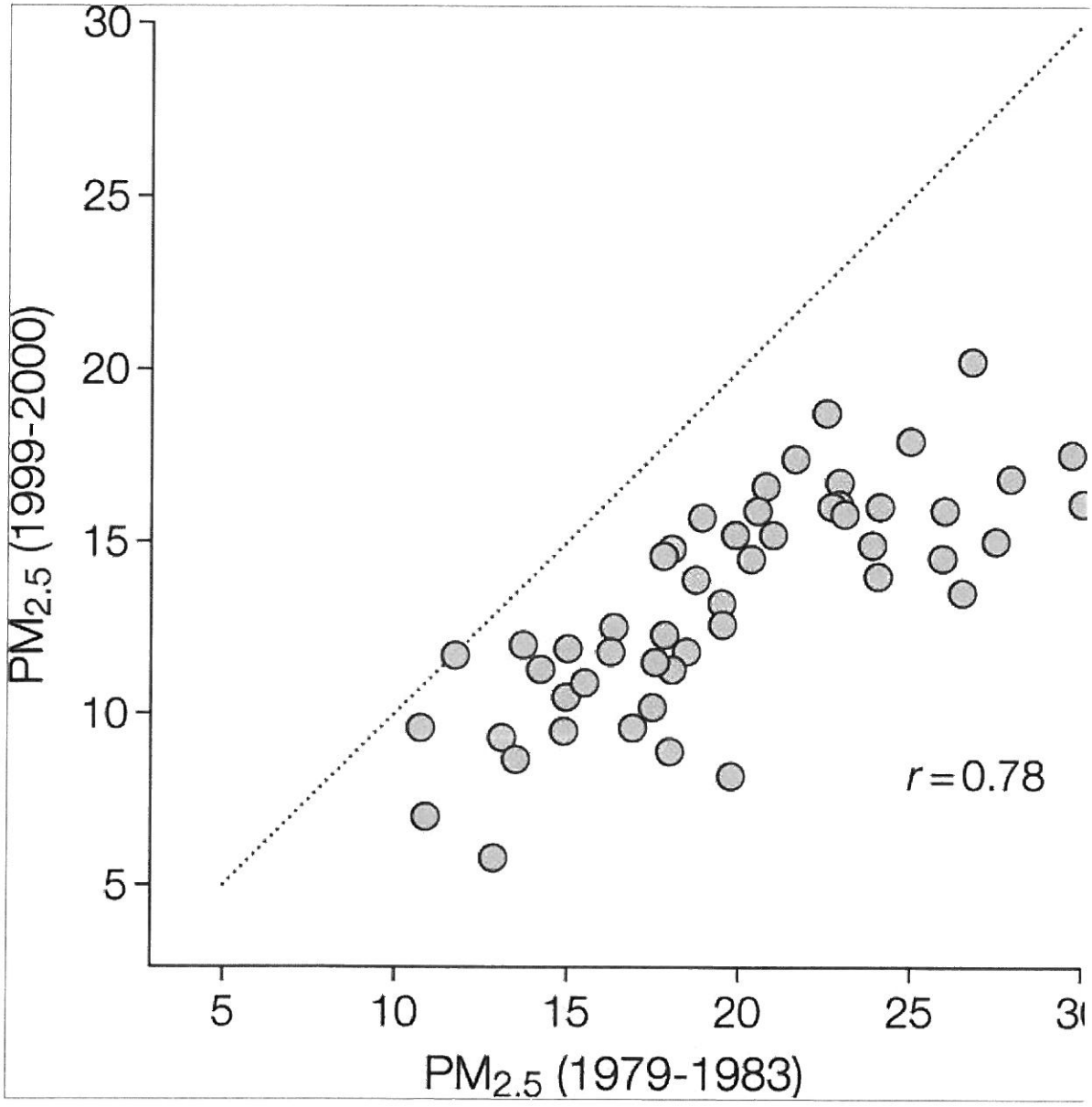
Figure 1



Mean Fine Particles Measuring Less Than 2.5 μm in Diameter ($PM_{2.5}$)

23

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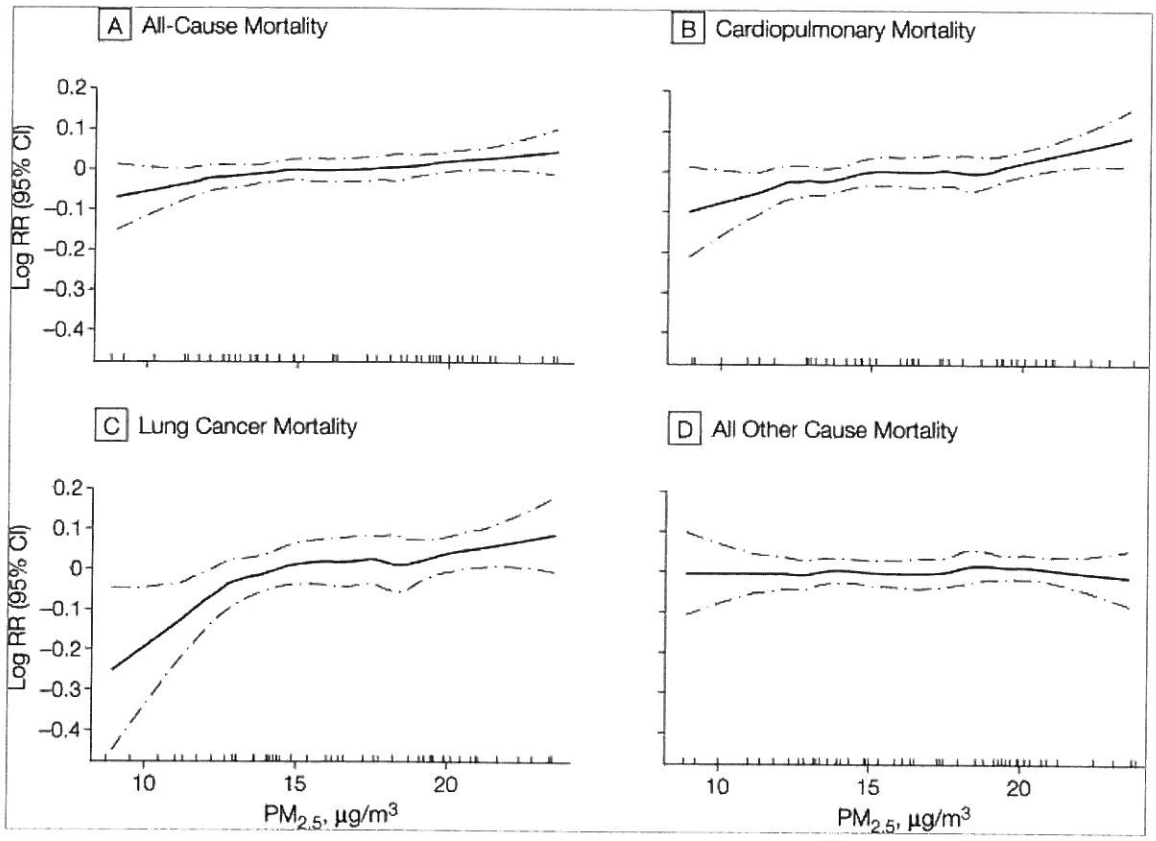
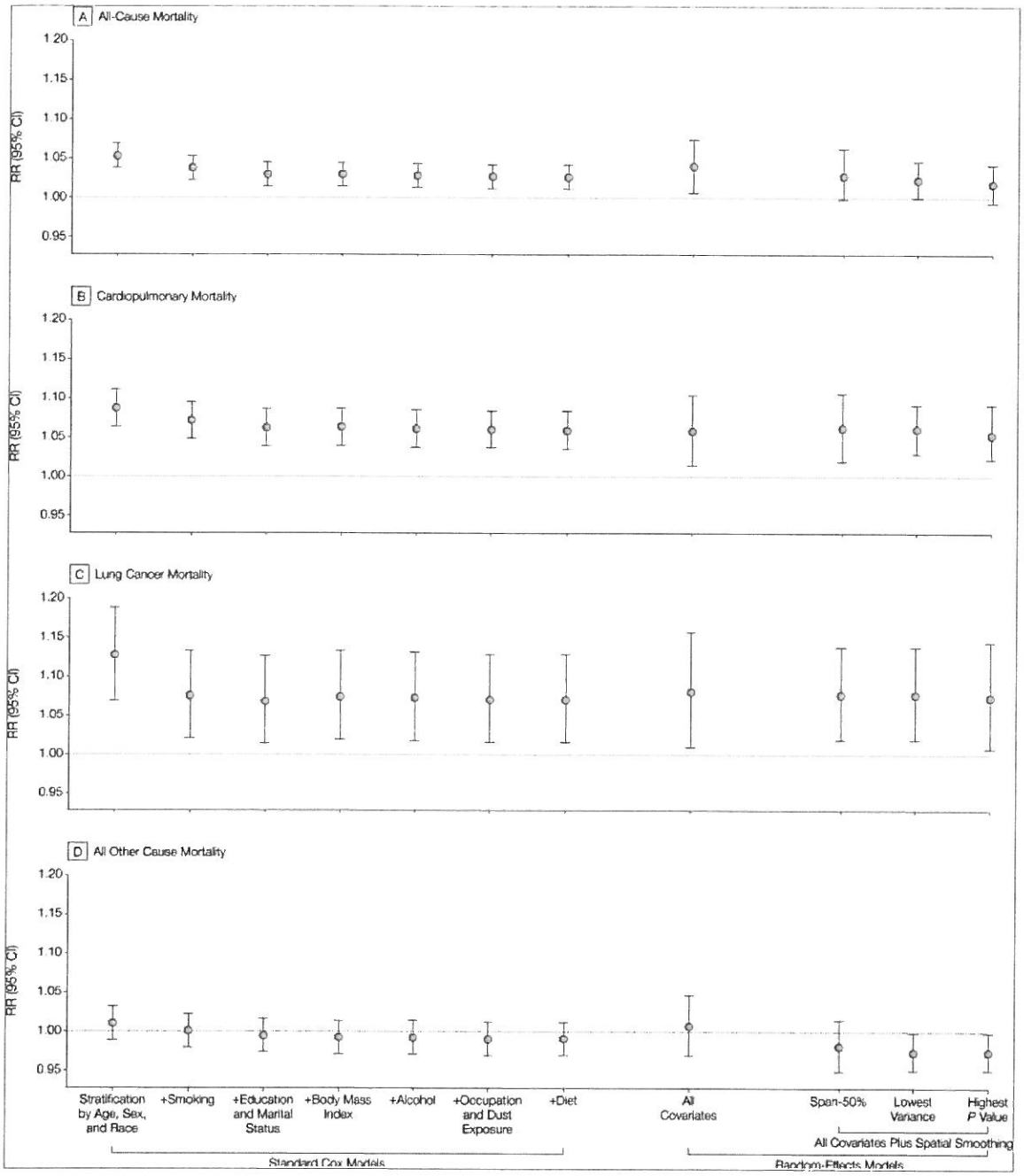
Figure 2



Nonparametric Smoothed Exposure Response Relationship

24

Figure 3



Mortality Relative Risk (RR) Ratio Associated With 10-μg/m^3 Differences of $PM_{2.5}$ Concentrations

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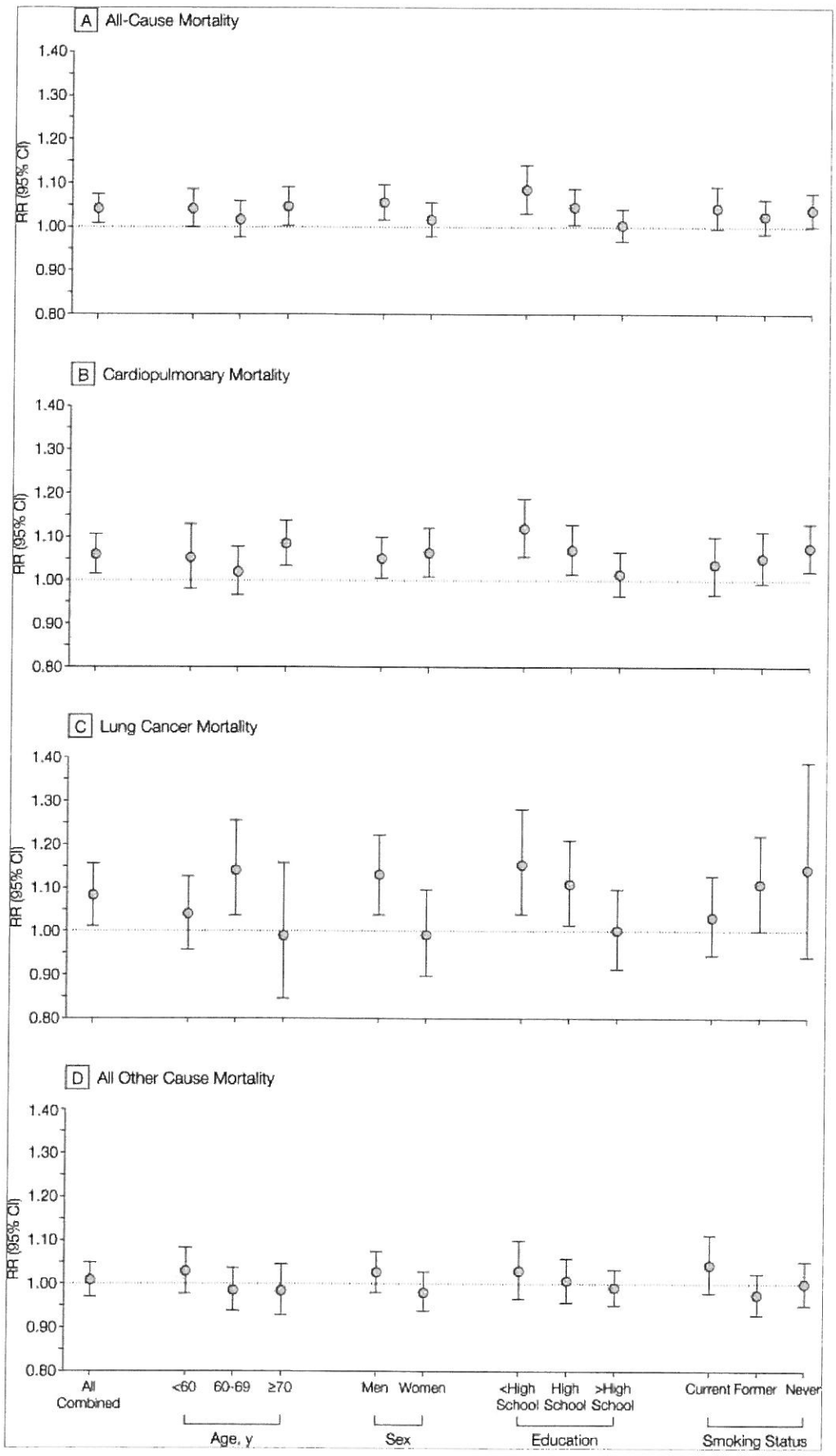
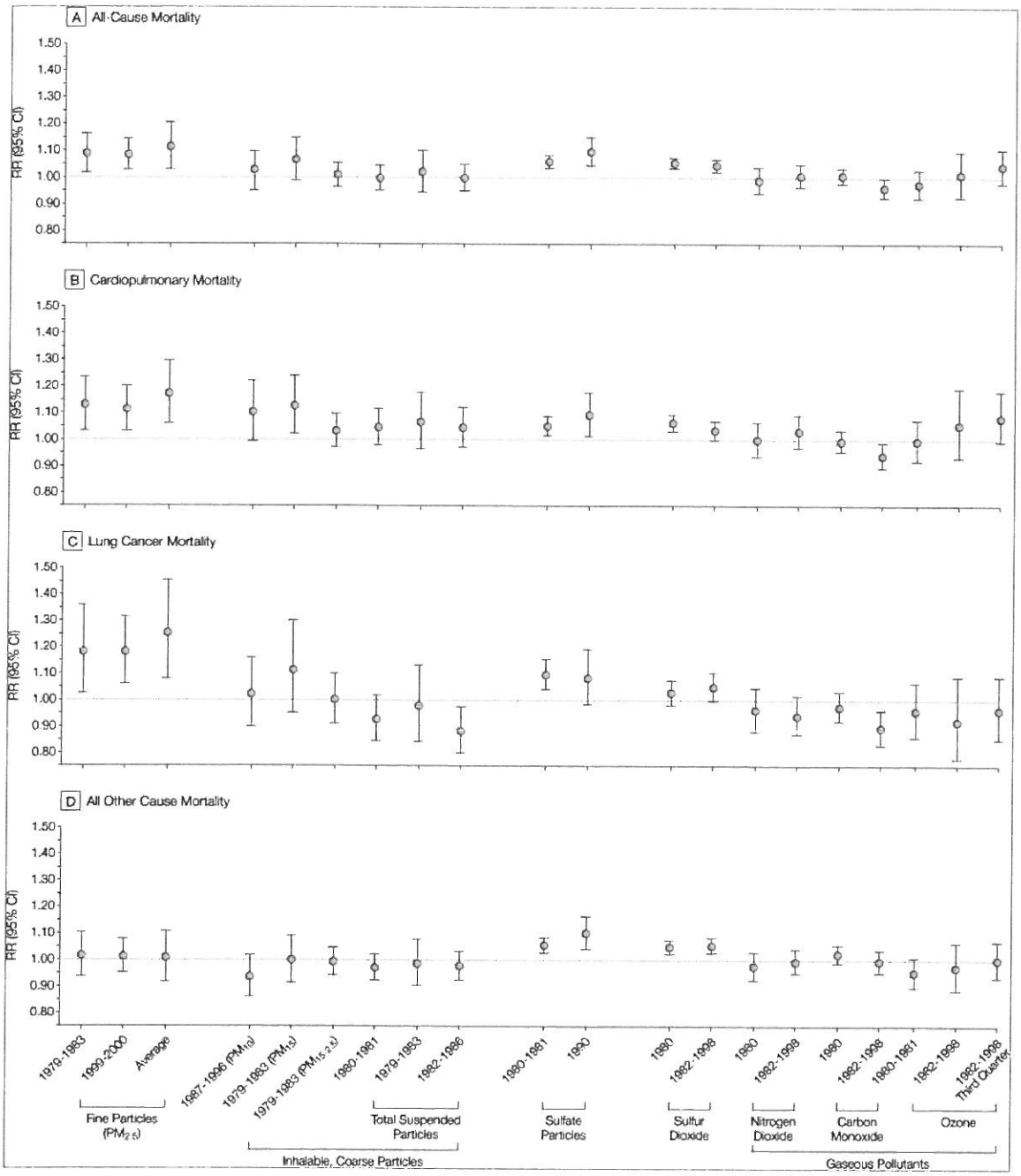

A All-Cause Mortality
B Cardiopulmonary Mortality
C Lung Cancer Mortality
D All Other Cause Mortality
RR (95% CI)
1.40
1.30
1.20
1.10
1.00
0.90
0.80
All Combined
<60
60-69
≥70
Age, y
Men
Women
Sex
<High School
High School
>High School
Education
Current
Former
Never
Smoking Status

26

Figure 5



Adjusted Mortality Relative Risk (RR) Ratio Evaluated at Subject-Weighted Mean Concentrations

27

Melissa Kaplan's

Chronic Neuroimmune Diseases

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Last updated January 1, 2014

Burning Wood is a Stinky Idea

Council may ban fireplaces, wood stoves

Citizen complaint raises issue of health problems caused by smoke

Mike McCoy, *The Press Democrat*, January 4, 2001

Traditional open-hearth fireplaces in new homes and remodels in Santa Rosa could be outlawed if the Santa Rosa City Council follows through with a proposal it is considering.

The council agreed to consider the legislation after a citizen complained about the unhealthy fireplace-generated smoke he said hovers over his Rincon Valley neighborhood, and earlier inquiries by Councilwoman Marsha Vas Dupre about whether fireplaces were allowed in new construction.

While council members agreed to move ahead Tuesday, Councilwoman Janet Condron said the proposal comes at an inopportune time, when people are seeking cheaper heating sources in the face of escalating energy bills.

"It's an interesting time to bring this forward," she said.

Mayor Mike Martini, who originally thought the idea would go nowhere, said he began to change his mind after recently learning about the inefficiency of the traditional fireplace and the health impacts of fireplace smoke.

"An open-face, old-fashioned fireplace is one of the worst sources of heat. It looks nice, but most of the heat goes up the chimney," said Martini.

And, according to the nine-county Bay Area Air Quality Management District, so does much of the smoke, ash and other particulates that

help make winter air hazardous to breathe.

Air Quality District spokesman Will Taylor said fireplaces and wood-burning stoves generate 4 tons of airborne particulates in Sonoma County on the average winter day, 10 times more than that generated by all the motorized vehicles in the county.

Taylor said Santa Rosa's airborne particulate count exceeded state standards six days during the winter of 1999.

Jenny Bard, spokeswoman for the Redwood Empire Chapter of the American Lung Association, said particulate matter is most worrisome for those with breathing problems and lung diseases.

"It is among the most harmful of all air pollutants. It gets inhaled deep into the lungs," she said.

Martini said the council likely will consider the ban in two to three weeks. "At this point, we need to gather all the information we can," he said.

So far, the cities of Petaluma and Cotati, as well as those areas under the jurisdiction of the North Sonoma Air Quality Management District -- Windsor, Cloverdale, Healdsburg and the west county -- have adopted some form of ban on wood-burning fireplaces and stoves.

The bans generally outlaw the installation of open-hearth, wood-burning fireplaces and wood stoves in new construction.

They do allow use of low-polluting wood stoves and inserts in fireplaces approved by the federal Environmental Protection Agency, as well as fireplaces fueled by natural gas.

Charlie Carson, executive director of the Home Builders Association of Northern California, said fireplaces are like mom and apple pie to homeowners.

"Nationally, a fireplace is among the top five amenities new home purchasers expect to see," he said.

That includes those living in the milder climates of California, Carson said.

"Even out here, while people don't need it for heat, they expect to see it. It literally adds a warmer touch to any home, whether the flame comes

from burning wood or burning gas," he said.

Carson said a quick survey of Santa Rosa builders indicates nearly all of the homes being built include a fireplace, mostly the wood-burning variety.

Martini said the council's focus likely will be to balance comfort and health.

"What we are saying is not that you can't have an alternative source of heat but that your alternate source of heat can't impact the rest of the community," he said.

Martini said the chances are minimal the council would enact a law that would retroactively affect current open-hearth fireplaces, but said it would have severe consequences for him if it was.

"It will knock out the romance in our home," he said.

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Letter to the Mayor and City Council:

January 5, 2001

The Honorable Michael Martini
Santa Rosa City Hall
P.O. Box 1678
Santa Rosa, CA 95402

Dear Mr. Mayor,

It is with interest that I read the article in yesterday's The Press Democrat about the City Council's considering the ban on wood burning stoves and open fireplaces in new construction and remodels (Council may ban fireplaces, wood stoves, January 4, 2001).

I used to enjoy the smell of a wood fire and the sight of glowing logs. Then, I developed an autoimmune disease that irrevocably altered my life. Among the many changes, I am now highly susceptible to respiratory tract inflammation due to wood smoke.

For the past several years, once the chilly days and nights of fall settle

in, just going back and forth from my front door to the sidewalk to get my mail results in my coughing for several minutes. I have to keep my windows closed all the time to keep out the smoke from my neighbors' fireplaces. On the coldest of days, or weekends when everyone is home, the smell of smoke is so strong inside my closed-up home that I move restlessly from room to room to make sure nothing in my house is on fire. At times I actually have to wear a filtration mask inside just to be able to breathe.

It is bad enough that I usually have to wear a mask when I go out into public because of the fragranced products people wear. I should not have to wear a mask just to be able to breathe safely in my home or in my own backyard.

Along with banning fireplaces and wood-burning stoves in new construction and remodels, why not institute an incentive program to upgrade to less polluting devices for people who currently have highly polluting wood-burning sources. Without such a program, there will be no incentive for people who are currently responsible for the smoke pollution to make any changes, and so there will effectively be no change in the existing air quality problems associated with such point sources of pollution. This will mean that those of us who currently suffer health problems related to the smoke pollution will continue to be sicker during the cold weather and the city and county will continue to have problems meeting the state air quality standards.

Finally, another thing to take into consideration is that what goes up, must come down. Particulate matter doesn't stay airborne forever. It not only settles as "dust" in our homes, but it settles on our rivers and streams causing pollution, and it falls on our crops and works its way into the soil in which they are grown. Just because wood is a plant doesn't make its particulates safe when it lands on organic and other farm soil and crops.

Thank you for your attention to this matter.

Melissa Kaplan

cc: City Council

Follow-up Letters to City Council

(31)

The Honorable Michael Martini
Santa Rosa City Hall
P.O. Box 1678
Santa Rosa, CA 95402

Dear Mayor Martini and Members of the City Council,

I previously wrote to you in January of last year about the health problems many Santa Rosa residents experience every fall, winter and spring as a result of wood being burned in fireplaces and woodburning devices that do not adequately remove particulate pollutants from the vented smoke and fumes.

In November, the council's appointed committee presented its recommendations that you then ordered to be drafted into an ordinance. From what I have read of the recommendations and proposed ordinance, the city council members' discussion and public comments by interested business parties, the council seems to be losing sight of some key issues:

1. Air pollution caused by existing woodburning fireplaces and appliances has already caused new health problems in previously healthy individuals, and continues to exacerbate existing health problems or create new ones for people already suffering from respiratory and heart disease.

2. Efforts need to be made to prevent further increases in air pollution not only by regulating the types of appliances that may be placed in new construction but by mandating such devices be placed in homes that are being remodeled.

3. Air pollution created by Santa Rosa residents doesn't stay within the city boundaries of Santa Rosa. Pollution crosses city and county borders, and so efforts also need to be made to work with other cities and county agencies to not only prevent increases over the current level of woodburning-related pollution, but to reduce the existing level of such pollution.

Our county is largely agricultural, with vineyards and farms and a variety of farm animals, some raised for food, others for pets or use in tourist-related businesses. It would be more than foolish to think that the existing high level of smoke-related particulate matter is not affecting crops and animals outside our city limits.

(32)

You have both read letters and articles, and heard testimony about woodburning pollution and its now being linked with increases in the onset of child and adult asthma. You have heard from residents such as myself being harmed by the smoke, not only when we venture outside, but the smoke invades our home, getting in through vents and closed windows. It is bad enough being unable to participate in any outdoor activities during the peak woodburning season, but we have been for over a year now unable to escape the smoke in our own homes.

In my public statement to the Council last year, I mentioned a fact I found startling when I came across it when writing a research paper on air pollution in 1991: it takes only 24 hours for pesticides sprayed on Georgia cotton crops to land on Isle Royale in the northern end of Lake Superior. Those of us affected by woodsmoke are not just being affected by our immediate neighbor's wood fire, but by all wood fires upwind of us.

If that doesn't give those of you who have voted to weaken the proposed ordinance, and who gave scant attention to those citizens whose quality of life has already been harmed by woodburning pollution, perhaps you will be interested in the following two studies recently published:

1. Lung Cancer, Cardiopulmonary Mortality, and Long-term Exposure to Fine Particulate Air Pollution, recently published in the Journal of the American Medical Association:

C. Arden Pope III, PhD; Richard T. Burnett, PhD; Michael J. Thun, MD; Eugenia E. Calle, PhD; Daniel Krewski, PhD; Kazuhiko Ito, PhD; George D. Thurston, ScD. Department of Economics, Brigham Young University, 142 FOB, Provo, UT 84602. cap3@email.byu.edu. JAMA. 2002;287:1132-1141

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.

33

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 500 000 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/m3 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. Conclusion: Long-term exposure to combustion-related fine particulate air pollution is an important environmental risk factor for cardiopulmonary and lung cancer mortality.

2. Use of an index to reflect the aggregate burden of long-term exposure to criteria air pollutants in the United States.
Kyle AD, Woodruff TJ, Buffler PA, Davis DL. School of Public Health, University of California Berkeley, Berkeley, California, USA. Environ Health Perspect 2002 Jan;110 Suppl 1:95-102

Air pollution control in the United States for five common pollutants--particulate matter, ground-level ozone, sulfur dioxide, nitrogen dioxide, and carbon monoxide--is based partly on the attainment of ambient air quality standards that represent a level of air pollution regarded as safe. Regulatory and health agencies often focus on whether standards for short periods are attained; the number of days that standards are exceeded is used to track progress. Efforts to explain air pollution to the public often incorporate an air quality index that represents daily concentrations of pollutants. While effects of short-term exposures have been emphasized, research shows that long-term exposures to lower concentrations of air pollutants can also result in adverse health effects. We developed an aggregate index that represents long-term exposure to these pollutants, using 1995 monitoring data for metropolitan areas obtained from the U.S. Environmental Protection Agency's Aerometric Information Retrieval System. We compared the ranking of metropolitan areas under the proposed aggregate index with the ranking of areas by the number of days that short-term standards were exceeded. The geographic areas with the highest burden of long-term exposures are not, in all cases, the same as those with the most days that exceeded a

34

short-term standard. We believe that an aggregate index of long-term air pollution offers an informative addition to the principal approaches currently used to describe air pollution exposures; further work on an aggregate index representing long-term exposure to air pollutants is warranted.

I thank you for allowing me to once again take your time with this matter. If you have any questions, please feel free to contact me.

Melissa Kaplan
When one tugs at a single thing in nature, he finds it attached to the rest of the world. - John Muir

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Related Articles

American Lung Association Fact Sheet: Why is Wood Burning and Air Pollution Problem?

ALA® Fact Sheet: Particulate Matter Air Pollution

Woodburning (ALA)

Proposed EPA Air Pollution Index Sends Mixed Message About Health Hazards

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Why is wood burning an air pollution problem?

County limits new rural fireplaces

http://www.anapsid.org/cnd/mcs/fireban.html

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35

AMERICAN LUNG ASSOCIATION.
Fighting for Air

American Lung Association Says Wood Smoke Pollution Hazardous to Health

As cooler temperatures begin to mark the beginning of fall, the American Lung Association of California is urging the public to avoid wood burning and to consider cleaner burning alternatives. Burning wood emits harmful toxins and fine particles in the air that can worsen breathing problems and lead to heart and lung disease and even early death.

"Breathing particle pollution – or soot – can literally shorten life and send our most vulnerable residents to the emergency room. Study after study shows that when particle pollution levels increase, people can die," said David Pepper, MD, a volunteer of the American Lung Association in California.

Wood smoke poses a special threat to people with asthma and COPD (chronic obstructive pulmonary disease) and should be actively avoided by those with lung disease. The American Lung Association strongly recommends using cleaner, less toxic sources of heat, such as natural gas. Converting a wood-burning fireplace or stove to use either natural gas or propane will eliminate exposure to the dangerous toxins wood burning generates including dioxin, arsenic and formaldehyde.

Wood smoke is the largest source of wintertime particle pollution in the Bay Area. Numerous studies have linked wood smoke exposure with exacerbation of lung disease. In the greater Bay Area, nearly one million residents have lung disease. "When they breathe wood smoke, they suffer even more," said Dr. Pepper.

As with any pollution, children are especially at risk. Their lungs do not fully form until the age of 18 and cumulative exposures of wood smoke can lead to reduced lung function and risk of future disease. For asthmatic children, breathing wood smoke can lead to immediate harm, including asthma attacks and respiratory distress.

Although both natural gas and propane stoves are much cleaner than their wood-burning alternatives, these devices must be directly vented outside the home to reduce exposure to carbon monoxide, nitrogen oxide and other emissions produced by these energy sources. The American Lung Association warns that gas and propane stoves can be a threat to any family's health without proper outdoor ventilation.

Indoor exposures to wood smoke can also pose health risks to residents. "Studies have also shown that people using wood burning devices to heat their homes can be routinely exposed to excessive levels of fine particulate matter in their indoor air," added Dr. Pepper.

Many air districts in California have adopted regulations to control wood burning. The Bay Area Air Quality Management District's (BAAQMD) wood burning regulation will be in effect in all nine Bay Area counties beginning November 1. The Air District will call a winter Spare the Air Alert making wood burning illegal until the alert is lifted. Individuals can make a complaint

about wood smoke pollution by calling the air district's hotline at 1-877-4NO-BURN or sign up to receive air alerts on the District's website at www.spareheair.org

When a wood burning curtailment is called, the regulation bans the use of all wood burning appliances, including US EPA certified stoves. Currently, a US EPA Phase 2 certified device is any device built on or after July 1, 1992. While new US EPA Phase 2 certified devices burn much more cleanly, older ones may not. A study conducted by the US EPA found that Phase 2 Certified devices can emit significant levels of pollution above certified values, due to improper operation and maintenance, and through normal equipment degradation.

"Wood smoke pollution is a huge problem in the wintertime, both at the neighborhood and regional level," Dr. Pepper said. "So many people are suffering health effects from breathing smoke in their homes and neighborhoods. Every single chimney and wood-burning stove can have an impact on air quality so we are urging the public to burn less."

For more information about the health effects of wood burning and cleaner burning alternatives to heating, please visit our website at www.lungusa.org or call 1-800-LUNG-USA.

Smoke from wood fireplaces, stoves raises new health concerns

Story by Cheryl Katz
Environmental Health News

Photos by Michael Macor, San Francisco Chronicle

March 14, 2011

Peter Lehmkuhl, general manager of the Sierra Club's Clair Tappaan Lodge, high in the Sierra Nevada, tends to the fireplace.

NORDEN, Calif. - On a frosty evening in the Sierra Nevada, smoke curling from the chimney of the Clair Tappaan Lodge is a welcome sight to chilly snowshoers and cross-country skiers. Gathering by the massive stone hearth at this landmark Sierra Club mountain hostel, guests relax in the warmth and aroma of the crackling log fire.

Those same woodsy scents waft across the wintry north, as millions of fireplaces and wood stoves are lit by people seeking an environmentally friendly source of heat and ambience. But recent research raises new concerns over the toxic substances borne aloft in wood smoke.

The tiny airborne specks of pollution known as particulate matter, or PM, produced by wood-burning stoves appear to be especially harmful to human health. Small enough to penetrate deep into the lungs, they carry high levels of chemicals linked to cardiopulmonary diseases and cancer, and they can damage DNA and activate genes in hazardous ways comparable to cigarette smoke and car exhaust.

"We found that wood smoke PM has similar toxicity and effects on DNA as that of vehicle exhaust particles," said University of Copenhagen researcher Steffen Loft, who led a new study of air pollution from wood stoves.

Another new study, conducted in Canada, found that infants and toddlers living in areas with a lot of wood stoves and fireplaces were significantly more likely to get ear infections, one of the leading causes of childhood trips to the doctor.

Early humans began building wood fires hundreds of thousands of years ago, providing protection from predators, expanding sources of food and allowing migration to colder climates. Because wood is a "natural" material and has

been an integral part of human existence for so long, many view it as a benign, cheap and renewable energy alternative.

"It's the cave man's television," said John Walsh, an engineer who heats his 3,000-square-foot home with a wood stove during the brisk winters in Bozeman, Mont., describing how the graceful gyre of flames has enthralled people through the ages.

Small enough to penetrate deep into lungs, particulates in wood smoke contain chemicals capable of damaging DNA and activating genes in ways comparable to cigarettes and car exhaust.

Walsh, who burns mostly lodgepole pines killed by pine beetles, enjoys the exercise of cutting and splitting the logs, as well as saving about $2,000 in energy bills a year. In addition, "wood heat is carbon neutral," he said, because "burning it releases the same amount of carbon as having it decay."

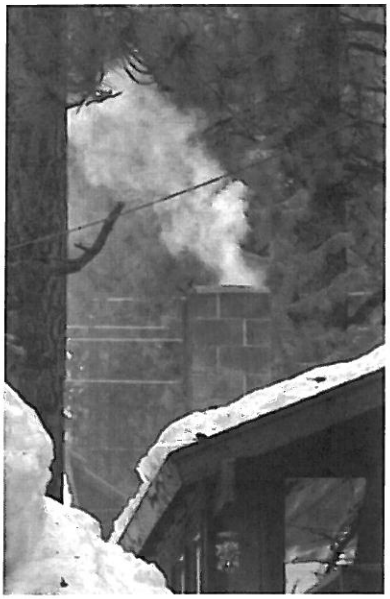
Wood-burning fits in with a rustic ethic. In Northern California's nine-county Bay Area Air Quality Management District, the most frequent violations of the region's fireplace and wood-stove restrictions tend to come from bucolic Sonoma County, home to vineyards, ranches and farms.

"These are places that are somewhat rural," said Bay Area AQMD spokesperson Aaron Richardson, "and there does tend to be a kind of a culture of relying on wood for additional heating needs."

However, that woodsy "link to the land" is also linked to potentially serious health risks. Vented outdoors, the smoke can pose a bigger threat to people in the community than to those sitting fireside.

Exposure to the particulates in smoke irritates the lungs and air passages, causing swelling that obstructs breathing. Wood smoke can worsen asthma, and is especially harmful to children and older people. It also has been linked to respiratory infections, adverse changes to the immune system, and early deaths among people with cardiovascular or lung problems.

"We know there's a lot of bad stuff released when wood is burned," said Dr. John Balmes, a professor of medicine at the University of California, San Francisco, and professor of environmental science at the University of California, Berkeley School of Public Health. "It's actually not that far away from tobacco smoke and smoke from fossil fuel combustion engines. They're in the same ball park."



Staff at the Clair Tapaan lodge use dried wood and small pieces in an effort to reduce smoke.

Until recently, little was known about the specific harmful agents in wood smoke. While the components of particulate matter produced by vehicles and tobacco have been studied fairly extensively, the University of Copenhagen project is one of the first to characterize the minute particles and droplets of pollution released by wood fires.

The researchers analyzed the particulate matter in air samples from a Danish village where most homes were heated by wood-burning stoves, and compared it to background particles in air outside the smoke area. They also sampled emissions at the stoves' flues.

The results, published in the January issue of Chemical Research in Toxicology, determined that wood smoke was more likely to comprise extremely small "fine" and "ultrafine" particles – PM 2.5 (.0001 inch) or less, which can lodge deep in the lungs or even pass into the bloodstream. The wood smoke particles had higher levels of polycyclic aromatic hydrocarbons, including some of the same ones found in tobacco smoke and classified by the U.S. Environmental Protection Agency as "probable human carcinogens."

When added to cultures of human lung cells, the wood smoke particulates induced large amounts of reactive oxygen species – powerful oxidants able to injure cells, damage DNA and trigger oxidative stress. Although they are a normal product of metabolism, overexposure can set off an inflammatory reaction that interferes with the body's ability to fight infection and can spread to other parts of the body.

39

The Danish researchers found that wood smoke particulates were more powerful than the background air particles at damaging DNA, causing potentially cancerous changes, and activating genes linked to inflammation and oxidative stress, which is a possible mechanism for atherosclerosis, asthma and other diseases.

The new findings provide important information on the toxic agents in wood smoke, Balmes said. In addition to the fine particles, wood smoke contains gases such as nitrogen oxides, and carcinogenic compounds like benzene, formaldehyde and dioxins.

Identifying the specific harmful components in particulate matter is key to understanding how to protect human health, said Doug Brugge, a Tufts Medical School professor who studies the effects of particles produced by traffic.

"The evidence is pretty clear that fine and ultrafines get into the lungs much better," he said, describing the particles as so tiny that as many as 100,000 packed together would be the size of a sugar cube. The tiniest – ultrafine particles with diameters of 100 nanometers (.000004 inch) or less – appear to be especially toxic.

"There is growing evidence that the ultrafines are able to pass through the lungs and get into other parts of the body," Brugge said. Because they are light, they may be able to travel greater distances.

This may be the case with the otitis media – a painful middle ear infection that is responsible for many a midnight trip to the emergency room with a screaming baby, and source of the greatest number of antibiotic prescriptions for children under five.

> **"We know there's a lot of bad stuff released when wood is burned. It's actually not that far away from tobacco smoke and smoke from fossil fuel combustion engines. They're in the same ball park."**
>
> ***Dr. John Balmes, University of California, San Francisco***

A study conducted at the University of British Columbia and reported in the January issue of Epidemiology analyzed visits to the doctor for 45,000 children aged two and under in Vancouver and surrounding areas, comparing it to data on wood smoke pollution levels during the same period. The study is the first to show a connection between ear infections and neighborhood wood stoves and fireplaces.

Children with the highest exposures to wood smoke "were 32 percent more likely to visit the doctor for otitis media" compared to children with the lowest exposures, said lead author Elaina MacIntyre, a scientist at the University of British Columbia's School of Environmental Health.

MacIntyre cautioned that the association may be due to some other factor not accounted for in the study, but said the correlation between wood smoke exposure and otitis media was as strong as the well-known link between tobacco smoke and this disease. The study proposes that substances in the smoke suppress children's ability to fight off common upper-respiratory tract viruses and bacteria, which then migrate to the ear, causing infections.

Overall, few numbers are available on the amount of disease and deaths attributed to wood smoke pollution, say Loft and other researchers.

"In the Third World, more than a million women and children die annually… due to massive exposure [from cooking on indoor] open wood fires," Loft pointed out. "However, that cannot be translated to use of wood stoves in the developed world. We lack proper population-based data to estimate the risks of that like we can for traffic emissions."

Data on fine particulates from all sources show a considerable health toll. In California, the state Air Resources Board estimates that they contribute to about 9,200 premature deaths from cardiopulmonary disease every year. Wood smoke is a major component of fine particulates in many areas, especially in winter. And the smoke going up the chimney can find its way back inside: more than 70 percent of indoor particulate concentrations come from sources outside the house, a University of Washington study shows.

Lehmkuhl reaches for kindling at the Sierra Club lodge.

Many municipalities, such as Denver, Albuquerque and the San Francisco Bay Area, have taken steps to clean up wood smoke. In the Bay Area for instance, home to an estimated 1.7 million fireplaces and wood stoves, burning wood is prohibited on winter "Spare the Air" days when air

(10)

quality is forecast to be poor, and all new wood-burning devices sold or installed in new or remodeled homes must meet EPA emission standards.

Southern California, which has some of the worst fine particle pollution in the nation, will begin banning fires on poor-air-quality days next winter. The South Coast Air Quality Management District estimates that winter residential wood-burning in the region belches out 10 tons of fine particulates per day. Other cities, such as Fairbanks, Alaska and Tacoma, Wash., offer cash incentives for residents to replace their old wood-burning appliances with new, less-polluting ones.

"Parents should be aware that wood smoke is an important risk factor in the development of childhood respiratory infections."

Elaina MacIntyre, University of British Columbia

In one of the most successful programs, nearly all the wood stoves in Libby, Mont., were replaced with new models that burned as much as 15 times cleaner. The fine particulate levels in this mountain town, which has a long tradition of heating with wood, dropped an average of 20 percent, and residents reported fewer cases of respiratory infections, bronchitis and children's wheezing, according to recent studies by University of Montana.

So is it safe to cozy up to that inviting hearth at the mountain lodge or your own little fireplace or woodstove at home?

"If you don't have asthma I think it's okay," said Balmes. "If it's an efficient fireplace with good updraft, it doesn't expose the people sitting near the fireplace. Rather, "it's the people downwind" who are exposed, he said.

MacIntyre cautioned that families with young children in wood-burning areas should keep the windows closed and use a HEPA air filter.

"Parents should be aware that wood smoke is an important risk factor in the development of childhood respiratory infections and that wood-burning increases the risk of these infections, not only for their own children, but also for children in their neighborhood," she said. "If parents choose to continue using their fireplace or wood stove it is important that they ensure proper maintenance and consider upgrading to cleaner technologies wherever possible."

According to Walsh, who uses a high-efficiency, EPA-approved, tightly sealed wood stove in his Bozeman home, "If your stove is designed properly and you have a proper chimney, you don't really have a lot of reason to be concerned about smoke."

At the Sierra Club's Clair Tappaan lodge, guests use the fireplace almost every night. The wood is dried for a season or two and cut into small pieces to keep the smoke to a minimum, said general manager Peter Lehmkuhl.

"There's just something about a fire," he said. "It's very meditative to sit there and think about your day and stare at that fire and reflect on where you are. Dealing with a very primal element there, the fire."

For more information on cleaner-burning fireplaces and wood stoves, go to http://www.epa.gov/burnwise/

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(41)

Health Effects Of WoodSmoke

Updated March 1997

Health Effects of Wood Smoke

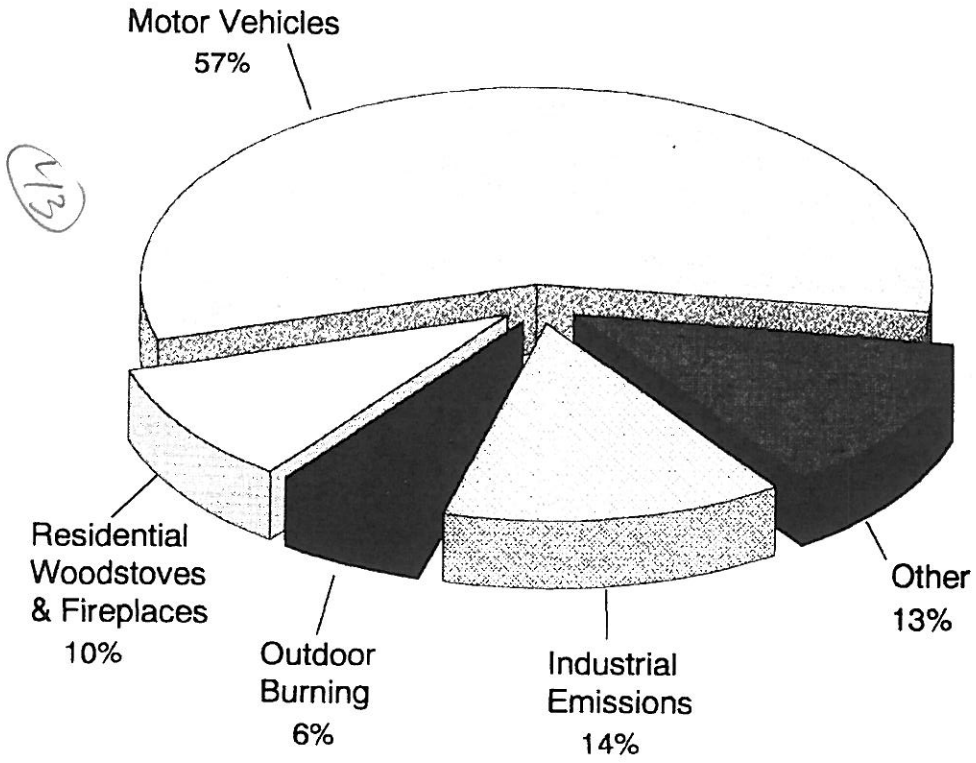
INTRODUCTION

The smell of wood smoke evokes fond memories for many people, but for others it has become a danger signal. Wood smoke, largely from wood stoves, has become a major part of the air pollution problem in the United States. Smoke is composed of many small particles of carbon compounds from the burning of organic matter such as wood or coal. These small pieces of Organic matter from smoke, along with dust and other small particles of solid and liquid matter suspended in the air, are called particulate matter. Particulate matter is regulated by the federal government as one of the principal air pollutants.

In Washington State wood stoves and fireplaces release 10 percent of the total air pollution (see pie chart).[13] Particulate pollution from wood stoves is produced mostly in the winter when stagnant air and temperature inversions limit air movement, "trapping" the pollution close to the ground and keeping it in our breathing space.

A growing body of evidence suggests that we cannot ignore the medical consequences of extensive exposure to wood smoke. This booklet presents some current air quality data, and information extracted from medical research on the health effects of household and neighborhood wood smoke.

AIR POLLUTION SOURCES AND THEIR CONTRIBUTIONS



The category "Other" in this pie chart includes sources such as boats and other recreational vehicles, lawn mowers, etc.

TABLE OF CONTENTS

Glossary

ACUTE. Having a quick start and relatively serious characteristics, as an acute illness.

AIR. A mixture of gases containing about 78 percent nitrogen, 21 percent oxygen, and less than one percent carbon dioxide and other nonreactive gasses, with varying amounts of water vapor.

AIRSHED. The air supply of a given area defined by natural or topographic features, as well as by political or legal boundaries.

ALVEOLI. The numerous tiny air sacs at the end of the bronchioles in the lungs, where exchange of oxygen and carbon dioxide takes place across membranes with the blood.

ARTERIOSCLEROSIS. A chronic disease where thickening or hardening of the artery walls interferes with blood circulation.

ATMOSPHERE. The layer of air surrounding the Earth.

BACKDRAFTING. A downward flow of air into a fireplace or wood stove when outside air enters the exhaust opening. Most common in airtight dwellings where this becomes the only available source of combustion air, or when chimneys develop a creosote buildup, or when wood stoves are not properly installed.

BENZENE. A toxic chemical found in coal tar and used as an industrial solvent, as a gasoline additive, and in some paints or varnishes; a known carcinogen.

BRONCHIOLES. Small thin-walled branches of the bronchus, which branch further and lead to the tiny air sacs within the lungs.

BRONCHITIS. Inflammation of the mucous membrane of the bronchial tubes.

BRONCHIOLITIS. Inflammation of the mucous membrane of the bronchioles.

BRONCHUS. Either of two primary branches of the trachea or other branches with cartilage in their walls, which lead to the bronchioles in the lungs.

CARBON MONOXIDE (CO). A colorless, odorless gas produced by incomplete burning of carbon containing substances, especially by gasoline-powered vehicles. It is one of the six major air pollutants for which there is a national air quality standard.

CARCINOGEN. A substance or activity that causes cancer.

CARCINOGENIC. Capable of causing cancer.

CARDIAC. Of, near, or relating to the heart.

CERTIFIED WOOD STOVE. Models certified as relatively clean burning by the U.S. Environmental Protection Agency based on carbon monoxide and particulate matter emission testing at accredited laboratories.

CHRONIC Marked by long duration or frequent recurrence, such as a chronic disease.

CILIA. Hairlike cells that line the passageways to the lungs. The motion of cilia propels tiny dirt particles and germs out of the respiratory tract.

COMBUSTION. The process of burning. More specifically, when a substance reacts with oxygen to produce heat and often light.

EMISSIONS. Waste substances or pollutants discharged into the air from sources such as automobiles, power plants, or wood stoves.

EMPHYSEMA. A swelling of the tiny air sacs or alveoli in the lungs resulting from destruction of the membranes which line the inside of the air sacs -- characterized by breathing difficulties and greater susceptibility to infections.

ENVIRONMENT. The aggregate of all outside conditions and influences affecting life, development, and survival of organisms.

ENVIRONMENTAL PROTECTION AGENCY (EPA). The independent federal government agency established in 1970 that regulates environmental matters and oversees environmental law enforcement.

HYDROCARBONS. A large family of compounds containing hydrogen and carbon. Can include many organic compounds in various combinations. Wood and fossil fuels are composed mainly of hydrocarbons. Many are air pollutants and some are carcinogenic.

INDOOR AIR POLLUTION. Air pollutants which are released inside buildings at concentrations often higher than in outdoor air. Common indoor air pollutants include radon, tobacco smoke, formaldehyde, and wood smoke.

INVERSIONS. An atmospheric condition occurring when a cool layer of air gets trapped below a layer of warm air and is unable to rise. This "ceiling" leads to a buildup of polluted air close to the ground and prevents vertical mixing and dispersion of smoke and other air pollutants.

LIFETIME CANCER RISK. The probability that someone will get cancer from a specific source at some time in their life.

LOWER RESPIRATORY TRACT INFECTIONS (LRTIs). Infections deeper in the respiratory tract than the nose and throat, such as bronchiolitis or pneumonia which involve the bronchioles or lung.

MICRON. A unit of length 1/1,000,000 of a meter.

MORTALITY RATE. Death rate.

MUCOUS MEMBRANE. The membrane lining all body channels that are exposed to air, such as the respiratory tract or the digestive tract. The glands of this membrane secrete mucus.

MUCUS. Organic compounds secreted by the mucous membrane, along with cells and inorganic salts suspended in water. This mixture acts as a lubricant and a protective coating.

MUTAGEN. An agent that causes biological mutation.

MUTATION. A change in the genes or chromosomes within the cells of living organisms.

MUTAGENICITY. A measure of the relative strength of different mutagens based on their ability to cause mutations.

NITROGEN OXIDES. Compounds of nitrogen and oxygen formed from high temperature burning. They contribute to acid rain and ozone formation. Nitric oxide (NO) and nitrogen dioxide (NO2) contribute to lung and respiratory health problems. Nitrogen dioxide is the most toxic and is one of the six major air pollutants for which there is a national air quality standard.

ORGANIC. Concerning, related to, or derived from living organisms; in chemistry, a carbon containing compound.

OZONE (03). A form of oxygen consisting of three oxygen atoms per molecule. Ozone is a pungent, colorless, toxic gas that is a major component of smog. It is formed from nitrogen oxides and hydrocarbons in the presence of sunlight and heat. Ozone is one of six major air pollutants for which there is a national air quality standard.

PARTICULATE MATTER (PM). Small particles of solid and liquid matter found in the atmosphere including soot, dust, organic matter, smoke, or smog.

PM10. Particulate matter that measures ten microns in diameter or less. One of six major air pollutants for which there is a national air quality standard.

PNEUMONIA. An acute or chronic disease caused by bacteria, viruses, or particulate matter and chemical agents characterized by inflammation of the lung tissue.

PULMONARY. Of or relating to the lungs.

RADON. A colorless, odorless radioactive gas formed by the decay of uranium, found in rocks and soil.

RESPIRATORY SYSTEM. The body's system for breathing, including the nose, throat, and lungs.

SINUS. Any of the air-filled cavities in the skull connected to the nostrils or external openings of the nose.

SINUSITIS. Inflammation of a sinus membrane, especially in the nasal or nose region.

SMOG. Air pollution consisting of fog mixed with smoke and or ozone, also called haze, or, in Los Angeles, smog ozone.

SLASH BURNING. The burning of woody debris left over from logging or land clearing operations.

STAGNATION. Lack of motion in a mass of air or water which tends to hold pollutants in place.

TOXIC. Poisonous, carcinogenic, or directly harmful to life.

TRACHEA. A passage through which air passes to and from the lungs.

VOLATILE. Unstable substances that evaporate rapidly at normal temperatures, such as benzene or gasoline.

WHAT IS WOOD SMOKE?

Wood smoke is a complex mixture of substances produced during the burning of wood. The major emissions from wood stoves are carbon monoxide, organic gases (containing carbon or derived from living organisms), particulate matter, and nitrogen oxides. Wood smoke contains many organic compounds known to cause cancer (such as benzopyrenes, dibenzanthracenes, and dibenzocarbazoles), and other toxic compounds (such as aldehydes, phenols, or cresols). The particulate fraction is composed of solid or liquid organic compounds, carbon char (elemental or soot carbon -- similar to charcoal), and inorganic ash.[1]

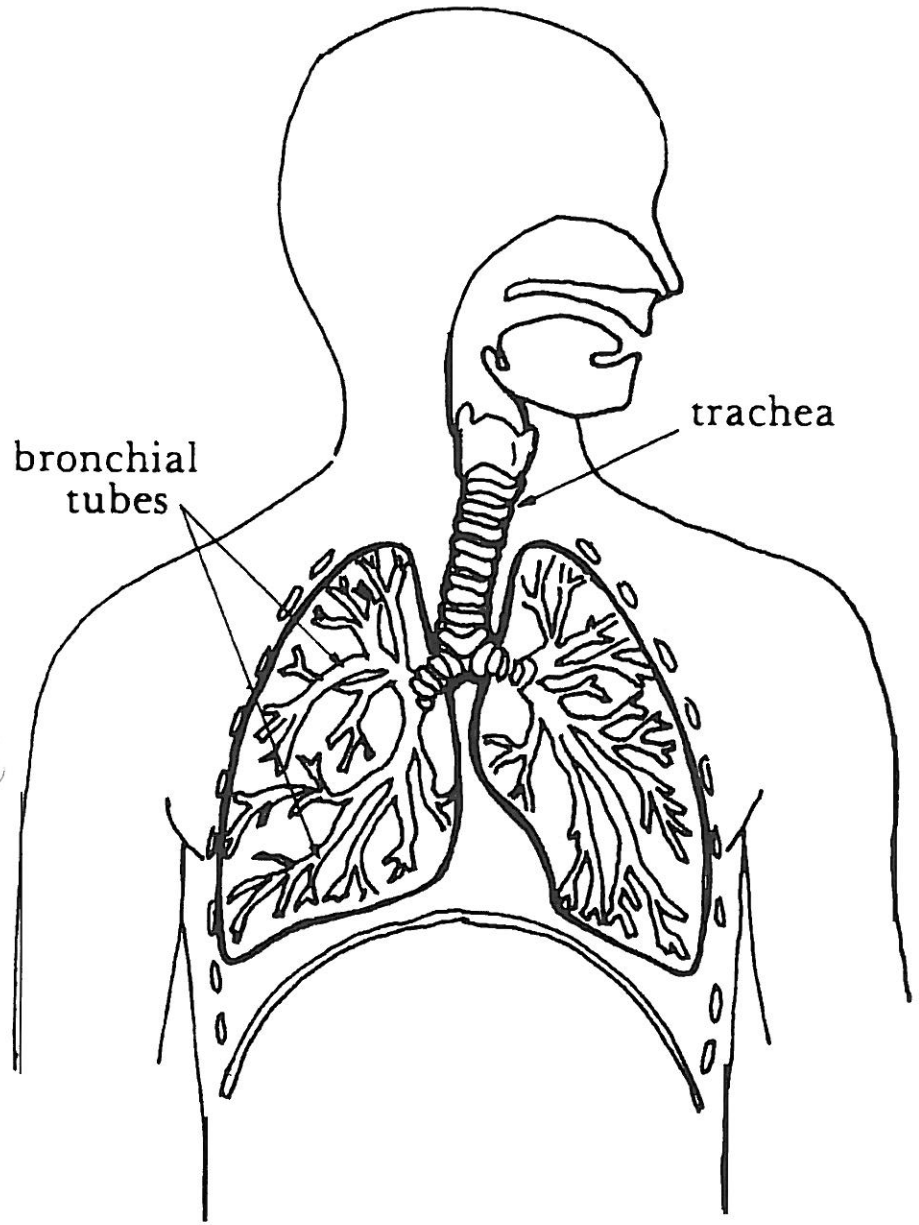
WHY IS WOOD SMOKE A PROBLEM?

The particles in wood smoke are too small to be filtered by the nose and upper respiratory system, so they wind up deep in the lungs. They can remain there for months causing structural damage and chemical changes. Poisonous and cancer-causing chemicals often enter the lungs by adhering to tiny particulate matter (such as wood smoke particles).

These tiny particles are emitted in neighborhoods, both indoors and out, where people spend most of their time. Unfortunately, wood smoke is not only in the outdoor air we breathe. The particulate matter in wood smoke leaving chimneys is so small that it is not stopped by closed doors and windows, and often seeps into neighbors' houses. Even more smoke is sometimes released inside homes which heat with wood.

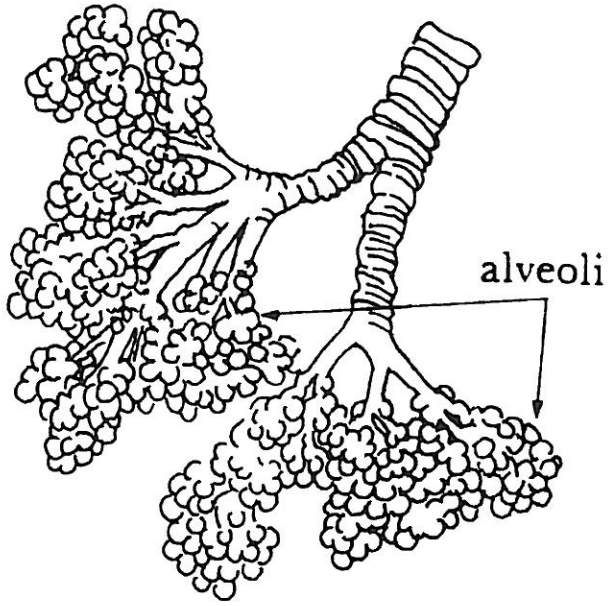
GENERAL EFFECTS OF WOOD SMOKE

Wood smoke exposure causes a decrease in lung function and an increase in the severity of existing lung disease with increases in smoke concentration or exposure time.[2] It also aggravates heart conditions and carbon monoxide (a component of wood smoke) causes heart pain. The occurrence of respiratory illness in children has been shown to increase with increased exposure to wood smoke. This includes lower respiratory infections such as acute pneumonia, or bronchiolitis, which are major causes of disease and death in young children.[20] Wood smoke aggravates asthma, emphysema, pneumonia, and bronchitis. It irritates the eyes and triggers headaches and allergies. Long-term exposure may lead to emphysema, chronic bronchitis, arteriosclerosis, and nasal, throat, lung blood, and lymph system cancers (based on animal studies). [2,4,17,25,27]



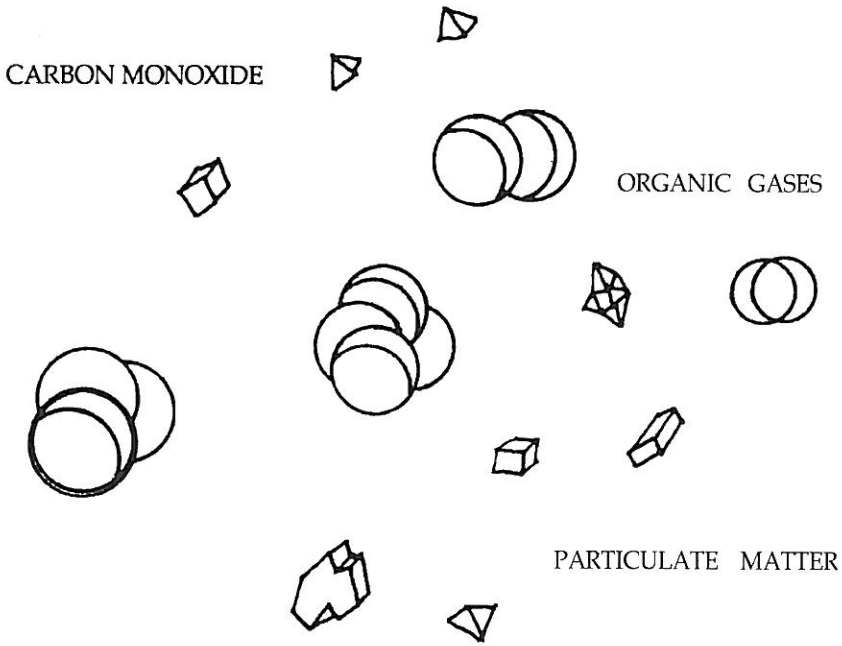
WHAT PARTS OF WOOD SMOKE CAUSE PROBLEMS?

Many organic compounds are produced by combustion of wood. Some burn completely, some are changed chemically, and some leave the stove without burning. Some of these compounds deposit in the chimney as creosote, some condense as very tiny particles of smoke, and some are released into the air as gases. Some of these organic compounds are poisonous, some irritate the respiratory tract, and some may cause cancer or mutations. The primary pollutants from wood smoke are carbon monoxide, particulate matter, and organic gases (including aldehyde gases such as acrolein, which is a serious respiratory irritant).



Carbon monoxide is a colorless, odorless gas produced when any carbon-containing fuel such as gasoline or wood is burned. It reduces the ability of blood to carry oxygen to body tissues. High exposures can lead to death. Lower levels, common in highly polluted urban areas, lead to increased hospitalizations for individuals with heart and circulatory disease, lower birth weights, and increased deaths of newborns.[3]

Particulate matter larger than 10 microns in diameter collects in the upper respiratory system (throat and nose) and is eliminated by sneezing, coughing, noseblowing, spitting, or the digestive system. The particulate matter from wood smoke is a much more serious health threat due to the small particle size. Wood smoke particles are less than 10 microns (a micron is one millionth of a meter) in diameter. Most of them are less than 2.5 microns in diameter. The period at the end of this sentence is about 500 microns in diameter.



Wood smoke particles are so small that they get past the cilia or hair-like structures on the respiratory tract cells. Cilia clear mucous and catch and help remove larger particulate matter from the lungs with a rhythmic motion. Tiny wood smoke particles evade the cilia and collect in the most remote portions of the lungs, called alveoli - the tiny air sacs where oxygen enters the blood stream. Wood smoke particles cause structural and chemical changes deep in the lungs. Other toxic and cancer causing compounds can attach to the smallest smoke particles and enter the lungs at the same time.

Because of the health threat from tiny particulate air pollution, the federal government regulates all particulate matter less than 10 microns in diameter (PM_{10}) as one of six major air pollutants.

Irritants in wood smoke (such as phenols, aldehydes, quinones, nitrogen oxides, and sulphur oxides) contribute to health problems in the respiratory tract. Irritants interfere with the cilia and disrupt the flow of the particle-trapping mucus stream, resulting in more particulate matter entering the lungs. Exposure to wood smoke irritants can lead to inflammation and pulmonary edema (swelling of lung tissue). Irritants can also cause allergic reactions and may contribute to long-term health effects.[1]

WOOD SMOKE AND CANCER

The cancer threat from air pollution is a serious public health concern. Most of the wood smoke cancer research before 1985 focused on identifying the components of soot or the particulate portion of wood smoke, including carcinogens such as benzo(a)pyrene, best known from tobacco smoke research. The first known human carcinogens were from coal tars and chimney soot. The particle or soot component of air pollution has been clearly implicated as a human carcinogen from studies of human cancer victims.[22]

Many substances on the U.S. Environmental Protection Agency's (EPA) priority pollutant list, many suspected human carcinogens, co-carcinogens (cancer initiators or promoters), and cilia-toxic agents (poisonous to the hair-equipped cells which filter most particles out of the respiratory tract) have been identified from wood smoke particles.[14] However, many of the compounds in wood smoke particles have not been identified and even less is known about the toxic organic gases which are also released by wood burning.

Burning of fossil fuels, wood, tobacco, or garbage produces hundreds of different compounds associated with the soot, or particle phase of air pollution. Also produced are many gaseous compounds which are carcinogenic, such as benzene, aldehydes, alkenes, and numerous semi-volatile organic compounds.[21] Recent research has focused on the health effects from wood smoke as a whole, rather than further studies of its component parts.

In 1985 the EPA started a major long-term research program to clarify the sources of air pollution and population exposure, and to estimate future cancer risk (the Integrated Air Cancer Project.[11] Studies include human cancer victims, as well as laboratory mice, and bacteria and mammal cells exposed to the total mixture of particulate matter from urban air samples.

This research found motor vehicles and wood stoves to be the major sources of cancer risk from particulate air pollution in all the urban airsheds studied.[8,34]

Human cancer risks have now been estimated for lifetime exposure to diesel vehicle, leaded and catalyst-equipped gas vehicle, wood stove, cigarette smoke, coke oven (coal), and roofing tar emissions.[23] EPA researchers suggest that the lifetime cancer risk from wood stove emissions may be 12 times greater than the lifetime cancer risk from exposure to an equal amount of cigarette smoke. We must keep in mind that this is not actual cancer risk, but rather an estimate based on bacteria and animal studies comparing the potency of wood smoke to cigarette smoke and other better documented carcinogens. The lifetime human cancer risk estimates from exposure to wood smoke and motor vehicle emissions are theoretical based on such comparative potency tests.

The lifetime cancer risk estimate from exposure to motor vehicle emissions is more than three times that from equal exposure to wood stove emissions, based on recent EPA research in Boise, Idaho.[23] However, we also know that wood stoves produce much more particulate air pollution in the winter than motor vehicles in all Pacific Northwest cities studied by the EPA.[20,31,33,34]

With all of these cancer risk estimates we must also keep in mind that we do not yet have much information on actual yearly levels of human exposure to various types of particulate air pollution.

MUTAGENS IN WOOD SMOKE

Mutagens cause biological mutations or changes in cells such as chromosome defects or genetic damage. Mutagenicity is often used as a screening test for human cancer risk from compounds in air pollution. However, mutagens and carcinogens are not the same thing and not all mutagenic substances cause cancer. Motor vehicles and wood heating emissions cause mutations. These two sources are also major contributors to the human cancer risk from air pollution. [8]

A 1988 EPA study found that wood heat and motor vehicle emissions account for nearly all of the mutagenicity in winter air samples from Albuquerque, NM, Raleigh, NC, and Juneau, AK, over a wide range of climate and wood species. [34] This study found that biological mutations in bacteria exposed to winter air samples increased with higher concentrations of fine particulate matter and were most numerous at times of coldest temperatures, weekends and holidays -- when many wood stoves were in use. One would expect this to be true in Washington State as well.

In the United States more than 30% of mutagenic material emitted to the atmosphere each year comes from wood combustion, according to 1981 calculations. [15,24] In Washington State the contribution from wood smoke is greater than this national average since Washington has the third highest percentage of households burning wood in the United States, behind Oregon and Maine, according to a 1983 U.S. Forest Service survey. [30]

PARTICULATE AIR POLLUTION AND DEATH RATES

London's "Black Fog" in December 1952 killed 4,000 people and led to the British Clean-Air Act. Tiny particulate air pollution from coal stoves was largely to blame. London is once again in the news with a new study linking particulate air pollution to death rates.

This year an EPA researcher applied statistical techniques to daily particulate air measurements and daily death records in London, as well as U.S. cities where daily particulate measurements were available.[28] These cities vary vastly in size, climate, and mixes of air pollutants, including wood smoke. This study found an increase in deaths by 6% for each 100 micrograms of total particulate air pollution measured.

This same EPA study also found that, for every 100 micrograms of total particulate per cubic meter of air, the risk of dying goes up 32% from emphysema, 19% from bronchitis and asthma, 12% from pneumonia, and 9% from cardiovascular disease. Sulfur dioxide, an air pollutant which has often been suspected of causing deaths, showed no effect on death rates. These new findings suggest that particulate matter is more toxic than ozone (commonly called smog). Confirmation of these findings would make particulate air pollution the largest known "involuntary environmental insult" and should encourage a stricter federal particulate standard. [28]

INDOOR AIR QUALITY

Wood smoke does not rise and disperse during winter temperature inversions. At these times, wood smoke hangs close to the ground and enters neighbors' yards and houses, schools and hospitals. Areas with winter atmospheric inversions and valley locations with poor air circulation are most affected. Wood smoke particles are so tiny that they remain suspended for long periods of time and readily penetrate into buildings with incoming cold air, even with today's "air-tight" construction.

Wood stove fumes are released into the outdoor air through chimneys, but fumes are also released directly into the homes where stoves are used. Wood smoke and fumes also enter homes where stoves are not used. Indoor PM_{10} levels from wood smoke in homes without wood stoves reach at least 50% to 70% of outdoor levels, according to a recent University of Washington study in Seattle and an EPA study in Boise, Idaho neighborhoods. [16,24]

Wood stove use not only degrades the air quality inside neighbors' homes, but causes additional air quality impairment in the home using the stove. Klamath Falls, Oregon has the highest measured PM_{10} pollution from wood stoves in the United States. A 1991 study in Klamath Falls by the Oregon Health Division found that children with wood stoves in their own homes have even greater declines in lung function than other children living in the same smoky neighborhood.[12]

Indoor and outdoor air can be degraded significantly by the use of poorly designed noncertified airtight stoves and non airtight stoves.[16,32] Poor burning practices, improper stove operation, improper stove installation, lack of maintenance, and burning wet wood create much indoor and outdoor air pollution even with the newest certified stove models. Backdrafting and "accidents" while loading fuel or opening stove doors can cause substantial amounts of indoor air pollution with any wood stove. Backdrafting can usually be eliminated by proper stove installation and/or regular chimney sweeping.

POPULATIONS MOST AT RISK

Infants, children, pregnant women, senior citizens, cigarette smokers and ex-smokers, and all those suffering from allergies, asthma, bronchitis, emphysema, pneumonia, or any other heart or lung illness are most affected by wood smoke and are sensitive to low levels.[14,17,27,28] Even the most healthy citizens should refrain from heavy outdoor physical exercise (such as jogging) during periods of very poor air quality or high levels of particulate air pollution.

Lung ailments are the leading cause of disease and death in the United States among newborns, accounting for 37% of all deaths in the first year of life, according to the American Lung Association. Lung diseases accounted for 28.3% of all hospitalizations of children under 15 in the United States in 1988. A total of 738,000 children in this age group were hospitalized for lung disease in 1988.[5]

Wood smoke interferes with normal lung development in infants and young children. In addition, several studies have found that home use of wood burning stoves increases the risk of lower respiratory tract infections (LRTIs) such as bronchiolitis and pneumonia in young children.[26] LRTIs are a major cause of early childhood disease and death. Parental smoking, especially during the first year of life, is another well-demonstrated risk factor for lower respiratory tract infections.[9]

A 1990 study of American Indian children found that those living in homes with a wood burning stove have a higher risk of bronchiolitis and pneumonia (lower respiratory tract infections) than children living in homes without wood stoves.[6,11,26] Childhood LRTIs have also been linked with chronic lung disease in later life. Wood smoke exposure causes a chronic reduction in lung function, increasing the rate of decline with age in adults.[25]

Researchers at the University of Washington in 1990 documented more symptoms of respiratory disease in Seattle preschool children living in high wood smoke residential areas than in children living in areas with lower wood smoke levels.[7] This demonstrates the effect of community wood smoke pollution on the occurrence of respiratory illness. Tests have also demonstrated measurable reductions in lung function among both healthy children and asthmatics in smoky Seattle neighborhoods in the winter.[18]

HIDDEN COSTS OF WOOD HEAT

Most people realize that direct costs of wood heat include the purchase price, installation, and maintenance of the heater. Floor protection, the building permit, installation, fuel supply, and chimney sweeping are other direct costs. Time spent cutting, splitting, stacking, and moving wood (as well as cleaning out ashes and maintaining the stove) are also direct costs. However, few people realize the possible extent of hidden or indirect costs.

Indirect costs of wood heat include both fire insurance and health insurance. Fire insurance costs have been climbing. The 1984 statistics from the Washington State Fire Marshall show that nearly half of all fires reported in one and two-family dwellings were caused by wood stoves or fireplaces. These insurance losses help push up the cost of insurance on a home.

Health insurance costs have been rising rapidly in recent years. Health insurance claims raise the cost of health insurance. Wood smoke exposure leads to more frequent and extended hospital visits, as well as more emergency ward or physician visits, and increased use of medications. Lung diseases are second only to heart disease as a cause of disability under social security.[5] Wood smoke adversely affects the cardiovascular system and heart patients, as well as those suffering from lung disease.[1,3,4,25,28]

In 1989 an estimated 85 million people -- more than one-third of the U.S. population -- were afflicted with chronic respiratory disease, from chronic sinusitis to chronic bronchitis and emphysema.[5] The direct and indirect costs of lung disease were about $45 billion in 1988 according to the American Lung Association.

The National Health Interview Survey estimates 25.6 million people suffer from severe lung diseases such as chronic bronchitis, emphysema, or asthma. The total number of deaths attributed to these diseases increased 57% between 1979 and 1987. Lung cancer now accounts for more cancer deaths in the United States for both men and women than any other form of cancer.[5]

Obviously wood smoke does not account for all of the lung disease in this country. Tobacco, radon, car and truck exhaust, asbestos, and other substances share much of the blame. However, the medical evidence is growing each year that wood smoke plays a larger role than was previously thought, and the tiny particulate matter from wood smoke cannot be kept out of your home if you live in a neighborhood where there is much wood stove use.

WHAT ABOUT OUTDOOR BURNING AND CERTIFIED WOOD STOVES?

Outdoor burning in Washington contributes roughly two-thirds as much particulate air pollution as wood stoves do each year (see graph). Agricultural burning, slash burning, backyard burning, and burn barrels all contribute to air pollution and are becoming more closely regulated under Washington's new clean air legislation. Outdoor burning is not as common in the winter when cooler temperatures lead to inversions and trap wood stove smoke close to the ground in our neighborhoods; but any time that citizens are exposed to smoke there is cause for concern.

About 85% of wood stoves in Washington State are not certified to meet federal particulate emission standards (based on a 1990 survey by the Washington State Energy Office). These noncertified stoves release most of the particulate air pollution statewide. The newer EPA certified models release much less smoke although they still require dry wood, and proper installation, operation, and maintenance to minimize air pollution. All smoke from any source adds to the air pollution problem.

HOW YOU CAN HELP FIGHT SMOKY AIR

Noncertified stoves should be scrapped if possible. Cleaner heating methods include the best technology certified wood stoves, gas stoves or gas inserts, and high efficiency gas and oil central heating systems. Energy conservation, insulation and weatherstripping can save money with any heating system and reduce pollution. Support burning bans and smoke regulations. Learn about composting, mulching, recycling, or other smokeless disposal strategies. Any smoke or odor from open fires or from home heating which affects our neighbors' health or enjoyment of their property is illegal and should be reported to the local county air pollution control authority.

We take pride in our quality of life in Washington. In light of current and ongoing health research, the smell of wood smoke no longer has the pleasant connotation it once had. Thoughts of sick children, and lung and heart disease victims are not pleasant. Less smoke means cleaner air for you, your family, and the environment.

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If you need this information in another format, please contact Ecology's Air Quality Program at (360) 407-6800. If you are a person with a speech or hearing impairment, please call 711, or 1-800-833-6388 for TTY.

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Olympia, WA 98504-7600
or call the Washington State Energy Office toll-free energy hotline at 1-800-962-9731.

92-46 *(Updated August 2004)*

Mike Branson

From: kathleen bang [kathybang@mac.com]
Sent: Thursday, June 26, 2014 9:53 PM
To: Sharon Friedrichsen; Mike Branson
Subject: Fwd: the dangers of campfire smoke: The Dangers of Campfire Smoke

For F and B

here are some EPA and NIH publications (plus one from U. Washington that does review animal studies conducted prior to 1994) (plus another from an environmental health nonprofit) that may interest you:
http://www.airnow.gov/index.cfm?action=smoke.index
http://www.epa.gov/airnow/particle/pm-color.pdf
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1523269/pdf/envhper00354-0064.pdf
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3272333/
http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1026893/pdf/westjmed00121-0093.pdf
http://ehs.sph.berkeley.edu/krsmith/CRA/tb/LarsonTimothy%20V_1994.pdf
http://www.ehhi.org/woodsmoke/health_effects.shtml

And below are published papers I had found before but do not have the ability anymore to get full text articles of them (my Stanford library access expired when I retired from there). In the research that I did, I could find nothing about wood smoke from beach fires. I suspect that it has not been studied since it is a relatively uncommon phenomena. But there is no reason to think that beach fires are any safer qualitatively than the smoke from indoor wood stoves. And probably the beach fires are qualitatively worse since people burn paper and other materials in these fires and these materials contain chemicals such as one of the carcinogens in Agent Orange (dioxin). See http://www.greentownconference.com/wp-content/uploads/2013/10/Trash-Fact-Sheet.pdf

Bull Acad Natl Med. **2013** Jan;197(1):187-90.

[The health impact of woodsmoke].

[Article in French]
Masse R, Boudène C.

Abstract

Biomass is an important renewable resource of primary energy. Wood burning is expected to expand in future as a cheap means of residential heating, especially in wood-producing rural areas. The health consequences of wood smoke are taken in account far less actively in Europe than in North America, where stringent limitations have been introduced, such as those of the EPA Burnwise project. Recently, wildfires were estimated to release 2 petagrams of carbon into the atmosphere worldwide, contributing to more than 300 000 deaths annually. More effective measures are thus needed to prevent indoor and outdoor exposure to wood smoke. Ultrafine particles are the biggest concern, but the volatile phase also contains many toxicants. Ultrafine particles are among the most dangerous carbonaceous particles found in polluted atmospheres. They can be identified by using C14, levoglucosan, crystallography and spectrometry. Any expansion of the use of wood as a fuel source should take into account its health impact, which is similar to that of fossil fuels.

PMID:

24672990

[PubMed - indexed for MEDLINE]

Indoor Air. **2008** Oct;18(5):408-15. doi: 10.1111/j.1600-0668.2008.00541.x. Epub 2008 Jul 28.

Results of a residential indoor PM2.5 sampling program before and after a woodstove changeout.

Ward T1, Palmer C, Bergauff M, Hooper K, Noonan C.
Author information

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Erratum in

- Indoor Air. 2008 Dec;18(6):529. Palmer, C [added]; Bergauff, M [added]; Hooper, K [added].

Abstract

During 2005-2007, a woodstove changeout program was conducted in a Rocky Mountain valley community in an effort to reduce ambient levels of PM(2.5). In addition to changes in ambient PM(2.5), an opportunity was provided to evaluate the changes in indoor air quality when old stoves were replaced with US Environmental Protection Agency (EPA)-certified woodstoves. PM(2.5) samples were measured in 16 homes prior to and following the changeout. For each sampling event, PM(2.5) mass was continuously measured throughout the 24-h sampling periods, and organic/elemental carbon (OC/EC) and associated chemical markers of woodsmoke were measured from quartz filters. Results showed that average PM(2.5) concentrations and maximum PM(2.5) concentrations were reduced by 71% and 76%, respectively (as measured by TSI DustTraks). Levoglucosan was reduced by 45% following the introduction of the new woodstove. However, the concentrations of resin acids, natural chemicals found in the bark of wood, were increased following the introduction of the new woodstove. There were no discernible trends in methoxphenol levels, likely due to the semi-volatile nature of the species that were measured. Although there is some uncertainty in this study regarding the amount of ambient PM infiltration to the indoor environment, these findings demonstrated a large impact on indoor air quality following this intervention.

PRACTICAL IMPLICATIONS:

Emissions from residential woodstoves are an important air quality issue (both indoors and ambient) in many regions throughout the US and the world. More specifically, woodstoves have been identified as a major source of PM(2.5) in valley locations throughout the Northern Rocky Mountains, where biomass combustion is the predominant source of home heating. In this study, we present results that demonstrate the dramatic reduction in PM(2.5) concentrations (as measured by TSI, Inc. DustTrak PM(2.5) air samplers) inside homes following the replacement of old, polluting woodstove with new EPA-certified woodstoves.

PMID:

18665872

[PubMed - indexed for MEDLINE]

Inhal Toxicol. 2007 Jan;19(1):67-106.

58

Woodsmoke health effects: a review.

Naeher LP1, Brauer M, Lipsett M, Zelikoff JT, Simpson CD, Koenig JQ, Smith KR

- 1Department of Environmental Health Science, College of Public Health, University of Georgia, Athens, Georgia, USA.

Abstract

The sentiment that woodsmoke, being a natural substance, must be benign to humans is still sometimes heard. It is now well established, however, that wood-burning stoves and fireplaces as well as wildland and agricultural fires emit significant quantities of known health-damaging pollutants, including several carcinogenic compounds. Two of the principal gaseous pollutants in woodsmoke, CO and NOx, add to the atmospheric levels of these regulated gases emitted by other combustion sources. Health impacts of exposures to these gases and some of the other woodsmoke constituents (e.g., benzene) are well characterized in thousands of publications. As these gases are indistinguishable no matter where they come from, there is no urgent need to examine their particular health implications in woodsmoke. With this as the backdrop, this review approaches the issue of why woodsmoke may be a special case requiring separate health evaluation through two questions. The first question we address is whether woodsmoke should be regulated and/or managed separately, even though some of its separate constituents are already regulated in many jurisdictions. The second question we address is whether woodsmoke particles pose different levels of risk than other ambient particles of similar size. To address these two key questions, we examine several topics: the chemical and physical nature of woodsmoke; the exposures and epidemiology of smoke from wildland fires and agricultural burning, and related controlled human laboratory exposures to biomass smoke; the epidemiology of outdoor and indoor woodsmoke exposures from residential woodburning in developed countries; and the toxicology of woodsmoke, based on animal exposures and laboratory tests. In addition, a short summary of the exposures and health effects of biomass smoke in developing countries is provided as an additional line of evidence. In the concluding section, we return to the two key issues above to summarize (1) what is currently known about the health effects of inhaled woodsmoke at exposure levels experienced in developed countries, and (2) whether there exists sufficient reason to believe that woodsmoke particles are sufficiently different to warrant separate treatment from other regulated particles. In addition, we provide recommendations for additional woodsmoke research.

PMID:

17127644

[PubMed - indexed for MEDLINE]

J Toxicol Environ Health B Crit Rev. 2002 Jul-Sep;5(3):269-82.

The toxicology of inhaled woodsmoke.

Zelikoff JT1, Chen LC, Cohen MD, Schlesinger RB.
Author information

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Abstract

In addition to developing nations relying almost exclusively upon biomass fuels, such as wood for cooking and home heating, North Americans, particularly in Canada and the northwestern and northeastern sections of the United States, have increasingly turned to woodburning as an alternate method for domestic heating because of increasing energy costs. As a result, the number of households using woodburning devices has increased dramatically. This has resulted in an increase in public exposure to indoor and outdoor woodsmoke-associated pollutants, which has prompted widespread concern about the adverse human health consequences that may be associated with prolonged woodsmoke exposure.

59

This mini-review article brings together many of the human and animal studies performed over the last three decades in an attempt to better define the toxicological impact of inhaled woodsmoke on exposed children and adults; particular attention is given to effects upon the immune system. General information regarding occurrence and woodsmoke chemistry is provided so as to set the stage for a better understanding of the toxicological impact. It can be concluded from this review that exposure to woodsmoke, particularly for children, represents a potential health hazard. However, despite its widespread occurrence and apparent human health risks, relatively few studies have focused upon this particular area of research. More laboratory studies aimed at understanding the effects and underlying mechanisms of woodsmoke exposure, particularly on those individuals deemed to be at greatest risk, are badly needed, so that precise human health risks can be defined, appropriate regulatory standards can be set, and accurate decisions can be made concerning the use of current and new woodburning devices.

PMID:

12162869

[PubMed - indexed for MEDLINE]

On Jun 15, 2014, at 4:39 PM, Karen Ferlito <ferlito@pacbell.net> wrote:

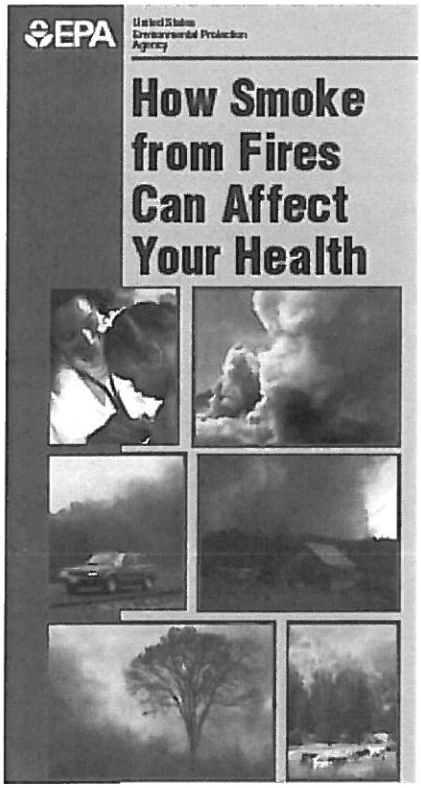
http://campingmyway.blogspot.com/2009/04/dangers-of-campfire-smoke.html

Karen Ferlito
Sent from my iPad.

Local Air Quality Conditions

Zip Code: Go **State :** Alabama

Forest Fire Smoke



Contents

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Links A-Z

About AirNow	Health	For Partne
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Go

Local Air Quality Conditions

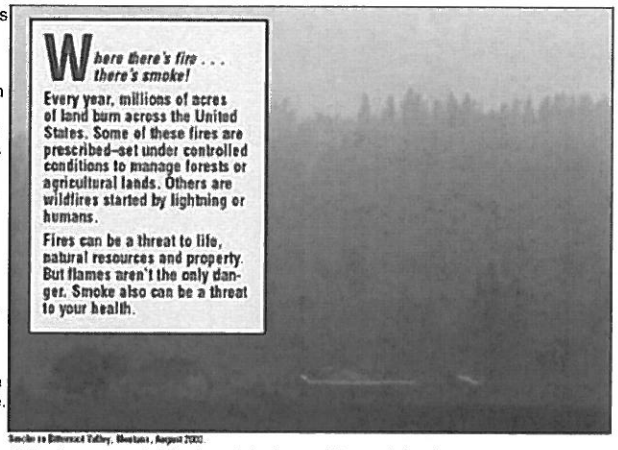
Zip Code: Go State : Alabama Go National Summary

How Smoke from Fires Can Affect Your Health

Smoke may smell good, but it's not good for you.

If you are healthy, you're usually not at a major risk from smoke. Still, it's a good idea to avoid breathing smoke if you can help it.

Smoke is made up of a complex mixture of gases and fine particles produced when wood and other organic matter burn. The biggest health threat from smoke comes from fine particles. These microscopic particles can get into your eyes and respiratory system, where they can cause health problems such as burning eyes, runny nose, and illnesses such as bronchitis. Fine particles also can aggravate chronic heart and lung diseases - and even are linked to premature deaths in people with these conditions.



Smoke in Bitterroot Valley, Montana, August 2000.

Some people are more susceptible than others:

If you have heart or lung disease, such as congestive heart failure, angina, chronic obstructive pulmonary disease, emphysema or asthma, you may experience health effects earlier and at lower smoke levels than healthy people.

Older adults are more likely to be affected by smoke, possibly because they are more likely to have heart or lung diseases than younger people.

Children also are more susceptible to smoke for several reasons: their respiratory systems are still developing; they breathe more air (and air pollution) per pound of body weight than adults; and they're more likely to be active outdoors.

How to tell if smoke is affecting you:

If smoke is affecting your area, check your local media for information on air quality and how to protect your health.

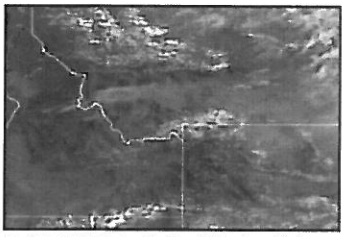
Children with respiratory diseases should be monitored closely during smoke alerts.

Smoke can irritate the eyes and airways, causing coughing, a scratchy throat, irritated sinuses, headaches, stinging eyes or a runny nose. If you have heart or lung disease, smoke might make your symptoms worse.

People with heart disease might experience chest pain, palpitations, shortness of breath, or fatigue. People with lung disease may not be able to breathe as deeply or as vigorously as usual, and they may experience symptoms such as coughing, phlegm, chest discomfort, wheezing and shortness of breath.

When smoke levels are high enough, even healthy people may experience some of these symptoms.

Protect yourself!



Smoke from a fire can travel rapidly, affecting air quality in areas hundreds of miles downwind.

It's important to limit your exposure to smoke - especially if you may be susceptible. Here are some steps you can take to protect your health:

Pay attention to local air quality reports. Stay alert to any news coverage or health warnings related to smoke. Also find out if your community reports EPA's Air Quality Index (AQI). The AQI, based on data from local air quality monitors, tells you about the daily air quality in your area and recommends precautions you can take to protect your health. As smoke gets worse, the concentration of particles in the air changes - and so do the steps you should take to protect yourself.

Use visibility guides, where they're available. Not every community has a monitor that measures particle levels in the air. In the western United States, some areas without air quality monitors have developed guidelines to help people estimate the AQI based on how far they can see. Check with your local air quality agency to find out if there's a visibility guide for your area.

Use common sense. If it looks smoky outside, it's probably not a good time to mow the lawn or go for a run. And it's probably not a good time for your children to play outdoors.

If you are advised to stay indoors, take steps to keep indoor air as clean as possible. Keep your windows and doors closed - unless it's extremely hot outside. Run your air conditioner, if you have one. Keep the fresh air intake closed and the filter clean to prevent bringing additional smoke inside. Note: If you don't have an air conditioner, staying inside with the windows closed may be dangerous in extremely hot weather. In these cases, seek alternative shelter.

Help keep particle levels inside lower. When smoke levels are high, try to avoid using anything that burns, such as wood fireplaces, gas logs, gas stoves - and even candles! Don't vacuum. That stirs up particles already inside your home. And don't smoke. That puts even more pollution in your lungs, and in the lungs of people around you.

If you have asthma or other lung disease, make sure you follow your doctor's directions about taking your medicines and following your asthma management plan. Call your doctor if your symptoms worsen.

If you have heart or lung disease, if you are an older adult, or if you have children, talk with your doctor about whether and when you should leave the area. When smoke is heavy for a prolonged period of time, fine particles can build up indoors even though you may not be able to see them.

If you have heart or lung disease, if you are an older adult, or if you have children, talk with your doctor about steps you should take to protect yourself if smoke affects your community. If you live in a fire-prone area, plan ahead! Talk with your doctor before fire season, so you'll know what to do in a smoky situation.

Only your doctor can advise you about your specific health situation. But EPA's Air Quality Index can help you protect yourself when particle levels are high. Check the table below for specific steps you can take.

Air cleaners can help indoors-but buy before a fire.

Some room air cleaners can help reduce particle levels indoors, as long as they are the right type and size for your home. If you choose to buy an air cleaner, don't wait until there's a fire - make that decision beforehand. Note: Don't use an air cleaner that generates ozone. That just puts more pollution in your home.

For more information about home air cleaners, go to: www.epa.gov/iaq/pubs/residair.html

Dust masks aren't enough!

Paper "comfort" or "dust" masks - the kinds you commonly can buy at the hardware store - are designed to trap large particles, such as sawdust. These masks generally will not protect your lungs from the fine particles in smoke.

For more information:

- If there is an active fire in your area, follow your local news or fire web sites for up-to-date information.
- About wildfires, including current status:http://www.nifc.gov/ EXIT AIRNOW ▶

About indoor air quality: http://www.epa.gov/iaq/ia-intro.html

Air Quality Guide for Particle Pollution

Good	0-50	None
Moderate	51-100	Unusually sensitive people should consider reducing prolonged or heavy exertion
Unhealthy for Sensitive Groups	101-150	People with heart or lung disease, older adults, and children should reduce prolonged or heavy exertion.
Unhealthy	151 to 200	People with heart or lung disease, older adults, and children should avoid prolonged or heavy exertion. Everyone else should reduce prolonged or heavy exertion.
Very Unhealthy Alert	201 to 30	People with heart or lung disease, older adults, and children should avoid all physical activity outdoors. Everyone else should avoid prolonged or heavy exertion.

Contents

Links A-Z

About AirNow
AirNow International
Air Quality Action Days / Alerts
AirCompare
Air Quality Index (AQI)
Calculator: AQI to Concentration
Calculator: Concentration to AQI
Canada Air Quality
EnviroFlash E-mail
EPA Burn Wise
FAQs

Health
Health Providers
Links to International Air Quality Sites
Kids
Movies
NAQ Conferences
NOAA
Older Adults
Ozone
Particle Pollution (PM2.5, PM10)
List of Partners

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Smoke2003_version1.qxd 5/1/2003 3:51 PM Page 3

If you have asthma or other lung disease, make sure you follow your doctor's directions about taking your medicines and following your asthma management plan. Call your doctor if your symptoms worsen.

If you have heart or lung disease, if you are an older adult, or if you have children, talk with your doctor about whether and when you should leave the area. When smoke is heavy for a prolonged period of time, fine particles can build up indoors even though you may not be able to see them.

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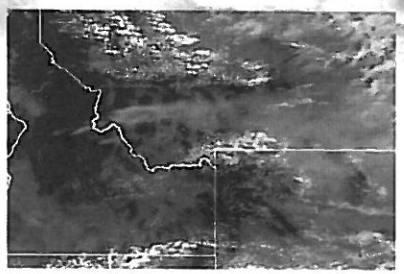
Dust masks aren't enough!

Paper "comfort" or "dust" masks – the kinds you commonly can buy at the hardware store – are designed to trap large particles, such as sawdust. These masks generally will not protect your lungs from the fine particles in smoke.

Air Quality Index	Air Quality	Protect Your Health
0 to 50	Good	None.
51 to 100	Moderate	Unusually sensitive people should consider reducing prolonged or heavy exertion.
101 to 150	Unhealthy for Sensitive Groups	People with heart or lung disease, older adults, and children should reduce prolonged or heavy exertion.
151 to 200	Unhealthy	People with heart or lung disease, older adults, and children should avoid prolonged or heavy exertion. Everyone else should reduce prolonged or heavy exertion.
201 to 300	Very Unhealthy	People with heart or lung disease, older adults, and children should avoid all physical activity outdoors. Everyone else should avoid prolonged or heavy exertion.
301 to 500	Hazardous	Everyone should avoid all physical activity outdoors; people with heart or lung disease, older adults, and children should remain indoors and keep activity levels low.



Smoke from a fire can travel rapidly, affecting air quality in areas hundreds of miles downwind.

If you have heart or lung disease, if you are an older adult, or if you have children, talk with your doctor about steps you should take to protect yourself if smoke affects your community. If you live in a fire-prone area, plan ahead! Talk with your doctor *before* fire season, so you'll know what to do in a smoky situation.

Only your doctor can advise you about your specific health situation. But EPA's **Air Quality Index** can help you protect yourself when particle levels are high. Check the table to the left for specific steps you can take.

For more information:

- **If there is an active fire in your area, follow your local news or fire web sites for up-to-date information.**
- **About smoke and health:** *http://www.epa.gov/airnow/smoke*
- **About wildfires, including current status:** *http://www.nifc.gov/*
- **About indoor air quality:** *http://www.epa.gov/iaq/ia-intro.html*

Top right cover photo (billowing smoke) courtesy of Ravalli Republic News.
Office of Air and Radiation
EPA-452/F-02-002
www.epa.gov/air
May 2003

United States
Environmental Protection
Agency

How Smoke from Fires Can Affect Your Health

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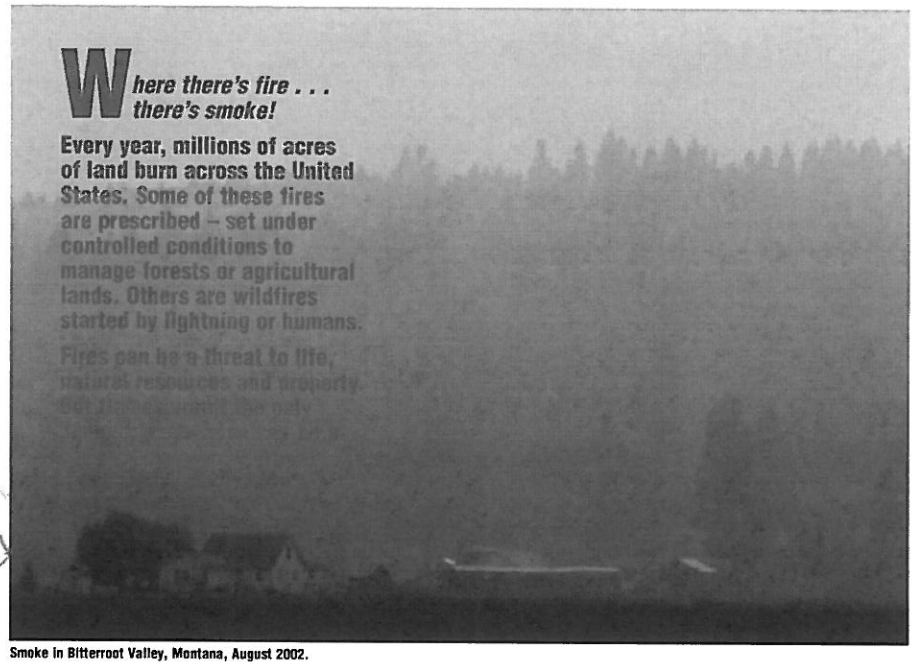


Smoke in Bitterroot Valley, Montana, August 2002.

Smoke may smell good, but it's not good for you.

If you are healthy, you're usually not at a major risk from smoke. Still, it's a good idea to avoid breathing smoke if you can help it.

Smoke is made up of a complex mixture of gases and fine particles produced when wood and other organic matter burn. The biggest health threat from smoke comes from fine particles. These microscopic particles can get into your eyes and respiratory system, where they can cause health problems such as burning eyes, runny nose, and illnesses such as bronchitis. Fine particles also can aggravate chronic heart and lung diseases – and even are linked to premature deaths in people with these conditions.

Some people are more susceptible than others:

If you have heart or lung disease, such as congestive heart failure, angina, chronic obstructive pulmonary disease, emphysema or asthma, you may experience health effects earlier and at lower smoke levels than healthy people.

Older adults are more likely to be affected by smoke, possibly because they are more likely to have heart or lung diseases than younger people.

Children also are more susceptible to smoke for several reasons: their respiratory systems are still developing; they breathe more air (and air pollution) per pound of body weight than adults; and they're more likely to be active outdoors.

How to tell if smoke is affecting you:

Smoke can irritate the eyes and airways, causing coughing, a scratchy throat, irritated sinuses, headaches, stinging eyes or a runny nose. If you have heart or lung disease, smoke might make your symptoms worse.

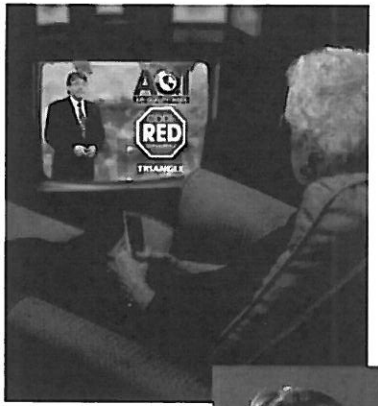
People with heart disease might experience chest pain, palpitations, shortness of breath, or fatigue. People with lung disease may not be able to breathe as deeply or as vigorously as usual, and they may experience symptoms such as coughing, phlegm, chest discomfort, wheezing and shortness of breath.

When smoke levels are high enough, even healthy people may experience some of these symptoms.

Protect yourself!

It's important to limit your exposure to smoke – especially if you may be susceptible. Here are some steps you can take to protect your health:

Pay attention to local air quality reports. Stay alert to any news coverage or health warnings related to smoke. Also find out if your community reports EPA's Air Quality Index (AQI). The AQI, based on data from local air quality monitors, tells you about the daily air quality in your area and recommends precautions you can take to protect your health. As smoke gets worse, the concentration of particles in the air changes – and so do the steps you should take to protect yourself.



If smoke is affecting your area, check your local media for information on air quality and how to protect your health.

Use visibility guides, where they're available. Not every community has a monitor that measures particle levels in the air. In the western United States, some areas without air quality monitors have developed guidelines to help people estimate the AQI based on how far they can see. Check with your local air quality agency to find out if there's a visibility guide for your area.

Use common sense. If it looks smoky outside, it's probably not a good time to mow the lawn or go for a run. And it's probably not a good time for your children to play outdoors.

If you are advised to stay indoors, take steps to keep indoor air as clean as possible. Keep your windows and doors closed – unless it's extremely hot outside. Run your air conditioner, if you have one. Keep the fresh air intake closed and the filter clean to prevent bringing additional smoke inside. *Note:* If you don't have an air conditioner, staying inside with the windows closed may be dangerous in extremely hot weather. In these cases, seek alternative shelter.

Help keep particle levels inside lower. When smoke levels are high, try to avoid using anything that burns, such as wood fireplaces, gas logs, gas stoves – and even candles! Don't vacuum. That stirs up particles already inside your home. And don't smoke. That puts even more pollution in your lungs, and in the lungs of people around you.

Children with respiratory diseases should be monitored closely during smoke alerts.

Airborne particles, the main ingredient of haze, smoke, and airborne dust, present serious air quality problems in many areas of the United States. This particle pollution can occur year-round—and it can cause a number of serious health problems, even at concentrations found in many major cities.

Particles contribute to haze, such as this brown haze over Boston.

What is particle pollution?

Particle pollution is a mixture of microscopic solids and liquid droplets suspended in air. This pollution, also known as particulate matter, is made up of a number of components, including acids (such as nitrates and sulfates), organic chemicals, metals, soil or dust particles, and allergens (such as fragments of pollen or mold spores).

The size of particles is directly linked to their potential for causing health problems. Small particles less than 10 micrometers in diameter pose the greatest problems, because they can get deep into your lungs, and some may even get into your bloodstream. Exposure to such particles can affect both your lungs and your heart. Larger particles are of less concern, although they can irritate your eyes, nose, and throat.

Small particles of concern include "fine particles" (such as those found in smoke and haze), which are 2.5 micrometers in diameter or less; and "coarse particles" (such as those found in wind-blown dust), which have diameters between 2.5 and 10 micrometers.

Are you at risk from particles?

People with heart or lung disease, older adults, and children are considered at greater risk from particles than other people, especially when they are physically active. Exercise and physical activity cause people to breathe faster and more deeply—and to take more particles into their lungs.

People with heart or lung diseases—such as coronary artery disease, congestive heart failure, and asthma or chronic obstructive pulmonary disease (COPD)—are at increased risk, because particles can aggravate these diseases. People with diabetes also may be at increased risk, possibly because they are more likely to have underlying cardiovascular disease.

Older adults are at increased risk, possibly because they may have undiagnosed heart or lung disease or diabetes. Many studies show that when particle levels are high, older adults are more likely to be hospitalized, and some may die of aggravated heart or lung disease.

Children are likely at increased risk for several reasons. Their lungs are still developing; they spend more time at high activity levels; and they are more likely to have asthma or acute respiratory diseases, which can be aggravated when particle levels are high.

It appears that risk varies throughout a lifetime, generally being higher in early childhood, lower in healthy adolescents and younger adults, and increasing in middle age through old age as the incidence of heart and lung disease and diabetes increases. Factors that increase your risk of heart attack, such as high blood pressure or elevated cholesterol levels, also may increase your risk from particles. In addition, scientists are evaluating new studies that suggest that exposure to high particle levels may also be associated with low birth weight in infants, pre-term deliveries, and possibly fetal and infant deaths.

How can particles affect your health?

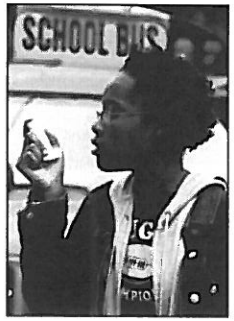
Particle exposure can lead to a variety of health effects. For example, numerous studies link particle levels to increased hospital admissions and emergency room visits—and even to death from heart or lung diseases. Both long- and short-term particle exposures have been linked to health problems.

Long-term exposures, such as those experienced by people living for many years in areas with high particle levels, have been associated with problems such as reduced lung function and the development of chronic bronchitis—and even premature death.

Short-term exposures to particles (hours or days) can aggravate lung disease, causing asthma attacks and acute bronchitis, and may also increase susceptibility to respiratory infections. In people with heart disease, short-term exposures have been linked to heart attacks and arrhythmias. Healthy children and adults have not been reported to suffer serious effects from short-term exposures, although they may experience temporary minor irritation when particle levels are elevated.

What are the symptoms of particle exposure?

Even if you are healthy, you may experience temporary symptoms, such as irritation of the eyes, nose, and throat; coughing; phlegm; chest tightness; and shortness of breath.



If you have lung disease, you may not be able to breathe as deeply or as vigorously as normal, and you may experience coughing, chest discomfort, wheezing, shortness of breath, and unusual fatigue. If you have any of these symptoms, reduce your exposure to particles and follow your doctor's advice. Contact your doctor if symptoms persist or worsen. **If you have asthma,** carefully follow your asthma management plan when particle levels are high. Your doctor can help you develop a plan if you don't have one.

If you have heart disease, particle exposure can cause serious problems in a short period of time—even heart attacks—with no warning signs. So don't assume that you are safe just because you don't have symptoms. Symptoms such as chest pain or tightness, palpitations, shortness of breath, or unusual fatigue may indicate a serious problem. If you have any of these symptoms, *follow your doctor's advice.*

How can you avoid unhealthy exposure?

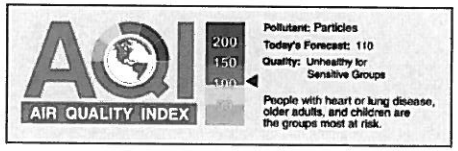
Your chances of being affected by particles increase the more strenuous your activity and the longer you are active outdoors. If your activity involves prolonged or heavy exertion, reduce your activity time—or substitute another that involves less exertion. Go for a walk instead of a jog, for example. Plan outdoor activities for days when particle levels are lower. And don't exercise near busy roads; particle levels generally are higher in these areas.

Particle levels can be elevated indoors, especially when outdoor particle levels are high. Certain filters and room air cleaners can help reduce indoor particle levels. You also can reduce particle levels indoors by not smoking inside, and by reducing your use of other particle sources such as candles, wood-burning stoves, and fireplaces.

How can the Air Quality Index help?

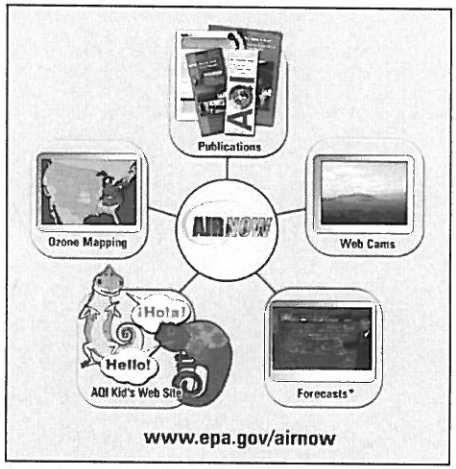
In many areas, local media provide **air quality forecasts** telling you when particle levels are expected to be unhealthy. Forecasts use the same format as EPA's Air Quality Index, or AQI, a tool that state and local agencies use to issue public reports of actual levels of particles, ground-level ozone, and other common air pollutants.

Using the AQI's color-coded scale, these forecasts help you quickly learn when air pollution is expected to reach unhealthy levels in your area. In the newspaper forecast below, for example, the black arrow points to the "orange" range, indicating that particle levels are expected to be unhealthy for sensitive groups. On television, you might hear a meteorologist say something like this: *"Tomorrow will be a code orange air quality day, with particle pollution at levels that are unhealthy for sensitive groups. If you have heart or lung disease, or if you're an older adult or a child, you should plan strenuous activities for a time when air quality is better."*



AIR QUALITY INDEX FOR PARTICLE POLLUTION

Air Quality Index	Air Quality	Health Advisory
0 to 50	Good	None.
51 to 100	Moderate	Unusually sensitive people should consider reducing prolonged or heavy exertion.
101 to 150	Unhealthy for Sensitive Groups	People with heart or lung disease, older adults, and children should reduce prolonged or heavy exertion.
151 to 200	Unhealthy	People with heart or lung disease, older adults, and children should avoid prolonged or heavy exertion. Everyone else should reduce prolonged or heavy exertion.
201 to 300		People with heart or lung disease, older adults, and children should avoid all physical activity outdoors. Everyone else should avoid prolonged or heavy exertion.



Daily air quality and health information are available on the AIRNOW Web site.

AIRNOW

AIRNOW (*www.epa.gov/airnow*) is a Web site that gives daily information about air quality, including ground-level ozone and particles, and how they may affect you. AIRNOW contains:

- Real-time particle levels for many locations.
- Air quality forecasts for many cities across the country.
- Kids' Web page and associated teacher curriculum.
- Smoke Web page.
- Links to state and local air quality programs.
- Ideas about what you can do to reduce particles. For example, you can keep your car, boat, and other engines well-tuned, and avoid using engines that smoke. You can also participate in local energy conservation programs.

**Photo courtesy of The Weather Channel*

Office of Air and Radiation
www.epa.gov/air
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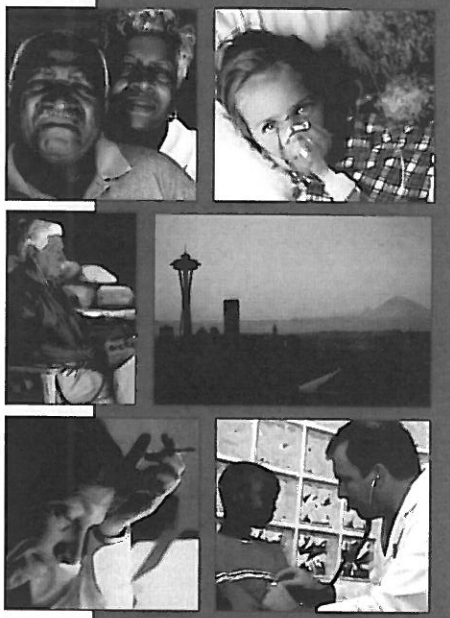
EPA United States Environmental Protection Agency

Particle Pollution and Your Health



What Is Particle Pollution?

Are You at Risk?

How Can You Protect Yourself?

Health Effects of Particulate Air Pollution: Time for Reassessment?

C. Arden Pope III,[1] David V. Bates,[2] and Mark E. Raizenne[3]

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Numerous studies have observed health effects of particulate air pollution. Compared to early studies that focused on severe air pollution episodes, recent studies are more relevant to understanding health effects of pollution at levels common to contemporary cities in the developed world. We review recent epidemiologic studies that evaluated health effects of particulate air pollution and conclude that respirable particulate air pollution is likely an important contributing factor to respiratory disease. Observed health effects include increased respiratory symptoms, decreased lung function, increased hospitalizations and other health care visits for respiratory and cardiovascular disease, increased respiratory morbidity as measured by absenteeism from work or school or other restrictions in activity, and increased cardiopulmonary disease mortality. These health effects are observed at levels common to many U.S. cities including levels below current U.S. National Ambient Air Quality Standards for particulate air pollution. *Key words:* air pollution, particulate pollution, health effects, respiratory disease. *Environ Health Perspect* 103:472–480 (1995)

Many investigators have reported associations between particulate air pollution and respiratory disease. Early studies focused on severe air pollution episodes including an episode in Meuse Valley, Belgium, in December 1930 (*1*); an episode in Donora, Pennsylvania, in 1948 (*2*); and several episodes in London, the most notable occurring in December 1952 (*3*). Although few data were available regarding concentrations of air pollutants during these episodes, large increases in sickness and death demonstrated severe health effects of air pollution.

Two other important observations were made. First, Martin noted that in the greater London region, overall annual respiratory mortality (as opposed to episodic mortality increases) was significantly related to smoke level (*4*). Second, Holland and Reid (*5*) made a cross-sectional comparison of British male postal employees in London and in smaller country towns, where levels of SO_2 and particulate pollution were about half those in the metropolis. Accounting for cigarette smoking, significant decrements of FEV_1 (forced expiratory volume in 1 sec) in London employees compared to those in the provinces were reported. With our present knowledge of the remarkable predictive capability of FEV_1 for survival (*6*), this finding also suggests that longevity was adversely affected by pollution levels prevailing at that time. This type of air pollution from coal burning has been greatly reduced in the western industrialized nations, but it is still present in Eastern Europe and China. A recent study (*7*) of nonsmoking women in different areas around Beijing shows similar decrements of FEV_1 in areas of higher pollution as were first demonstrated by Holland and Reid (*5*).

By the 1970s, a link between respiratory disease and particulate air pollution and/or sulfur oxide pollution had been well established. There remained disagreement about what levels of pollution significantly affected human health. For example, Holland and several other prominent British scientists (*8*) reviewed research on the health effects of particulate pollution that had been published between 1968 and 1977. They concluded that particulate and related air pollution at high levels pose hazards to human health, but that health effects of particulate pollution at lower concentrations that existed in the United States and Britain by the 1970s could not be "disentangled" from health effects of other factors (*8*). Shy (*9*) responded that this review systematically discounted evidence of pollution-related health effects at contemporary pollution levels. Shy and other reviewers (*9–12*) contended that the cumulative weight of evidence suported the belief that particulate pollution may adversely affect human health even at relatively low concentrations. There has been a great increase in vehicular traffic over the past 20 years. The larger particles resulting from uncontrolled coal burning have been replaced in the urban environment by relatively high concentrations of much smaller particles, commonly 0.2 μm in size.

Much additional research has been conducted since the mid 1970s. While earlier research was useful in documenting the substantial health effects associated with large, dramatic pollution episodes, recent epidemiologic studies have provided more quantification of subtle health effects associated with particulate pollution common to contemporary cities in the developed world. Recent epidemiologic research has generally had better definitions and measures of pollution exposures and health endpoints. Advanced biostatistical and econometric techniques for longitudinal or cross-sectional analysis have greatly expanded opportunities to evaluate health effects of particulate pollution and have increased the analytical rigor of recent studies. For example, recent advances in autoregressive Poisson and logistic regression analysis have permitted the evaluation of pollution associations in panels or other small populations.

This review focuses on approximately 80 recently published epidemiologic studies, most since 1985, which evaluated effects of particulate pollution at concentrations commonly observed in contemporary cities in the developed world. For convenience, these studies are subdivided into general categories based on health endpoint and research methodology and are summarized in Tables 1–5. Separate critiques of each of the key papers are beyond the scope of this review. Although individual studies necessarily have limitations imposed by data deficiencies or by problems in analytical methods, when the whole body of contemporary research is viewed, it is possible to form a judgment concerning the validity of adverse health outcomes as a consequence of particulate pollution.

Acute Morbidity

Numerous studies evaluated acute morbidity effects of particulate pollution by examining short-term temporal associations between lung function measures and/or respiratory symptoms and pollution. Measures of lung function including forced vital capacity (FVC), FEV_1, and peak expiratory flow (PEF) were used. Most of these studies used formal daily time-series analysis, but some of them used periodic time-series analysis across one or more pollution episodes. A summary of the results of many of these studies is presented in Table 1.

Negative associations with particulate pollution and lung function measures were often observed. The particulate pollution effect on lung function was generally physiologically small but statistically signifi-

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This work was done partially while C.A.P. was on leave as a visiting scientist at the Environmental Epidemiology Program, Harvard School of Public Health, Boston, Massachusetts.

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cant. Because various measurements of particulate pollution were used, precise comparisons between the studies are difficult. Results of most of the studies suggest, however, that a 10 $\mu g/m^3$ increase in respirable particles (particulate matter ≤10 μm; PM_{10}) resulted in less than a 1% decline in lung function. Nevertheless, in several studies, 24-hr PM_{10} concentrations would occasionally exceed 150 $\mu g/m^3$. During such episodes lung function declined by as much as 7%. In addition to declines in lung function, many of these studies observed increases in respiratory symptoms. A 10 $\mu g/m^3$ increase in PM_{10} was typically associated with a 1–10% increase in symptoms such as cough, combined lower respiratory symptoms, and asthma attacks. These effects were also observed at comparable PM_{10} levels near or even below 150 $\mu g/m^3$.

An important limitation of these studies is that acute effects of particulate pollution on lung function are on average physiologically small and transient, remaining for a few days up to a few weeks. The importance of these small transient effects on a person's long-term well being is unclear. Furthermore, there is concern that these pollution effects may be due to confound-

Table 1. Selected time-series studies on acute effects of particulate pollution on lung function and respiratory symptoms

References	Health endpoints	Study area	Summary of findings
Braun-Fahrlander et al., 1992 (*13*)	Respiratory symptom episodes of 625 children	Basel and Zurich, Switzerland	Particulate pollution (TSP) was associated with incidence and duration of respiratory symptom episodes. No such associations were observed with SO_2 or ozone.
Dassen et al., 1986 (*14*)	Lung function (FVC, FEV_1, PEF) of 636 children	Ijmond, The Netherlands	Significant declines in lung function of 3–5% were observed during an episode of moderately elevated TSP and SO_2. Reductions remained for 16 (but not 25) days.
Dockery et al., 1982 (*15*); Brunekreef et al., 1991 (*16*)	Lung function (FVC, FEV_{75}) of approx. 200 children	Steubenville, OH	Small, significant decreases in lung function were observed after TSP/SO_2 pollution episodes. The association was strongest with previous 5-day mean TSP concentrations.
Johnson et al., 1982 (*17*)	Lung function (FVC, FEV_1, FEF_{25-75}) of children	Missoula, MT	Declines in lung function of 1–3% were associated with a "normal" urban air pollution episode. Comparable declines were not associated with a volcanic ash episode.
Johnson et al., 1990 (*18*)	Lung function (FVC, FEV_1, FEF_{25-75}, PEF) of children	Five Montana cities	Transient declines in lung function were associated with particles. In some cities sources were mostly wood smoke and entrained dust with low levels of SO_2 or O_3.
Hoek and Brunekreef, 1993 (*19*)	Lung function (FVC, FEV_1, PEF, MMEF) of children	Wageningen, The Netherlands	Negative associations between lung function and particulate pollution were observed. Consistent associations with respiratory symptoms were not observed.
Koening et al., 1993 (*20*)	Lung function (FVC, FEV_1) of 326 children	Seattle, WA	Lung function declines were associated with fine particulate air pollution for asthmatic children, but not for nonasthmatic children.
Kinney et al., 1989 (*21*)	Lung function (FVC, FEV_{75}, MMEF, $Vmax_{75}$) of 154 children	Kingston/Harriman, TN	Small decrements in lung function were associated with O_3 but not with particulate pollution, which had low concentrations with little variability.
Krupnick et al., 1990 (*22*)	Respiratory symptoms of 1000+ children/adults	Los Angeles, CA	Respiratory symptoms in both adults and children were associated with particulate pollution. An association with O_3 was observed for adults but not children.
Lebowitz et al., 1985 (*23*)	Lung function (PEF) and symptoms of 229 children/adults	Tucson, AZ	Declines in PEF were associated with elevated concentrations of TSP and O_3. Associations with smoking, gas stove use, and outdoor NO_2 concentrations were also observed.
Ostro et al., 1991 (*24*)	Respiratory symptoms of 207 adult asthmatics	Denver, CO	Several respiratory symptoms including cough and shortness of breath were associated with airborne acidic particulate pollution (H^+). Cough was also associated with $PM_{2.5}$.
Pope et al., 1991 (*25*); Pope and Dockery, 1992 (*26*)	Lung function (PEF) and symptoms of 100+ children and asthma patients	Provo/Orem, UT	Elevated PM_{10} levels were associated with significant declines in PEF, increases in respiratory symptoms, and increased use of asthma medication. Lagged associations were observed for up to 5 days. Particle acidity, SO_2, and O_3 levels were low.
Pope and Kanner, 1993 (*27*)	Lung function (FVC, FEV_1) of smokers with COPD	Salt Lake City, UT	Small transient negative associations between PM_{10} levels and lung function (FEV_1, FEV_1/FVC) were observed. This association was not entirely obscured by participants' smoking habit.
Raizenne et al., 1989 (*28*)	Lung function (PEF, FEV_1) in 112 girls	Girl's camp in Ontario	Maximum average declines in FEV_1 and PEF equal to 3.5 and 7%, respectively, were associated with elevated acid aerosol episodes.
Schwartz et al., 1994 (*29*)	Respiratory symptoms in children	Six U.S. cities	Cough and lower respiratory symtoms were positively associated with PM_{10}.
Whittemore and Korn, 1980 (*30*)	Asthma attacks of 443 asthmatics	Los Angeles, CA	Increased asthma attacks were observed on days with elevated levels of TSP and/or O_3.

Abbreviations: TSP, total suspended particulates; FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; PEF, peak expiratory flow; FEF, forced expiratory flow; PM_{10}, particulate matter ≤10 μm; COPD, chronic obstructive pulmonary disease.

ing by weather or some other factor that is not adequately accounted for in the analysis. This concern is partially mitigated by the fact that similar effects have been observed in locations with differing weather conditions including locations where particulate concentrations are high in the summer and where they are high in the winter. For example, effects were observed in studies from Switzerland, the Netherlands, Ohio, Montana, southern California, Arizona, Colorado, Utah, and southern Ontario. Effects were observed in communities where most of the pollution was from wood burning, steel mills, and related industry, or was composed of a complex mixture of aerosols and other particles that are characteristic of urban air pollution. Effects were observed in studies both with and without high concentrations of acid aerosols, sulfur dioxide, and ozone. Effects were observed for both children and adults. Effects were generally observed across areas where the primary source of particulate pollution was combustion, but very small or no effects were associated with large volcanic ash particulate concentrations (*17*).

Numerous studies have also evaluated acute morbidity effects of particulate pollution by examining short-term temporal associations between particulate air pollution and hospital admissions, health care visits, or other measures of restricted activity due to illness (Table 2). These studies usually used formal time-series analytical methods. Statistically significant associa-

Table 2. Selected studies on acute effects of particulate pollution on respiratory morbidity as measured by hospital admissions, health care visits, or other measures of restricted activity due to illness

References	Health endpoints	Study area	Summary of findings
Bates and Sizto, 1987, 1989 (*31,32*); Lipfert and Hammerstrom, 1992 (*33*)	Hospital admissions for 79 hospitals	Southern Ontario	Statistically significant associations between respiratory hospital admissions and sulfates and ozone were observed during summer months. No such associations with nonrespiratory admissions were observed.
Bates et al., 1990 (*34*)	Hospital emergency visits to 9 hospitals	Vancouver, British Columbia	Particulate concentrations were low, and associations with pollution variables were inconsistent. For ages 15–60, asthma and all respiratory visits were significantly correlated in summer with SO_2 and SO_4 levels.
Burnett et al., 1995 (*35*)	Admissions for 168 hospitals	Ontario	Significant positive associations were observed between respiratory and cardiac hospital admissions and previous-day sulfate levels.
Schwartz et al., 1993 (*36*)	Asthma emergency visits	Seattle, WA	Asthma visits were associated with PM_{10} (but not SO_2 or O_3) even at PM_{10} levels below 100 μg/m^3. An increase in PM_{10} equal to 30 μg/m^3 was associated with a 12% increase in asthma visits.
Thurston et al., 1992 (*37*)	Respiratory emergency visits	Buffalo, Albany, New York, NY	Associations between elevated summer haze pollution, including acid particles, particulate sulfate, and ozone, and total respiratory and asthma admissions were observed.
Lutz, 1983 (*38*)	Outpatient clinic visits	Salt Lake City, UT	Strong positive associations were observed between weekly particulate pollution levels and the percentage of patients with a diagnosis of respiratory tract or cardiac illnesses.
Ponka, 1991 (*39*)	Hospital admissions for asthma attacks	Helsinki, Finland	Hospital admissions for asthma were associated with NO_2, NO, SO_2, CO, O_3, and TSP. Associations were strongest for adults of working age, followed by the elderly.
Pope, 1989, 1991 (*40,41*)	Respiratory hospital admissions	Provo/Orem, UT	Strong, statistically significant associations between PM_{10} and respiratory admissions (especially children's bronchitis and asthma admissions) were observed.
Samet et al., 1981 (*42*)	Emergency room visits	Steubenville, OH	Statistically significant but small increase in respiratory emergency room visits were associated with elevated levels of TSP and SO_2.
Schwartz, 1994 (*43*)	Hospital admissions for elderly	Birmingham, AL	Admissions for pneumonia and COPD were associated with particulate air pollution and less strongly associated with ozone.
Schwartz, 1994 (*44*)	Hospital admissions for elderly	Minneapolis/St. Paul, MN	Admissions for pneumonia and COPD were associated with both particulate and ozone air pollution.
Schwartz et al., 1991 (*45*)	Medical visits for croup or obstructive bronchitis	Five areas in Germany	Associations between croup cases and TSP and NO_2 were observed. An increase in TSP from 10 to 70 μg/m^3 was associated with a 27% increase in croup cases. No associations with obstructive bronchitis and pollution were observed.
Schwartz, 1994 (*46*)	Hospital admission for elderly	Detroit, MI	Admissions for pneumonia and COPD (other than asthma) were associated with particulate and ozone air pollution.
Sunyer et al., 1991 (*47*)	Hospital emergency room visits for COPD	Barcelona, Spain	Emergency visits for COPD were consistently associated with particulate pollution (black smoke) and SO_2, even at 24-hr levels less than 100 μg/m^3.
Ostro, 1983, 1987, 1990 (*48–50*); Ostro and Rothschild, 1989 (*51*)	Restricted activity days of up to 12,783 adult workers	Cities in U.S. Health Interview Survey (HIS)	Respiratory morbidity was consistently associated with particulate pollution. Morbidity was often more strongly associated with the fine, respirable, or sulfate component of particulate pollution. Lagged pollution effects of 2–5 weeks were observed.
Ransom and Pope, 1992 (*52*)	Grade school absences	Provo/Orem, UT	Significant robust associations between grade-school absenteeism and PM_{10} were observed. These effects persisted for up to 3–4 weeks.

Abbreviations: PM_{10}, particulate matter ≤10 μm; TSP, total suspended particulates; COPD, chronic obstructive pulmonary disease.

tions between hospital/health care visits for respiratory illnesses and particulate pollution were observed in most, but not all, of these studies. Most of the studies suggested that a 10 μg/m^3 increase in PM_{10} on the day of the visit or 1 or 2 days before the visit was typically associated with a 1–4% increase in hospital visits. Significant associations between respiratory hospital admissions or related health care visits and particulate pollution were observed in many study areas including southern Ontario, British Columbia, Washington, Utah, Finland, New York, Ohio, Germany, and Spain.

A major concern about using hospital databases to evaluate effects of air pollution is the reliability of diagnoses and other information. Some studies have concluded that when only broad diagnostic classes are used, hospital databases provide reliable information for research on air pollution effects (*53*). Nevertheless, most air pollution studies have used existing databases without specifically evaluating data reliability.

Another concern about interpreting these studies is that the length of the lead-lag relationship of particulate pollution effects differed across studies. These differences may be partially due to disparities in type of health care visit and health care delivery systems across study areas. It may also be due to data analysis that only evaluated short-term effects. The study of inpatient admissions for respiratory disease in Utah Valley (*40,41*) found associations between admissions and air pollution levels up to a month or more before the visit. Several studies that used other measures of respiratory morbidity also observed long lead-lag times. Associations between particulate pollution in Utah Valley and absences of elementary schoolchildren had lead-lag relationships up to 4 weeks (*52*). Also, a series of studies by Ostro (*48–51*) observed that the association between particulate air pollution and days of respiratory morbidity serious enough to restrict activity (including loss of work, confinement to bed, or other restrictions) had a lead-lag time of 2 or more weeks.

Chronic Morbidity

Measures of lung function and incidence rates of respiratory symptoms have also been compared across communities or neighborhoods with different levels of particulate air pollution. Given the cross-sectional design of these studies and because pollution measures are averages over relatively long periods of time (1 year or more), these studies are often interpreted as evaluating chronic or cumulative effects of exposure rather than acute effects. Approximately 35 cross-sectional studies of lung function and/or respiratory symptoms were initially reviewed. Most reported associations between particulate pollution and either respiratory symptoms, lung function, or both. However, many of these studies included only a small number of communities and did not have individual data but relied only on aggregate measures of illness, limiting the amount of confidence that can be given to their results. In this paper, only studies using more sophisticated analysis and individual data for several or more study areas are included and are summarized in Table 3.

Small deficits in lung function (FVC, FEV_1, PEF) were generally associated with higher levels of particulate pollution and were often statistically significant. The results suggest that a 10 μg/m^3 increase in PM_{10} was typically associated with a decline of less than a 2% in lung function. Respiratory disease, including emphysema and chronic bronchitis, and the incidence of respiratory symptoms were also associated with particulate pollution. The results suggest that a 10 μg/m^3 increase in PM_{10} was typically associated with a 10–25% increase in bronchitis or chronic cough.

The major limitation of these studies may be the lack of true long-term individual exposure data. An individual's cumulative or long-term exposure to particulate pollution can only be estimated by using available data for the individual's area of residence. Even when available pollution databases allow for adequate estimation of pollution concentrations for a given area, there may be bias in exposure estimates due to selective migration from or into more polluted areas. For example, if persons most sensitive to pollution are more likely to move from polluted areas to less polluted areas, pollution effects would be underestimated.

Acute Mortality

Some of the most striking studies of health effects of particulate air pollution are those that observed changes in daily death counts associated with short-term changes in particulate air pollution (Table 4). Because various measurements of particulate pollution were used, precise comparisons between studies is difficult, but results of most of the studies suggest that a 10 μg/m^3 increase in

Table 3. Selected cross-sectional studies on chronic effects of particulate pollution on respiratory morbidity

References	Health endpoints	Study areas	Summary of findings
Chestnut et al., 1991 (*54*)	Lung function (FVC, FEV_1) of adults	49 U.S. cities from NHANES I	Small, statistically significant associations between lung function and TSP were observed.
Schwartz, 1989 (*55*)	Lung function (FVC, FEV_1, PEF) age 6–24	44 U.S. cities from NHANES II	Lung function was negatively associated with TSP, NO_2 and O_3, but not with SO_2
Vedal et al., 1991 (*56*)	Lung function and symptoms in children	Port Alberni, British Columbia	Particulate pollution was associated with prevalence of chronic respiratory symptoms, but not lung function.
Dockery et al., 1989 (*57*); Ware et al., 1986 (*58*); Speizer, 1989 (*59*)	Lung function (FVC, FEV_1, FEV_{75}, MMEF), respiratory symptoms of 5422 children	Portage, WI; Topeka, KS; Watertown, MA; Kingston, TN; St. Louis, MO; Steubenville, OH	Chronic cough, bronchitis, and chest illness were associated with particulates. Associations were stronger for children with a history of wheeze or asthma. Associations with lung function were negative but statistically insignificant.
Euler et al., 1987 (*60*)	Nonsmoking adult COPD symptoms	Areas in California	COPD symptoms were associated with particulate pollution and less strongly with SO_2 pollution.
Portney and Mullahy, 1990 (*61*)	Chronic respiratory disease	Cities in U.S annual Health Interview Survey (HIS)	Particulate pollution was associated with emphysema, chronic bronchitis, and asthma. Ozone was more associated with sinusitis and hay fever.
Schwartz, 1993 (*62*)	Chronic respiratory disease	53 U.S. Cities from NHANES I	Chronic bronchitis and respiratory diagnosis by a physician were associated with particulate pollution.

Abbreviations: FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 sec; NHANES, National Health and Nutrition Examination Survey; TSP, total suspended particulates; PEF, peak expiratory flow; MMEF, maximal midexpiratory flow rate; COPD, chronic obstructive pulmonary disease.

PM_{10} was associated with an increase in daily mortality equal to 0.5–1.5%. In some studies, lagged pollution effects of up to approximately 5 days were observed.

A few studies divided mortality by cause of death. Figure 1 presents relative risk ratios of mortality associated with similar increases in particulate air pollution in Philadelphia (*69*) and Utah Valley (*77*). Respiratory disease deaths were most strongly associated with particulate pollution levels, but statistical associations were also observed for cardiovascular disease deaths. Why cardiovascular mortality is associated with particulate air pollution is unclear. Reasons may include diagnostic misclassification, acute bronchiolitis precipitating heart failure, and/or effects of pollutants on lung permeability. A recent detailed examination of cardiovascular deaths on days with high particulate air pollution reported that most of the increase in cardiovascular deaths also had respiratory disease as a contributing factor (*70*).

These daily time-series studies have been partially summarized and reviewed (*81,82*). The daily time-series studies suggest that the association between particulate pollution and mortality are not due to confounding by weather, SO_2, or ozone. They also provide information on the age pattern of early deaths and causes of death. Most of them used Poisson regression analysis, allowing a comparison of effect-size estimates on a common scale. Time-series studies have observed effects in varied locations such as California, Utah, Michigan, Missouri, Tennessee, and Alabama in the United States, as well as England, Germany, Greece, and Brazil. It is highly unlikely that such concordance across so many locations could have occurred due to confounding or by chance. Many studies are in locations where particulate concentrations peak in the summer while others are in areas with winter peaks. In locations with winter peaking of particulate concentrations, ozone can be eliminated as a potential confounding factor. Most of the studies examined SO_2. The relationship between mortality and particles was generally independent of SO_2, while the SO_2 relationship disappeared when particles were considered. In Utah Valley and Santa Clara, California, SO_2 concentrations were low. Almost all the studies examined nonlinear relationships with weather factors. Both warm and cold climates and dry and humid locations have reported positive associations between air-

Table 4. Selected daily time-series studies on acute effects of particulate pollution on mortality

References	Study area	Summary of findings
Ostro, 1984 (*63*); Schwartz and Marcus, 1990 (*64*); Ito et al., 1993 (*65*)	London	Daily mortality was associated with particulate pollution (British smoke) and SO_2. The association seemed to be primarily due to particulate pollution but the overall air pollution effect could not be assigned to a specific pollutant with certainty. No threshold level was observed.
Schwartz, 1993 (*66*)	Birmingham, AL	Daily mortality was associated with PM_{10}. The association was strongest for respiratory deaths. An increase in PM_{10} equal to 100 $\mu g/m^3$ was associated with an 11% increase in mortality.
Ozkaynak and Spengler, 1985 (*67*)	New York, NY	Daily mortality was associated with particulate pollution (COH) and SO_2. Data limitations provided little opportunity to estimate separate effects of particles and SO_2.
Wyzga, 1978 (*68*)	Philadelphia, PA	An association between daily mortality and particulate pollution (COH) was observed. The estimated total deaths due to pollution equaled approximately 6%.
Schwartz and Dockery, 1992 (*69*) Schwartz, 1993 (*70*)	Philadelphia, PA	A 100 $\mu g/m^3$ increase in TSP was associated with an increase in mortality due to COPD, pneumonia, and cardiovascular disease equal to 19, 11, and 10%, respectively. On high pollution days, COPD, pneumonia, and dead-on-arrival deaths were disproportionately increased.
Shumway et al., 1988 (*71*) Kinney and Ozkaynak, 1991 (*72*)	Los Angeles, CA	Associations between daily mortality and particulate pollution were observed. Because of multicollinearity between pollutants, independent effects could not be estimated but mortality was not significantly associated with SO_2.
Mazumdar and Sussman, 1983 (*73*)	Three areas in Pittsburgh, PA	Small associations between daily mortality and particulate pollution (COH) were observed only for the area with the highest pollution levels. Emergency hospital admissions were also significantly associated with particulate pollution.
Dockery et al., 1992 (*74*)	St. Louis, MO; Kingston, TN	Mortality was associated with PM_{10}. The association was statistically significant only in St. Louis, yet in both areas an increase in PM_{10} of 100 $\mu g/m^3$ was associated with an approximately 17% increase in mortality. Associations with SO_2, aerosol acidity, or O_3 levels were not observed.
Schwartz, 1991 (*75*)	Detroit, MI	Daily mortality was associated with TSP levels. The association seemed to be independent of SO_2 and existed even at very low levels of pollution. A 100 $\mu g/m^3$ increase in PM_{10} was associated with a 6% increase in mortality.
Fairley, 1990 (*76*)	Santa Clara County, CA	Daily mortality was associated with particulate pollution (COH) at levels below 150 $\mu g/m^3$. Lagged effects were observed for at least 2 days. The association was stronger for respiratory mortality than for mortality due to other causes.
Pope et al., 1992 (*77*)	Provo/Orem, UT	Daily mortality was associated with PM_{10} pollution. The strongest association was with 5-day moving average PM_{10}. An increase in PM_{10} equal to 100 $\mu g/m^3$ was associated with an increase in deaths/day equal to 16%. The association was largest for respiratory disease deaths, next largest for cardiovascular deaths, and smallest for all other deaths.
Schwartz and Dockery, 1992 (*78*)	Steubenville, OH	An association between daily mortality and TSP was observed. This association seemed to be independent of SO_2 and was observed at particulate levels below current standards.
Saldiva et al., 1995 (*79*)	Sao Paulo, Brazil	Mortality of elderly persons was associated with PM_{10}, NO_x, SO_2, and CO. Only the association with PM_{10} was independent of other air pollutants.
Wichmann et al., 1989 (*80*)	North Rhine-Westfalia Germany	During a moderate pollution episode, mortality in the more polluted area was elevated by 8%. Hospital admissions were also elevated. Effects on cardiovascular diseases were larger than on respiratory disease.

Abbreviations: PM_{10}, particulate matter ≤10 μm; TSP, total suspended particulates; COPD, chronic obstructive pulmonary disease.

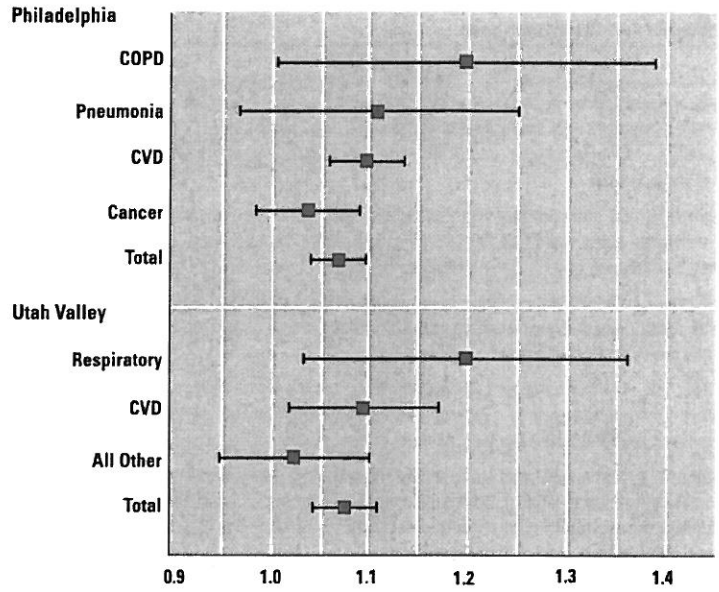

Figure 1. Relative risks of mortality in Philadelphia associated with a 100 μg/m^3 increase in total suspended particulates and in Utah Valley associated with a 50 μg/m^3 increase in suspended particles ≤10 μm diameter. COPD, chronic obstructive pulmonary disease; CVD, cardiovascular disease.

borne particles and mortality. They also covered over an order of magnitude range in airborne particle concentrations. Despite substantial variations in all of the potential confounding factors, the quantitative relationships between particles and daily mortality were essentially the same.

A particulate pollution threshold was not generally observed in these studies. Regression results remained relatively consistent even when pollution episodes that exceed air quality standards were excluded. Several studies, such as those in St. Louis and eastern Tennessee, were conducted in locations that never exceeded two-thirds of the ambient air quality standard, and all provided evidence of an exposure-dependent increase in mortality with particle concentration.

Chronic Mortality

Mortality effects of long-term or chronic exposure to particulate air pollution have been studied using two basic cross-sectional study designs. Population-based (ecologic) cross-sectional studies have correlated city-specific mortality rates with particulate air pollution (Table 5). These studies generally observe a positive association between mortality and various measures of particulate pollution. Most of the population-based cross-sectional studies observed strongest associations with fine particulate pollution or sulfate particulate matter. One of these studies evaluated health effects based on sources of particulate pollution and suggested that particles from the iron/steel industry or coal combustion are relatively more hazardous than soil-derived particles (*87*). These studies typically estimate that 2–9% of total mortality was associated with particulate pollution.

The major limitation of the cross-sectional population-based studies is their ecologic design, which does not permit direct control of individual differences in cigarette smoking and other risk factors. The strength of the relationship between mortality and particulate pollution was often sensitive to model specification, socioeconomic, demographic, and other risk factors included in the analysis and the choice of study areas included in the analysis.

Cross-sectional differences in mortality and air pollution were also studied in a prospective cohort study of 8,111 adults in 6 cities (*92*) and a larger study of over 500,000 adults in 151 cities (*93*). The prospective cohort design allowed for direct control of individual differences in age, sex, cigarette smoking, and other risk factors. In both studies fine particulate air pollution or sulfate particle concentrations were associated with mortality. Adjusted risk of mortality was approximately 15–25% higher in cities with the highest levels of fine particulate pollution compared to cities with the lowest levels. The results suggest that a 10 μg/m^3 increase in average PM_{10} exposure was associated with an increase in daily mortality equal to 3% or more. The strongest associations were observed with cardiopulmonary disease and lung cancer deaths, with only small, insignificant associations with death due to other causes.

Discussion

There are important concerns pertaining to these studies that reflect legitimate skepticism about inherent limitations imposed upon epidemiologic studies. Limitations and concerns relating to these studies tend to fall in three categories: 1) issues related to methodological or analytical bias, 2) issues relating to biological significance or plausibility, and 3) concerns about confounding.

It is unlikely that the overall effects of particulate air pollution are due to systematic methodological or analytical bias because the reasonably consistent findings from many differing study designs, data sets, and analytical techniques used. Many of the studies used simple, straightforward comparative statistical analysis coupled with more sophisticated statistical modeling techniques. Generally, the simple and the more sophisticated analysis observed similar associations between particulate air pollution and the health endpoint. Furthermore, recent reviews have noted considerable consistency across studies, especially the daily time-series mortality studies (*81,82,94*).

The epidemiologic studies, taken individually or as a whole, are severely limited with regard to establishing biological plausibility or providing information on specific biological mechanisms responsible for the observed effects (*95*). Recently, Bates (*96*) has noted the importance of biological plausibility but suggested that the coherence of epidemiologic studies of the health effects of particulate air pollution provide a convincing pattern. A recent review of the acute health effects of particulate air pollution also noted substantial coherence across various health endpoints (*94*). Clearly, biological plausibility is enhanced by the observation of a coherent cascade of respiratory health effects and by the fact that non-cardiopulmonary health endpoints were not typically associated with particulate pollution. Also, several authors have offered biological explanations for the observed relationship between fine-particulate air pollution and cardiopulmonary disease (*6,97*).

The most fundamental concern about the validity of these epidemiologic studies pertains to issues of confounding. Confounding may result when another risk factor that is correlated with both exposure and disease is not adequately controlled for in the analysis, resulting in spurious correlations. One proposed confounder is cigarette smoking. Although cigarette smoking contributes to baseline or underlying respiratory disease rates in a population, it could not be a common confounder across all the studies. For example, cigarette smoking would not be a confounder in the short-term time-series studies because 1) lung function, respiratory symptoms, and school absences studies were generally conducted among nonsmoking children; 2) the largest association between respiratory hospitalizations and pollution was often

Table 5. Selected cross-sectional studies on chronic effects of particulate pollution on mortality

References	Study areas	Summary of findings
Lave and Seskin, 1970 (*83*); Chappie and Lave, 1982 (*84*); Lipfert, 1984 (*85*); Evans et al., 1984 (*86*)	U.S. SMSAs	Statistical associations between mortality and particulate pollution were observed. The strength of the relationship between mortality and particulate pollution was sensitive to model specification, choice of social, demographic, and other variables included in the models, and the choice of SMSAs used in the analysis.
Ozkaynak and Thurston, 1987 (*87*)	U.S. SMSAs	Associations between mortality and particulate concentrations were relatively strong and consistent with sulfate and fine particles. Particles from the iron/steel industry and coal combustion seemed to be larger contributors to human mortality than soil-derived particles.
Mendelsohn and Orcutt, 1979 (*88*)	U.S. county groups	Statistically significant associations between mortality and sulfate particulates were observed. Smaller, less consistent associations with CO and SO_2 were observed. An estimated 9% of total mortality was associated with air pollution.
Lipfert et al., 1988 (*89*)	U.S. cities	SO_4, SO_2, NO_x, fine particles, and particulate trace metals (Fe and Mn) were associated with mortality. The data did not allow estimation of independent effects of these pollutants, but effects of SO_4 and fine particles were fairly consistent.
Bobak and Leon 1992 (*90*)	Czech Republic districts	Infant mortality, especially post-neonatal infant mortality, was consistently associated with particulate air pollution (PM_{10}). After adjusting for differences in socioeconomic characteristics, the relative risk of respiratory post-neonatal respiratory mortality was approximately 3.00 for most polluted areas versus least polluted areas.
Archer, 1990 (*91*)	Three counties in Utah	Spatial and longitudinal differences in death rates in three counties with low smoking rates and the introduction of a major pollution source were evaluated. It was estimated that 30–40% of respiratory disease deaths (approximately 5% of all deaths) were associated with the air pollution in the most polluted county.
Dockery et al., 1993 (*92*)	Six U.S. cities	Prospective cohort study directly controlled for individual differences in age, sex, cigarette smoking, and other risk factors. Statistically significant and robust associations between particulate air pollution and mortality were observed.
Pope et al., 1995 (*93*)	151 U.S. cities	Prospective cohort study included over >500,000 subjects followed up for 8 years. After controlling for individual differences in age, sex, cigarette smoking, and other risk factors, fine and sulfate particulate pollution was associated with mortality (mostly cardiopulmonary mortality).

Abbreviations: SMSA, standard metropolitan statistical area; PM_{10}, particulate matter ≤10 μm.

between young, nonsmoking children; and 3) cigarette smoking does not change day-to-day, week-to-week, or month-to-month in positive correlation with air pollution. Cigarette smoking is a more plausible confounder in some of chronic morbidity and mortality studies. However, even in these studies, smoking is an unlikely common confounder because the estimated pollution effects were observed after analytically controlling for cigarette smoking or restricting the analysis to never-smokers.

Another set of proposed confounders are socioeconomic factors. As with cigarette smoking, socioeconomic status in a population does not change day-to-day in correlation with air pollution. Therefore, socioeconomic status was not a potential confounder in the short-term time-series studies looking at lung function, respiratory symptoms, school absences, outpatient visits, and mortality.

Temporal multicollinearity makes confounding by weather and seasonal variables a concern. The studies often observed various weather and seasonal effects. Several observations pertaining to the studies as a whole mitigate the prospect of weather and/or seasonal variables being common confounders: 1) daily, seasonal, or annual changes in weather were not potential confounders in the chronic mortality and morbidity studies; 2) in most of the shorter-term time-series studies, at least some attempts to control for weather and/or seasonal effects were part of the analysis; 3) the study period for some of the acute studies were conducted only during single seasons, eliminating potential confounding by seasonal or annual changes in weather; and 4) the estimated pollution effects are reasonably consistent for areas with different climates and weather conditions.

It is extremely unlikely that several different confounders for the different studies would coincidentally cause spurious correlations that were coherent across the different studies and different health endpoints. The most likely common confounder that could be responsible for the effects observed is another pollutant or combination of pollutants that are highly correlated with particulate pollution. However, it is difficult to determine which other pollutant(s) may be the confounder. Similar particulate-related health effects have been observed in areas where sulfur dioxide, ozone, and aerosol acidity levels are low or poorly correlated with particulate pollution compared with areas with relative high levels of these pollutants. Given current data, proposing additional potential confounding pollutants is largely speculative. It may be that the true culprit pollutant is a constituent of particulate mass such as combustion particles, sulfate particles, fine- or ultra-fine particles. It may also be possible that various measures of particulate exposure are serving as proxies for an unknown or unmeasured pollutant or combination of pollutants.

Conclusions

There is substantial body of contemporary epidemiologic research that has explored health effects of particulate air pollution at levels common to contemporary cities in the developed world. Observed health effects of respirable particulate pollution include: increased incidence of respiratory symptoms, decreased lung function, increased hospitalizations and other health care visits for cardiopulmonary disease, increased respiratory morbidity as measured by absenteeism from work or school or other restrictions in activity, and increased cardiopulmonary disease mortality. Health effects are observed at levels common to many U.S. and Canadian cities, including levels well below current U.S. National Ambient Air Quality Standards. There is no clear evidence of a safe threshold level. Many studies observe that health effects increase monitonically with pollution levels, often with a near-lin-

ear dose–response relationship.

When a substantial body of epidemiologic evidence indicates that a material to which people are commonly exposed may be having serious adverse health effects the burden of proof may be deemed to have shifted from those who draw a causal inference, to those who maintain no causal inference is possible (*98*). The latter should be required to explain the consistency and coherence of the large body of evidence and put forward alternative hypotheses to explain the findings.

Based on our evaluation of this recent research, there is enough consistency and coherency of results across a large number of studies and a wide range of expected outcomes, methodologies, study areas, and researchers to merit a reassessment of the importance of fine and/or respirable particulate pollution on cardiopulmonary health. A research emphasis should be directed at elucidating the mechanisms behind the epidemiological data. It is unclear why morbidity and mortality should be so closely linked to respirable particulate pollution. Nonetheless, Sir Austin Bradford Hill (*99*), in his famous lecture in 1965, warned us that we should not require mechanistic understanding before making the inference of causality from associative epidemiological studies.

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Respiratory Health Effects of Air Pollution: Update on Biomass Smoke and Traffic Pollution

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Abstract

Go to:

Mounting evidence suggests that air pollution contributes to the large global burden of respiratory and allergic diseases including asthma, chronic obstructive pulmonary disease, pneumonia and possibly tuberculosis. Although associations between air pollution and respiratory disease are complex, recent epidemiologic studies have led to an increased recognition of the emerging importance of traffic-related air pollution in both developed and less -developed countries, as well as the continued importance of emissions from domestic fires burning biomass fuels primarily in the less-developed world. Emissions from these sources lead to personal exposures to complex mixtures of air pollutants that change rapidly in space and time due to varying emission rates, distances from source, ventilation rates, and other factors. Although the high degree of variability in personal exposure to pollutants from these sources remains a challenge, newer methods for measuring and modeling these exposures are beginning to unravel complex associations with asthma and other respiratory disease. These studies indicate that air pollution from these sources is a major preventable cause of increased incidence and exacerbation of respiratory disease. Physicians can help to reduce the risk of adverse respiratory effects of exposure to biomass and traffic air pollutants by promoting awareness and supporting individual and community-level interventions.

Keywords: biomass, traffic, COPD, asthma, air pollutants, particulate matter

77

Introduction

Go to:

Worldwide increases in rates of asthma and COPD over the past several decades have motivated intensive investigation of the role of environmental factors, including air pollution, in their causation. Recent research also suggests that air pollution contributes to the substantial worldwide burden of disease from acute lower respiratory infections and possibly tuberculosis. While the health effects of air pollution have been an international public health concern since at least the 1950's, recent research has heightened the focus on two broad sources of air pollution: biomass fuels (BMF) and motor vehicles. Understanding of the health effects of BMF and traffic -related air pollution (TRAP) has lagged behind that of ambient air pollution, at least in part due to challenges in estimating highly-variable individual exposure from these widespread, but very localized, air pollution sources.

Of course, air pollution is only one of many environmental (non-genetic) factors for which a causative role in exacerbation or incidence of complex respiratory diseases has been suggested. Indeed, based on ecological analyses from the International Study of Asthma and Allergies in Childhood (ISAAC)[1], generally less-polluted developed countries (DCs) have much higher rates of asthma than many countries with higher levels of air pollution. However, studies with individual-level analyses that control for potential confounding have demonstrated associations between air pollutants, including TRAP, and asthma exacerbation, as well as possible links to increased asthma incidence. Additional evidence suggests that exposure to TRAP is correlated with the rising rates of allergic respiratory disease.[2] Although tobacco smoke is clearly the dominant cause of COPD worldwide, BMF smoke is now recognized as a major cause of COPD, especially among women in less developed countries (LDCs). Current evidence also indicates that BMF smoke plays a causative role in mortality from lower respiratory infections among children living in homes where BMF is used. The effects of indoor air exposures and individual ambient pollutants on asthma have recently been discussed in this forum.[3] Here, we emphasize the growing body of recent research pertaining to the relationship between respiratory health effects and exposure to TRAP and biomass air pollution. For recent reviews of the respiratory health effects of traffic exposures see Kelly and Fussell (2011)[4] and Salam et al. (2008)[5], and for BMF see Torres-Duque C (2008)[6], Balmes (2010)[7], Fullerton (2008).[8]

Exposures to Biomass and Traffic Pollutants

Go to:

Exposures to BMF smoke and TRAP are widespread. Domestic fires burning biomass (wood, charcoal, dung, crop residues, and other raw plant materials) for cooking and/or heating remain the most pervasive and important source of exposure to air pollution for much of humanity. About 2.4 billion people live in households in which BMF is the primary cooking and/or heating fuel[9–10], with more than 90% of individuals in rural areas of LDCs using BMF.[9] Exposures are often exacerbated by use of open fires or traditional stove designs that lack flues or hoods to exhaust emissions away from the living area.[7]

While stationary industrial "smokestacks" continue to be a major source of outdoor air pollution from the burning of fossil fuels throughout the world, TRAP from motor vehicles are a growing

78

concern in both DCs and LDCs.[11] Regulation of ambient (widespread, regional) 'criteria' pollutants in the US and other DCs has resulted in relatively effective stationary and mobile source controls. However, an increased number of vehicles and vehicle-miles-driven has lessened the impact of vehicle emissions controls. At the same time, heavy industry has moved to LDCs, resulting in a higher relative contribution from mobile sources in DCs, while in LDCs both total stationary and mobile source emissions have been increasing. Most of the worldwide growth in fleets of gasoline and diesel vehicles is occurring in LDCs.[11] As discussed below, in addition to making large contributions to background ambient air pollution, mobile sources may dominate exposures near roadways in urban areas, where a growing proportion of the world's population lives.

Although one is rooted in longstanding cultural practices and the other arises from modern economic development, emissions from domestic fires and from motor vehicles have similarities in composition, toxicity, and exposure characteristics. Complex aerosol emissions from use of BMF share many components with TRAP and other outdoor PM, including particulate matter, carbon monoxide, nitrogen oxides, and scores of toxic organic compounds, such as formaldehyde, acrolein, and polynuclear aromatic hydrocarbons (PAHs).[12] However, the physical and chemical characteristics of both BMF smoke and TRAP can vary substantially depending on the type of fuel burned and combustion conditions.[13]

Based on robust epidemiological associations between ambient particulate matter (PM) and respiratory and cardiovascular health effects, much attention has focused on the PM component of BMF and motor vehicle emissions.[14] PM of respirable size is classified by size fractions based on aerodynamic diameter. Ultrafine PM (UFP) with diameters ≤ 0.1 μm (PM0.1) is a major component of emissions near fires and tailpipes, but in seconds to minutes accumulates into somewhat larger "fine PM," or $PM_{2.5}$ with diameters ≤ 2.5 μm, within short distances from the point of release. PM_{10}, with diameters ≤ 10 μm, consists of $PM_{2.5}$ and larger particles of mainly crustal or biological origin, including many aeroallergens. Based on epidemiological and laboratory studies, $PM_{2.5}$ appears to be more potent for respiratory and cardiovascular disease effects compared to PM10.[15] According to the "ultrafine hypothesis," ultrafine PM may be still more toxic due to increased surface area and other characteristics.[16] Although they contribute little to the mass concentration of PM due to their small size, ultrafine particles emitted by combustion dominate the particle number concentrations near these sources.

In contrast to large-scale industrial sources of air pollution, the sources of biomass and traffic emissions tend to be in close proximity to individual "receptors." Biomass emissions occur primarily indoors, where women and children are most highly exposed during cooking and other domestic activities. Exposures are exacerbated by reduced ventilation in homes where biomass in used[7], or under conditions in which vehicle emissions may be concentrated as in urban street canyons or tunnels.[17] Concentrations of TRAP have steep gradients near roadways, with heightened exposure to individuals living, attending school, or working near major roads in urban areas, and return of TRAP to background levels within several hundred meters away from roadways.[18–19] For both BMF emissions and TRAP, time-activity patterns are a critical determinant of exposure. Household members who cook have high peak exposures, as when

standing over the fire, as well as high time-averaged exposures to biomass pollutants.[20] Individuals living and working in urban areas may have a substantial part of their daily air polllutant exposure during usually relatively brief commuting times on roadways where TRAP are concentrated.[21–22] Substantial differences in TRAP concentrations and in inhaled doses as a consequence of travel mode, biking vs car vs bus, have been demonstrated, with bicyclists generally having the highest doses and electric bus riders the lowest.[23] Moreover, with distance and time away from sources, both BMF and vehicle emissions undergo complex "aging" processes that include oxidation, other chemical reactions and physical processes that alter exposure and toxic properties in ways that are not fully understood.[24]

The levels of BMF air pollutants measured in homes are typically far higher than ambient air pollutants, but they have received less attention from the international research community. Concentrations of PM and other air pollutants in indoor air during biomass burning can be orders of magnitude higher than levels that occur in ambient air in developed cities.[20] Levels of PM_{10} in homes using BMF often exceed several thousand $\mu g/m^3$ [20], compared to the EPA 24-hr ambient air quality standard of 150 $\mu g/m^3$ PM_{10} and the WHO guideline of 50 $\mu g/m^3$ PM_{10}.[25]. Little data is available on $PM_{2.5}$ and UFP indoors from BMF burning. The near-roadway microenvironment is mainly impacted by freshly-emitted UFPs and gas-phase compounds such as carbon monoxide, nitrogen oxides, and VOC's although resuspended road dust, mainly in the "coarse" mode of the PM_{10} fraction, may be an important exposure.[11]

The uneven distributions of exposure to BMF smoke and TRAP leads to uneven distribution of health risks, and environmental justice considerations at local, regional, national, and global scales.[26] There are age, gender, and socioeconomic differences in who is most exposed and most vulnerable to the health effects of BMF emissions and TRAP.[8] Exposure to BMF smoke is greatest among women and among young children who may be carried on mother's back during cooking activities, or spend more time indoors with mother.[26] BMFs that are least expensive and more affordable for impoverished households also burn less efficiently, increasing pollutant emissions.[8] TRAP exposures are concentrated in areas of greater traffic density, which, at least in the US, tend to be inner city communities of lower socioeconomic status with a higher burden of environmental contamination/impacts.

Epidemiology of Health Effects of BMF and TRAP

Go to:

Individual exposure assessment has been a major challenge for epidemiological studies of both BMF and TRAP, in contrast to studies of ambient air pollution, in which assigning personal exposure based on central air monitoring data has had demonstrated utility. Few epidemiologic studies of respiratory effects of BMF smoke have measured exposure, relying instead on self-report of fuel use, despite evidence for wide variation in exposure depending on combustion and ventilation conditions and time-activity patterns. More recent studies, including controlled trials of stove interventions, have begun to measure exposure. As described above, the spatial and temporal distributions of urban air pollutants are characterized by significant variability with steep gradients in intensity near sources.[27–28] Thus, the use of land use regression and other techniques for modeling microenvironmental exposures for various particle and gas pollutants

has become widespread and is featured in a number of the newer studies of TRAP to be described below. Land-use regression (LUR) uses the monitored levels of the pollutant of interest as the dependent variable and variables such as meteorology, traffic, topography, building shapes and sizes, and other geographic variables as the independent variables in a multivariate regression model.[29] Levels of pollution may then be predicted for any other geographic locations, such as residences or schools, using the parameter estimates derived from the regression model. A limitation is that LUR often captures only one time period, and may miss prior or neonatal exposures, however some studies have overcome this.[30–31] Further details are available in recent reviews.[32–33]

The most firmly established health effects of BMF emissions are acute lower respiratory infections in children and COPD in adults. While studies of ambient pollution effects have repeatedly demonstrated increased cardiovascular and respiratory morbidity and mortality for a variety of outcomes[14], those which have studied TRAP specifically have largely focused on asthma and related phenomena, with some investigations of allergy. The World Health Organization has estimated that BMF smoke exposure is responsible for about 1.5 million premature deaths per year,[34] and a global burden of disease of approximately 2.5% of all healthy life-years lost. Most of this burden of disease is due to respiratory infections, mainly among children less than 5 years of age, and COPD among adult women[35]. Several case-control and cross-sectional studies have evaluated associations between use of biomass fuels and prevalence of asthma with equivocal results among children and women.[36–43] Other studies have found strong associations between BMF smoke and COPD among non-smoking women.[9, 20, 44] Important new studies, discussed below, have strengthened the link between TRAP and asthma incidence in children,[30, 45–47] incidence in adults[48] and severity in adults.[49]

BMF and COPD

Go to:

Given that cigarette smoke is a type of biomass smoke, a causal association between exposure to BMF smoke and COPD would not be surprising. Three recent meta-analyses have evaluated associations between BMF smoke and COPD.[50–52] In a systematic review and meta-analysis of 23 studies, Kurmi et al found that exposure to all types of BMF smoke was consistently associated with COPD with risk more than doubled, with greater risk suggested for woodsmoke compared to other fuels.[50] Hu et al analyzed 15 studies and found that BMF smoke was associated with increased risk of COPD among both women and men, and in both Asian and non-Asian populations.[52] In a meta-analysis of 6 studies that evaluated COPD among women using biomass compared to alternative fuels, Po et al also found a statistically significant pooled estimate of greater than 2-fold increased risk.[51] In another 6 studies that assessed chronic bronchitis, the pooled risk estimate was also greater than 2-fold. However, most studies have lacked direct exposure measurements, none have described a dose-response relationship, and estimated effect sizes have varied widely. This variation may be due to heterogeneity in fuel types and conditions of use, as well as in study design and differences in control of confounders such as exposure to mainstream or second-hand cigarette smoke, occupational exposures, socioeconomic factors, and changes in fuel use over time.[50] In a study of 841 nonsmoking

women in Mexico that was notable for objective exposure and outcome measurements, Regalado et al found that peak PM_{10} over 2,600 μg/m3 among those using biomass fuels was related to small, but significant, reductions in FEV1 (81 ml), FVC (122 ml), and FEV1% predicted (4.7) compared to women who cooked with gas.[53] In an accompanying editorial, Jaakola noted that these effects were comparable to the estimates from environmental tobacco smoke exposure in adults.[54] Cigarette smoking rates are relatively low in most LDCs, especially among women.[55] Among women living in rural Turkey, the fraction of COPD attributed to exposures to BMF smoke was 23% after adjusting for possible confounders.[56]

TRAP AND COPD

Go to:

A number of studies have established that children living in more polluted areas,have reduced lung growth compared to those living in cleaner areas, and that moving from a more polluted to a cleaner area demonstrates improved growth.[57] Similar findings for lung function have been reported in adults, as well as a limited data base of studies documenting an association between ambient air pollutants and objectively defined COPD.[57]

Most recently, a 35 year prospective study of over 57,000 Danes, used individual modeled assignments of traffic pollution and extensive control of confounders, with the end point of first hospital admission for COPD.[58] This outcome was associated with chronic NO_2 exposure (HR=1.08, 1.02–1.14) with a stronger association in asthmatics. This is the first longitudinal study of COPD with hard outcomes in association with modeled TRAP exposures, and seems to confirm the previous findings of cross-sectional studies that TRAP is likely to be a cause of COPD.

TRAP and Asthma Overview

Go to:

Studies have long shown that asthma can be exacerbated, often measured as visits to emergency rooms, on days with higher levels of ozone and other pollutants.[59] More complex cohort study designs have been required to understand whether or not traffic-related pollutants play a role in the genesis, or causation, of asthma. To date all such investigations in a non-occupational setting have related to chronic, rather than acute, air pollution exposures. More sophisticated designs have been used in recent years and this part of the review will focus on those reported since 2009.

TRAP and Childhood Asthma

Go to:

Initial studies of air pollution and asthma examined associations in children, looking first at exacerbations and more recently, through cohort studies, at incidence. Initial reports of incidence in children were variable in their results. Investigators interpreted these inconsistencies to result from misclassification of pollution exposure to the individual cases, likely due to reliance on central or regional air monitoring stations that do not reflect urban microgeographies. Subsequent studies using LUR or dispersion modeling produced significant associations in both children. and adults, with refinement of exposure through use of LUR techniques yielding larger and significant associations with chronic pollution exposure.

(82)

Two recent studies, one a birth cohort, add substantially to our confidence that TRAP exposure of young childen contributes to the development of asthma.[30, 45] Both used sophisticated exposure assessment in the form of LUR or a related technique to study the association between childhood asthma and TRAP exposure at home and/or school. Increased risk of for childhood asthma incidence demonstrated significant increases of 26% up to 51%, with good control of relevant confounders. Interestingly the birth cohort study did not find corresponding associations of potentially explanatory mechanistic variables such as atopic eczema, allergic sensitization, and bronchial hyperresponsiveness, leaving open questions about pathophysiology and roles of irritancy versus allergy. For the Southern California Children's Study[60] non-freewway pollutants demonstrated a stronger effect than those from freeways, possibly reflecting an effect of frequent acceleration and deceleration on TRAP characteristics.

In a smaller study, Carlsten et al (2011)[31] recruited infants at high familial risk for asthma and examined birth year home exposures to NO, NO_2, black carbon, and $PM_{2.5}$ by land use regression with follow up at 7 years of age. Birth year $PM_{2.5}$ (IQR=4.1ug/m3) was associated with a significantly increased risk of asthma with an OR of 3.1 (1.3–7.4). NO and NO_2 demonstrated similar associations but black carbon did not. This dramatic finding with relatively small exposure magnitude is intriguing but needs replication.

A study of self-reported allergic disease (using the ISAAC questionnaire) and home traffic density based on distance to major roadways, found approximately 1.5 to 3 fold prevalence ratios for heavy traffic density for wheeze, asthma, rhinitis and rhinoconjunctivitis, with no associations for children who slept in air conditioned homes,[46] with obvious important implications for prevention in atopics and others at heightened risk.

Using a cross sectional design and an "enhanced" ISAAC protocol for outcomes,[47] 6683 children in the French Six Cities Study were studied, with exposures based on a 3-year dispersion model for each school address to assign individual school exposures,. Asthma (either past year, or lifetime) was significantly associated with benzene, SO_2, PM_{10}, NOx, and CO. All of those but SO_2 were associated with eczema, and allergic rhinitis with PM_{10},. Sensitization to pollens was associated with benzene and PM_{10}, The findings for benzene and CO are somewhat surprising, and given their presence as constituents of motor vehicle fuel and/or exhaust, uncontrolled confounding may be present.

TRAP and Adult Asthma

Go to:

The recent data base on asthma and traffic is less robust in adults. The Swiss Cohort Study on Air Pollution and Lung Diseases in Adults (SAPALDIA) is a Swiss population-based cohort of adult lung disease-free non-smokers initiated in 1991 with 11y follow in 2002.[48] Using a dispersion model that included hourly meteorological and emission data on industrial, construction, heating, agricultural and forestry, and traffic emissions, the latter separated by type of vehicle (truck vs. car), each participant was assigned an exposure to PM_{10}. Outcomes were adjusted for age, gender, baseline atopy, BMI, bronchial reactivity, and maternal allergies. They found a hazard ratio for doctor-diagnosed asthma of 1.30 (1.05–1.61) new cases for a given

(1ug/m3 as PM_{10}) change in traffic pollution over the 10 years, more frequent in those with baseline atopy or bronchial hyperreactivity.

Trupin et al looked at the simultaneous impact on FEV1 percent and an asthma severity score of diverse social and physical environmental exposures on adult asthma in 176 subjects. Their final model had an R^2 of 0.30 for FEV1 percent predicted and 0.16 for Severity of Asthma Score. Distance to nearest road was a significant predictor of FEV1 but not Severity of Asthma Score. The importance here is that even when other variables strongly associated with usual clinical management of asthma are accounted for, a role for roadway traffic still persists.

Based on high quality studies discussed herein there is an increasingly robust literature that supports a causal relationship between various aspects of TRAP and new onset asthma or worsened asthma in children and adults. These risks need to be both incorporated into both public policy and explored for their role in medical decision making at the individual level.

BMF and asthma

Go to:

In contrast to the abundance of studies showing exacerbation of asthma from increased exposure to ambient air pollution and TRAP, asthma prevalence has been the main outcome considered in studies of BMF smoke. Using Burden of Obstructive Lung Disease (BOLD) data on self-reported prevalence of asthma among 508 individuals in Southeastern Kentucky, increased odds of reporting current asthma was associated with cooking indoors with wood or coal for more than 6 months of one's life (OR 2.3, CI 1.1–5.0), but not with history of domestic heating with wood or coal (OR 0.8, CI 0.4–1.8).[61] However, a handful of earlier studies had not found compelling evidence of an increased risk of asthma among women or children in households using BMF.[36, 40] Among six studies that examined risk of asthma among women using BMF in rural India, Iran and Turkey, two found a statistically significant increased risk,[62] and two showed increased risks that were not statistically significant.[38, 41] Using national health survey data, Mishra (2003)[62] found that elderly Indian men and women who lived in households using BMF had a higher prevalence of self-reported asthma compared to those who used cleaner fuels (OR=1.59, CI 1.30–1.94). Also in India, Padhi[63] found increased physician-diagnosed asthma and decline in lung function among rural biomass burners. In a recent meta-analysis, Po et al (2011)[51] found that pooled risk estimates did not provide evidence of overall increased risk of asthma in children or women using BMF. Limitations of the available studies include likely exposure misclassification, outcome misclassification, low power, and/or incomplete control for confounding.

Respiratory Infection

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The WHO has concluded that exposure to indoor air pollution doubles the risk of pneumonia and other acute lower respiratory infections, and may account for half of the roughly 800,000 annual worldwide deaths in children under 5 years of age attributed to pneumonia.[25, 64] Dherani et al conducted a meta-analysis of 24 studies, and found that exposure to BMF increased the risk of pneumonia by almost 2-fold.[65] In a meta-analysis that included 8 studies, Po et al found a greater than 3 fold increased risk of acute respiratory infection in children.[51] Deaths among

84

children contribute disproportionately to years of life lost in global burden-of-disease calculations. Increased rates of chronic bronchitis and viral infection have been associated with both gaseous and particulate ambient pollutants, although not specifically with TRAP.[4, 66]

Tuberculosis

Go to:

Greater use of BMFs appears to be correlated with higher rates of TB infection in global geographic regions, but few studies have evaluated associations at the individual level. In a cross-sectional study of a large national sample of Indian households, Mishra 1999 found increased risk of self-reported TB infection with BMF use (OR 2.58, CI 1.98–3.37), but neither active nor passive smoking was measured. In a later well-designed case-control study in India, Shetty et al 2006 found that risk of bacteriologically or radiographically-confirmed TB was not increased after adjustment for smoking, education, income and other possible confounders.[67] Although a recent systematic review concluded that there was not sufficient epidemiological evidence to support an association between BMF and TB infection,[68] and no epidemiological associations between TRAP and TB have been reported, ambient particles and diesel exhaust particles have been shown to impair macrophage function in animal models, suggesting that such associations are biologically plausible.[69–70]

Mechanistic Insight

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As discussed above, oxidative stress is a commonly cited mechanism for the relationship between air pollutants, many of them with oxidant constituents, and asthma worsening or onset. Both particles and gases may produce oxidative stress and may act in concert. Polymorphisms of GSTM1, GSTP1, and TNF-alpha are all reported to have associations/interactions with asthma and air pollution, but data are not consistent enough to allow firm causal conclusions. [71–72] Further support for oxidative stress as an explanatory mechanism as to how TRAP exerts effects on intracellular regulation of inflammation and the oxidative stress response comes from an experimental study of air pollution aerosols, including fresh diesel exhaust demonstrating approximately a 10% decease in WBC proteasome activity following 2 hr of aerosol exposure. [73]

Diesel exhaust inhalation is frequently used as a model for acute inhalation of TRAP. Acute exposure to diesel exhaust in a real-world street canyon setting has been shown to significantly reduce pulmonary function in asthmatics (up to 6% decline in FEV1) along with an increase in sputum inflammation as measured by myeloperoxidase[74]. However, experimental exposures to diesel exhaust, despite showing increased airway reactivity in asthmatics, have not elicited evidence of airway inflammation in asthmatic subjects, in surprising contrast to elicitation of inflammatory changes in healthy subjects.[75–77] Diesel exhaust particles have been shown to have adjuvant effects on IgE synthesis in atopics, so that allergen-specific IgE production upregulates by as much as 50-fold with a skew towards a TH2 profile.[78] Diesel exhaust has been shown to acutely produce human bronchial epithelial inflammation characterized by inflammatory cell recruitment, increased expression of vascular endothelial adhesion molecules, cytokines, mitogen-activated protein kinases, and transcription factors. It has been proposed that

85

epithelial damage from diesel exhaust may lead to decreased mucociliary clearance and consequent increased access of allergens to immune cells in the mucosa.[76–77] Another recent study examined diesel exhaust produced under realistic conditions to simulate actual driving conditions and emissions.[79] Evaluating inflammatory markers 6 hours after a 1 hour exposure, they found increased expression of p-selectin ($p=0.036$) and vascular cell adhesion molecule-1 ($p=0.030$) in bronchial mucosal biopsies as well as the novel finding of increased eosinophils in bronchial alveolar lavage ($p=0.017$), not previously seen under idling engine conditions. The implications for diesel potentiation of allergic respiratory disease are substantial, especially in light of previous experimental work.[80]

To improve understanding of biological pathways underlying respiratory and cardiovascular effects, a number of panel or experimental studies have measured biomarkers of oxidative stress and inflammation following exposure to TRAP.[63] Examining respiratory effects associated with studies in commuters, there were modest effects of two hour commuting exposures on peak flow, eNO, and airway resistance.[81] Particle number (PN) doses were associated with decreased maximum mid-expiratory flow (MMEF) and FEV1 6 hours after exposure. PN and soot were associated with decreased MMEF and FEV1 immediately after exposure, and increased FeNO after car and bus but not bicycle trips. PN was also associated with an increase in airway resistance immediately but not 6 hours following exposure. There were no associations of exposures or doses with symptoms. They interpreted these findings in healthy individuals to show modest effects of a 2-hour in-traffic exposure on peak flow, eNO, and airway resistance. Examining inflammation and coagulation as a consequence of TRAP exposure, an accompanying study in the same subjects found no consistent associations in blood cell counts, CRP, IL-6, IL-8, IL-10, and TNFa, aPTT, fibrinogen, Factor VII, vWF, and CC 16 6 hours after the commute.[82] Thus these data do not indicate that short term changes predict the serious long-term consequences seen with chronic exposure.

Pollution Intervention studies

Go to:

Beijing Olympics Intervention Studies

Natural (or politically organized) changes in the environment are viewed by researchers as great opportunities to study the effects on human health of greater than usual degrees of independent variable (pollution) change. This has been applied to the effects of sudden or dramatic changes in air pollution.[83–84] When these changes are anticipated, detailed clinical studies can be designed.[85] One such example was the Beijing Olympics of 2008. (see Figure 1) One study that came out of this examined visits for outpatient treatment of asthma at a Beijing Hospital.[86] During the Olympics, the Chinese government endeavored to reduce air pollution by substantial amounts.[87] While somewhat sparse in clinical detail, they reported a reduction from 12.5 visits per day to 7.3 visits per day, a 41.6% reduction during the Olympic Games.

Figure 1

Cooking with wood biomass fuel in Nigeria.

Also based on the Beijing Olympics, Lin et al (2011)[88] measured serial FeNO as a function of ambient black carbon, a marker of diesel exhaust, in 36 fourth grade Beijing children, before, during, and after the 2008 Beijing Olympics.[88] FeNO was significantly lower during the Olympic period, and increased 16.6% (14.1%–19.2%) per interquartile range increase in BC, particularly in the first hours after exposure, suggesting rapid changes in inflammation. Asthmatics were not significantly different from healthy children.

Intervention Studies for BMF

Romieu (2009)[89] randomized an improved stove (Patsari stove) among 668 households in central Mexico where open wood-burning fires were used for cooking. The stoves had been shown in previous studies to reduce indoor air pollution levels by 70%.[90] At one-year follow-up, among the 50% of households still using the stove, there was a significant reduction in respiratory symptoms and a significantly lower decline in FEV1.[89] Using a randomized controlled trial, Smith et al found that an improved cooking stove halved average exposure to carbon monoxide, but did not significantly reduce physician-diagnosed pneumonia among infants in Guatemala.[91] However, there was a significant reduction in severe pneumonia, and a 50% reduction in exposure was significantly associated with a lower rate of diagnosis of pneumonia. Other randomized controlled trials of improved stoves are underway, but results are yet to be published (Bruce 2007).[92]

Clinical Guidance

Go to:

Reducing the impact of BMF smoke and TRAP on respiratory health will require both public policy and the actions of individual patients. Consensus standards recognize the importance of air pollutants in the prevention and management of asthma and COPD, and have recommended that clinicians counsel patients to become aware of, and avoid, exposures to air pollution (GOLD and NHLBI Expert Panel 3 report on asthma). Interventions at the individual level may include recommendations by clinicians that patients avoid exercising or cycling near busy roadways to reduce exposure toTRAP, and to improve ventilation in homes were BMF are used. Public policy can encourage or mandate engineering solutions that drastically reduce emissions from cook stoves and vehicles, but adoption of new technologies can be slowed by lack of awareness of health risks, traditional cultural practices, and economic costs.[8] In some respects public health and regulatory approaches to traffic emissions may be considered the low hanging fruit for opportunities toward health improvement on a societal scale. "Experiments" such as the Beijing Olympics have demonstrated how such changes may have health implications.

Figure 2

A and B Two photographs in Beijing taken from the same vantage point and time of day. Figure 2A was taken in early June of 2008, while the one on Figure 2B was taken in mid- July during the height of the Olympic reductions in heavy industry, power generation, ...

87

Acknowledgements

Go to:

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Abbreviations

Go to:

TRAP Traffic-related air pollution
DC Developed countries
LDC Less developed countries
PM Particulate Matter
BMF Biomass fuel
LUR Land use regression
GST G;utathione S-Transferase
FeNO Fractional exhaled Nitric Oxide

Footnotes

Go to:

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Environmental Health

Potential Adverse Health Effects of Wood Smoke

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The use of wood stoves has increased greatly in the past decade, causing concern in many communities about the health effects of wood smoke. Wood smoke is known to contain such compounds as carbon monoxide, nitrogen oxides, sulfur oxides, aldehydes, polycyclic aromatic hydrocarbons, and fine respirable particulate matter. All of these have been shown to cause deleterious physiologic responses in laboratory studies in humans. Some compounds found in wood smoke—benzo[a]pyrene and formaldehyde—are possible human carcinogens. Fine particulate matter has been associated with decreased pulmonary function in children and with increased chronic lung disease in Nepal, where exposure to very high amounts of wood smoke occurs in residences. Wood smoke fumes, taken from both outdoor and indoor samples, have shown mutagenic activity in short-term bioassay tests. Because of the potential health effects of wood smoke, exposure to this source of air pollution should be minimal.

(Pierson WE, Koenig JQ, Bardana EJ Jr: Potential adverse health effects of wood smoke. West J Med 1989 Sep; 151:339-342)

Homeowners have turned to the use of wood as a heating fuel because of the increasing cost of oil and natural gas. This trend has been especially striking in the northeastern and northwestern United States.[1] The Washington State Department of Ecology estimated in a study in 1984 that wood was burned in 60% of Washington households, with about 2.2 million cords of wood consumed per year. Several studies have shown the potential for wood-burning stoves and fireplaces to pollute indoor as well as outdoor air. Although wood-burning stoves and fireplaces are vented to the outside, many circumstances facilitate the access of combustion products into the indoors, including improper installation, such as insufficient stack height, cracks, leaks in or poor fitting of the stovepipe, negative air pressure indoors, downdrafts, and accidents, such as wood spilling from the fireplace.[1] Also, about 70% of the outdoor wood smoke reenters the house (T. V. Larson, PhD, University of Washington, Department of Civil Engineering, unpublished data). Combustion products of wood are highly irritating to the eyes, nose, and respiratory system. Duncan and co-workers have developed data on the types of pollutants associated with wood burning (Table 1).[2]

A wood-burning stove functions differently than a fireplace. In a fireplace, as much as 90% of a fire's heat is lost up the chimney along with exhaust gases. With a stove, the air supply is controlled and the rate of combustion is also controlled so that as much as 60% of the heat produced can be delivered indoors.[3]

Sexton and associates compared particle mass, size distribution, and chemical composition of indoor and outdoor air in a residential neighborhood.[4] They found that indoor concentrations of particles were often higher than outdoor, and although wood smoke contributed greatly to the mass, other sources also were important. They also found great variation among residences in the same neighborhood.

Traynor and colleagues measured indoor air pollution and found that both airtight and nonairtight stoves produced measurable particulate matter and polycyclic aromatic hydrocarbons within the home.[5] Nonairtight stoves emitted as much as 650 μg per m^3 for a 24-hour period. Polycyclic aromatic hydrocarbons, including benzo[*a*]pyrene, are also expelled into the indoor environment. Benzo[*a*]pyrene is a known carcinogen, and any exposure above local background levels should be avoided when possible. Other pollutants can cause both acute and toxic adverse health effects. Thus, it seems appropriate to highlight our understanding of the toxic exposures involved.

Effects of Individual Pollutants

Carbon Monoxide

Carbon monoxide is one of the most ubiquitous indoor pollutants. It is a major product of tobacco combustion, and concentrations from 2 to 110 ppm have been measured in dwellings, depending on the size of the space, the number and type of tobacco products smoked, and the adequacy of ventilation.[6] An indoor level of no higher than 5 ppm has been recommended by the American Society for Heating, Refrigeration, and Air Conditioning Engineers (ASHRAE). Incomplete combustion in fuel-rich flames due to wood burning also can produce substantial amounts of carbon monoxide, an odorless, colorless gas that has the potential to be an invisible and silent hazard. Carbon monoxide competes with oxygen on the hemoglobin molecule, forming carboxyhemoglobin. The current outdoor standard is 9 ppm for an eight-hour period, or 35 ppm for any given hour. Research on persons with coronary artery disease has shown that the amount of exercise that could be done before an attack of angina was notably shortened after carbon monoxide exposure.[7]

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96

Nitrogen Oxides

Nitrogen is capable of forming several types of gaseous oxides. Nitric oxide and nitrogen dioxide are found in very hot wood combustion flames, and both are toxic.[8(p103)] Nitrous oxide is not formed in normal combustion. Nitric oxide and nitrogen dioxide result from the oxygen-rich combustion of wood, coal, natural gas, or oil in a variety of stoves. These gases are very reactive and can interconvert. Nitric oxide binds to hemoglobin to produce methemoglobin. Many of the adverse effects attributed to carbon monoxide in the past may be related to the combined effect of carboxyhemoglobin and methemoglobin.[1] An indoor level of no higher than 2.5 ppm has been recommended by ASHRAE. The National Ambient Air Quality Standard for nitrogen dioxide set by the Environmental Protection Agency is 0.05 ppm averaged over one year. Nitrogen oxides may produce hematologic aberrations, affect the activity of several enzyme systems, and may also cause vascular membrane injury and leakage leading to edema. Exposures to nitrogen dioxide have been associated with toxicologic effects including pulmonary edema, bronchoconstriction, and increased infection rates. An indoor level of no higher than 0.5 ppm has been recommended by ASHRAE. Some epidemiologic evidence indicates that an increased prevalence of respiratory tract infections in young children and adult men as well as lower pulmonary function performance are associated with a history of exposure to gas stove emissions.[9] Consistent lung effects in children due to nitrogen dioxide exposure have been difficult to characterize, however.[9]

Sulfur Oxides

Sulfur dioxide is a common air pollutant from wood-burning stoves that has known airway irritating effects. Kerosene heaters have been shown to emit sulfur dioxide levels that can exceed certain occupational health standards. An indoor level of not greater than 0.5 ppm has been recommended by ASHRAE. Koenig and co-workers[10-12] have shown that adolescent subjects with asthma or exercise-induced bronchospasm or both experience large changes in pulmonary function after exposure to either 1.0 or 0.5 ppm sulfur dioxide during moderate exercise. Symptoms related to lower airway dysfunction such as dyspnea and chest tightness are generally confined to those with asthma, but healthy subjects usually complain of an unusual taste or odor.

Aldehydes

Formaldehyde is ubiquitous in our environment, and the primary indoor source of this and other aldehydes is the combustion of tobacco products. Polyurea foam insulation, particle board, and other construction products also can release formaldehyde into the indoor environment. The indoor levels reported with wood-burning stoves range from 0.3 to 1.0 ppm. The Department of Housing and Urban Development has recommended indoor levels not higher than 0.4 ppm, but ASHRAE has recommended 0.1 ppm. Formaldehyde is associated with an annoying odor and at higher concentrations—generally more than 0.8 ppm—can produce a transient irritation of the eyes and mucous membranes of the upper respiratory tract.[13] It is so soluble and rapidly metabolized that it rarely reaches the lower respiratory tract to inflict damage, except when inhaled in cigarette smoke. Formaldehyde may, on rare occasions, induce bronchial asthma at relatively high exposure doses. It appears to be carcinogenic at exceptionally high cumulative doses in rodents, but there are no conclusive studies proving its carcinogenic effects in humans.

TABLE 1.—*Emissions From Residential Wood-Fired Stoves**

Pollutant	*Emission Range, lb/cord†*
Particulates	3-93
SO_x	0.5-1.5
NO_x	0.7-2.6
Hydrocarbons	1-146
Carbon monoxide	300-1,220
Polycyclic organic materials	0.6-1.22
Formaldehyde	0.3-1.0
Acetaldehyde	0.1-0.3
Phenols	0.3-8
Acetic acid	5-48

*Adapted from Duncan et al.[2]
†The factor used to convert 1 lb per ton was 1.65 lb per cord.

Polycyclic Aromatic Hydrocarbons

Incompletely burned hydrocarbons from wood stoves are frequently found in indoor air. Usually they are all the gaseous or vaporizable hydrocarbons, such as hydrocarbons with 1 to 16 carbon atoms. Toxic hydrocarbons are produced if plastic materials are incinerated in wood stoves. Resulting polycyclic aromatic hydrocarbons have been shown to be carcinogenic in animal studies. Coke oven workers have exposure to polycyclic aromatic hydrocarbons in levels similar to those measured in wood smoke fumes; these hydrocarbons can serve as a surrogate for wood smoke exposure. Studies have shown that coke oven workers with 15 years' or more exposure have a 16-fold excess risk of having lung cancer as compared with the general population.[14] One of the polycyclic aromatic hydrocarbon compounds, benzo[*a*]pyrene, is a proven carcinogen in animals.

Effects of Pollutant Mixtures

Most experimental studies, because of experimental design constraints, investigate the health effects of exposure to only one agent at a time. In real life, however, people are exposed to many pollutants. The effect of the described pollutants in combination with one another is reason for additional concern. Only a few studies have been conducted with wood smoke itself. One has investigated the incidence of respiratory illness among 31 preschool children living in homes heated with wood-burning stoves as compared with 31 children living in homes heated by other means.[15] Moderate to severe respiratory symptoms such as wheezing and cough at night were notably greater in the wood stove group compared with the control group. Possible confounding factors were investigated. Approximately the same proportion of children in each group had exposure to parental cigarette smoking in the home. There was no significant difference between groups in terms of the presence of urea formaldehyde insulation or the use of humidifiers. On the other hand, another study with older children—kindergarten through sixth grade—indicated that having a wood stove in the home did not increase significantly the frequency of acute respiratory episodes.[16] In this study formaldehyde exposure, estimated from construction or remodeling products, showed a small association with respiratory symptoms. Controlling for wood stove use did not diminish the formaldehyde effect.

Epidemiologic studies of long-term exposure to wood smoke have found an increased prevalence of respiratory illness in both children and adults. A study by Anderson[17] suggested an association between wood smoke exposure and chronic lung disease in adults in Papua New Guinea. Another study with children in the same region found no difference dependent on exposure except for an excess of wheeze in boys.[18] In a study in the hill region of Nepal the prevalence of chronic bronchitis among nonsmoking women increased substantially with the duration of time per day spent near the fireplace.[19] Houses there are poorly ventilated and have no chimneys. The absence of chronic bronchitis in men was suggested to be due to the lesser amount of time they spent indoors with the burning wood.

A unique case was reported of a 61-year-old woman who had shortness of breath. She was evaluated for interstitial lung disease of an unknown cause. Bronchoalveolar lavage revealed the presence of numerous particles and fibers that were identified as wood. The patient lived in a home heated with a solid-fuel radiant room heater that roasted wood to produce heat.[20]

A review of the health effects of indoor air pollution in general with some mention of wood smoke was published in 1987.[9] Also in 1987, Dockery and colleagues[21] concluded that wood stove use was associated with an increased relative risk for respiratory illness in children selected from grades 2 through 5 in six cities where wood stove use ranged from 46% to 5%. The odds ratio was 1.32 (95% confidence intervals 0.99 to 1.76).

Particulate Matter

Wood stoves have been shown to emit substantial amounts of fine particulate matter of less than 10 μm in size. Fine particulate matter ranging in size from 0.02 to 10 μm is of concern to public health because it has been shown to be readily inspired and deposited into lungs. The finest particles are deposited more deeply in lungs where some can remain indefinitely and cause morphologic and biochemical changes.

In a study in Steubenville, Ohio, decreases in lung function in children correlated with elevated concentrations of total suspended particulate matter.[22] In a similar study in the Netherlands, lung function measurements were followed in 179 children aged 7 to 11 years during a winter season in which total suspended particulate matter was being monitored and 3% to 5% reductions in lung volume measurement were found during air pollution episodes when high concentrations of particulate matter were present.[23] Taken together, these studies suggest that declines in lung function associated with episodic exposures to total suspended particulates occur rapidly and persist for as long as two to three weeks before recovery. Particles of less than 10 μm were measured during the winter of 1985-1986 in Olympia, Washington. For a period of five days, the concentration was greater than 150 μg per m^3. Wood smoke concentration was high and responsible for 80% to 90% of the fine particles. For comparison, the concentration at the same sampling site during the summer of 1986 was approximately 20 μg per m^3.

Airborne wood smoke fumes, collected both inside and outside homes using wood stoves, have been analyzed for their toxic properties. Kamens and associates[24] have shown that wood smoke fumes contain mutagens according to the Ames short-term mutagenicity assay. The volatile organic compounds, polycyclic aromatic hydrocarbons, and especially semivolatile compounds all showed mutagenic activity, with some having as much as 100 times the activity of some well-known carcinogens. Alfheim and co-workers showed that the polar fraction of organic extracts from emissions of wood combustion had direct mutagenic activity in a modified Ames *Salmonella* assay.[25] Using another test of mutagenicity, sister chromatid exchange, Hytonen and colleagues showed the capacity of emission from an airtight residential wood stove to induce sister chromatid exchange.[26] Burnet and Insley reported that emissions from both traditional and advanced technology wood stoves caused sister chromatid exchange in mammalian cells.[27] Even though the newer stoves produced less particulate matter and carbon monoxide, the emissions from these stoves did give a positive response to the sister chromatid exchange test. In another study, air samples were collected from occupied homes using wood as an energy source and mutagenic activity was found in the air of 8 of the 12 homes sampled.[28]

Other Combustion Sources of Indoor Pollution

Whenever unvented combustion takes place indoors or venting systems attached to stoves, boilers, or heaters are malfunctioning, a wide variety of combustion products can be emitted directly indoors. Besides tobacco combustion, the primary sources of combustion by-products in residential buildings are usually space heaters, gas stoves, and gas water heaters, as well as wood stoves. Exhaust from automobiles in homes with attached carports or garages and oil and kerosene lamps and candles can be additional sources of combustion by-products.

Summary

Concern over the quality of outdoor air has been an active issue in the United States for many years. A substantial portion of the Environmental Protection Agency's budget of $300 million is devoted to concern about the contamination of our outdoor environment, such as the selenium contamination of water in California, acid rain in the Great Lakes, or a toxic waste dump in New York.[29] Though these are important issues, most people spend 80% to 90% of their time indoors, each taking well over 10,000 breaths per day to provide the necessary oxygen for human metabolism. More time is spent indoors in the harsh winter months.

The increasing use of wood as a heating fuel has precipitated concern for its potential to further pollute the outdoors but, perhaps more important, to contaminate the home. Many irritating and potentially carcinogenic compounds have been identified in wood smoke, including carbon monoxide, nitrogen and sulfur oxides, fine particulate matter, and various aromatic hydrocarbons, including benzo[*a*]pyrene. In addition to wood smoke, various other combustion sources have been identified that could directly augment the problem. Several studies have identified dozens of toxic chemicals that all appear to originate in common household activities and practices in homes.

With respect to burning wood, it is advised as well that only dry, cured wood—less than 20% water content—be burned because combustion will be more complete and fewer products of incomplete combustion will be released into the air. All stoves should be operated in a manner to produce efficient burning with minimum smoke emissions. It is important to emphasize that even the newest airtight wood

98

stoves emit a considerable amount of fine particles to the outdoor air.[26] Plastic and other synthetic products should never be incinerated in wood stoves. We also strongly recommend curtailing all indoor wood burning during air pollution episodes or stagnations. Finally, we propose that public health authorities increase their research and monitoring efforts directed at this important issue.

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WOOD SMOKE: EMISSIONS AND NONCANCER RESPIRATORY EFFECTS

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INTRODUCTION

During the past twenty years, the use of wood has become popular as an alternative to conventional home heating fuels. Part of this movement has been due to uncertainty about the availability of fossil fuels. About ten percent of space heating in urbanized areas of the northern United States is from wood burning, with up to fifty percent in some smaller, rural towns (42, 63, 75, 85). Wood is obviously a renewable resource. This attraction is offset, however, by the increased air pollution emissions from wood heating devices compared with devices fueled with oil or gas. As noted initially by Cooper (20), particle and organic carbon emission rates can be as much as one to two orders of magnitude larger in wood heating devices than in oil or gas heating units. Legislation restricting the sale of conventional wood stoves first appeared in Oregon in 1984, followed by nationwide restrictions in 1988. Although rapid progress has reduced emissions in some types of modern wood heaters, older "conventional" wood stoves and fireplace inserts are still the predominant appliance in use today. As discussed later in this document, a number of communities continue to experience elevated levels of wood smoke during the winter heating season. In addition, elevated indoor air pollution levels have been observed in homes with non-airtight or improperly operated wood stoves. As a result, there has been an ongoing interest in the potential health effects of exposure to wood smoke. Several reviews of the health effects of wood smoke have been

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prepared (2, 3, 28, 53, 66, 75). The present review attempts to summarize the information available on the chemical composition of wood smoke, concentrations of wood smoke in both indoor and outdoor environments, emission data, and the adverse respiratory responses in animal toxicity studies and epidemiological studies of human populations. A more detailed discussion of the emissions and the chemical and physical properties of wood smoke can be found in Larson & Koenig (58).

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WOOD COMBUSTION AND WOOD SMOKE

Most wood burned for heat is cordwood with some increasing use of wood pellets. Cordwood heaters burn wood with a deficit of oxygen and readily generate products of incomplete combustion, including carbon monoxide and numerous organic compounds. If these vapors are not immediately oxidized, they cool as they are exhausted to the atmosphere with subsequent formation of fine particles rich in relatively high molecular weight organic compounds. A "conventional" wood stove fits this description. To reduce emissions from cordwood heaters, these vapors are oxidized directly downstream of the combustion zone either by using a noble metal catalyst to more completely combust vapors at the lower exhaust temperatures, or by using an insulated secondary combustion chamber to maintain a high exhaust temperature while mixing the gases with a separate stream of additional combustion air. The former method is employed in "catalytic" stoves, the latter in "non-catalytic" or "high technology" stoves. In contrast to cordwood heaters, pellet stoves take advantage of the larger wood surface area per unit mass of wood. Consequently, the higher heat transfer rates from the combustion gases to adjacent wood result in efficient vaporization of the wood prior to combustion. Mixing these vapors with excess combustion air at the top of a pellet bed results in much more complete combustion than that in conventional stoves.

Wood consists of approximately 50 to 70 weight percent cellulose, which are polysaccharides, and about 30 weight percent lignin, which is a skeletal network of branch-chain polymers that provide structural integrity. In addition, there are small amounts of resinous materials and inorganic salts. The lignin polymer consists of two main monomers, a guaiacylpropane structure and a syringylpropane structure. Upon heating, these structures break apart producing a large variety of smaller molecules, many of which are part of the general class of oxygenated monoaromatics (89). Included in this class are methoxy phenols and methoxy benzenes, as well as phenols and catechols. This decomposition also produces benzene and alkyl benzenes. The presence of guaiacol, syringol and their derivatives as a group are unique to the burning of wood because they are a direct consequence

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of the destruction of the unique lignin structure. In contrast, phenols, catechols, benzene, and alkyl benzenes are not unique to wood combustion and have been found in the exhaust gases of other combustion sources. Table 1 summarizes the reported constituents in wood smoke and provides quantitative information on their emission rates.

Of the trace elements, potassium is found at relatively high concentrations in wood smoke. Combustion of hardwoods produces more ash (and thus higher concentrations of trace elements) than does combustion of softwoods. The particulate elemental carbon concentrations reported in wood smoke are somewhat controversial. Some researchers claim that up to 95 weight percent of the total particle mass is extractable in dichloromethane and/or methanol (88), while others claim that up to 50 percent of the total particulate carbon is elemental carbon as measured by optical and thermal methods (78). It seems reasonable to conclude that 5 to 20 percent of the total particulate mass is unextractable and that this unextractable fraction contains elemental carbon.

The size distribution of wood smoke particles has been measured by several investigators (23, 47, 49). The particle volume peaks at between 0.15 and 0.4 μm, with essentially no particles greater than 1 μm. This is consistent with the fact that the majority of the mass is formed by condensation processes in the exhaust. Owing to their relatively small size, they are very efficient at reducing visibility and are not readily removed by inertial and gravitational processes.

Upon release to the environment, many compounds in wood smoke are expected to undergo some degree of chemical transformation in the atmosphere. However, there have been relatively few studies of these transformations (see ref. 58 for a more complete discussion).

ATMOSPHERIC CONCENTRATIONS OF WOOD SMOKE

In the past ten years, a number of studies have documented the outdoor concentrations of airborne particles resulting from wood burning. These studies are summarized in Table 2. We have included only those studies in this table that quantified the levels of airborne particles using one of several chemical tracer methods. These studies document that wood smoke has been identified in airsheds in many areas throughout the United States.

Numerous other studies have documented elevated levels of particulate matter in residential communities where wood burning is prevalent, but have not employed receptor models to estimate the wood burning fraction. Perhaps the most notable of these are the measurements taken in Klamath Falls, Oregon, that have exceeded 600 $\mu g/m^3$ on a 24-hr basis during the winter (43). Based upon inventories of fuel use, wood smoke may account

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102

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Table 1 Chemical composition of wood smoke

Specie[1]	g/kg wood[2]	Physical state[3]	Reference
Carbon monoxide	80–370	V	25, 74
Methane	14–25	V	74
VOCs (C_2–C_7)	7–27	V	74
Aldehydes	0.6–5.4	V	25, 62
Formaldehyde	0.1–0.7	V	25, 62
Acrolein	0.02–0.1	V	62
Propionaldehyde	0.1–0.3	V	25, 62
Butryaldehyde	0.01–1.7	V	25, 62
Acetaldehyde	0.03–0.6	V	25, 62
Furfural	0.2–1.6	V	30, 34
Substituted furans	0.15–1.7	V	30, 34
Benzene	0.6–4.0	V	74
Alkyl benzenes	1–6	V	89
Toluene	0.15–1.0	V	89
Acetic acid	1.8–2.4	V	30
Formic acid	0.06–0.08	V	30
Nitrogen oxides (NO,NO_2)	0.2–0.9	V	25, 74
Sulfur dioxide	0.16–0.24	V	25
Methyl chloride	0.01–0.04		48
Napthalene	0.24–1.6	V	89
Substituted napthalenes	0.3–2.1	V/P	89
Oxygenated monoaromatics	1–7	V/P	89
Guaiacol (and derivatives)	0.4–1.6	V/P	35
Phenol (and derivatives)	0.2–0.8	V/P	35
Syringol (and derivatives)	0.7–2.7	V/P	35
Catechol (and derivatives)	0.2–0.8	V/P	35
Total particle mass	7–30	P	74
Particulate organic carbon	2–20	P	21
Oxygenated PAHs	0.15–1	V/P	89
PAHs			
Fluorene	4×10^{-5}–1.7×10^{-2}	V/P	1, 19, 21, 46, 92, 99
Phenanthrene	2×10^{-5}–3.4×10^{-2}	V/P	1, 19, 21, 46, 92, 99
Anthracene	5×10^{-5}–2.1×10^{-2}	V/P	1, 19, 21, 46, 92, 99
Methylanthracenes	7×10^{-5}–8×10^{-3}	V/P	1, 19, 21, 92, 99
Fluoranthene	7×10^{-4}–4.2×10^{-2}	V/P	1, 19, 21, 92, 99
Pyrene	8×10^{-4}–3.1×10^{-2}	V/P	1, 19, 21, 92, 99
Benzo(a)anthracene	4×10^{-4}–2×10^{-3}	V/P	1, 19, 21, 92, 99
Chrysene	5×10^{-4}–1×10^{-2}	V/P	1, 19, 21, 92, 99
Benzofluoranthenes	6×10^{-4}–5×10^{-3}	V/P	1, 19, 21, 92, 99
Benzo(e)pyrene	2×10^{-4}–4×10^{-3}	V/P	1, 19, 21, 92, 99
Benzo(a)pyrene	3×10^{-4}–5×10^{-3}	V/P	1, 19, 21, 92, 99
Perylene	5×10^{-5}–$3 - 10^{-3}$	V/P	1, 19, 21, 92, 99
Ideno(1,2,3-cd)pyrene	2×10^{-4}–1.3×10^{-2}	V/P	1, 19, 21, 92, 99
Benz(ghi)perylene	3×10^{-5}–1.1×10^{-2}	V/P	1, 19, 21, 92, 99
Coronene	8×10^{-4}–3×10^{-3}	V/P	1, 19, 21, 92, 99

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Table 1 (*Continued*)

Specie[1]	g/kg wood[2]	Physical state[3]	Reference
Dibenzo(a,h)pyrene	$3 \times 10^{-4}-1 \times 10^{-3}$	V/P	1, 19, 21, 92, 99
Retene	$7 \times 10^{-3}-3 \times 10^{-2}$	V/P	21, 46
Dibenz(a,h)anthracene	$2 \times 10^{-5}-2 \times 10^{-3}$	V/P	19, 21, 46, 92, 99
Trace elements			
Na	$3 \times 10^{-3}-1.8 \times 10^{-2}$	P	21, 46, 96
Mg	$2 \times 10^{-4}-3 \times 10^{-3}$	P	21, 46, 96
Al	$1 \times 10^{-4}-2.4 \times 10^{-2}$	P	21, 46, 96
Si	$3 \times 10^{-4}-3.1 \times 10^{-2}$	P	21, 46, 96
S	$1 \times 10^{-3}-2.9 \times 10^{-2}$	P	21, 46, 96
Cl	$7 \times 10^{-4}-2.1 \times 10^{-1}$	P	21, 46, 96
K	$3 \times 10^{-3}-8.6 \times 10^{-2}$	P	21, 46, 96
Ca	$9 \times 10^{-4}-1.8 \times 10^{-2}$	P	21, 46, 96
Ti	$4 \times 10^{-5}-3 \times 10^{-3}$	P	21, 46, 96
V	$2 \times 10^{-5}-4 \times 10^{-3}$	P	21, 46, 96
Cr	$2 \times 10^{-5}-3 \times 10^{-3}$	P	21, 46, 96
Mn	$7 \times 10^{-5}-4 \times 10^{-3}$	P	21, 46, 96
Fe	$3 \times 10^{-4}-5 \times 10^{-3}$	P	21, 46, 96
Ni	$1 \times 10^{-6}-1 \times 10^{-3}$	P	21, 46, 96
Cu	$2 \times 10^{-4}-9 \times 10^{-4}$	P	21, 46, 96
Zn	$7 \times 10^{-4}-8 \times 10^{-3}$	P	21, 46, 96
Br	$7 \times 10^{-5}-9 \times 10^{-4}$	P	21, 46, 96
Pb	$1 \times 10^{-4}-3 \times 10^{-3}$	P	21, 46, 96
Particulate elemental carbon	0.3–5	P	21, 78
Normal alkanes ($C_{24}-C_{30}$)	$1 \times 10^{-3}-6 \times 10^{-3}$	P	21
Cyclic di- and triterpenoids			
Dehydroabietic acid	0.01–0.05	P	87
Isopimaric acid	0.02–0.10	P	87
Lupenone	$2 \times 10^{-3}-8 \times 10^{-3}$	P	87
Friedelin	$4 \times 10^{-6}-2 \times 10^{-5}$	P	87
Chlorinated dioxins	$1 \times 10^{-5}-4 \times 10^{-5}$	P	72
Particulate acidity	$7 \times 10^{-3}-7 \times 10^{-2}$	P	72

[1]Some species are grouped into general classes as indicated by italics.
[2]To estimate the weight percentage in the exhaust, divide the g/kg value by 80. This assumes that there are 7.3 kg combustion air per kg of wood. Major species not listed here include carbon dioxide and water vapor (about 12 and 7 weight percent, respectively, under the assumed conditions).
[3]At ambient conditions; V = vapor, P = particulate, and V/P = vapor and/or particulate (i.e. semivolatile).

for as much as 80 percent of the airborne particle concentrations during the winter (36). The Klamath Falls studies emphasize another important point—the location of the air monitoring device. There is up to a fourfold difference between various parts of town, with the highest readings in the residential area (36). This same spatial variability was observed by Larson et al (56) using a mobile nephelometer. They found that the nighttime drainage flow

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104

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tended to concentrate the wood smoke at valley floors, with a consistent factor of two to three difference between ridge line and valley smoke levels. With this caveat in mind, we can see from Table 2 that the average nighttime concentrations of fine particle wood smoke vary from location to location. As expected, concentrations are higher in residential areas than in downtown urban or industrial areas and generally higher at night than during the day. The agreement between different source apportionment methods, when compared, is good.

Several studies are of interest to later discussions of the health effects of wood smoke. In addition to the Klamath Falls studies discussed above, the limited measurements by Carlson (15) in Missoula, Montana, indicated that most of the fine particle mass was due to wood burning. The measurements taken in Boise, Idaho, also found that most of the extractable organic material found in fine particles was from wood burning, with the remainder due to mobile sources (50, 61). Finally, the measurements taken in Seattle, Washington, (56, 57) indicate not only that there are elevated concentrations of wood smoke particles during winter evening periods at a residential location, but also that most of the fine particle mass at this location is due to wood burning all weeks of the year. The fine particle mass concentrations at this site are low in the summer, and therefore the absolute concentrations due to wood burning are about an order of magnitude less in summer than in winter. Open burning restrictions did not go into effect in this area until September, 1992. Thus some wood burning is expected in the summer from burning of yard waste and land-clearing fires.

Although smoke levels in outdoor air are important, most people spend a majority of their time indoors, especially at night in residential areas. Indoor exposure can occur not only from infiltration of outdoor air, but also from emissions into the home from a wood burning appliance. Table 3 summarizes current knowledge of the effects of wood burning appliances on indoor air quality. The table is arranged by pollutants. Emissions occur into the home during fueling of the stove and may also occur during stove operation. More modern, airtight stoves generate fewer emissions directly into the home than older, nonairtight stoves or improperly operated and/or maintained stoves. To put the emission rates listed in Table 3 into perspective, consider a 100 cubic meter room (approximately 20 by 20 feet). If seven tenths of the volume of air in the room is exchanged with outside air every hour, and if 70 percent of the fine particles from the outside air penetrate into the home, then for a typical outdoor concentration of 20 $\mu g/m^3$ of wood smoke particles there is an effective infiltration rate of $20 \times 0.7 \times 0.7 \times 100 = 1$ mg per hour of fine particle mass. This value is comparable to estimated emission rates of fine particles into the home due to leakage from "airtight" stoves (2.3–3.6 mg/hr; see Table 3). Higher

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Table 2 Summary of measured wood burning effects on airborne particle concentrations

Investigator[a]	Location	Measurement[b]	Concentration ($\mu g/m^3$) Mean	Range	Wood smoke (wt %)	Method[c]	Comments
Cooper (20)	Portland, OR	$PM_{2.5}$	68	—	36	^{14}C	Single sample at residential location in winter
		total carbon	31.3	—	51		
Wolff et al (97)	Denver, CO	$PM_{2.5}$	39.5	—	12	K/Fe	
		total carbon	27	7–43	33	^{14}C	Five samples during winter
Carlson (15)	Missoula, MT	$PM_{3.5}$	—	—	68	CMB	Average of winter samples
Imhoff (44)	Petersville, AL	$PM_{2.5}$	45	13–86	85	CMB	Seven residential samples in winter
Core et al (22)	Spokane, WA; Seattle, WA; Tacoma, WA; Portland, OR; Boise, ID	$PM_{2.5}$	57	—	71	CMB	Sixty-one 24-hr samples in autumn and winter from 8 sites in WA, 1 site in ID and 1 site in OR
	Medford, OR	$PM_{2.5}$	17.5	8.8–30.2	55		Annual average values for 3 sites
	Portland, OR		3.0	1.5–3.9	14		Annual average values for 4 sites
Ramdahl et al (76)	Elverum, Norway	total carbon (<3 μm)	20	5–50	65	^{14}C	Ten 24-hr winter samples; avg PM_{10} = 51 $\mu g/m^3$ (range 31–101)
Naylor (71)	Las Vegas, NV	total carbon	36	25–46	47	^{14}C	Four 12-hr winter samples (day and night)
Lewis et al (60)	Denver, CO	$PM_{2.5}$	19	?–47	8	MLR	Seventeen 12-hr daytime samples in winter
		$PM_{2.5}$	12	?–41	17		Nineteen 12-hr nighttime samples in winter
Klouda et al (51)	Raleigh, NC	total carbon	—	23–80	95	^{14}C	Four 12-hr daytime samples in winter
		elemental carbon	3.2	—	68		One 12-hr daytime sample in winter
	Albuquerque, NM	total carbon	—	11–71	75	^{14}C	Six 12-hr samples (day & night) at residential site in winter
		elemental carbon	4.6	—	41		Four 12-hr samples (day & night) at residential site in winter

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Table 2 (*Continued*)

Investigator[a]	Location	Measurement[b]	Concentration ($\mu g/m^3$) Mean	Range	Wood smoke (wt %)	Method[c]	Comments
Lewis et al (59)	Albuquerque, NM	total carbon	—	—	67	MLR	Six 12-hr samples (day or night)
			—	—	68	$\times 1^4C$	in winter
		EOM	18.9	—	78	MLR	Forty-four 12-hr samples (day & night) in winter
Chow et al (16)	Sparks, NV	PM_{10}	41	?–154	30[d]	CMB	Fifty seven 24-hr samples every 6th day for one year at a residential site
			76		44[d]		Subset of above samples from Oct–Dec period (n = 15)
	Reno, NV		30	?–99	3[d]		Fifty six 24-hr samples every 6th day for one year at an urban site
			46		9[d]		Subset of above samples from Oct–Dec period (n = 15)
Benedict & Naylor (8)	Las Vegas, NV	$PM_{2.5}$	12.5	—	27	CMB	One 24-hr sample during winter
Magliano (65)	Bakersfield, CA	PM_{10}	8.7	—	12.9	CMB	Nine month average of every 6th day
		$PM_{2.5}$	13.8	—	62.8		24-hr samples (March–Dec)
	Fresno, CA	PM_{10}	7.1	—	16.8		
		$PM_{2.5}$	5.3	—	35.5		
Dresser & Baird (29)	Telluride, CO	PM_{10}	—	—	33	CMB	Four 24-hr average spring samples
			205	—	58		Two 24-hr holiday winter samples
Larson et al (56)	Seattle, WA	PM_{10}	39	9–123	6	CMB	Seven 12-hr daytime samples at industrial site in winter of 1987–88
			30	8–61	11		The corresponding seven 12-hr nighttime samples at above site

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			45	12–104	54		Ten 12-hr daytime samples at residential site in winter of 1987–88
			75	5–144	82		The corresponding ten 12-hr nighttime samples at above site
			116	75–139	82		The sixteen highest 12-hr nighttime samples at the same residential site in the winter of 1988–89
Klouda et al (50)	Boise, ID	EOM	—	—	72–89	^{14}C	Reported range of values (average not reported) for nine 12-hr daytime samples at a residential site in winter
		EOM	—	—	52–83		Range of values (average not reported) for nine 12-hr nighttime samples at residential site in winter
Lewis et al (61)	Boise, ID	EOM	22	—	67[d]	MLR	Forty 12-hr samples (day & night)
Larson et al (57)	Seattle, WA	$PM_{2.5}$	14.8	6.0–32.9	71	CMB	Forty eight one-week average composite samples (Jan–Nov) at a residential site. The composite consisted of sampling for 15 min every 2 hr for the entire study period. Wood burning was the dominant source all seasons of the year, ranging from 60% in summer to 90% in winter

[a] Other investigators have measured elevated concentrations of particulate matter in wood burning communities, but did not use one of the methods cited above to quantify the fraction attributable to wood burning. Methods not listed above include emission inventory/atmospheric dispersion modeling (13, 42, 44, 55, 69, 71, 76, 80, 84), gaseous methyl chloride tracer measurement (47), time series of particle light scattering coefficient (54, 55) and thermography (54).

[b] PM_x = mass concentration of particles ≤ x μm in aerodynamic diameter; EOM = mass concentration of extractable organic matter from particles with aerodynamic diameters ≤ 2.5 μm; total carbon = total organic and elemental carbon mass concentration in particles ≤ 2.5 μm in aerodynamic diameter unless otherwise noted.

[c] ^{14}C = isotopic carbon measurement to determine biogenic carbon concentration, i.e., contemporary carbon from biogenic material ~ 40 years or less old; CMB = chemical mass balance regression model; K/Fe = tracer enrichment method based upon the mass ratio of potassium to iron; MLR = multiple linear regression of individual tracer elements (e.g. potassium for wood and lead for motor vehicles) against mass concentration of relevant measurement listed above.

[d] Estimated from reported average concentration of wood smoke divided by average concentration of total mass

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outdoor concentrations or more rapid air exchange rates would give larger infiltration rates. For most studies of fine particle mass in homes with airtight stoves, the indoor outdoor ratios are at or below 1.0, implying that infiltration is important even in homes with stoves. As also shown in Table 3, the indoor/outdoor ratios are much higher for carbon monoxide and formaldehyde, two species that have a number of indoor sources in addition to wood stoves. The data for formaldehyde are particularly striking, implying that although wood stoves emit formaldehyde, their emissions are not a major determinant of overall exposure to this specie.

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HEALTH EFFECTS

Many of the constituents of wood smoke described earlier aggravate respiratory disease and irritate mucous membranes. Knowledge of the toxicity of a compound usually depends on data from three sources: animal toxicology, controlled human studies, and epidemiology. In this section, we review the available information on wood smoke exposures, with emphasis on studies of human subjects.

Animal Toxicology

We restrict our discussion of animal toxicology to those studies employing whole wood smoke; we do not discuss data on individual compounds found in wood smoke. Unfortunately few data are available on acute effects and none on chronic effects of inhalation of wood smoke in animals. One study found an overall depression in macrophage activity, increases in albumin and lactose dehydogenase (LDH) levels (both indicating damage to cellular membranes), and a large increase in red blood cell numbers (7). A morphological study of injury from inhalation of white pine wood smoke in rabbits showed a reproducible, necrotizing tracheobronchial epithelial cell injury (90). Another pathological result from wood smoke exposure comes from an investigation of smoke from burning pine wood delivered to the lungs of dogs (10). Significant increases in angiotensin-1-converting enzyme, a substance that regulates vasomotor activity in endothelial cells, was measured immediately after exposure and was even higher 30 min. post exposure. This pathological change could be an initial step toward pulmonary hypertension which is a suggested risk factor for a myocardial infarction. Fick and co-workers (32), in a study of young adult New Zealand white rabbits, reported significant changes in the numbers and functions of the macrophages after exposure to wood smoke from Douglas fir compared with control. Wong and co-workers (98) demonstrated a blunted respiratory response to CO in guinea pigs exposed to wood smoke, which may indicate disruption of respiratory neural control. Clark and co-workers (17) studied the distribution of extravascular lung water after acute smoke inhalation in

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Table 3 Effects of wood burning appliances on indoor air quality[1]

Specie	Source[2]	Indoor concentration ($\mu g/m^3$) with source	Indoor concentration ($\mu g/m^3$) without source	Average indoor:Outdoor ratio with	Average indoor:Outdoor ratio without	Estimated indoor source strength (mg/hr)[3]	Reference
$PM_{2.5}$	a	7–40	8–32	<1	<1	—	37, 38
	a,i	50–80	10–30	1.3	0.4	—1	37, 38
	a	3–27	—	0.3	—	—	22
	a,i	18	—	0.8	—	—	22
	a	28–38	—	1.1	—	2.3–3.6	52
	n	44–91	—	2.1	—	5.5–11.1	52
$PM_{3.5}$	—	13–15	8–32	>1	>1	—	18
	—	25 (19)	24 (22)	1.7	1.4	—	84
Total suspended particles	a	24–71	—	3.1	—	2.5–8.7	91
	n	28–1500	—	33.5	—	16–230	91
	b	19–24	—	1.3	—	1.1–1.6	91
Total suspended particles	a	52–65	—	1.2	—	4.1–7.5	52
	n	119–166	—	2.6	—	12–21	52
Carbon monoxide	a	1100–1960	—	2.4	—	55–182	52
	n	2450–4410	—	6.8	—	210–530	52
	a	490–3430	—	2.2	—	12–170	91
	n	2200–17,000	—	24.5	—	270–2200	91
	a	1100–2700	—	1.0	—	—	22

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Table 3 (*Continued*)

Specie	Source[2]	Indoor concentration ($\mu g/m^3$) with source	Indoor concentration ($\mu g/m^3$) without source	Average indoor:Outdoor ratio with	Average indoor:Outdoor ratio without	Estimated indoor source strength (mg/hr)[3]	Reference
Nitric oxide	a	27–47	—	1.6	—	0.4–3.2	52
	n	26–67	—	4.4	—	2.0–9.4	52
Nitrogen dioxide	a	12–25	—	0.05	—	<0–2.4	52
	n	18–26	—	0.7	—	2.1–3.6	52
Formaldehyde	a	12–51	18–25	8	5	—	100
	a,i	15–33	17–34	3	4	—	37, 100
Benzo(a)pyrene	a	3×1^{-4}–3.5×10^{-3}	—	1.6	—	2×10^{-5}–7.6×10^{-4}	91
	n	2×10^{-3}–4.9×10^{-1}	—	84	—	2×10^{-3}–5.7×10^{-2}	91
	b	4×10^{-5}–1.6×10^{-3}	—	0.2	—	1×10^{-5}	91
Extractable organic matter	a	1.4–2.3	—	0.6	—	—	22
particulate phase	a,i	2.0	—	0.6	—	—	22
	a	12–20	16–20	3.0	3.5	—	67
vapor phase (semi volatile)	a	14.4–14.5	—	2.1	—	—	22
	a,i	29.1	—	2.6	—	—	22
	a	268–308	296–308	3.0	3.5	—	67

[1]Studies listed here include simultaneous measurements of indoor and outdoor concentrations. Other studies of note not listed include (4, 9, 73, 95). These latter studies document the impacts on indoor air quality of wood burning devices by comparison with "no-burn" periods, but do not specifically control for outdoor pollution variability.

[2]a = airtight wood stove; i = improperly operated wood stove; n = non-airtight wood stove; f = fireplace; b = background test (no stove present).

[3]Reported estimates via mass balance from indoor and outdoor levels as well as air exchange rates.

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mongrel dogs. The exposures were for two hours. Extravascular lung water, determined by using Evans dye, which binds quickly to plasma albumin, was increased in the smoke-exposed dogs compared with controls. Wood smoke was generated by burning a standard mixture of fir plywood sawdust and kerosene. Whether the plywood contained epoxys and the contribution of toxicity from kerosene were not discussed.

Extrapolation of the results of these animal studies to human populations living in areas with elevated wood smoke concentrations is very difficult. Instillation of material directly into the lung certainly is different from inhalation. Also, inhalation from a smoke stream would result in breathing considerably higher concentrations of smoke than seen in neighborhoods in the human breathing zone. None of the animal studies evaluated pulmonary function or symptoms of respiratory illness, the endpoints assessed in epidemiological studies. Some individual components of wood smoke, such as formaldehyde (5) and various products of incomplete combustion (25a), have been studied more extensively in animals.

Controlled Laboratory Studies

No controlled laboratory studies of human subjects exposed to wood smoke per se have been reported. There are some related studies with formaldehyde and environmental tobacco smoke but these are not discussed here.

Studies in Developing Countries

The health effects of inhalation of wood smoke have been documented in developing countries where women spend many hours close to an open unvented indoor fire used for cooking (2, 66). Increased respiratory symptoms, decreased pulmonary function, and large increases in the prevalence of chronic bronchitis have been reported in New Guinea, India, and Nepal. However, measurements of particulate matter have not been reported in most of these studies. Other studies, such as one in Nepal, compared concentrations of indoor particulate matter in huts where traditional cooking methods were practised to those where an improved cookstove was used (79). Concentrations of total suspended particulate matter in the former averaged 2.7 mg/m^3; a similar average concentration associated with the improved cook stove was 1.0 mg/m^3, still much higher than concentrations of particulate matter to which US populations currently are exposed. A recent report of indoor air pollution in a similar situation in China (indoor use of an open cooking fire) measured concentrations up to 25 mg/m^3 PM_{10} (33).

A recent clinical report describes a group of 30 nonsmoking patients whose lung disease may be due to wood smoke exposure (81). These individuals were seen in Mexico City and all had a history of living in the

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countryside away from urban air pollution. The smoke exposure was the result of the use of wood and biomass for home cooking. These patients had abnormal chest X-ray scans showing a diffuse, bilateral, reticulonodular pattern, and evidence of pulmonary arterial hypertension. Their pulmonary function tests were consistent with a mixed restrictive-obstructive disease diagnosis. The authors suggest that this group of patients was suffering from wood-smoke inhalation-associated lung disease (WSIALD) (81).

Epidemiology

Reports of lung function decreases in children associated with fine particulate matter in the early 1980s (24, 27) inspired investigations of one source of those fine particles, wood smoke. Epidemiological investigations of adverse respiratory effects of wood smoke emissions in the US have centered on either symptomatology or pulmonary function. The symptoms measured have been the traditional respiratory disease outcomes; cough, wheeze, upper or lower respiratory infection. Pulmonary functions measured have been FEV_1, a measure of air flow limitation caused by obstruction in the airways, or FVC, a measure of the total amount of air that can be forcibly exhaled from the lungs. All but one of the available studies have been carried out in children, most likely on the assumption that children are most at risk for adverse effects from inhaled irritants due to the small size of their lungs and also due to the immature nature of their immune system. Other advantages of children as subjects in studies of respiratory effects of air pollution are the relative lack of confounders such as years of cigarette smoking or occupational exposure. There is good precedence from the numerous studies of the effects of environmental tobacco smoke on children's respiratory health for suspecting that young children are vulnerable to inhaled agents. Eight reports have been published of associations between lung function and wood smoke in children studied in the field and one study of responses in adult subjects, summarized in Table 4. An additional study of the association between visits to emergency departments for asthma and fine particulate matter (82) is included since this study was conducted in Seattle where a considerable percentage of fine particles are produced by residential wood burning.

The earliest report of adverse health effects from exposure to wood smoke in the US came from Michigan. Honicky and co-workers studied 31 young children who lived in homes with wood stoves and compared them to 31 children who lived in homes with other sources of home heating (41). They recorded respiratory symptoms over the telephone using a modified Epidemiology Standardization Project Children's Questionnaire (31). The occurrence of cough and wheeze was much greater in children from the homes with stoves and, in general, both moderate and especially severe symptoms

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Table 4 Summary of studies of respiratory effects of exposure to wood smoke

Reference	Age (yr)	Number of subjects	Endpoints measured	Results
41	1–7 yr	34 w/stoves 34 without	Symptoms	More symptoms in children with stoves $p < 0.001$
93	5–11*	258 w/stoves 141 without	Symptoms	Risk ratio = 1.1, showing no significant effect
11	1 and older	455 high smoke** 368 low smoke	Symptoms disease prevalance	No significant effects. Trend in children aged 1–5
54	8–11	296 healthy*** 30 asthmatic	Spirometry	Significant association between fine particles and lung function in asthmatics, $p = 0.05$
14	1–5 ½	59	Symptoms	Significant correlation between woodstove use and wheeze and cough frequency $p = 0.01$
68	<24 mo	58 pairs	Respiratory disease	Woodstove significant risk factor for lower resp infection
36	8–11	410***	Spirometry	Significant decrease in PFTs with elevated wood smoke
45	8–11	495	Spirometry	Significant relation of functional decrease with elevated wood smoke
64	$\bar{X} = 46$	182	Symptoms	Significant association
82	all ages	2955, asthma 3810, gastroenteritis	Emergency	Significant association between room visits asthma visits fine particles

*Described as kindergarten through grade 6.
**Geographical areas with high or low wood smoke pollution.
***Grades 3 through 6.

of respiratory diseases were significantly greater in the wood smoke-exposed children ($p < 0.001$). No measurements of wood smoke were reported.

Previously, Tuthill reported the results of an investigation of symptoms of respiratory illness and respiratory disease prevalence associated with wood smoke and formaldehyde exposure (93). Symptoms were collected using a questionnaire; chronic respiratory illness was defined as physician-diagnosed chronic bronchitis, asthma, or allergies. The subjects were 399 children from kindergarten through the sixth grade. Two hundred fifty eight lived in homes with wood stoves and 141 lived in homes without stoves. Although he found increased risk ratio = 2.4 (confidence intervals 1.7–3.4) for exposure to formaldehyde (from off-gassing of building materials after new construction or remodeling, from foam insulation, or from wood burning), the risk ratio for exposure to wood smoke of 1.1 (0.76–1.7) was not significant. The difficulty assigning formaldehyde exposure to sources other than wood burning was not discussed, although it is consistent with data from studies reviewed in Table 3.

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The effects of wood stoves on general respiratory health in preschool age children was studied by Butterfield and others (14). Ten symptoms of respiratory disease were tracked in 59 children during the 1985–86 winter heating season in the Boise, Idaho, area. The children ranged in age from 1 to 5 1/2 years. Symptoms significantly associated with living in a home with a wood stove in use were frequency of wheeze, severity of wheeze, frequency of cough, and waking up at night with cough. An independent study of sources of extractable organic material in ambient particles in Boise during the 1986–87 heating season showed an average of 67% due to wood burning (61).

Another study compared the incidence of lower respiratory tract infection in Native American children with presence of a wood stove in the home (68). The children lived on the Navajo reservation in Arizona. Cases were children less than 24 months of age with lower respiratory tract infection (*bronchiolitis or pneumonia*) who were matched with a control case visiting the clinic as part of a well-child program. Fifty-eight age and gender matched pairs participated in the study. Forty-nine percent of the cases lived in homes using wood-burning for heat, whereas only 33 percent of the control children lived in such homes. In this study, living in a home with a wood burning source of heat was a risk factor for lower respiratory tract infection (odds ratio = 4.2, $p < 0.001$).

Heumann and co-workers (36) studied pulmonary function in a group of elementary school children in Klamath Falls, Oregon, using standard spirometric values. Pulmonary function test data were collected on 410 children in grades 3 through 6 at three time periods during the 1990 heating season. There was a significant decrease in average FEV_1 and FVC among children who had the highest exposure to wood smoke. A preliminary report of this study was presented (35); analysis is still ongoing.

The 1977 Montana legislature funded an extensive Montana Air Pollution Study (45), designed to evaluate whether air pollution was associated with adverse health effects in urban centers. The study involved third, fourth, and fifth grade children in five Montana cities. It measured lung function both within and between communities. Thus, lung function of school children living in communities with different levels of air pollution was ascertained. Also, comparisons of lung function changes of school children and air quality within a single community were evaluated. Each child served as his or her own control and analysis of covariance was used to test for statistical significance in the acute study within a single community. In the multicity study, linear regressions and principal components techniques were used and appropriate adjustments were made for factors such as altitude, which varied from city to city. Three-day averages of the pollutants were used. Both studies detected significant lung function effects associated with total

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suspended particulate matter (TSP) and both fine and coarse respirable particulate matter. Pulmonary function decrements ranged from 1% to 10%, 24-hr average TSP ranged from 24 to 128 $\mu g/m^3$ during the study period. Sources of the particulate matter were not identified, however the authors state that the particulate matter essentially was from wood burning and entrained dust. Measurements of fine particles ($PM_{3.5}$) during this period found 68% by weight attributable to wood smoke in Missoula, Montana (15).

One study in Denver, Colorado, of a panel of adult subjects with asthma was conducted evaluating the presence of a wood stove or fireplace in the home and symptoms of respiratory disease and shortness of breath (64). Using logistic regression analysis, the presence of a wood stove in the home was associated with shortness of breath in females and both shortness of breath and moderate or severe cough in males ($p<0.01$ in all cases).

Two studies of the health effects of wood smoke have been conducted at the University of Washington. The first was a questionnaire study of respiratory health in areas of high and low ambient wood smoke pollution (11). The communities were chosen based on extensive air monitoring of wood smoke distributions in the greater Seattle area (56). Six hundred residences in each community were sent questionnaires and asked to answer for one adult and one children at each address. The initial questionnaire asked about chronic symptoms of respiratory disease, in mild, moderate, and severe categories (31). Two follow-up mailings asked about acute symptoms over the past two weeks. During the study period, PM_{10} concentrations in the low wood smoke area averaged 33 $\mu g/m^3$; in the high wood smoke area, the average for the three months of the study was 55 $\mu g/m^3$. Questionnaire responses were stratified by age; 1–5; 6–14; 14–44; 45–64; and >65. No statistically significant differences emerged between residents of the high and low wood smoke communities, however there was a pattern of increased symptoms and chronic illness in children aged 1–5 in the area with high wood smoke.

These suggestive data stimulated another study in the same air shed. In this second study, pulmonary function was measured in third through sixth grade children in two elementary schools in the area characterized as being affected by wood smoke (54). FEV_1 and FVC were measured before, during, and after the heating season in 326 children during 1988–89 and in just 26 children with asthma in 1989–90. Wood smoke was assessed using an integrating nephelometer, a light scattering device. In this airshed there is a high correlation between light scattering coefficient and PM_{10}. Analyses show that greater than 80% of particles in residential neighborhoods are from wood burning during winter months (57). Random and mixed effects models of statistical association were used to evaluate the relationships

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between lung function and wood smoke concentrations. Lung function measurements were compared with wood smoke concentrations for the previous 12 hr period from 7 p.m. to 7 a.m. Statistically significant decrements in both FEV_1 and FVC were seen in young children with asthma, both at the $p < 0.05$ level. FEV_1 and FVC dropped an average of 34 ml and 37 ml, respectively, for each unit of light scattering coefficient (1×10^{-4} m^{-1}). During the study period the PM_{10} levels were over 90 $\mu g/m^3$ on four nights in 1988–89 but not above 110; the highest value during 1989–90 was 103 $\mu g/m^3$. Thus, the National Ambient Air Quality Standard for PM_{10} was not violated during either heating season. It was concluded that wood smoke is significantly associated with respiratory function decrements in young children with asthma.

A study of the relationship between fine particulate matter and emergency room visits for asthma in the metropolitan Seattle area was designed to help determine whether air pollution is a risk factor for asthma (82). Using Poisson regressions controlling for weather, season, time trends, age, hospital, and day of the week, a significant association ($p < 0.005$) was found between fine particles measured at the residential monitoring station used in the studies described above and visits to emergency departments in eight participating hospitals. Analyses show that between 60 and 90% of particles in residential neighborhoods measured either gravimetrically or by nephelometers are from wood burning year round (57).

Suspended particulate air pollution is associated with decreased lung function and increased prevalence of respiratory disease symptoms in young children under 12 years of age (27). In this two-year study of the relationship between pulmonary function changes in third and fourth grade children and air pollutant alerts in Steubenville, Ohio, researchers found a decline in pulmonary function tests associated with increasing 24-hr concentrations of total suspended particulate matter (TSP). Peak values of TSP ranged from 27 $\mu g/m^3$ to 422 $\mu g/m^3$. The pulmonary function declines were small but persisted for up to two weeks. The elimination of children with reported prevalence of coughs, colds, and other respiratory symptoms did not change the estimated mean effect. Similar findings were reported from the Netherlands (24) in a study of children aged 6–11 years before and during an air stagnation episode, although the effects of allergy and chronic respiratory disease were not evaluated. More recently, Dockery and co-workers (26) have reported increased rates of cough, bronchitis, and chest illness in children exposed to inhaled particulate pollution.

It certainly is biologically plausible that wood smoke could cause adverse respiratory effects. The average size of the particles (< 1 μm) is such that these will travel deep into the lower respiratory tract (2). Some chemical species in wood smoke are chemically reactive and thus present a risk to

(17)

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respiratory tissues. The complex mixture of wood smoke allows deposition of reactive chemical onto particles that then can be carried into the alveolar region of the lung. As stated by Ammann (2), "irritants such as phenols, aldehydes, and quinones, as well as nitrogen oxides and sulfur oxides, in smoke may also contribute to both acute and chronic health problems. Generally irritants interfere with ciliary activity ... and hence the flow of the particle-trapping mucous stream. Inflammation, with all of its sequelae, also results."

In the earlier Six City Study report of children in Steubenville (27), a group median estimate of the slope between FVC and total suspended particulate was -0.081 mL/μg/m^3 for all children. When the estimate of a similar relationship (FVC/measure of fine particle concentration) is made using the Seattle data (54), the estimated mean FVC decrease per unit increase of $PM_{2.5}$ is -1.8 mL/μg/m^3 and $+0.34$ mL/μg/m^3 for asthmatic and nonasthmatic children, respectively. The FVC change per unit increase in $PM_{2.5}$ for the asthmatic children in our study is sufficiently pronounced as to suggest that fine particulate matter measured with a nephelometer may be more irritating than general industrial TSP. However, the difference between the two studies may be due solely to a increased sensitivity to airborne irritants in children with asthma. Based on prior work by Larson (56), the fine particulate matter measured on winter nights in this Seattle residential area is almost exclusively the result of residential wood-burning.

SUMMARY

In conclusion, this review reveals much about the constituents and fate of wood smoke but not enough about the health effects. Animal toxicological studies show that wood smoke exposure can disrupt cellular membranes, depress macrophage activity, destroy ciliated and secretory respiratory epithelial cells, and cause aberrations in biochemical enzyme levels. With respect to the human epidemiological data, the literature summarized in Table 4 shows a coherence of the data from young children, with 7/8 studies especially in children with asthma, reporting increased respiratory symptoms, lower respiratory infection, and decreased pulmonary function as a result of exposure to wood smoke. As Bates (6) has discussed, coherence of the data, although not amenable to statistical tests, carries the weight of linkage and plausibility. These adverse respiratory effects associated with wood smoke exposure also comply with many of Brandon Hill's aspects of association necessary to establish causation (40). There is strength of association, consistency (7/8 studies showing positive associations), temporality, plausibility, coherence, and analogy (using ETS exposure; 70, 94). A biological gradient has not been shown, although one is suggested in the

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study of pulmonary function in wildfire fighters. We conclude that the preponderance of the data suggest a causal relationship between elevated wood smoke levels and adverse respiratory health outcomes in young children.

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Annual Review of Public Health
Volume 15 (1994)

CONTENTS

Annu. Rev. Public. Health. 1994.15:133-156. Downloaded from arjournals.annualreviews.org
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(Continued)

124

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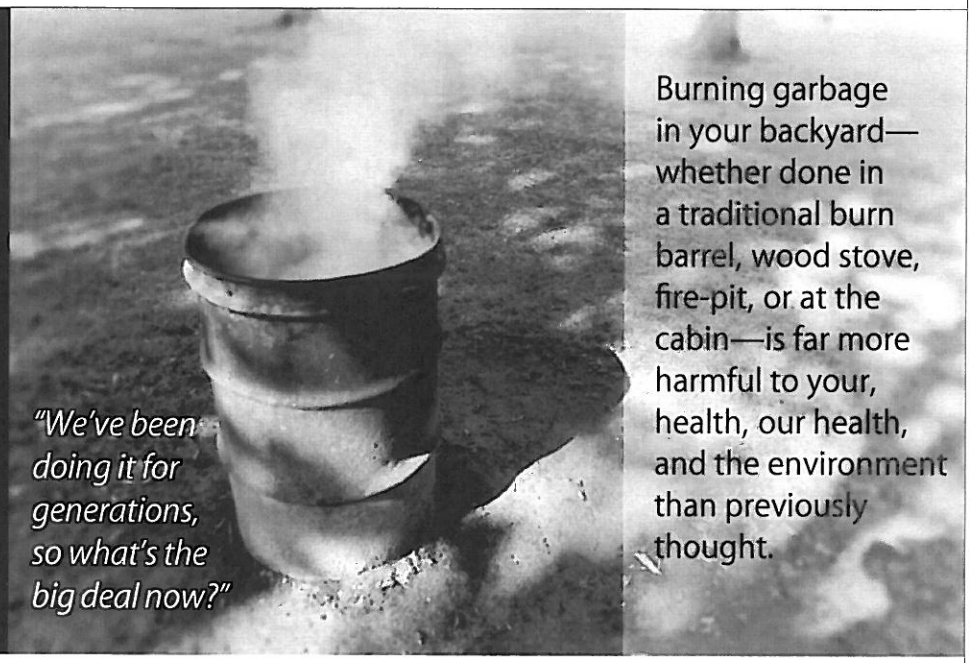
125

If you're burning garbage, you're making poison.



Burning garbage in your backyard—whether done in a traditional burn barrel, wood stove, fire-pit, or at the cabin—is far more harmful to your, health, our health, and the environment than previously thought.

Backyard garbage burning can affect your health

Garbage has changed

Until a few decades ago, burning garbage in the backyard was much less dangerous to your health. Fifty years ago, most household garbage contained only untreated paper, wood, and glass. Today's garbage contains paper, plastics, and other types of packaging waste that release a hazardous mixture of carcinogens and other toxics (such as lead, mercury, and arsenic) when burned. Even seemingly harmless items, like paper, mail, packaging, and cardboard boxes used for frozen pizzas and vegetables, can give off toxic emissions.

Smoke

Pollution created by backyard garbage burning increases the health risk to those exposed directly to the smoke, which is an irritant that especially affects people with sensitive respiratory systems, as well as children and the elderly. Exposure to smoke can also increase the risk of heart disease, cause rashes, nausea, and headaches.

Dioxin

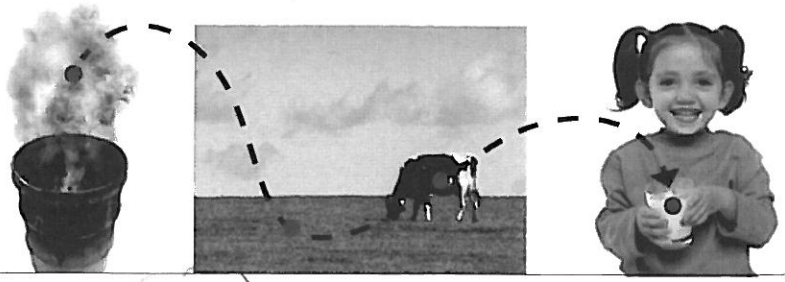
But the health concerns go well beyond those who are directly exposed—to those indirectly exposed to these toxic chemicals through their food. Among the health risks posed by backyard burning, dioxin—a known, potent human carcinogen and endocrine disrupter—is the main concern. Dioxin can have significant impacts on human immune, developmental, and reproductive systems. Dioxin exposure is especially harmful for children, pregnant women, and the elderly.

In Minnesota, dioxin is of particular concern since a recent survey shows that 45 percent of rural Minnesota residents still burn their garbage *(see our website)*. Because burn barrels are more common in the rural, agricultural areas of the state, there is particular concern about high levels of dioxin settling on crops and in our streams and lakes—and eventually winding up in the food we eat. Dioxin produced by backyard burning is deposited on plants, which in turn are eaten by animals. When people eat meat and dairy products, the dioxin is also absorbed. In fact, over 90 percent of all human dioxin uptake comes from meat and dairy consumption.

The EPA has been conducting exhaustive studies of dioxins for years. The good news is that as we've reduced the amount of dioxin in the environment, we've seen a corresponding reduction in the average level of dioxins in humans—from an average of 55 parts per trillion (ppt) in the 1980s to 25 ppt by the 1990s. The bad news, however, is that further studies show that health effects are detected at levels below 1 ppt.

An example of how dioxin in the smoke from burning garbage can end up in our food. When livestock eat feed that has been contaminated with dioxin, they concentrate the chemical in their milk and meat.

Dioxin and our food



Minnesota Pollution Control Agency

w-hhw1-17

www.pca.state.mn.us/burnbarrel

The arguments for reducing backyard burning are clear

- Largest remaining source of dioxin, a potent carcinogen, as well as other health and environmental risks.

- Nearly 45% of rural Minnesotans and an estimated 20 million Americans still burn their garbage on-site.

- Proximity to animal feed and food crops creates a serious risk to our food supply.

- Backyard burning contributes to nearly half of all wildfires in Minnesota each year.

Reduction efforts in Minnesota

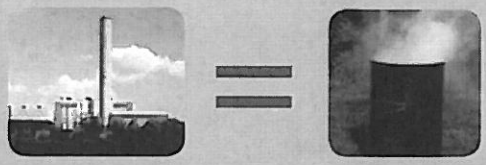
In Minnesota, open burning of household garbage is banned, with the exception of farms where regularly scheduled pick up of waste is not "reasonably available to the resident" (Minn. Stat. §§ 17.135 and 88.171). However, 28 of Minnesota's 87 counties have passed no-burn/bury resolutions to close this exemption.

Statewide, the MPCA is working on the first phase of its Burn Barrel Reduction Campaign, a multi-year effort to reduce backyard garbage burning throughout Minnesota. Based on recommendations to the Legislature in the 2005 Solid Waste Policy Report, the MPCA will work to eliminate burn barrels by 2010.

Partnerships with state and local government. The MPCA has also worked with counties, local units of government, haulers, and rural residents on programs designed to reduce the use and prevalence of burn barrels and backyard burning through education, incentives, enforcement, and infrastructure development. Many counties like Houston, Otter Tail, Carver, Chisago, Crow Wing, St. Louis, and the Western Lake Superior Sanitary District have seen significant reductions in burn barrel use as a result of programs developed in partnership with neighboring counties, the MPCA, and DNR. Many other counties are also concerned about backyard garbage burning; 24 counties were being awarded burn barrel reduction grants in northeast, southwest, and east central Minnesota in 2007 with more projects expected to begin throughout 2007 and 2008.

Minnesota Pollution Control Agency

Did you know?



The U.S. EPA estimates that one burn barrel (from an average family of four) can produce as much or more dioxin as a full-scale municipal waste incinerator burning 200 tons per day.

Burning garbage in burn barrels or fire pits creates low-temperature fires (less than 2,200° F), which receive very little oxygen and produce a lot of smoke. Under these conditions, a variety of toxic substances are produced and then released directly into the air without being treated or filtered.

Local governments leading the way

Chisago County reduced the number of residents who use burn barrels by 40 percent in four years after passing a no-burn resolution and conducting an education and incentive program called the Burn Barrel Buy-Back Campaign (4Bs). In a joint effort with local haulers, this program offered six months of garbage service at half price to residents who turned in their old burn barrels and signed up for garbage service. The haulers collected residents' old burn barrels and ash and disposed of them at no charge. This had the added benefit of increasing the number of customers for local haulers; and after a short time, the haulers were actively promoting the program to residents. This project was funded by a grant with the MPCA, and MPCA staff worked hand-in-hand with the county to develop the program.

Other local governments are using various education and reduction programs along with parts of Chisago's 4Bs program. Crow Wing and St. Louis have both conducted education campaigns through billboard displays, and St. Louis is in the process of measuring the impact on the number of people who no longer use burn barrels. With its successful Bernie the Burn Barrel TV ads, WLSSD has continued its education campaign, hosted a well-attended workshop for elected officials on burn barrels, and worked with the MPCA to update *Clearing the Air: Tools for Reducing Residential Garbage Burning*, a resource for Minnesota local governments.

For more about the dangers of burning or how to get involved in the campaign

www.pca.state.mn.us/burnbarrel

651-296-6300 | 800-657-3864

MEMORANDUM

TO: Lisa Boardman, Chairperson
Members of the Forest and Beach Commission

FROM: Mike Branson, City Forester

DATE: 3 July 2014

SUBJECT: Consideration of an Arbor Day Event for 2014

The City of Carmel does not have an officially designated "Arbor Day". A day to recognize and promote the value of trees to the community fulfills a duty and responsibility of the Forest and Beach Commission, as well as, meeting one of the requirements for designation as a Tree City USA.

The Friends of Carmel Forest have a lecture program and tree walks with Dr. Matt Ritter scheduled for July 10th and 11th. The City can designate these activities as the City's required Arbor Day event, even though they are not presented or produced by the City, or decide to do another program later this year.

If the Commission decides to do another program, please bring any ideas for discussion and consideration at this meeting noting some previous suggestions regarding this annual event:

- Attract a diverse audience
- Consider a Saturday program
- Possible children's program
- Interactive or hands-on program
- Tree care program with an environmental component

Supporting Information

The City of Carmel does not have an officially designated "Arbor Day". A day to recognize and promote the value of trees to the community fulfills a duty and responsibility of the Forest and Beach Commission, as well as, meeting one of the requirements for designation as a Tree City USA. The Tree City USA standards are attached for reference.

Previous "Arbor Day" events:

2005 - Tree seedling giveaway in December at Devendorf Park.

2006, 2007, and 2008 - Guest speaker presentations on tree care, pruning, and landscaping were co-sponsored with the Friends of Carmel Forest at Sunset Center.

2009 – Tree care workshop in February at Vista Lobos. Presentations were made by the City Forester and arborist Peter Quintanilla from the Friends of Carmel Forest.

2010 – Trees and Art event at the Sunset Center, Carpenter Hall. Guest speakers on the

Carmel forest management plan and the use of local trees in the art history of Carmel and the Monterey Peninsula. The event also featured a juried art and photo competition. This event was co-sponsored with the Friends of Carmel Forest.

2011 – Tree forum featuring local architect Paul Byrne speaking on sustainable building, Monterey Pine Watch presented an overview of their new book on the Monterey pine forest. Sustainable Carmel introduced their organization and their plans for the future, and the city forester spoke about Carmel's tree rules.

2012 – Japanese cherry tree planting ceremony in Devendorf Park. This was attended by the Mayor, City Council members, Forest and Beach Commissioners and representatives of the Japanese embassy in San Francisco.

2013 – Tree care workshop was presented by the City Forester at the Vista Lobos community room.

The Four Standards for Tree City USA Recognition

To qualify as a Tree City USA community, a town or city must meet four standards established by The Arbor Day Foundation and the National Association of State Foresters.

These standards were established to ensure that every qualifying community would have a viable tree management plan and program.

It is important to note that they were also designed so that no community would be excluded because of size.

1. A Tree Board or Department
2. A Tree Care Ordinance
3. A Community Forestry Program With an Annual Budget of at Least $2 Per Capita
4. An Arbor Day Observance and Proclamation

1) A Tree Board or Department

Someone must be legally responsible for the care and management of the community's trees. This may be a professional forester or arborist, an entire forestry department, or a volunteer tree board. Often, both a professional staff and advisory tree board are present, which is a good goal for most communities.

A tree board, or commission, is a group of concerned volunteer citizens charged by ordinance with developing and administering a comprehensive tree management program. Balanced, broad-based community involvement is encouraged. Boards function best if not composed entirely of tree-related professionals such as forestry professors, nursery operators, arborists, etc. Fresh ideas and different perspectives are added by citizens with an interest in trees that is entirely

avocational. Limited, staggered terms of service will prevent stagnation or burnout, while at the same time assuring continuity.

2) A Tree Care Ordinance

The tree ordinance must designate the establishment of a tree board or forestry department and give this body the responsibility for writing and implementing an annual community forestry work plan. Beyond that, the ordinance should be flexible enough to fit the needs and circumstances of the particular community.

A tree ordinance provides an opportunity to set good policy and back it with the force of law when necessary. Ideally, it will provide clear guidance for planting, maintaining and removing trees from streets, parks and other public places. For tips and a checklist of important items to consider in writing or improving a tree ordinance, see Bulletin No. 9.

3) A Community Forestry Program With an Annual Budget of at Least $2 Per Capita

Evidence is required that the community has established a community forestry program that is supported by an annual budget of at least $2 per capita. At first, this may seem like an impossible barrier to some communities. However, a little investigation usually reveals that more than this amount is already being spent by the municipality on its trees. If not, this may signal serious neglect that will cost far more in the long run. In such a case, working toward Tree City USA recognition can be used to re-examine the community's budget priorities and re-direct funds to properly care for its tree resource before it is too late.

Ideally, this standard will be met by focusing funding on an annual work plan developed after an inventory is completed and a report is approved by the city council. Such a plan will address species diversity, planting needs, hazardous trees, insect and disease problems and a pattern of regular care such as pruning and watering.

4) An Arbor Day Observance and Proclamation

This is the least challenging and probably the most enjoyable standard to accomplish. An Arbor Day celebration can be simple and brief or an all-day or all-week observation. It can be a simple tree planting event or an award ceremony that honors leading tree planters. For children, Arbor Day may be their only exposure to the green world or a springboard to discussions about the complex issue of environmental quality.

The benefits of Arbor Day go far beyond the shade and beauty of new trees for the next generation. Arbor Day is a golden opportunity for publicity and to educate homeowners about proper tree care. Utility companies can join in to promote planting small trees beneath power lines or being careful when digging. Smokey Bear's fire prevention messages can be worked into the event, as can conservation education about soil erosion or the need to protect wildlife habitat.

Still another way to develop Arbor Day is to link it with a tree-related festival. Some that are currently celebrated include dogwood festivals, locust blossom festivals and Macon, Georgia's Cherry Blossom Festival that annually brings more than $4.25 million into the local economy. In meeting the four standards, help is available! The urban and community forestry coordinator in your state forester's office will be happy to work with communities in taking these first steps toward better community forestry.

MEMORANDUM

TO: Lisa Boardman, Chairperson
Members of the Forest and Beach Commission

FROM: Mike Branson, City Forester

DATE: 6 July 2014

SUBJECT: Update on oak tree diseases and tree care.

During public appearances of the June 25, 2014 special meeting, Barbara Livingston asked for some information on new oak disease and proper care of trees. I provided Mrs. Livingston the information she requested via email following the meeting, but it is also something that the Commission should be aware of for their own benefit.

I have attached an article on the new oak disease and some tree care information from the Britton Foundation and the California Oak Foundation.

132

California's oaks — more bad news

A FUNGUS ASSOCIATED with the western oak bark beetle is causing a decline in coast live oak trees in Southern California by spreading "foamy bark canker disease." It has also been reported as far north as Monterey County. At this time, only coast live oaks have been infected, but other oaks species may be susceptible.

According to Akif Eskalen, UC Cooperative Extension specialist in the Department of Plant Pathology and Microbiology at the University of California, Riverside, "a decline of coast live oak trees have seen throughout urban landscapes in Los Angeles, Orange, Riverside, Santa Barbara, Ventura and Monterey counties,"

Eskalen's lab recovered the fungal species, *Geosmithia pallida*, from tissues of infected coast live oak trees and performed pathogenicity tests on it in his laboratory at UC Riverside. The tests showed that the fungus is pathogenic to coast live oak seedlings and produces symptoms of foamy canker.

Figure 1. Bark cankers (dark brown areas) caused by the fungus. (*Photo Akif Eskalen*)

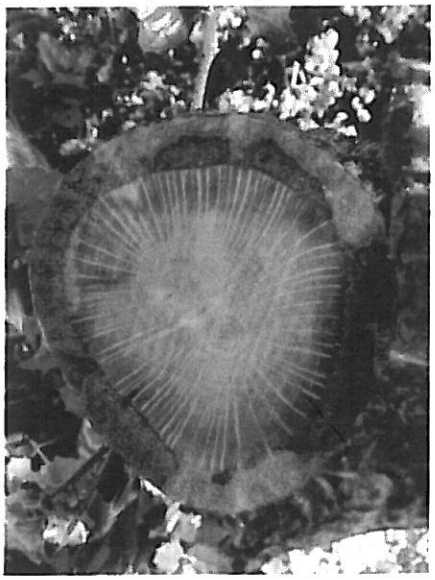


Figure 2. (Left) A coast live oak colonized by the western oak bark beetle that carries the new pathogen (*Photo Akif Eskalen*)

Figure 3. (Right) Symptoms also include a cinnamon-colored gum followed by a creamy, foamy sap, running down the bark. (*Photo Rebecca Latta*)

Arborists are asked to report suspicious cases of oak decline to their local UC Cooperative Extension advisor.

The western oak bark beetle (*Pseudopityophthorus pubipennis*), which is native to California has been implicated in the pathogen's spread. Female beetles burrow through the bark of the coast live oak tree, excavating shallow tunnels under the bark across the grain of wood (Fig. 1). Female beetles lay their eggs in the tunnels. It is not known at this time if the beetle infects trees other than coast live oak trees.

A related species, *Geosmithia morbida*, is a serious pathogen of walnuts, both native and commercial species. This pathogen, known for causing thousand canker disease, is native to Arizona, but is now established in California and the other western states. This disease is spread by the walnut twig beetle.

Initial symptoms of foamy bark canker disease on infected trees include multiple wet spots on the trunk and main branches, surrounding the entry holes made by the oak bark beetle. As the disease advances, a reddish sap may be seen oozing

(133)

from the entry hole (Fig. 2), followed by a prolific foamy or frothy mass. This pinkish foamy liquid, the cause of which remains unknown, may run as far as two feet down the trunk (Fig. 3). When dissected, the bark (phloem) surrounding the entry hole is dead and brown. Symptoms may be confused with those caused by the *polyphagous shot hole borer* (PSHB) associated with *Fusarium dieback*.

If just the branches are infected, they can be pruned to stop the spread of the disease, however once the trunk is infested the tree dies. Currently, no control methods are in place to control the fungus or the beetle.

It should be pointed out that the western oak bark beetle is a secondary pest, colonizing dying, stressed-weakened trees, windfall, and recently cut oak wood. Beetle populations, at present, particularly in southern California and central coast, are high due to severe drought conditions. Many oaks there are declining and therefore subject to attack. Healthy trees typically are not subject to attack unless beetle populations are inordinately high (Fig. 4). Pruning of stressed trees can attract beetles from the surrounding area, and therefore should be avoided unless done to remove diseased branches. Pesticide applications may be of value to protect high value trees. Irrigating drought-stressed trees, even minimally, can help reduce a tree's attractiveness and increase it natural resistance.

Figure 4. Branch dieback symptoms of foamy canker disease. (*Photo Akif Eskalen*)

Arborists are asked to report suspicious cases of oak decline with the symptoms listed above to their local UC Cooperative Extension advisor, pest control adviser, county agricultural commissioner's office, or Eskalen at **akif.eskalen@ucr.edu**.

Utility Vegetation Management Opportunities

WESTERN ECI

ECI and Western ECI Currently Has Openings for Entry Level and Experienced Utility Arborist Positions

Work Planners
Auditors
Ticket Inspectors
Consulting Utility Foresters
Supervisors
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Managing Trees During Drought & Water Restrictions

The BRITTON Fund
www.thebrittonfund.org

Drought and water restrictions are a part of life in the Western States. Arborists must cope with these adverse conditions by offering clients drought survival strategies when water budgets are implemented and irrigation is curtailed.

The west is an arid region. Although Hawaii enjoys greater rainfall in many areas, prolonged drought and water restrictions are still an issue.

Large trees are an easy target when water is restricted. However, the benefits trees provide usually pay back the water they use. Trees protect water sources by reducing topsoil erosion and intercepting rain and reducing runoff during storms. Tree roots capture nutrients and act as pollution filters. Couple that with additional tree services of energy reduction, air quality improvement and aesthetics and these tree benefits "pay" for the water applied.

Dieback is an advanced symptom of drought. Photos courtesy: Russ Thompson

SURVIVING WATER RESTRICTIONS

Many water districts offer incentives to remove turfgrass, often around mature trees. This has led to the decline of tree canopies in some urban areas.

Depending on the tree species, symptoms of drought can be slow and subtle or quick and deadly. Symptoms include wilted, yellowing and/or scorched leaves. Whole branches can die back, usually starting at the top. Drought stress can cause secondary disease and insect infestations that can accelerate decline.

Large trees can thrive even with turf removal if they receive adequate irrigation. Drip or low-flow irrigation can keep trees healthy with little or no water waste.

A ***drip (or "low flow") irrigation system*** is comprised of a valve, pressure regulator, filter, delivery tubing or piping and emitters. New technology in drip emitters can prevent clogging and pressure loss, two major drawbacks in a drip system.

Subsurface irrigation tubing has emitters built in at regular intervals. These can be installed in spirals, radial patterns or grids around trees. Subsurface systems can provide good uniformity using less components and labor.

Drip systems are capable of providing adequate water for tree health with little or no waste, but caution must be used. Too many times, only one or two emitters are installed. On a new installation, the tree can decline and fail just as it is reaching a beneficial size. On retrofits, using too few emitters can lead to rapid decline and death.

Traditional irrigation systems, using PVC pipe and bubblers, are still a viable choice. Since they are aboveground, it's easy to see if they are functioning properly. They are also less prone to breakage and clogging.

START WITH SELECTION

The best way to deal with drought is to select trees that are well adapted to the site. Start with native trees. Keep in mind, though, that just because a tree is native to your state, it does not necessarily mean it is the right choice for your particular location. Obviously, a coastal or riparian tree is not a good choice for an inland, high elevation, exposed location. Improper tree selection requires more applied water.

When selecting trees, matching them to the soil and exposure of the site is critical. Trees from similar geographic regions can be a better choice than "natives" that may prefer a very different climate zone and soil texture Plant lists for specific regions are available from several sources. Try your local water district, Cooperative Extension or even the local utility.

SMART SCHEDULING

Once a new tree has been properly planted, sound irrigation practices are critical to its survival and how well it can handle drought in the future. ***Newly planted trees need their existing rootballs to be evenly moist, with the surrounding soil irrigated to encourage rooting.*** As the tree matures, the wetted area should extend past the canopy to ensure that its water needs are met.

Managing Trees During Drought

www.thebrittonfund.org

Arborists need to be familiar with two key concepts of irrigation—frequency and duration. As irrigation frequency (the number of times a day, week or month the system is run) decreases, the duration (amount of "run time") should generally increase. Run times cannot be standardized because soil textures and the depth of soil also determine the amount of potential wetted area and potential soil water content and thus the run time.

Care must be taken when scheduling to be sure that water does not runoff, especially in heavy clay or "tight" soils. Tree berms may be required to minimize runoff.

Be sure that a tree's entire root system will be wetted when using drip irrigation. Photo courtesy: Joseph Fortier

PRUNING AND FERTILIZING

Once trees are established, providing adequate soil moisture is still one of the most important factors in their health. Pruning and fertilization, while viable, are not as much of a concern.

In times of drought, fertilization, especially with high nitrogen levels, should be reduced if not eliminated. As soils dry out the concentration of salts increases. Fertilized trees are more susceptible to salt burn during drought. If water is in limited supply, eliminate fertilizations. Try to apply longer run times less frequently to move salts lower in the soil profile.

Pruning can also encourage release of dormant buds and stimulate new growth. Heavy pruning can leave branch and bark tissue exposed to the sun and elements, resulting in sunburn of exposed stems. ***Prune only to remove dead wood, remove crossing competing branches and to maintain overall health.*** Light thinning (no more than 25% live foliage) that removes foliage will also cut transpiration and prevent water loss from water-stressed trees.

MULCH MATTERS

Another important tool for droughty times is mulch. The benefits of mulching are well established. Mulching a young tree will save about every other irrigation once established in the site soil. Mulching trees after turfgrass removal will help prevent water loss from the soil surface, but will not take the place of applied water. Mulched trees still need irrigation.

Organic mulch is preferred. As it decomposes, it improves soil quality. Tree trimmings run through a well-maintained chipper makes an ideal mulch. Avoid diseased or infested materials. This mulch can be laid up to a depth of up to 12 inches; it will rapidly compress and meld to form a "mat" that will retard moisture loss while also allowing rain and irrigation to reach the soil surface and prevent runoff. Mulched soils also take water in at a higher rate, so irrigation can be applied with higher flow if necessary; useful when irrigation time and days are restricted.

Organic mulches should be replenished annually. Avoid composts; because of their fine texture they hold moisture, allow for weed germination, and can also promote water loss from underlying soils. The best mulches are coarse chips.

Inorganic mulches are a popular option in desert landscapes. Rock, gravel and decomposed granite are used to a depth of one to three inches. Although they are durable and can be aesthetically pleasing, they can also reflect heat, driving up temperatures.

In times of drought, arborists must acquaint themselves with the numerous benefits trees provide. Unfortunately, sometimes trees lose the battle, and replacement is the only option. Mature trees in arid climates that were planted when water was plentiful might not survive water restrictions. In that case, arborists can provide safe removal and offer options for replacement using water-efficient trees.

Further Reading/Links:

Managing Trees During Drought (Dr. Ed Gilman)
http://hort.ifas.ufl.edu/woody/drought.shtml

Tree Irrigation Overview
http://www.snwa.com/land/install_drip_trees.html

Fifth in a series of 10 Technical Information sheets by Helen M. Stone and Dr. AJ Downer. Funded by The Britton Fund, Inc. and supported by the Western Chapter of the International Society of Arboriculture. Copyright 2013.

Caring for Old Native Oaks

The BRITTON Fund
www.thebrittonfund.org

Oak trees are beloved throughout the world, with more than 600 species found in the northern hemisphere. In the West, they occur throughout California and Arizona, from coastal climates to the mountains – in almost all ecosystems with the exception of the high desert.

Mature specimens are prized and add substantial value to a home or commercial property. Yet too often we see a stately giant in a slow state of decline. How can arborists make sure that these priceless treasures survive and thrive?

As a general rule, less is more. ***The closer a tree's native surroundings can be simulated, the better off it will be.*** Keep in mind that the tree's most active roots are close to the surface of the soil—no deeper than one to three feet, with absorbing roots prolific in the top six to 12 inches. Roots can also extend far beyond the "drip line" or canopy of the tree—an astounding two to three times the distance in optimum conditions.

PRESERVATION DURING CONSTRUCTION

Preserving mature oaks starts before any construction project, with a plan and design that keeps the trees' needs in mind. Provide room for leaves and mulch to accumulate on an undisturbed root system. ***Traffic should be directed away from specimen trees when possible, and raising or lowering existing grades avoided***. Placing soil on top of the roots will literally suffocate them, while scraping away the soil will destroy absorbing roots.

Once construction begins, the area beneath the tree(s) should be fenced off and strictly off-limits to vehicles, equipment and personnel. Chain link fence is preferable to the commonly seen orange plastic fencing, which can be easily breached. ***Fencing should extend beyond the trees' canopies if possible***. Mulching with a thick layer of wood chips also helps protect the root zone. The site should be monitored and any restrictions enforced immediately.

Chain link fence is preferable to orange plastic fencing to protect oaks during construction. Photo courtesy: HortScience, Inc.

LANDSCAPING UNDER OAKS

Once the building construction is complete, landscape construction is the next challenge. Again, caring for oaks starts at the design phase. If at all possible, ***landscaping and planting under established oaks should be kept to a minimum***.

If supplemental landscaping is necessary, try to limit plantings to accent plants, rather than blanketing the soil surface beneath the trees' canopies. Aim to disturb the roots as little as possible.

Turfgrass should be avoided, as should plants that require copious summer water such as azaleas, rhododendrons and the like. Ornamental grasses and native, drought-tolerant plants such as *Mahonia* or *Heuchera* (coral bells) are suggested.

IRRIGATION ISSUES

When rainfall is at normal levels, native oaks do not need supplemental irrigation. If landscape plantings require irrigation, use drip lines with emitters placed at each plant rather than sprinkler systems that flood the root zone. Avoid sprays on the tree's trunk at all costs.

During drought years, oaks will benefit from supplemental irrigation, especially during the normal rainy season. If the winter has been dry, irrigate during early spring. Irrigation can continue into summer, but limit cycles to once a month or less. During warmer months take care not to irrigate near the base of the tree as this can exacerbate any possible oak root fungus infections.

The "irrigation zone" should be approximately half way between the tree's drip line and the trunk and extend a few feet beyond the canopy. Irrigate deeply. A soil probe should be used to test the soil moisture content. Avoid woody roots when inserting the probe. The soil should be moist to the depth of approximately 12 inches. Proper irrigation at a slow

Caring for Old Native Oaks

www.thebrittonfund.org

rate can take several hours per tree to avoid runoff and ensure that the water penetrates deeply.

It was long thought that younger mature oak trees would adapt to frequent irrigation. This is false. Although young trees tolerate irrigation, their lives will be shortened if root diseases set in.

PRESERVING ROOT ZONE BIOTA

Oak trees require organic matter around their root systems. ***Do not remove leaf litter if at all possible.*** Fallen leaves provide nutrients slowly as they decompose, help moderate soil temperatures, prevent water loss and encourage earthworms and other beneficial insects in the soil. Oaks are unique in that they can absorb forms of nitrogen in organic litter that other trees cannot uptake.

Mushrooms at a tree's base are a sign that Oak Root Fungus is present. Photo courtesy: Jim Downer.

With sufficient mulch and water, oaks should not require supplemental fertilization. However, if they are located where leaf litter is regularly removed or there is extensive landscaping, supplemental nitrogen might be required.

Broadcast one to three pounds of actual nitrogen per 1,000 square feet in the irrigation zone and water in. Calculate the actual nitrogen in a fertilizer by multiplying the weight of the bag by the percentage nitrogen.

Do not fertilize stressed or declining trees in the mistaken notion that fertilizer is curative. Determine the cause of the stress and make corrections.

COMMON PESTS AND DISEASES

Oak trees are highly susceptible to root and crown rots. When carelessly irrigated, a seemingly healthy tree can suddenly topple over with no warning. Examining the failure will show no visible roots past the trunk.

The two major oak diseases are Crown Rot (*Phythophthora* spp.) and Oak Root Fungus (*Amillaria mellea*). In both cases, signs include slow or reduced growth, dieback, premature leaf drop and general symptoms of decline. ***By the time symptoms such as trunk cankers, canopy dieback, or defoliation occur, it is usually too late to save the tree.***

The best treatment for these oak-destroying diseases is to avoid them in the first place. ***Improper irrigation, root cutting and fill over the root ball are primary predisposing factors for oak diseases.*** Although a variety of insects can appear on oaks, treatment is usually not recommended or necessary with the exception of ambrosia beetles in Northern California and Oak bark beetles and polyphagous shot hole borers in Southern California, which can destroy weakened oaks.

PROPER PRUNING PRACTICES

Oaks need pruning to structure their canopies as young trees in urban settings. ***Mature oaks need little pruning and old specimens require little or no pruning.*** Pruning removes leaves and stored carbohydrates in wood that mature oaks depend on for annual growth. Even deadwood removal is not necessary if that deadwood can become a part of the litter/mulch under the tree.

Mature oak canopies shade the main stem or bole of the tree and rely on that shade to prevent stress. Do not "skirt up" oaks as this places them in higher stress. Let branches provide shade as much as the site will allow.

Many oaks are also reliant on their inner canopies of "shade" leaves which continue to transpire and function during hot summer months. Crown cleaning is deleterious to most oaks if green leaves are removed.

Most oaks are easily attacked by decay fungi and so large cuts should be avoided. Frequent light prunings as the trees reach maturity will ensure good structure. As trees enter old age, respect for their canopies and litterfall zones will ensure their survival.

Further Reading/Links:

Compatible Plants Around Oaks
http://www.californiaoaks.org/ExtAssets/CompatiblePlantsUnder&AroundOaks.pdf

California Oak Disease and Arthropod (CODA) host index database
http://phytosphere.com/coda/

A Field Guide to Insects and Disease of California Oaks
http://www.fs.fed.us/psw/publications/documents/psw_gtr197/psw_gtr197.pdf

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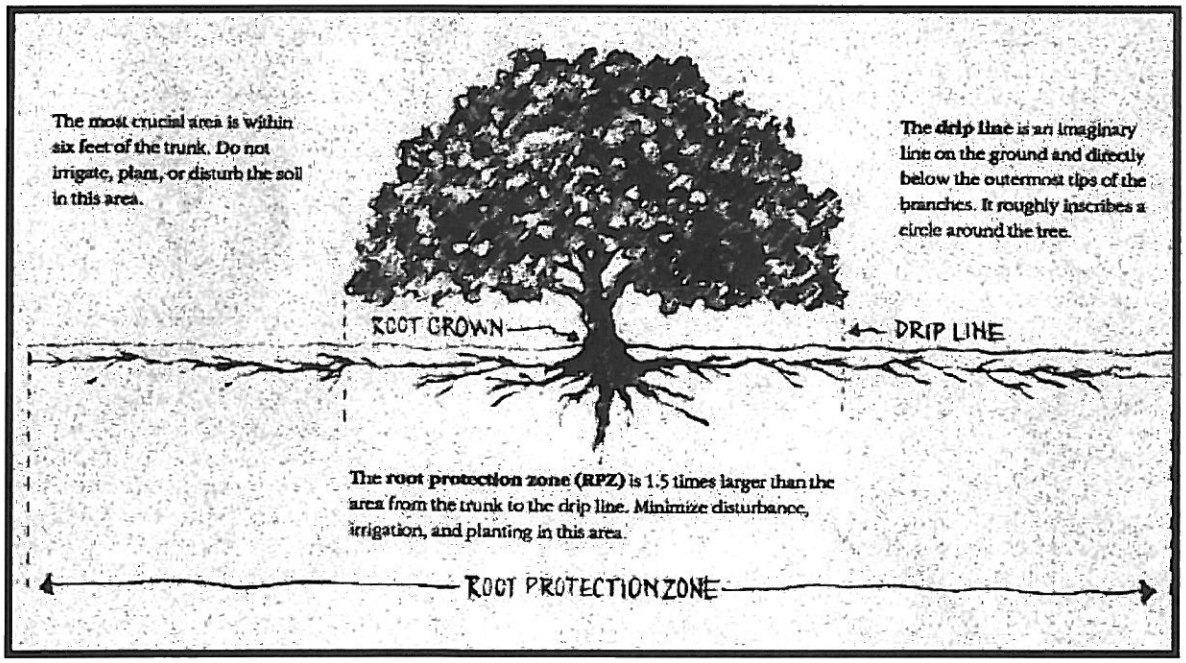
Care of California's Native Oaks
Bulletin of the California Oak Foundation

Native oaks, when young trees, are very tolerant of their environment and make excellent and adaptable landscape assets. The mature native oak is an invaluable part of our environment but does not tolerate many changes once established.

Architects, builders, homeowners, and others should be very careful in fitting their plans with these magnificent giants. Any substantial change in the mature oak's environment can weaken or kill an oak, even a healthy specimen.

A good rule of thumb is to leave the tree's **root protection zone (RPZ)** undisturbed. This area, which is half again as large as the area from the trunk to the dripline, is the most critical to the oak. Many problems for oaks are initiated by disturbing the roots within this zone.



A Word About Roots

Our native oaks have developed survival adaptations to the long, dry summers of most of California. Primary to this survival is the development and characteristics of its root system. When an acorn first sprouts, there is rapid root development and very little growth above ground.

This initial root is a tap root extending deep underground for dependable moisture. In fact, the tree's first few years are focused on establishing a deep sustaining root system. Once this has happened, greater foliage and above-ground growth takes place.

As the oak grows, the tap root is outgrown by an extensive lateral root system that spreads horizontally out from the trunk to and well beyond the dripline, sometimes as much as 90 feet. For

a mature oak, this horizontal root system is the primary supporter of the tree for the rest of its life. It includes the important fine roots, which absorb moisture and nutrients. Most of the root system occurs within the top three feet of soil. In shallower soil the root system is concentrated in an even shallower zone, typically one to two feet below the surface.

As the oak matures, particularly in areas naturally dry in summer, deep-growing vertical roots form off the laterals, usually within ten feet of the trunk. These sinker roots exploit deeper soil moisture and add stability to an increasingly massive tree.

By the time a mature oak has established its elaborate root system – so well designed for its environment and particular site conditions – it has lost the vigor of youth. It is less tolerant of change and can less easily recover to support a fully developed living structure.

To protect a mature oak, pay particular attention to drainage, and avoid filling, trenching, or paving near its root zone.

Fill Around Oaks

Soil and other materials placed on top of the natural soil level, called fill, are usually compacted. They make the soil less permeable, thereby restricting or prohibiting the exchange of gases and movement of water. Excessive moisture trapped by fill can also cause root and crown rot. Because there is no guarantee that fill can be safely added around an oak tree, it is best to avoid tampering with the natural grade, or to leave the natural grade within the root zone alone and use retaining walls.

Drainage

Poor drainage is a common cause of oak tree deaths, since adequate drainage is critical to ensure a proper balance of moisture, air, and nutrient to grow and survive. Too much moisture, particularly in the warm months when natural conditions are dry, can smother the roots and encourage the proliferation of crown and root rot fungi.

Another moisture threat to oak roots is presented by barriers such as concrete foundations and footings, streets, and swimming pools downhill of oaks. These structures can dam underground water, causing water to back up into a tree's root zone and drown it.

Trenching

Trenching is an often-overlooked cause of tree death. Trenching usually occurs when underground utilities are installed. Digging a trench for utilities within the RPZ of an oak can sever a significant portion of a tree's roots. Often, several trenches are opened by separate utilities. This multi-trenching is particularly destructive since it impacts a greater portion of the root system.

If utilities must impinge on the root protection zone of a native oak, the trench should be dug by hand, avoiding roots, or utilities bored through the ground at least three feet below the surface.

Paving

Paving can cause the same problems associated with soil compaction. Paving, such as asphalt and concrete, prevents water from soaking into the soil and impedes the exchange of gases between roots, soil, and the atmosphere. In addition, paving usually requires excavation to create a stable base and to allow for depth of paving material. This process compacts the soil and damages roots.

Decking placed on piers is much more compatible with mature oaks than paving.

Care of Established Oaks on Home Grounds

Oaks on home grounds require certain conditions to survive and prosper. Activities of concern to the homeowner are planting near oaks, irrigation and feeding, pruning, installation of home improvements, and disease and insect infestations.

Most native oaks in California evolved and prospered in an environment typified by a cool, moist winter and a hot, dry summer. Under natural conditions, surface soils are wet during the cooler months and become dry by summer. Natural vegetation growing beneath oaks flourishes during the winter and spring and dies by early summer, creating the well-known golden-brown landscape of California's valleys and foothills.

Native oaks, however, remain green because their thick, leathery leaves and other adaptive features reduce their water use. The homeowner should attempt to approximate the natural environment in which these magnificent trees are originally found.

Planting Near Oaks

Only drought-tolerant plants that require no summer water should be planted around old established oaks, and they should be planted no closer than six feet from the base of the tree. Do not plant exotic grasses, ivy, azaleas, rhododendrons, or any other vegetation that needs summer irrigation. Such plants develop thick mats of roots and thus inhibit the exchange of air and water the established oak has grown used to.

There are a number of plants, some of which are native to California, that can be grown beneath oaks. For an extensive listing of compatible plants useful for landscaping around oaks, contact the California Oak Foundation.

In place of plants, other types of ground cover can be used to landscape beneath oaks. When installed properly, cobbles, gravel, and wood chips are good examples of ground covers that do not interfere with the roots' ability to obtain oxygen and appropriate moisture.

Irrigating and Fertilizing

Native oaks usually do not require irrigation as they are well adapted to dry summer conditions. Healthy oaks are even able to survive the excessively dry summers sometimes brought on by California's variable climate. But if an oak has been compromised, as when impervious surfaces have been placed in the RPZ, occasional water may be helpful if done properly.

Oaks should be irrigated only outside of the RPZ. Under no circumstances should the ground near the base of a native oak be allowed to become moist during warm weather periods. Moist, warm soil near the base of a mature oak promotes crown and root rot.

Irrigation, if done, should be by the "deep watering method," which consists of a slow, all-day soaking only once or twice during the summer dry period. Frequent, shallow watering not only encourages crown and root rot, it also results in the growth of ineffective shallow roots near the surface, a needless waste of the tree's energy.

If oaks need supplemental watering, it is best to apply the water at times that lengthen the normal rainy season, so the normal dry period in the middle to the end of summer is preserved. For example, additional irrigation would be appropriate in May and September, while leaving the area under the tree dry in July and August.

Mature oaks usually need little or no supplemental fertilization. Light fertilization may be appropriate in landscaped situations to replace nutrients supplied by leaves and other litter that normally accumulates under an oak in its native environment. If leaves are allowed to remain under trees, they eventually break down and supply nutrients.

Fertilization should only be done if growth is poor. Fertilizers should be applied to the entire RPZ, ideally in late winter or early spring. Trees that have recently undergone severe pruning or root damage should not be fertilized for at least six months.

Often, when an oak tree shows yellowing leaves, one thinks it lacks nutrients. Generally, this is not the case. More likely, the tree is suffering from root or crown rot. When an oak appears unhealthy, consult a certified arborist to determine the cause.

Pruning

Excessive pruning or thinning of limbs may expose interior branches to sun damage, may stimulate the tree to produce succulent new growth that is subject to mildew, and, in some cases, may cause a decline in vigor or may kill a tree. *Only dead, weakened, diseased, or dangerous branches should be removed.* Necessary pruning should be done during the winter dormant period for deciduous species and during July and August for evergreen species. Recent research has shown that tree paint, wound dressings, and sealing compounds do more harm than good.

Pruning should be performed by a certified arborist according to the pruning standards of the Western Chapter of the International Society of Arboriculture.

Home Improvement

The installation of home improvements should be done with caution when oaks are located nearby. Trenching severs roots, and impervious surfaces placed over roots may result in the death of the oak. A swimming pool placed downhill of oaks can act as a dam and cause an oak to drown in saturated soil.

Great caution should be taken and a certified arborist consulted before proceeding with improvements that impact on the root protection zone of any valued native oak.

142

Diseases

When growing under natural conditions, native California oaks are relatively tolerant of most diseases. However, they are subject to several problems when disturbed or hampered by frequent summer watering.

The two oak diseases most often encountered in irrigating settings are crown rot and oak root fungus. Both attack trees weakened by disturbance or improper care.

Crown Rot
This is one of the most common and serious diseases of oaks in home plantings. Infected trees decline slowly over a period of years. The disease, caused by a microscopic fungus, is made worse by saturated soil and poor soil aeration.

Symptoms of this disease are a general decrease in tree vigor, twig die-back and wilting, abnormally yellow leaves, and formation of lesions on the bark accompanied by oozing of dark-colored fluid.

In most cases people notice crown rot too late for successful treatment. However, if the disease is caught in the early stages a tree can be saved. Comprehensive treatment is best left to a qualified expert. The following measures usually benefit the tree:

1) Remove lawn and other plants that require summer irrigation from within the RPZ.
2) Remove soil and all other debris that has accumulated against the trunk.
3) Do not water within the RPZ during the summer except under unusual conditions when advised by a certified arborist.
4) Improve drainage around the tree, and make sure all water drains away from the trunk.

Oak Root Fungus
This oak fungus, also known as *Armillaria* root rot, is found in the root systems of most oaks in California. Our oaks experience little damage from this fungus under natural, dry summer conditions. However, when oaks are watered in the summer or weakened by other impacts, the tree can suffer damage from the fungus.

Symptoms shown by an infected oak include die-back of branches and yellowing and thinning of foliage. The fungus itself may appear as a white, fan-like growth with rhizomorphs and mushrooms.

Prevention of damaging conditions is the only sure action that can be taken against this disease. Avoid summer irrigation near oaks. Prevent mechanical damage to major roots or root crown. As with crown rot and other tree diseases, it is recommended that a certified arborist be consulted.

Mistletoe
This parasitic plant grows on the branches of many oaks and can cause structural weaknesses that make branches more vulnerable to breakage. Its sticky seeds are spread from one tree to another by birds. The seeds germinate under favorable conditions, and rootlike structures find their way through the bark, ultimately becoming attached to the oak and tapping into the water-and-mineral-conducting tissues of the tree.

143

Small infestations can be controlled by removing the mistletoe and cutting back the oak's bark around the spot where the mistletoe stem entered the oak branch. Major infestations are difficult to control, however, and an arborist specializing in oaks should be consulted.

Other diseases

The health and vigor of oaks can also be compromised by a number of other afflictions that are not discussed here. Since 1980, for example, die-back and decline, particularly among the coast live oak (*Quercus agrifolia*), has been observed in widespread areas of California. Several fungi may be involved in this condition, and treatments are still experimental. Seek professional advice whenever you notice serious, unexplained decline in your oaks.

INSECTS

Innumerable insects find their livelihoods in the branches and leaves of oaks, usually without much consequence to the healthy tree. The oak gall, for example, is a harmless swelling of leaves and twigs in reaction to enzymes released where a wasp lays its eggs. Some galls are large and round, others resemble small wads of fuzz, stars, or tops; one, which looks like a tiny seed, falls from leaves in the late summer and occasionally jumps into the air like a Mexican jumping bean.

Some infestations, however, can cause serious damage. Insects such as pit scales (which appear as pinhead-sized scales on the bark of twigs), oak moth and other leaf-eaters can weaken oaks, making them susceptible to disease.

Whenever an insect infestation causes substantial leaf loss, changes in leaf color, twig die-back, sticky or sooty foliage and branches, or other significant changes in appearance, intervention may be required. Consult a certified arborist for assistance.

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The California Oak Foundation is dedicated to the conservation and perpetuation of California's native oak woodlands. The California Oak Foundation educates the general public and decision-makers about the importance of oak woodlands to California's wildlife habitat, watersheds, and quality of life through its newsletters, website, bulletins, books, symposia, and workshops.

Founded in 1988, the California Oak Foundation is a non-profit 501(c)(3) corporation that relies on memberships and donations to continue its work. Join us today and invest in the future of California's oak heritage.

144