HHS ITEM #1
June 23, 2011

Worksession

MEMORANDUM

June 21, 2011

TO: Health and Human Services Committee

FROM: Amanda Mihill, Legislative Analyst

SUBJECT: **Worksession:** Resolution to adopt Board of Health Regulation prohibiting smoking in certain common areas of multiple-family residential dwellings and certain playground areas

A resolution to a adopt Board of Health Regulation prohibiting smoking in certain common areas of multiple-family residential dwellings and certain playground areas, sponsored by Councilmembers Leventhal and Berliner, was introduced on March 29, 2011. A public hearing by the Health and Human Services Committee was held on May 5.

Public Hearing Testimony

At its May 5 public hearing, the Committee heard from constituents both supportive, including Rita Turner of the Center for Tobacco Regulation, and opposed, including local cigar shop owner Paul Spence and Bruce Bereano, on behalf of the Maryland Association of Tobacco and Candy Distributors, to the regulation. Additionally, the Council has received several e-mails from constituents supportive of the proposed regulation, including from an owner of apartments (©64). The Council did receive some correspondence from those opposed to the regulation, including the Board of Directors of Fairways South, a condominium in Leisure World (©60).

Several constituents urged the Council to expand the proposed regulation to include other areas such as the dwelling units inside the multiple-family residential buildings. Council staff cautions that the scope of the regulation is limited to common areas and playground areas and believes that expanding the regulation beyond these areas would be beyond the scope of the advertisement of the regulation. Therefore, if Councilmembers are interested in pursuing smoking limitations in these areas, a new regulation should be introduced.

Background

What Health Concerns are Associated with Secondhand Smoke? The United States Surgeon General first raised the topic of involuntary smoking in a 1972 Surgeon General's report entitled "The Health Consequences of Smoking". In 1986, the Surgeon General devoted an entire report on the health issues regarding involuntary smoking. The most recent version of this report, "The Health Consequences of Involuntary Exposure to Tobacco Smoke," was issued in 2006 and is available from Council staff or at the following website: http://www.surgeongeneral.gov/library/secondhandsmoke/. According to the Surgeon General, exposure to secondhand smoke can cause a variety of health ailments, including heart disease, lung cancer, respiratory diseases such as asthma and pneumonia, and sudden infant death syndrome. A pamphlet describing the health risks of secondhand smoke is on ©7-17. The Surgeon General has stated that there is no risk-free level of exposure to secondhand smoke. But, see ©18-28 for a different view.

What would the Proposed Regulation do? As introduced, the proposed regulation would prohibit smoking in indoor common areas of multiple-family dwellings. The proposed regulation would define a common area as "any indoor area of a multiple-family residential dwelling which is accessible to the occupants of more than one dwelling, including a hall, lobby, or laundry room" (©2, lines 2-6). The regulation would also prohibit smoking within 25 feet of a playground area. Playground area would be defined as any outdoor area of a multiple-family residential dwelling with playground equipment which is intended to be used by minors, such as a swing set, sandbox, slide, seesaw, or playhouse (©2, lines 9-15).

The proposed regulation would require the owner or person in control of a common area to post a sign prohibiting smoking in each common area and playground area and also notify each unit in the multiple-family residential dwelling in writing that smoking is prohibited in each common area and playground area (©2-3, lines 22-29). A violation would be a Class C violation (©3, lines 31-32). The Board of Health regulation would take effect 30 days after it is adopted.

What is the applicable state law? In 2007, the Maryland General Assembly enacted the Clean Indoor Air Act, which is enforced by the Maryland Department of Health and Mental Hygiene (DHMH). The Act prohibits smoking in indoor areas open to the public except in limited circumstances. The Act specifically prohibits smoking in public meeting places, public transportation vehicles, and indoor places of employment. Private homes and residences are generally exempt from the Act, but it is unclear how common areas in multi-family residential buildings are treated. Ms. Turner's testimony indicates that DHMH enforces the Clean Indoor Air Act as it relates to these indoor common areas. Prior to the Clean Indoor Air Act, the Maryland Occupational Safety and Health banned smoking in most workplaces. Under the Clean Indoor Air Act, the Department of Labor, Licensing and Regulation continues to be responsible for smoking in indoor places of employment not normally open to the public. Of note, the Clean Indoor Air Act specifically states that a county can enact and enforce more stringent measures to reduce involuntary exposure to environmental tobacco smoke.

What fiscal impacts could be associated with this proposed regulation? The Office of Management and Budget's fiscal impact statement estimates that the regulation will cost the

County approximately $11,000 per year. This assumes that 25 complaints will be investigated per year at a rate of 8 hours of staff time per complaint. The impact could be greater or less, depending on the number of complaints the Department receives and investigates. The statement indicates that the Department will be able to absorb the cost, but it could impact their ability to respond to all activities that inspectors currently undertake (©4-5).

Issues for Committee Discussion

1. *Should the regulation be expanded to include additional parks other than those only associated with multi-family dwellings?* As introduced, the proposed regulation would prohibit smoking within 25 feet of any playground area. "Playground area" would mean any outdoor area of a multiple-family residential dwelling with playground equipment which is intended to be used by minors." Council staff recommends that this definition be expanded to include all public and private outdoor playground areas and recommends the following amendment:

> "*Playground area*" means any outdoor area **[[**of a multiple-family residential dwelling**]]** with playground equipment which is intended to be used by minors, such as a:
>
> (1) swing set;
> (2) sandbox;
> (3) slide;
> (4) seesaw; or
> (5) playhouse.
>
> (b) **Smoking prohibited.** **[[**A**]]** Except as provided in paragraph (c), a person must not smoke:
> (1) in any common area in a multiple-family residential dwelling; or
> (2) within 25 feet of a public or private playground area.
>
> (c) **Applicability.**
> (1) This regulation does not apply to any playground area owned by the Maryland-National Capital Park and Planning Commission.
> (2) This regulation does not apply to any playground area located at, and used by the residents of, a detached single-family home.

The County is prohibited by State law from imposing rules and regulations for parks owned by the Maryland-National Capital Park and Planning Commission without the affirmative vote of three Montgomery County commissioners (see ©6). Therefore, Council staff recommends exempting these playground areas from the scope of this regulation.

2. *Should the scope of regulation be expanded to include other outdoor common areas?* As proposed, the regulation would prohibit smoking in indoor common areas. The Council received requests from several constituents urging that the regulation be broadened to include outdoor common areas such as parking lots, garages, and sidewalks. Council staff believes these proposed amendments are within the scope of the regulation's advertisement and if Committee

members are interested in expanding the definition of common areas to include outdoor common areas, Council staff will draft an appropriate amendment.

3. *Should the enforcement provisions be amended to require more detailed information?* In a June 10 memorandum, Uma Ahluwalia, Director of the Department of Health and Human Services indicated that while they supported the underlying goal of the regulation, they urged the Committee to amend the regulation's enforcement provisions to minimize the burden on the Department's licensure and regulatory staff (©29).

As introduced, the regulation would allow the Department to issue a citation for a violation if a Department employee witnesses the violation or receives a complaint from at least two individuals with personal knowledge of the violation. Ms. Ahluwalia recommended amending the regulation to require that a complainant submit an affidavit with certain detailed information, including the identity of the person alleged to have violated the regulation and the time and place of the banned activity. Council staff has drafted the following amendment for the Committee's consideration:

> (3) The Department of Health and Human Services may issue a citation for violating this Section if a Department employee:
>
> (a) witnesses the violation; or
>
> (b) receives **[[**complaints**]]** <u>an affidavit</u> from at least 2 individuals who have personal knowledge of the smoking violation. <u>The affidavit must identify the individual alleged to have violated this regulation and the time and place of the banned activity.</u>

4. *What enforcement provisions should apply to homeowner and/or condominium associations?* The Council received concerns from a homeowner's association that the regulation could be interpreted to require the homeowner or condominium association to be fined if an individual is found to be in violation of the regulation. Under the proposed regulation, a homeowner or condominium association would not be required to pay the fine of an individual that violates the smoking provisions in the regulation. However, since the regulation requires the owner or person in control of a common area to post at least one sign prohibiting smoking in each common area and playground area, an association could be cited for failing to post the required signs. If Committee members believe additional clarity is required to address this association's concerns, Council staff can draft an appropriate amendment.

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Resolution No.: _____
Introduced: March 29, 2011
Adopted: _____

COUNTY COUNCIL
FOR MONTGOMERY COUNTY, MARYLAND
SITTING AS THE MONTGOMERY COUNTY BOARD OF HEALTH

By: Councilmembers Leventhal and Berliner

Subject: **Board of Health Regulation prohibiting smoking in certain common areas of multiple-family residential dwellings and certain playground areas.**

Background

1. County Code §2-65 provides that the County Council is, and may act as, the County Board of Health, and in that capacity may adopt any regulation which a local Board of Health is authorized to adopt under state law.

2. Maryland Code Health-General Article §3-202(d) authorizes the County Board of Health to adopt rules and regulations regarding any nuisance or cause of disease in the County.

3. On May 5, 2011, the Health and Human Services Committee held a public hearing on this regulation. As required by law, each municipality in the County and the public were properly notified of this hearing.

4. The County Council, sitting as the Board of Health, finds after hearing the testimony and other evidence in the record of the public hearing that prohibiting smoking in multiple-family residential common areas and playground areas is necessary to protect the health of residents in the County.

Action

The County Council for Montgomery County, Maryland, sitting as the County Board of Health, approves the following regulation:

Prohibition of Smoking in Common Areas of Certain Multiple-Family Residential Dwellings and Certain Playground Areas

(a) **Definitions.**

"*Common area*" means any indoor area of a multiple-family residential dwelling which is accessible to the occupants of more than one dwelling, including a:

(1) hall;

(2) lobby; or

(3) laundry room.

"*Multiple-family residential dwelling*" means a dwelling containing 3 or more multiple-family dwelling units, which may or may not share a common entrance.

"*Playground area*" means any outdoor area of a multiple-family residential dwelling with playground equipment which is intended to be used by minors, such as a:

(1) swing set;

(2) sandbox;

(3) slide;

(4) seesaw; or

(5) playhouse.

"*Smoking*" means the act of lighting, smoking, or carrying a lighted or smoldering cigar, cigarette, or pipe of any kind.

(b) **Smoking prohibited.** A person must not smoke:

(1) in any common area in a multiple-family residential dwelling; or

(2) within 25 feet of a playground area.

(c) **Signs and notice required.**

(1) The owner or person in control of a common area, including any condominium or homeowner's association, must conspicuously post at least 1 sign prohibiting smoking in each common area and playground area. Any sign need not be permanently attached to a structure.

(2) No later than [30 days after adoption], the owner or person in control of a common area, including a condominium or homeowner's association, must notify

each unit in the multiple-family residential dwelling in writing that smoking is prohibited in each common area and playground area.

(d) **Enforcement.**

(1) Any violation of this regulation is a Class C civil violation. Each day a violation exists is a separate offense.

(2) The County Attorney or any affected party may file an action in a court with jurisdiction to enjoin repeated violations of this regulation.

(3) The Department of Health and Human Services may issue a citation for violating this Section if a Department employee:

(a) witnesses the violation; or

(b) receives complaints from at least 2 individuals who have personal knowledge of the smoking violation.

(e) **Applicability.** This regulation applies Countywide.

(f) **Severability.** If the application of this regulation or any part of it to any facts or circumstances is held invalid, the rest of the regulation and its application to all other facts and circumstances is intended to remain in effect.

(g) **Effective Date.** This regulation takes effect 30 days after it is adopted.

This is a correct copy of Council action.

_____

Linda M. Lauer, Clerk of the Council

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OFFICE OF MANAGEMENT AND BUDGET

Isiah Leggett
County Executive

Joseph F. Beach
Director

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MONTGOMERY COUNTY
COUNCIL
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MEMORANDUM

June 15, 2011

TO: Valerie Ervin, President, County Council

FROM: Joseph F. Beach, Director

SUBJECT: Resolution to adopt Board of Health Regulation prohibiting smoking in certain common areas of multi-family residential dwellings and certain playground areas.

The purpose of this memorandum is to transmit a fiscal and economic impact statement to the Council on the subject legislation.

LEGISLATION SUMMARY

Council Resolution 17-XXX would:

- prohibit smoking in any common area in a multiple-family residential dwelling, defined as any indoor area of a multiple-family residential dwelling which is accessible to the occupants of more than one dwelling, including a hall, lobby, or laundry room;
- prohibit smoking within 25 feet of a playground area, defined as any outdoor area of a multiple-family residential dwelling with playground equipment which is intended to be used by minors, such as a swing set, sandbox, slide, seesaw, or playhouse;
- require the owner or person in control of a common area, including any condominium or homeowner's association, to post at least one sign prohibiting smoking in each common area and playground area.

FISCAL AND ECONOMIC SUMMARY

The department estimates that the resolution will impact approximately 1,200 properties. The fiscal impact of this legislation was determined based on previous experience within the department. The department estimates 25 complaints will be investigated per year, assumed eight hours per complaint, for a total cost of $11,000 per year. Based on current costs, and assuming a constant complaint rate, the six year projected impact is $66,000. The department will be able to absorb this cost but it will impact the department's ability to respond to all activities currently undertaken by inspectors. Given that additional inspection requirements have been added to their workload over the last four years, without additional resources being added, there is some reduced capacity on inspection staff to fulfill all their assigned job responsibilities. The department notes that enforcement costs may be greater depending on the initial response to the legislation by residents and specificity of complaints received. There will be no additional revenue realized.

Given that the resolution does not prohibit smoking in general but only prohibits smoking in specified areas, the economic impact to employment, personal income, investment, or other economic variables cannot be known with any degree of certainty.

The following contributed to and concurred with this analysis: Uma S. Ahluwalia, Dr. Ulder Tillman, Clark Beil, Department of Health and Human Services, Mike Coveyou, Department of Finance, and Anita Aryeetey, Office of Management and Budget.

JFB:aa

c: Kathleen Boucher, Assistant Chief Administrative Officer
Lisa Austin, Office of the County Executive
Fariba Kassiri, Assistant Chief Administrative Officer
Uma S. Ahluwalia, Director, Department of Health and Human Services
Clark Beil, Department of Health and Human Services
Beryl Feinberg, Office of Management and Budget
John Cuff, Office of Management and Budget
Amy Wilson, Office of Management and Budget
Anita Aryeetey, Office of Management and Budget

Annotated Code of Maryland
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*** Current through chapters of the 2011 Regular Session of the General Assembly that took effect through May 19, 2011 ***
*** Annotations through April 29, 2011 ***

ARTICLE 28. MARYLAND-NATIONAL CAPITAL PARK AND PLANNING COMMISSION
TITLE 5. PROPERTY; POWERS; RECREATION PROGRAM.
SUBTITLE 1. METROPOLITAN DISTRICT PROPERTY AND POWERS GENERALLY.

GO TO MARYLAND STATUTES ARCHIVE DIRECTORY

Md. Ann. Code art. 28, § 5-101 (2011)

§ 5-101. General powers to acquire and develop property within metropolitan district

(a) General powers. -- For the purpose of carrying out its general plans for the physical development of the metropolitan district, or any part thereof, the Commission may acquire land or other property located within the district for parks, parkways, forests, streets, roads, boulevards, or other public ways, grounds, or spaces, by means of donations, purchases, or condemnation. The Commission may improve and develop land or other property so acquired by it for these purposes and has the control of the maintenance and operation thereof. No general regulation governing these public ways, grounds, or open spaces within either Montgomery or Prince George's County may go into effect unless and until it receives the affirmative vote of at least three members of the Commission from that county.

(b) Recreation facilities. -- The purposes for which the Commission may acquire lands or other properties, whether by purchase or condemnation, are enlarged to include the purposes of public recreation or the construction of public recreation centers, community buildings, or other public buildings necessary to house the public recreation program or any part thereof.

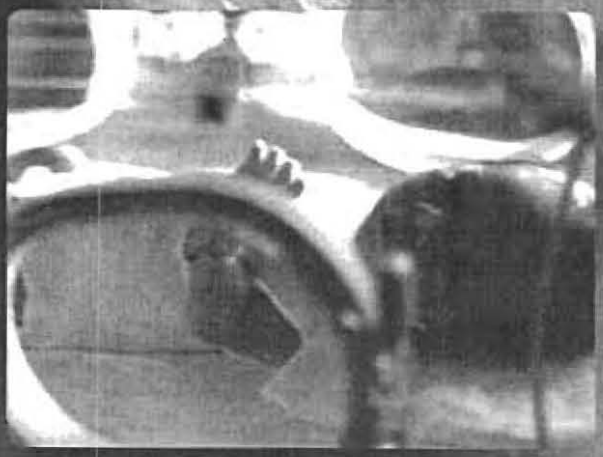
(c) Rock Creek restrictions. -- Notwithstanding any power given to the Commission to acquire land or other property, for the purpose of preserving and enhancing the scenic beauty of Rock Creek, the Commission may not construct without the approval of the majority of the members of the Montgomery County Council any new parkway, boulevard, street, or road adjacent to Rock Creek within 1500 feet from the banks of the creek. However, nothing in this section prohibits the construction of any bridge, parkway, boulevard, street, or road necessary to cross the creek.

HISTORY: 1975, ch. 892; 1983, ch. 57, § 1.

QUOTED IN Maryland-National Capital Park & Planning Comm'n v. Town of Wash. Grove, 408 Md. 37, 968 A.2d 552 (2009).

The Health Consequences of Involuntary Exposure to Tobacco Smoke

A Report of the Surgeon General



Secondhand Smoke

what it means to you

Secondhand Smoke

It hurts you.

It doesn't take much.

It doesn't take long.

The 2006 Surgeon General's report has new information about how breathing secondhand smoke hurts your health. You can find more information about this report by going to the Surgeon General's website at www.surgeongeneral.gov.

More information is also available by going to the Centers for Disease Control and Prevention (CDC) website at www.cdc.gov/tobacco.

Secondhand smoke is dangerous.

The Surgeon General of the United States, working with a team of leading health experts, studied how breathing secondhand tobacco smoke affects you.

This booklet explains what scientists have learned about the dangers of secondhand smoke. It also tells you how to protect youtself and your family.

What is secondhand smoke?

When a person smokes near you, you breathe secondhand smoke. Secondhand smoke is the combination of smoke from the burning end of the cigarette and the smoke breathed out by smokers. When you breathe secondhand smoke, it is like you are smoking.

Whether you are young or old, healthy or sick, secondhand smoke is dangerous.

What we now know:

- There is no safe amount of secondhand smoke. Breathing even a little secondhand smoke can be dangerous.
- Breathing secondhand smoke is a known cause of sudden infant death syndrome (SIDS). Children are also more likely to have lung problems, ear infections, and severe asrhma from being around smoke.
- Secondhand smoke causes heart disease and lung cancer.
- Separate "no smoking" sections DO NOT protect you from secondhand smoke. Neither does filtering rhe air or opening a window.
- Many states and communities have passed laws making workplaces, public places, restaurants, and bars smoke-free. But millions of children and adulrs still breathe secondhand smoke in their homes, cars, workplaces, and in public places.

No amount of secondhand smoke is safe.

When you are around a person who is smoking, you inhale the same dangerous chemicals as he or she does. Breathing secondhand smoke can make you sick. Some of the diseases that secondhand smoke causes can kill you.

Protect yourself: do not hreathe secondhand smoke. But completely avoiding secondhand smoke is very hard to do. Most of us breathe it whether we know it or not. You can breathe secondhand smoke in restaurants, around the doorways of buildings, and at work. When someone smokes inside a home, everyone inside breathes secondhand smoke. Some children even breathe smoke in day care.

There is no safe amount of secondhand smoke. Children, pregnant women, older people, and people with heart or breathing problems should be especially careful. Even being around secondhand smoke for a short time can hurt your health. Some effects are temporary. But others are permanent.

WHAT CAN YOU DO?

Make your environment smoke-free.

- *Make your home and car smoke-free.*
- *Visit smoke-free restaurants and public places.*
- *Ask people not to smoke around you and your children.*

Secondhand smoke contains poisons.

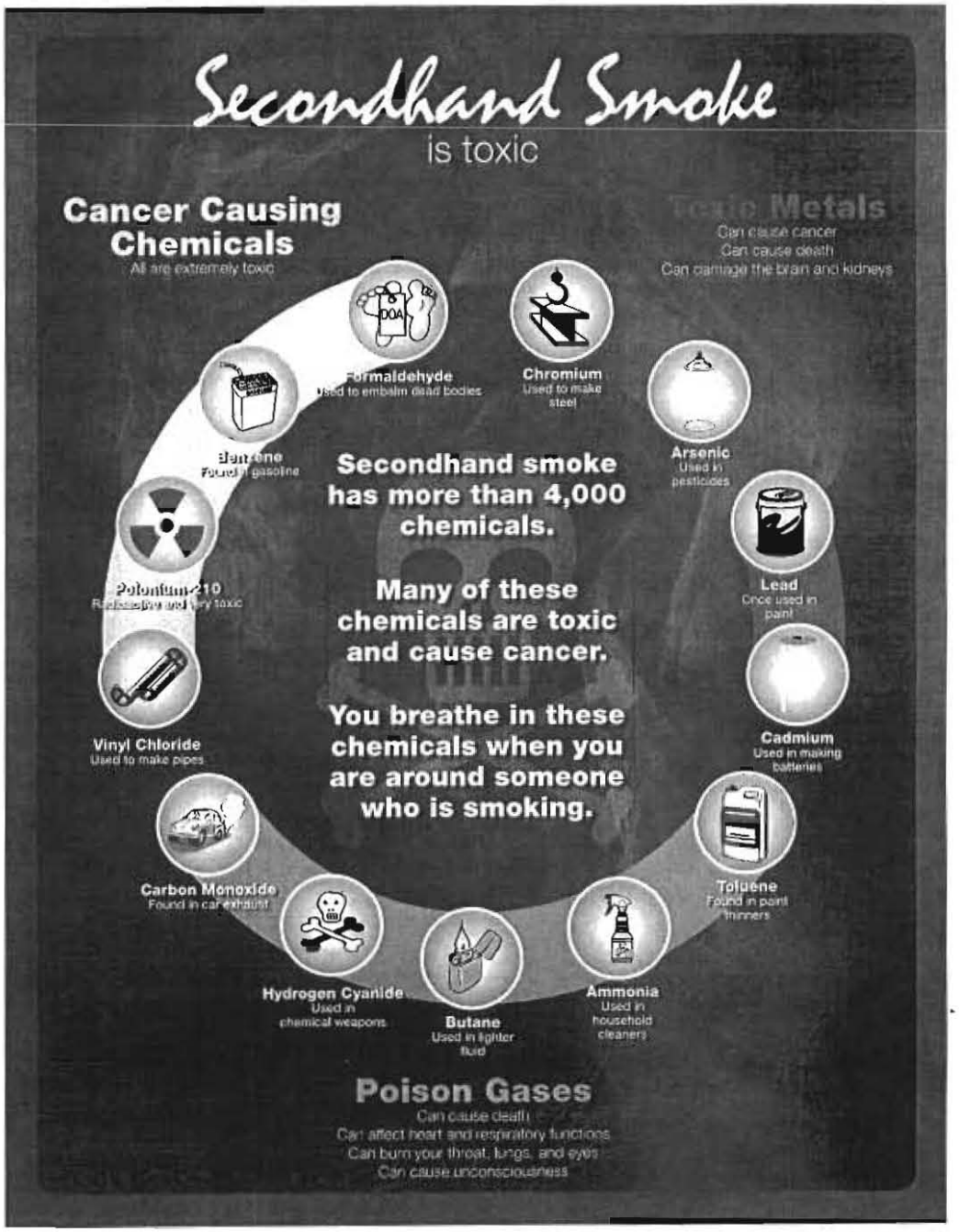
The chemicals found in secondhand smoke hurt your health and many are known to cause cancer. You breathe in thousands of chemicals when you are around someone who is smoking.

WHAT THE SCIENCE SAYS

How do scientists measure exposure to secondhand smoke?

Researchers measure

- how many people are smoking
- how many cigarettes they smoke
- time spent in the room
- levels of nicotine in the air, and
- levels of nicotine by-products in the body



Secondhand Smoke

causes death and sickness in children.

Breathing secondhand smoke is a known cause of sudden infant death syndrome (SIDS).

Children are also more likely to have lung problems, ear infections, and severe asthma.

Babies are hurt by secondhand smoke.

Tobacco smoke harms babies before and after they are born. Unborn babies are hurt when their mothers smoke or if others smoke arouud their mothers. Babies also may breathe secondhand smoke after they are born. Because their bodies are developing, poisous in smoke hurt babies even more than adults. Babies under a year old are in the most danger.

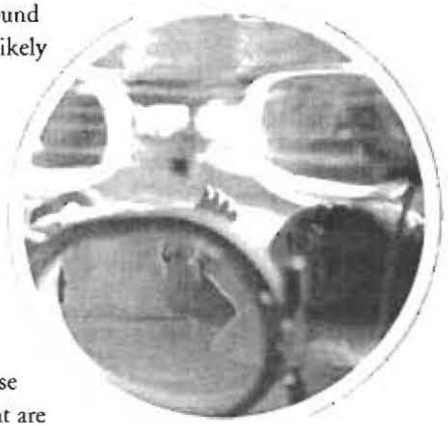
Secondhand smoke is a known cause of sudden infant death syndrome (SIDS).

The sudden, unexplained, unexpected death of an infant before age 1 year is knowu as SIDS. The exact way these deaths happen is still not known. We suspect it may be caused by changes in the brain or lungs that affect how a baby breathes. During pregnancy, many of the compounds in secondhand smoke change the way a baby's brain develops. Mothers who smoke while pregnant are more likely to have their babies die of SIDS.

Babies who are around secondhand smoke—from their mother, their father, or anyone else—afrer they are born, are also more likely to die of SIDS than ehildren who are not around secondhand smoke.

Secondhand smoke causes low birth weight and lung problems in infants.

Babies whose mothers are around secondhand smoke are more likely to have lower birth weights. These babies can have more health problems because they breathe smoke. For example, they are more likely to have infections than babies who are not around secondhand smoke.



Studies show that babies whose mothers smoke while pregnant are more likely to have lungs that do not develop in a normal way. Babies who breathe secondhand smoke after birth also have weaker lungs. These problems can continue as they grow older and even when they become adults.

WHAT THE SCIENCE SAYS

The main place young children breathe secondhand smoke is in their homes. Almost 3 million children in the United States under the age of 6 years old breathe secondhand smoke at home at least 4 days per week.

Older children are in danger, too.

Studies show that older children whose parents smoke get sick more often. Like babies, their lungs grow less than children who do not breathe secondhand smoke. They get more bronchitis and pneumonia. Wheezing and coughing are also more common in children who breathe secondhand smoke.

Secondhand smoke can trigger an asthma attack in a child. Children with asthma who are around secondhand smoke have worse asthma attacks and have attacks more often. More than 40 percent of children who go to the emergency room for asthma live with smokers. A severe asthma attack can put a child's life in danger.

Ear infections are painful. Children whose parents smoke around them get more ear infections. They also have fluid in their ears more often and have more operations to put in ear tubes for drainage.

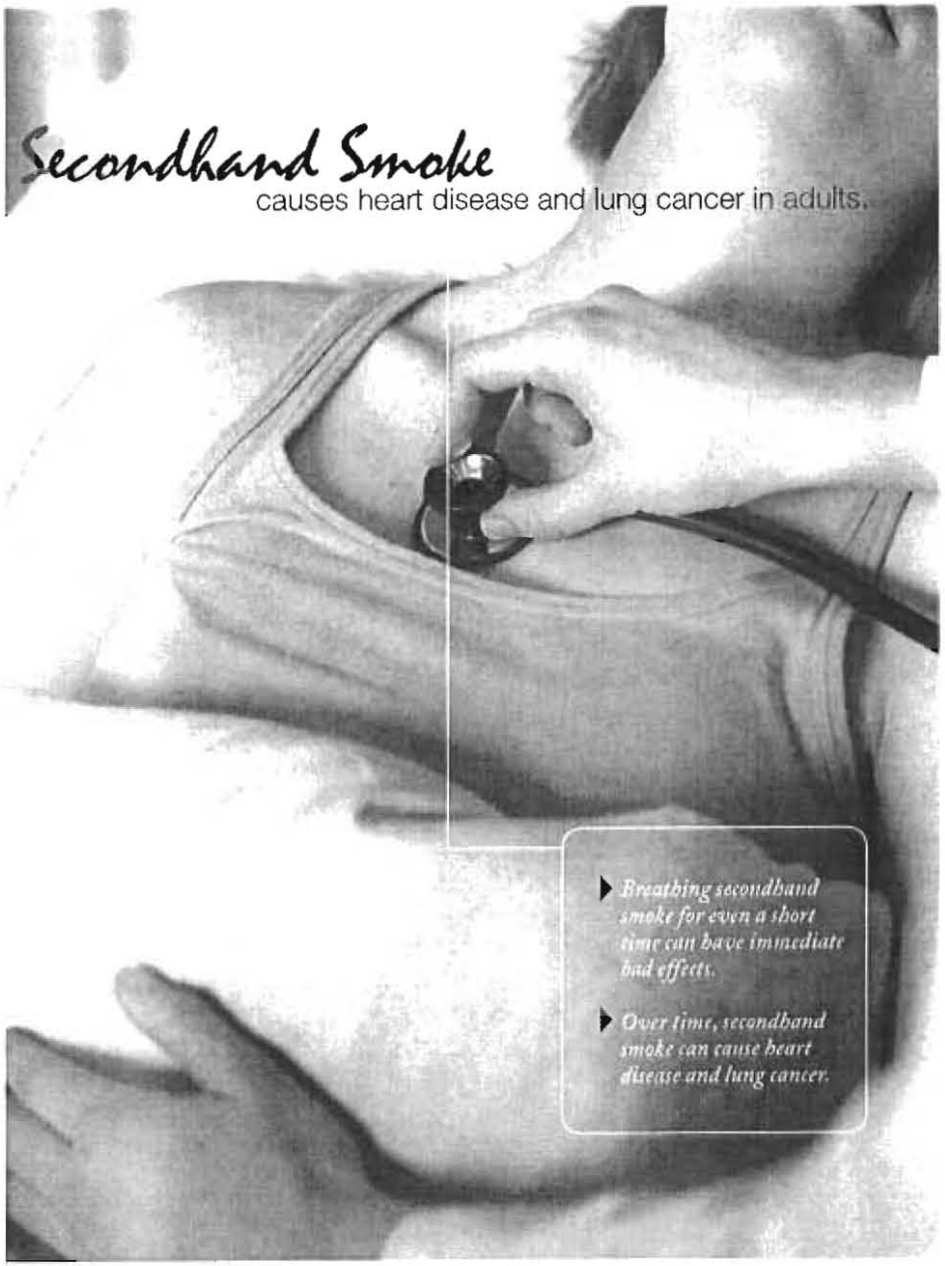
WHAT CAN PARENTS DO?

Protect your children's health.

- *Do not allow anyone to smoke near your child.*
- *Do not smoke or allow others to smoke in your home or car. Opening a window does not protect your children from smoke.*
- *Use a smoke-free day care center.*
- *Do not take your child to restaurants or other indoor public places that allow smoking.*
- *Teach older kids to stay away from secondhand smoke.*

Secondhand Smoke

causes heart disease and lung cancer in adults.



- *Breathing secondhand smoke for even a short time can have immediate bad effects.*
- *Over time, secondhand smoke can cause heart disease and lung cancer.*

Secondhand smoke hurts adults too.

The longer you are around secondhand smoke, the more likely it is to hurt you.

Nonsmokers who breathe smoke at home or at work are more likely to become sick and die from heart disease and lung cancer. Studies show that secondhand smoke may cause other serious diseases, too.

Secondhand smoke is bad for your heart.

Breathing secondhand smoke makes the platelets in your blood behave like those of a regular smoker. Even a short time in a smoky room causes your blood platelets to stick together. Secondhand smoke also damages the lining of your blood vessels. In your heart, these bad changes can cause a deadly heart attack.

Secondhand smoke changes how your heart, blood, and blood vessels work in many ways. Adults who breathe 5 hours of secondhand smoke daily have higher "bad" cholesterol that clogs arteries.

WHAT CAN YOU DO?

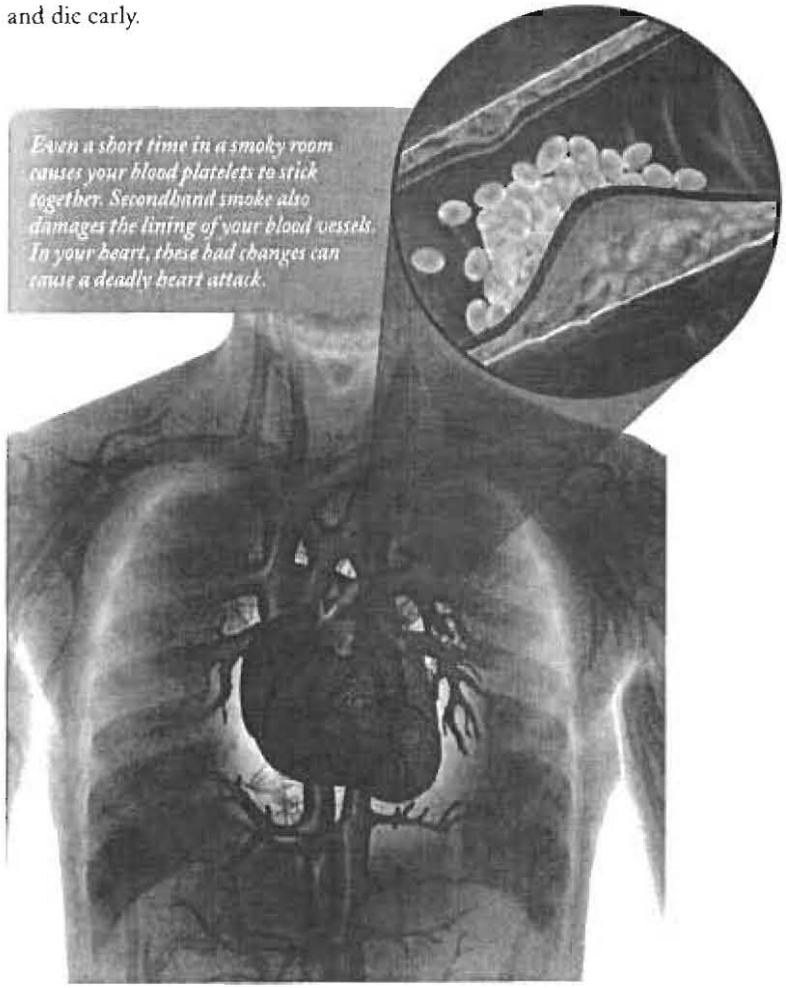
Protect your health.

More restaurants and bars are smoke-free than ever. New York City restaurants and bars increased business by 9 percent after becoming smoke-free.

- *Choose restaurants and bars that are smoke-free. Thank them for being smoke-free.*
- *Let owners of businesses that are not smoke-free know that smoke bothers you. Tell them a "no smoking" section is not good enough.*

People who have heart disease should be very careful not to go where they will be around secondhand smoke.

The bottom line is that breathing secondhand smoke makes it more likely that you will get heart disease, have a heart attack, and die early.



Even a short time in a smoky room causes your blood platelets to stick together. Secondhand smoke also damages the lining of your blood vessels. In your heart, these bad changes can cause a deadly heart attack.

Secondhand smoke hurts your lungs.

Secondhand smoke includes many chemicals that are dangerous for your lungs. Secondhand smoke is especially dangerous for young children and adults with heart and lung disease.

Secondhand smoke causes lung cancer.

Secondhand tobacco smoke contains the same cancer-causing chemicals that smokers inhale.

Secondhand smoke causes lung cancer in adults who don't smoke. Breathing in secondhand smoke at home or work increases your chances of getting lung cancer by 20 percent to 30 percent.

WHAT CAN HEALTH CARE EXPERTS DO?

- *Ask patients if they smoke and if they are around secondhand smoke.*
- *Advise patients who smoke to stop, and help them quit.*
- *Advise patients who smoke not to smoke around others.*
- *Advise nonsmokers to protect themselves by avoiding all secondhand smoke.*
- *Remind parents to protect their children from secondhand smoke.*
- *Discuss the added dangers of secondhand smoke for adults who have heart disease or asthma.*
- *Offer special warnings to parents when treating children with respiratory infections, asthma, or ear disease.*

WHAT CAN EMPLOYERS DO?

Protect your workers.

Secondhand smoke is harmful for all workers. Restaurant and bar workers breathe more secondhand smoke than other workers and have higher rates of lung cancer.

- *Make sure your employees do not breathe secondhand smoke at work.*
- *Make all indoor places smoke-free.*
- *Don't allow smoking near doorways and entrances.*
- *Offer programs to help employees quit smoking.*

Secondhand smoke causes other breathing problems.
Secondhand smoke affects how well your lungs work, especially if you already have asthma or other breathing problems. Being around smoke makes you more congested and cough more.

Secondhand smoke also irritates your skin, eyes, nose, and throat. If you have allergies or a history of breathing problems, secondhand smoke can make you even sicker.

You should especially speak to your doctor or healthcare provider about the dangers of secondhand smoke if:

- **You have breathing or heart problems**
- **You are pregnant**
- **You are concerned about your children's health**

Secondhand smoke may cause disease in other parts of your body.
We know that smoking causes many forms of cancer. Scientists believe even a little tobacco smoke is dangerous. Scientists also believe secondhand smoke may cause other diseases throughout your body. They are doing studies on possible links to stroke, breast cancer, nasal sinus cancer, and chronic lung problems in children and adults.

Secondhand smoke may cause disease in other parts of your body.

There's no such thing as a

NO SMOKING

section

No amount of secondhand smoke is safe.

Here are some unexpected ways you may breathe secondhand smoke every day:

- *Sitting in the "no smoking" section, even if it doesn't smell smoky*
- *Riding in a car while someone else is smoking, even if a window is open*
- *Being in a house where people are smoking, even if you're in a another room*
- *Working in any restaurant, warehouse, or building that allows smoking inside, even if there is a filter or ventilation system*

Acknowledgments

This public document was prepared by the U.S. Department of Health and Human Services under the direction of the Office of the Surgeon General to make information in *The Health Consequences of Involuntary Exposure to Tobacco Smoke. A Report of the Surgeon General* available to everyone.

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About
the Surgeon General's Report

The Surgeon General is the nation's highest-ranking health officer. The President appoints the Surgeon General to help promote and protect the health of all Americans.

The Surgeon General gives Americans the best scientific information available on how to improve their health and reduce their risk of illness and injury.

The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General was prepared by many of the country's leading scientists and public health experts. The full report is more than 600 pages long. It took more than 4 years to complete. It is written for a scientific audience. However, Surgeon General Richard H. Carmona believes the findings are very important to everyone.

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Secondhand Smoke

It hurts you.

It doesn't take much.

It doesn't take long.

To download copies of this booklet or the full Surgeon General's report, *The Health Consequences of Involuntary Exposure to Tobacco Smoke: A Report of the Surgeon General*, go to **www.cdc.gov/tobacco**. To order single copies of these documents, call toll free **1-800-CDC-INFO**.

For more information

For more information on secondhand smoke, talk to your doctor, nurse, pharmacist, or other healthcare professional.

More information about the Surgeon General's report is available on the Surgeon General's website at

www.surgeongeneral.gov

More facts and advice are available from
Centers for Disease Control and Prevention
www.cdc.gov/tobacco
Toll free: **1-800-CDC-INFO** (1-800-232-4636)
In English, en Español
24 hours/day, 7 days/week
Text telephone for hearing impaired: **1-888-232-6348**
Other helpful information is available at www.smokefree.gov.
To access a telephone quitline serving your area, call
1-800-QUIT-NOW (1-800-784-8669).

THE AMERICAN COUNCIL
ON SCIENCE AND HEALTH

SPECIAL RELEASE

A Critical Assessment of "Lies, Damned Lies, & 400,000 Smoking-Related Deaths"

by Robert Levy and Rosalind Marimont Published in *Regulation*,* Fall 1998

** by Alicia M. Lukachko, M.P.H.
and Elizabeth M. Whelan, Sc.D., M.P.H.

October, 1999

Executive Summary

In the Fall 1998 issue of *Regulation* ("The Cato Review of Business and Government"), the Cato Institute*** published an article by Robert Levy and Rosalind Marimont titled "Lies, Damned Lies, & 400,000 Smoking-Related Deaths." In their article, Levy and Marimont contend that the U.S. government's estimate of approximately 400,000 annual premature deaths due to cigarette smoking is scientifically unsound and substantially inflated. The authors assert that "the war on smoking . . . has grown into a monster of deceit and greed, eroding the credibility of government and subverting the rule of law."

In this report, scientists at the American Council on Science and Health refute Levy and Marimont's key arguments (presented below) as unscientific and inflammatory. ACSH's critique concludes that the estimate of 400,000 annual deaths due to cigarette smoking is indeed reliable and may even be an underestimate.

The authors make four primary assertions leading to their conclusion that the figure of 400,000 is inflated. Those four assertions and a summary of ACSH's critique of each of them follow.

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* Regulation is a publication of the Cato Institute

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*** The Cato Institute, whose headquarters are in Washington, DC, is a nonpartisan public policy research foundation advocating limited government and free markets

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1. *Levy and Marimont discount over one-third of the estimated 400,000 annual deaths caused by smoking with the erroneous claim that "small" increases in the risk of disease or death—marked by relative risks less than 2.0—are "statistically insignificant," and "insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease."*

Contrary to the authors' misstatement, relative risks less than 2.0, while small, can indeed be statistically significant and reflect a causal relationship. A relative risk is a measure of the strength of an association between exposure (e.g. smoking) and a disease. Given the pervasiveness of a risk factor, such as smoking, and the prevalence of some of the diseases it causes, small relative risks can, and do, represent serious threats to public health.

Levy and Marimont's assumptions regarding small relative risks violate basic principles of epidemiology. The authors confuse two distinct concepts, that of relative risk and that of statistical significance. The size of a relative risk, alone, *does not* signify its statistical significance. Rather, research findings must undergo statistical tests to assess their "significance." Small relative risks suggest a weak association (or risk factor), not necessarily an insignificant finding.

Moreover, relative risks of *any* value, when considered alone, are insufficient to conclude that an association is causal. Relative risks and several other factors (i.e., the consistency of the finding across studies, the biologic plausibility of the hypothesis, the presence of a dose-response relationship and the time sequence of the cause and effect) must be considered when judging causality.

2. *Levy and Marimont argue that the American Cancer Society's Cancer Prevention Survey (CPS)—a widely used data set for the calculation of public health statistics—is unrepresentative of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.*

It is true that the American Cancer Society's Cancer Prevention Survey includes a greater proportion of white, older, more educated, married and middle-class participants than does the general U.S. population. However, this characteristic alone does not undermine the findings derived from this data set. The CPS, with over one million participants, is the largest study collecting data on smoking and mortality over an extended period of time. It has a uniquely strong study design from which valid estimations have been drawn.

Moreover, the relative risks of dying from smoking-related diseases derived from the CPS are within the range of those from other studies. This consistency lends to the reliability of the CDC's estimate of smoking-related deaths that use relative risks drawn from the CPS.

3. *The authors state that the Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its calculation of smoking-related deaths. They argue that after accounting for other factors that may contribute to deaths among smokers, the CDC's estimate should be greatly reduced.*

According to Levy and Marimont, "if a smoker who is obese; has a family history of high cholesterol, diabetes, and heart problems; and never exercises, dies of a heart attack, the government attributes his death to smoking alone." What the authors are reasonably questioning here is the role of potential confounders—other factors that may explain some of the deaths attributed to smoking—on estimates of smoking-related deaths.

The CDC's estimate of annual smoking-related deaths does control for age—the confounding variable that has the greatest impact on the association of smoking with disease and death. Analyses that have controlled for multiple factors (such as exercise and alcohol intake) indicate that the impact of potential confounders on the CDC's age-adjusted risk of death due to smoking would be minimal. Some studies have even found that controlling for certain confounders results in an increase in smoking-attributable mortality. For example, one study (which controlled for risk factors including age, education, alcohol intake, diabetes, and hypertension) found smoking-related mortality estimates to be 2 percent higher than the CDC's age-adjusted estimates.

Furthermore, it is important to note that only a fraction of the deaths from smoking-related diseases are attributed to smoking.

4. *Finally, Levy and Marimont purport that the impact of smoking-related mortality is overstated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.*

In fact, it has been estimated that over one-half of all smoking-related deaths occur between ages 35 and 69, which translates into an average loss of roughly 23 years of life. Cigarette smoking also accounts for approximately 30 percent of all deaths among this age group. That the majority of deaths caused by smoking occurs among adults does not mitigate the real risks that cigarettes pose to children.

Levy and Marimont insinuate that the deaths of older adults should not be considered premature or preventable. But many adults remain healthy into their eighties and nineties. It is inappropriate to set an arbitrary age limit on premature death.

In conclusion, Levy and Marimont's arguments do not present a scientifically sound and convincing case that the estimate of 400,000 annual smoking-related deaths is a specious, statistical gimmick. Their essay, however, does illustrate the importance of educating the public about basic epidemiological and biostatistical concepts, including the methods used to determine smoking-related deaths.

Introduction

In the Fall 1998 issue of *Regulation* ("The Cato Review of Business and Government"), the Cato Institute published an article by Robert Levy and Rosalind Marimont entitled "Lies, Damned Lies, & 400,000 Smoking-Related Deaths." In their article, Levy and Marimont contend that the government's estimate of 400,000 annual premature deaths due to cigarette smoking is scientifically unsound and substantially inflated. The authors assert: "The war on smoking . . . has grown into a monster of deceit and greed, eroding the credibility of government and subverting the rule of law."

In May 1999 Levy and Marimont's arguments resurfaced through an article written by *Boston Globe* columnist Jeff Jacoby. Mr. Jacoby's column has been widely circulated and cited in op-ed pages nationwide.

The Levy and Marimont article also served in the defense of American tobacco companies in the recent Florida "Engle case," the largest class action lawsuit filed, and the first won, against the tobacco industry.

For over 20 years, the American Council on Science and Health (ACSH), has relied on sound science to educate the public about real versus hypothetical risks to health. ACSH has paid particular attention to well-established and preventable causes of disease and death, especially cigarette smoking. For these reasons, ACSH is in a unique position to examine the veracity of claims made by Levy and Marimont.

In the following report, ACSH evaluates the plausibility of the estimate that 400,000 premature deaths are attributable to smoking. ACSH reviews the confirmed health problems caused by smoking and explains the scientific methods used to establish these risks. Lastly, ACSH evaluates the key arguments employed by Levy and Marimont to discount the fatalities caused by cigarette smoking each year.

ACSH considers this report a work in progress. As more information becomes available, the report will be updated.

About the Authors

Robert Levy, the lead author of "Lies, Damned Lies, & 400,000 Smoking-Related Deaths," is a senior fellow at the Cato Institute specializing in constitutional studies. With a J.D. degree, and a Ph.D. degree in business, he is also an adjunct professor at the Georgetown University Law Center. Although Levy does not have a background in science, he has written extensively about tobacco from a business perspective.

Rosalind B. Marimont, the article's co-author, is described as a mathematician and scientist. Before her retirement in 1979, Marimont worked for the Bureau of Standards (now the National Institute of Standards and Technology). A prominent contributor to the pro-tobacco group FORCES (Fight Ordinances and Restrictions to Control and Eliminate Smoking), Marimont has written several essays criticizing the focus of public health groups on tobacco. Marimont has also been an active member of the National Smokers' Alliance and has testified against local legislation in Maryland to restrict smoking in public places.

The Health Hazards of Smoking

Cigarette smoking has been recognized as a leading cause of disease and death for at least 40 years. Few subjects have received such thorough and extensive scientific scrutiny by both governmental and independent bodies. Thousands of scientific studies have confirmed that smoking is a major health hazard.[1] Besides the relationship between smoking and disease, many studies have found that the overall death rate among smokers is 2–3 times greater than that of nonsmokers.[2] Cigarettes also contain nicotine, a chemical proven to be highly addictive (which has been acknowledged in internal tobacco-industry documents).

Despite the overwhelming evidence to the contrary, Levy and Marimont state that the hazards of smoking remain largely speculative. They allege that the "war on smoking started with a kernel of truth—that cigarettes are a high risk factor for lung cancer." Ironically, it is Levy and Marimont's article that contains only a kernel of truth about the risks of smoking. In fact, while active cigarette smoking has been causally linked to lung cancer, it is also associated with an array of other diseases, listed below.[3]

- Cigarette smoking is a principal cause of cancer of the esophagus, larynx, lip, mouth, pharynx, tongue, kidney, pancreas, urinary bladder, and uterine cervix.

- Cigarette smoking has also been identified as a major cause of cardiovascular disease, including atherosclerosis, coronary heart disease (angina and heart attack), stroke, sudden death, and aortic aneurysm.

- Cigarette smoking causes chronic obstructive lung disease (emphysema, chronic bronchitis, and related conditions). Smokers have been found to suffer more respiratory problems (such as colds, pneumonia, influenza, and bronchitis) and their recovery from these illnesses is slower.

- For men under age 65, smoking has been shown to be an independent risk factor for impotence, including erectile dysfunction. For women, smoking can impair fertility, induce premature menopause and spontaneous abortion, and lead to a host of complications of pregnancy and childbirth.

- Cigarette smoking increases the risk for osteoporosis (a reduction in bone mass) and periodontal (gum) disease.

- Smoking precipitates premature hearing loss, and vision problems, including blindness secondary to cataracts and macular degeneration, and premature hearing loss.

- Smokers face a significantly greater chance than nonsmokers of suffering complications during and after surgery.

Evidence suggests that smoking also increases the risk for other diseases, such as rheumatoid arthritis, and cancers of the prostate and stomach. These relationships, however, have not yet been scientifically established.

Preliminary research also indicates that cigarette smoking may be associated with reduced risk for endometrial cancer and Parkinson's disease. Yet the harmful effects of cigarette smoking dramatically outweigh any of the potential benefits. By identifying and isolating the components of smoking that may have positive effects, potential benefits may be achieved while avoiding exposure to the many toxins contained in cigarette smoke.

Environmental Tobacco Smoke

A mounting body of scientific research reveals that exposure to environmental tobacco smoke (ETS) also poses health risks. The most common and firmly established adverse health effects associated with exposure to ETS are irritation of the eyes, nose, and respiratory tract; exacerbation of asthma and emphysema; and increased susceptibility to respiratory infections.[4] Furthermore, studies have consistently shown that ETS contributes to lung cancer and heart disease.[4]

As the Levy and Marimont article itself illustrates, the concerns over secondhand smoke extend far beyond public health. The political implications of finding a causal association between ETS and disease have fueled long and bitter struggles between interested parties (pro- and anti-tobacco organizations and individuals). To counter the growing trend toward indoor-smoking restrictions and to allay public fears, some parties, including the tobacco industry, have argued that ETS does not pose a "meaningful" lung cancer risk—and therefore does not present a threat to public health.

Similarly, authors Levy and Marimont focus their arguments about secondhand smoke exclusively on lung cancer in an attempt to dismiss all of the health effects associated with ETS. Their argument is simplistic, as it ignores ETS-related health risks other than lung cancer that should also be considered when

developing public health policy—heart disease and respiratory illnesses, for example.

Establishing Cause and Effect

Scientists rely on epidemiology ("the study of the distribution and determinants of disease frequency"[5]) to determine whether a factor, such as cigarette smoking, causes a particular health outcome (e.g., disease or death). They begin by suggesting and then establishing an association. In the case of smoking, the optimal way to evaluate the effect of smoking on health is to compare groups of smokers with groups of nonsmokers to assess the differences (if any) in health outcomes between them. Researchers try to assure that the groups being compared (i.e., smokers and nonsmokers) have similar characteristics in addition to their smoking behavior. By doing so, differences in health outcomes found between these groups are more likely attributable to smoking than to other factors. Dissimilarities between groups can also be accounted for in the analysis of the research data.

Once an association is found between an exposure (e.g., smoking) and a health outcome (e.g., disease), researchers must determine whether the apparent association is *valid*. A valid association is one that is unlikely to be due to chance, bias (on the part of researchers or study participants), or confounders—other factors that caused the disease and are independently associated with smoking.[5]

Statistical tests are routinely applied to research findings to assess the probability that the results are not merely coincidental. A "statistically significant" finding means that the result is unlikely to be due to chance. Factors such as the number of people being examined (sample size) and the strength of the association between the exposure and health outcome influence whether or not a finding will achieve "statistical significance." Generally, the larger the sample size and the stronger the effect of the risk factor, the more likely it is that the results will be found to be significant.

While finding a statistically significant result denotes that *chance* is unlikely to account for the results, *bias* and potential *confounders* must still be addressed to demonstrate a valid association. Furthermore, a statistically significant finding does not alone confirm a causal relationship.

To conclude that a particular disease is *caused* by smoking, five basic criteria must be considered.[5] Researchers must assess:

1. *the strength of the association found between smoking and disease;*

A *relative risk* is one measure of the strength of the association between a risk factor and disease (e.g., between smoking and disease) and is the ratio of disease among smokers to disease among nonsmokers. A relative risk equal to 1 indicates that there is no association between the exposure and the outcome. The closer the relative risk is to 1, the *smaller* or *weaker* the association.

A relative risk of 2.0 for example, indicates that those exposed to a particular agent (e.g., smokers) are twice as likely than those unexposed (e.g., nonsmokers) to develop the health outcome under study (e.g., death from heart disease). The larger the relative risk, the less likely the effect is due solely to bias or confounders. Still, small relative risks do not exclude the possibility that a causal relationship exists, nor do they preclude the possibility that a finding is statistically significant.

2. *the consistency of the finding across studies;*

If several well-designed studies replicate a finding, it is more likely that the relationship being studied is real. As stated previously, the enormous body of research examining the health effects of smoking corroborate the relationship between smoking and disease.[3]

3. *the biologic plausibility of the hypothesis;*

The relationship between exposure and disease must be consistent with what is currently known about biology and the disease process. Much is understood about the biological mechanisms by which smoking causes disease, though more remains to be learned. It is known that cigarette smoke contains approximately 4,000 chemical components, many of which are toxins and some of which are human carcinogens.

4. *the presence of a dose-response relationship; and*

In a dose-response relationship, increases in the degree of exposure are associated with increases in risk. Numerous studies have demonstrated that increases in cigarette use (duration and number of cigarettes smoked) increase the risk for smoking-related disease and death.[3]

5. *the time sequence of the cause and effect.*

The exposure or hypothesized cause must logically precede the effect. Research from multiple study designs affirm that cigarette use among study participants does in fact occur before the health outcomes that are measured.

The basic principles of epidemiology presented above are critical to evaluating the claims made by Levy and Marimont.

Calculating Premature Deaths Due to Cigarette Smoking

The number of deaths that are attributable to cigarette smoking may be thought of as the reduction in deaths that would be achieved if the entire population had never smoked. The basic calculation involves multiplying the death rates for smoking-related diseases among representative nonsmokers by the number of people in the entire population and then subtracting the resulting expected number of deaths from the actual number of these deaths.

Since a significant number of persons who have smoked in the past have quit, and thus have a greater risk of smoking-related disease than those who have never smoked, some formulas, such as that used by the CDC, distinguish between current smokers, former smokers and "never-smokers." The CDC estimates the proportion of deaths from a particular disease (e.g., lung cancer) by multiplying the difference in death rates between smokers and nonsmokers by the proportions of the population that smoke. The formula uses differences in death rates of current smokers compared to never-smokers, and of former smokers compared to never-smokers. These differences are then multiplied by the proportions of the population who currently smoke and used to smoke.

Estimations of this death toll can vary widely depending on the diseases considered to be smoking-related, the data sources used, the control for confounding variables (e.g. age) and variations in formulas.

For over two decades, the U.S. government has been estimating the number of Americans who die prematurely from smoking. According to current government calculations, approximately 430,000 deaths occur each year in the United States as a result of cigarette smoking.[6] Some larger estimates fall in the range of 600,000 to 700,000 annual deaths due to smoking.[7]

In "Lies, Damned Lies, & 400,000 Smoking-Related Deaths," Levy and Marimont challenge the veracity of the associations found between smoking and disease, and ultimately the estimate that 400,000 premature deaths are caused by smoking each year. By employing largely haphazard and unscientific methods, the authors try to minimize smoking's death toll.

The section below assesses the following key arguments maintained by Levy and Marimont, namely that:

- Relative risks less than two are "statistically insignificant" and "insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease."
- The Cancer Prevention Survey (CPS) is unrepresentative of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.
- The Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its calculation of smoking-related deaths.
- The impact of smoking-related mortality is overstated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.

Argument 1: **Relative risks less than 2.0 are "statistically insignificant" and "insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease." Based on this claim, Levy and Marimont subtract over 150,000 of the 400,000 annual deaths caused by smoking.**

Relative risks less than 2.0, while small, can indeed be statistically significant and reflect a causal relationship. Given the pervasiveness of a risk factor, such as smoking, and the prevalence of some of the diseases it causes, small relative risks can, and do, represent serious threats to public health. For example, cigarette smoking is a much greater risk factor for mortality from lung cancer than from heart disease.[5] But, because heart disease affects many more people than lung cancer, the number of smoking-related deaths from heart disease rivals those from lung cancer.[6]

Levy and Marimont's assumptions regarding small relative risks violate basic principles of epidemiology. The authors confuse two distinct concepts, that of relative risk and that of statistical significance.

The size of a relative risk, alone, does not signify its statistical significance. Rather, as explained in the previ-

ous section, research findings must undergo statistical tests to assess their "significance." Small relative risks suggest a weak association (or risk factor), not necessarily an insignificant finding. Again, small relative risks may have a substantial public health impact if the exposure and the health outcome affect a large proportion of the population.

Moreover, relative risks of *any* value, when considered alone, are insufficient to conclude that an association is causal. As discussed above, relative risks are one of many factors that must be considered when judging causality. An association of small magnitude can be judged as cause and effect after considering the totality of the evidence.

A good illustration of this point is offered by the authors themselves. In their derision of the risks associated with ETS, Levy and Marimont claim that "the relative risk of lung cancer for persons who drink whole milk is 2.4." Even if we accept this highly dubious association, the other criteria necessary to judge causality (i.e., biologic plausibility, consistency of findings, etc.) are not fulfilled. Thus, whole milk cannot be legitimately judged as a cause of lung cancer given the relative risk alone.

The authors mislead readers by misrepresenting a quotation from the National Cancer Institute, which qualifies relative risks, as the agency's "own guideline." In fact, the NCI has no such guideline about relative risks, and the quotation cited is taken from a 1994 NCI press release on abortion and the risk of breast cancer. Given its proper context, this so-called guideline makes a much different point from what the authors suggest.

The authors use the arbitrary and unscientific ceiling of relative risks less than 2.0 as a means of reducing the CDC's estimate of smoking by 163,071 deaths. But, based on the arguments presented above, their logic is fundamentally flawed.

Argument 2: The American Cancer Society's Cancer Prevention Survey (CPS)—a widely used data set for the calculation of public health statistics—is unrepresentative of the general population and is therefore "the wrong sample [to use] as a standard of comparison" when estimating smoking-related deaths in the US.

It is true that the American Cancer Society's CPS includes a greater proportion of white, older, more educated, married, and middle-class participants than the general U.S. population.[2] However, this characteristic alone does not undermine the findings derived from this data set. The CPS has a uniquely strong study design from which valid estimations have been drawn.

Moreover, and perhaps more important, the relative risks of dying from smoking-related diseases derived from the CPS are within the range of those from other studies. This consistency lends to the reliability of the CDC's estimate of smoking-related deaths that use relative risks drawn from the CPS.

The important issue of generalizability (whether the results are applicable to other populations) should be considered only after a study has been determined to be valid. Levy and Marimont overlook the overriding strengths of the CPS: its excellent study design and valid findings. With over one million study participants, the CPS is the largest U.S. study that collects data over an extensive period of time on the relationship between smoking and mortality.

After accepting that the results of the CPS reflect valid cause and effect relationships, the next important question is: How would the results from a mostly white and middle-class population differ, if at all, from those among the general U.S. population? The answer depends on how the data are used. The absolute mortality rates are lower in the CPS than in the general population. But, the CDC's estimation of smoking-related deaths relies on ratios—relative risks comparing smokers with nonsmokers and former smokers within the CPS. These relative risks of smoking-related disease have been found to be within a reasonable range of those from other studies, and therefore, enhance the reliability of the CDC's estimate.[8]

Levy and Marimont advocate substituting data from the National Center for Health Statistics (specifically the National Mortality Followback Survey and the National Health Interview Survey) for data from the CPS—an approach proposed by long-time tobacco industry consultant T.D. Sterling.[9] However, Sterling's approach has been justly criticized for its implausible findings (e.g., previous smoking was found to be protective against coronary heart disease and cerebrovascular disease among males over age 65), and for combining data from two surveys with largely dissimilar, and thus incompatible, study designs.[10,11]

On the other hand, the CPS uses the appropriate study design (a large prospective cohort*) to provide valid relative risk estimates used in the calculation of the number of premature deaths due to smoking.

* A prospective cohort study is one that follows a group of exposed (e.g. smokers) and unexposed (e.g. nonsmokers) subjects over an appropriate time period to observe the health outcome(s) under study.

Argument 3: **The Centers for Disease Control and Prevention (CDC) fails "to control for obvious confounding variables" in its estimation of smoking-related deaths. Levy and Marimont argue that after accounting for other factors that may contribute to deaths among smokers, the CDC's estimate should be greatly reduced.**

The CDC's estimate of annual smoking-related deaths does control for age, the confounding variable that has the greatest impact on the association of smoking with disease and death. Analyses that have controlled for multiple factors (such as exercise and alcohol intake) indicate that the impact of potential confounders on the age-adjusted risk of disease/death due to smoking is minimal.[8,12]

According to Levy and Marimont, "if a smoker who is obese; has a family history of high cholesterol, diabetes, and heart problems; and never exercises dies of a heart attack, the government attributes his death to smoking alone." What the authors are reasonably questioning here is the role of potential confounders—other factors that may explain some of the deaths attributed to smoking—on estimates of smoking-related deaths. For some diseases, such as lung cancer, which is almost wholly attributable to smoking (approximately 87 percent of lung cancers are caused by smoking), the influence of confounders is trivial. But, for diseases that have multiple notable risk factors (such as cardiovascular disease), the effect of confounders may indeed be significant.

As Levy and Marimont point out, failing to account for confounders can result in inaccurate estimates of smoking-related deaths. Yet, the authors incorrectly assume that by controlling for potential confounding factors, the CDC's age-adjusted calculation would be substantially diminished. In fact, controlling for some confounders has been shown to result in increases in attributable risk that would suggest that the CDC's estimate may be conservative.[8]

Moreover, only a fraction of the deaths from smoking-related disease are attributed to smoking.

When assessing the impact of controlling for confounding variables on the CDC's estimate, it is important to consider the results of studies that have examined the effects of confounders on smoking risk. The Nurses' Health Study is a well-designed prospective cohort study with 12 years of follow-up on registered nurses in the U.S. Results of this study reveal that after controlling for potential confounders (including hypertension, diabetes, high serum cholesterol, weight, parental history of heart attack before age 60, past use of oral contraceptives, postmenopausal estrogen use, and age at starting smoking), the multivariate relative risk of 1.87 for *total mortality* comparing current smokers with "never-smokers" was basically the same as the age-adjusted estimate of 1.86.[12] This study also showed a slight strengthening of the association between current smoking and mortality due to cardiovascular disease after adjusting for alcohol and exercise.[12]

Another analysis of the CPS data (used by the CDC)—which controlled for risk factors including age, education, alcohol intake, diabetes and hypertension—found smoking-related mortality estimates for the combined disease categories of lung cancer, ischemic heart disease, bronchitis/emphysema, chronic airway obstruction, and cerebrovascular disease to be 2 percent higher than the CDC's age-adjusted estimates.[8]

Contrary to Levy and Marimont's claim, the available data (presented above) strongly suggest that further adjustment for potential confounders (other than age) would have little impact on the CDC's estimate of roughly 400,000 smoking-related deaths.

Argument 4: **The impact of smoking-related mortality is overstated, particularly with respect to children, given that the majority of smoking-related deaths occur late in life.**

In fact, it has been estimated that over one half of all smoking-related deaths occur between ages 35 and 69, which translates into an average loss of roughly 23 years of life. Cigarette smoking also accounts for approximately 30 percent of all deaths among this age group.[13] That the majority of deaths due to smoking occur among adults does not mitigate the real risks that cigarettes pose to children.

Levy and Marimont aver that smoking "kill[s] people at an average age of roughly seventy-two—far closer to ninety-nine than to childhood or even young adulthood." This unreferenced assertion is inconsistent with studies suggesting that the average age of death among smokers is well below age 72.[13,14]

It is important to consider that what the authors are reporting is an average age of death. Cigarette smoking kills people at ages much younger than 72, as well as at ages older than 72. Long-term follow-up studies of smokers versus nonsmokers have found that smokers are three times more likely to die between the ages of 45 and 64, and two times more likely to die between the

ages of 65 and 84, than are nonsmokers.[2] Thirty-three percent of nonsmokers live to age 85, while only 12 percent of smokers live that long.[2]

Levy and Marimont insinuate that the deaths of older adults should not be considered premature or preventable. But many adults remain healthy into their eighties and nineties. It is inappropriate to set an arbitrary age limit on premature death. A premature, preventable death is a premature, preventable death at any age. The authors' underlying assumption is that deaths among the "old" are less consequential than deaths among the young. This "modest proposal" controverts the fundamental, humanitarian principle of medicine and public health: All human lives are valuable.

Levy and Marimont present smoking-related deaths in terms of years of potential life lost (YPLL) in an attempt to minimize its impact. The authors, however, rely on an outdated approach to calculating YPLL by considering only those years under age 65. YPLL is more accurately calculated from life expectancy, which extends well beyond age 65.

After inappropriately comparing smoking-attributed mortality with immediate deaths from motor vehicle accidents, suicide, and homicide, the authors state that "measured by YPLL, tobacco was . . . not 'the number one killer in America' as alarmists have exclaimed." Some causes of premature, preventable deaths do occur at a much younger age than those due to cigarette smoking. Still, considering the vast number of deaths caused by cigarette use, smoking remains the leading cause of preventable death.

It is important to note that YPLL is just one of many measures representing the public health impact of a risk factor. Aside from the mortality due to smoking, the authors fail to take into account smoking-related morbidity and the poor quality of life that often accompanies the chronic illnesses caused by cigarette smoking.

The authors assert that the concern about youth cigarette smoking is unfounded because the majority of cigarette-related deaths occur later in life. They suggest that alcohol and drug abuse are more legitimate threats to youth. However, the dangers from alcohol and drug abuse do not preclude those from cigarette smoking.

Cigarettes and cigarette smoke contain nicotine, a powerfully addictive drug. People who begin smoking as children are more likely to become lifetime smokers, and, therefore, to die from smoking-caused disease.[12] Smoking at a young age (or any age) causes irreversible genetic and cellular damage that may take years to surface as disease.[15,16] Furthermore, studies have found that cigarette smoking is associated with, and tends to precede, alcohol and illicit drug use—the very behaviors Levy and Marimont deem most threatening to children.[17,18]

Levy and Marimont's arguments obscure the real risks associated with cigarette smoking—effects that may not be immediately observed, but are harmful nonetheless.

Conclusion

Levy and Marimont fail to present a scientifically sound and convincing argument that the estimate of 400,000 annual smoking-related deaths is a specious, statistical gimmick. Their assumptions about the effect of potential confounders and their dismissal of relative risks less than 2.0 as a means to minimize smoking's death toll are unsupported. Moreover, their criticisms of the CPS data and their disregard for the long-term impact of cigarette smoking are misguided. Our assessment concludes that the estimate that 400,000 people die from cigarette smoking each year is indeed reliable and may even be an underestimate.

"Lies, Damned Lies, & 400,000 Smoking-Related Deaths" does, however, bring to light some reasonable questions that the public may share about the methods used to determine smoking-related deaths. The article clearly illustrates the importance of educating nonscientists about basic epidemiological and biostatistical concepts.

In their conclusion the authors make further misleading and unscientific claims, stating, for example, that "the actual damage from smoking is neither known nor knowable with precision." As stated previously, smoking and tobacco use is the single most-studied health risk factor in the history of human health research. In fact, the first report of diminished life span among smokers appeared in 1938. The pathological effects of chronic tobacco use in individuals are well documented. Using rigorous study designs and analytical methods, scientists have established with a high degree of certainty the causal role of tobacco in disease and death.

Levy and Marimont suggest that the "correctly calculated number of smoking-related deaths" nears 100,000 per year. Even if one were to accept this gross miscalculation, is not the premature, debilitating, often painful death of "only" 100,000 Americans (of any age) worthy of being addressed as a significant public health problem?

The authors might well heed their own advice when they criticize federal officials for "tainting science to

advance predetermined ends." By straying from the most basic epidemiological principles in their arguments, and by touting opinions that masquerade as facts, the authors have themselves strayed far from science.

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DEPARTMENT OF HEALTH AND HUMAN SERVICES

Isiah Leggett
County Executive

Uma S. Ahluwalia
Director

MEMORANDUM

June 10, 2011

TO: George Leventhal, Chair
Health and Human Services Committee
Montgomery County Council

FROM: Uma S. Ahluwalia, Director

SUBJECT: Proposed Board of Health Regulation Prohibiting Smoking in Certain Common Areas of Multi-Family Residential Dwellings and Playgrounds

The Health and Human Services Committee is scheduled to hold a worksession to discuss this proposed regulation on June 23, 2011. On behalf of the Department of Health and Human Services (DHHS), I would like to highlight a concern that we discussed with you earlier regarding the enforcement mechanism outlined in Section (d)(3) of the regulation.

The regulation provides that a citation may be issued if an employee witnesses the violation or if an employee receives complaints from at least two individuals who have personal knowledge of the smoking violation. As a practical matter, the activities to be regulated take place in facilities that DHHS staff currently neither visit nor inspect. Therefore, we anticipate that our staff will be issuing citations primarily on a complaint-driven basis.

In order to minimize the administrative burden on our staff, we recommend that the regulation be amended to require that any complaint be provided in the form of an affidavit which would include details regarding the identity of the alleged violator, as well as the time and place of banned activity. Upon receipt of such information, DHHS staff could then issue a warning or citation using Certified Mail.

This approach will minimize the demand on our licensure and regulatory staff. As you are aware, our staff has been tasked with additional statutory responsibilities in the last four years without any corresponding increase in staff resources. Our concern is that the cumulative impact of these new responsibilities will affect our ability to meet all of our statutorily-required obligations.

We support the underlying goal of the regulation, but urge you to consider the impact on DHHS staff as you establish the enforcement mechanism. Both Dr. Tillman and I will be available at the worksession to respond to any questions you or other Councilmembers may have.

USA:tjk

c: Councilmember Nancy Navarro
Councilmember Craig Rice
Dr. Ulder J. Tillman, Health Officer
Amanda Mihill, Council Staff

Testimony for Public Forum on Secondhand Smoke
May 5, 2011

My name is Gail Becker and I live at The Promenade apartments, 5225 Pooks Hill Rd in Bethesda, MD. The Board of Health's proposed regulation prohibiting smoking in certain areas of multi-family residential dwellings and certain playground areas should be EXPANDED to include all apartments, condos, and co-ops, both inside the apartments, balconies, all inside and outside common areas, including playgrounds, parking lots, swimming pools, and other common use facilities.

There are 1800 residents at The Promenade. Most of the residents are non-smokers, but many of the non-smokers where I live are currently exposed both day and night to secondhand smoke in our apartments. The tobacco smoke comes into our apartments through the windows, vents, doors, and walls. The non-smoking residents include people of all ages, from newborn babies to elderly adults, people with asthma, bronchitis, severe allergies, heart disease, and cancer.

Now that most workers in Montgomery County are covered by smoke-free policies at work, the home has become the predominant location for exposure of children and adults to secondhand smoke. Exposure of nonsmokers to secondhand smoke cannot be controlled by air cleaners or mechanical air exchange. The Surgeon General's 2010 report confirmed that "There is no risk-free level of exposure to tobacco smoke." The report says that inhaling even the smallest amount of tobacco smoke causes immediate damage to one's organs and poses risk of serious illness or death and can also damage your DNA, which can lead to cancer.

No one should be forced to breathe their neighbors' secondhand smoke at home. It is not possible to contain the smoke. The health hazards of secondhand smoke are well-documented in the Surgeon General's reports. The New York Times reported yesterday that Americans are suffering from asthma in record numbers. According to the Centers for Disease Control and Prevention, nearly 1 in 10 children and 1 in 12 American adults of all ages now have asthma.

Maybe you are able to avoid secondhand smoke coming into your home, but you were elected to act on behalf of the public interest. You are urged to stop the crime of death, disease, and sickness being caused by secondhand smoke exposure for the nonsmoking majority living in apartments, condos, and co-ops. No one wants to breathe secondhand smoke and you are empowered to expand our protection and demonstrate leadership, instead of taking baby steps.

Janet Buyer's Testimony at Public Hearing by the Health and Human Services Committee of the Montgomery County Council on May 5, 2011 regarding Resolution to Adopt Board of Health Regulation prohibiting smoking in certain common areas of multiple-family residential dwellings and certain playground areas

Good evening. My name is Janet Buyer and I live at 15109 Sunflower Ct., Rockville. I am testifying in favor of this legislation in memory of my mother Marjory Buyer who passed away 3 years ago today from emphysema stemming from many years of smoking. She began smoking as a teenager, just as many kids do today, because it not only was accepted but was considered cool. The difference today, though, is that tobacco's harmful effects and its highly addictive nature are more widely known. Anything the County can do to lessen tobacco's grip on our citizens is not only a benefit but an obligation to protect the health of all residents, smokers and nonsmokers, in the county. The societal cost associated with the effects of smoking must be staggering and I fear that the number of deaths associated with tobacco use is underreported. My mother's death certificate says it was unknown if her death was associated with tobacco use simply because the nurse who filled out the certificate didn't check her medical record to see she had lung disease associated with smoking and had been on home oxygen for a year before her death. When I question my mom's doctor about this error, he told me he signed the certificate without checking the veracity of the information on it. I wonder how frequently this simple error is made and how it affects our national statistics on deaths attributed to tobacco.

Limiting areas where people can smoke is one step the county can take that provides two benefits. The first is limiting the number of children who will witness the habit and this will hopefully help prevent them from becoming smokers themselves. In addition, this regulation will help prevent nonsmokers from being exposed to the harmful effects of secondhand smoke. Second hand smoke is a CLASS A carcinogen and the US Surgeon General states that there are no acceptable levels of second hand smoke. Because secondhand smoke comes through windows, doorways, hallways, vents, and walls, I ask that you include in this legislation a ban on smoking inside all units of multi-unit dwellings and all common areas,including balconies, parking lots, and adjacent sidewalks.

EVIDENCE IN **OPPOSITION** TO A MONTGOMERY COUNTY COUNCIL INITIATIVE **TO EXPAND THE SMOKING BAN**

Testimony by Michael Copperman
May 5, 2011
2225 Washington Avenue, 102
Silver Spring, MD 20910

United States: Epidemiologist Questions Science Of ETS Studies

Source : Washington Post

Source Date : January 30, 2007

Country: United States

Author : Gio Batta Gori

Abstract :
In an op-ed in the Washington Post, Gio Batta Gori, an epidemiologist and fellow of the Health Policy Center in Bethesda, Maryland, wrote that although numerous recent studies have made estimates on the risk of diseases posed by environmental tobacco smoke exposure, the so-called "science" calculated the risk using a "misleading marker of 'lifetime exposure'" and "data that are illusory." Former U.S. Surgeon General Richard Carmona said in July 2006 that there is no risk-free level of ETS exposure and children exposed to it will "eventually ... develop cardiovascular disease and cancers over time." Gori said since cancer and cardiovascular diseases develop at advancing ages, estimating the risk of those illnesses posed by ETS requires knowing the total of "momentary secondhand smoke doses" that nonsmokers have been exposed to over their lifetimes. Such summations are "obviously impossible" because ETS concentrations in the air, rates of inhalation, metabolic transformations and other factors vary depending on the moment, location, and time, he said. All ETS-related studies have circumvented this obstacle by estimating the ETS risk using a misleading marker of "lifetime exposure" using information obtained during brief phone interviews, he noted. He added that people with cancer of cardiovascular disease are prone to "amplify their recall" of ETS exposure. He called for an open discussion to "restore straight thinking in the legitimate uses of 'the science' of epidemiology" (Washington Post 1/30).

LIES, DAMNED LIES, & 400,000 SMOKING-RELATED DEATHS

by Robert A. Levy and Rosalind B. Marimont

TRUTH WAS AN EARLY VICTIM in the battle against tobacco. The big lie, repeated ad nauseam in anti-tobacco circles, is that smoking causes more than 400,000 premature deaths each year in the United States. That mantra is the principal justification for all manner of tobacco regulations and legislation, not to mention lawsuits by dozens of states for Medicaid recovery, class actions by seventy-five to eighty union health funds, similar litigation by thirty-five Blue Cross plans, twenty-four class suits by smokers who are not yet ill, sixty class actions by allegedly ill smokers, five hundred suits for damages from secondhand smoke, and health-related litigation by twelve cities and counties—an explosion of adjudication never before experienced in this country or elsewhere.

The war on smoking started with a kernel of truth—that cigarettes are a high risk factor for lung cancer—but has grown into a monster of deceit and greed, eroding the credibility of government and subverting the rule of law. Junk science has replaced honest science and propaganda parades as fact. Our legislators and judges, in need of dispassionate analysis, are instead smothered by an avalanche of statistics—tendentious, inadequately documented, and unchecked by even rudimentary notions of objectivity. Meanwhile, Americans are indoctrinated by health "professionals" bent on imposing their lifestyle choices on the rest of us and brainwashed by politicians eager to tap the deep pockets of a pariah industry.

The aim of this paper is to dissect the granddaddy of all tobacco lies—that smoking causes 400,000 deaths each year. To set the stage, let's look at two of the many exaggerations, misstatements, and outright fabrications that have dominated the tobacco debate from the outset.

THIRD-RATE THINKING ABOUT SECONDHAND SMOKE

"Passive Smoking Does Cause Lung Cancer, Do Not Let Them Fool You," states the headline of a March 1998 press release from the World Health Organization. The release begins by noting that WHO had been accused of suppressing its own study because it "failed to scientifically prove that there is an association between passive smoking . . . and a number of diseases, lung cancer in particular." Not true, insisted WHO. Smokers themselves are not the only ones who suffer health problems because of their habit; secondhand smoke can be fatal as well.

The press release went on to report that WHO researchers found "an estimated 16 percent increased risk of lung cancer among nonsmoking spouses of smokers. For workplace exposure the estimated increase in risk was 17 percent." Remarkably, the very next line warned: "Due to small sample size, neither increased risk was statistically significant." Contrast that conclusion with the hype in the headline: "Passive Smoking Does Cause Lung Cancer." Spoken often enough, the lie becomes its own evidence.

The full study would not see the light of day for seven more months, until October 1998, when it was finally published in the *Journal of the National Cancer Institute*. News reports omitted any mention of statistical insignificance. Instead, they again trumpeted relative risks of 1.16 and 1.17, corresponding to 16 and 17 percent increases, as if those ratios were meaningful. Somehow lost in WHO's media blitz was the National Cancer Institute's own guideline: "Relative risks of less than 2 [that is, a 100 percent increase] are considered small. . . . Such increases may be due to chance, statistical bias, or effects of confounding factors that are sometimes not evident." To put the WHO results in their proper perspective, note that the relative risk of lung cancer for persons who drink whole milk is 2.4. That is, the increased risk of contracting lung cancer from whole milk is 140 percent—more than eight times the 17 percent increase from secondhand smoke.

What should have mattered most to government officials, the health community and concerned parents is the following pronouncement from the WHO study: After examining 650 lung cancer patients and 1,500 healthy adults in seven European countries, WHO concluded that the "results indicate no association between childhood exposure to environmental tobacco smoke and lung cancer risk."

Robert A. Levy is a senior fellow in constitutional studies at the Cato Institute and an adjunct professor at Georgetown University Law Center where he teaches "Statistics for Lawyers." Rosalind B. Marimont is a mathematician and scientist now retired after a 37-year career with the National Institute of Standards and Technology (formerly the Bureau of Standards) and the National Institute of Health.

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EPA'S JUNK SCIENCE

Another example of anti-tobacco misinformation is the landmark 1993 report in which the Environmental Protection Agency declared that environmental tobacco smoke (ETS) is a dangerous carcinogen that kills three thousand Americans yearly. Five years later, in July 1998, federal judge William L. Osteen lambasted the EPA for "cherry picking" the data, excluding studies that "demonstrated no association between ETS and cancer," and withholding "significant portions of its findings and reasoning in striving to confirm its *a priori* hypothesis." Both "the record and EPA's explanation," concluded the court, "make it clear that using standard methodology, EPA could not produce statistically significant results." A more damning assessment is difficult to imagine, but here are the court's conclusions at greater length, in its own words.

> EPA publicly committed to a conclusion before research had begun; excluded industry [input thereby] violating the [Radon Research] Act's procedural requirements; adjusted established procedure and scientific norms to validate the Agency's public conclusion, and aggressively utilized the Act's authority to disseminate findings to establish a de facto regulatory scheme intended to restrict Plaintiff's products and to influence public opinion. In conducting the ETS Risk Assessment, EPA disregarded information and made findings on selective information; did not disseminate significant epidemiologic information; deviated from its Risk Assessment Guidelines; failed to disclose important findings and reasoning; and left significant questions without answers. EPA's conduct left substantial holes in the administrative record. While so doing, EPA produced limited evidence, then claimed the weight of the Agency's research evidence demonstrated ETS causes Cancer. [*Flue-Cured Tobacco Coop. Stabilization Corp. v. United States Environmental Protection Agency*, 4 F. Supp. 2d 435, 465-66 (M.D.N.C. 1998)]

Hundreds of states, cities, and counties have banned indoor smoking—many in reaction to the EPA report. California even prohibits smoking in bars. According to Matthew L. Myers, general counsel of the Campaign for Tobacco-Free Kids, "the release of the original risk assessment gave an enormous boost to efforts to restrict smoking." Now that the study has been thoroughly debunked, one would think that many of the bans would be lifted. Don't hold your breath. When science is adulterated and debased for political ends, the culprits are unlikely to reverse course merely because they have been unmasked.

In reaction to the federal court's criticism EPA administrator Carol M. Browner said, "It's so widely accepted that secondhand smoke causes very real problems for kids and adults. Protecting people from the health hazards of secondhand smoke should be a national imperative." Like *Alice in Wonderland*, sentence first, evidence afterward. Browner reiterates: "We believe the health threats . . . from breathing secondhand smoke are very real." Never mind science; it is Browner's beliefs that control. The research can be suitably tailored.

For the EPA to alter results, disregard evidence, and adjust its procedures and standards to satisfy agency prejudices is unacceptable behavior, even to a first-year science student. Those criticisms are about honesty, carefulness, and rigor—the very essence of science.

CLASSIFYING DISEASES AS SMOKING-RELATED

With that record of distortion, it should come as no surprise that anti-tobacco crusaders misrepresent the number of deaths due to smoking. Start by considering the diseases that are incorrectly classified as smoking-related. The Centers for Disease Control and Prevention (CDC) prepares and distributes information on smoking-attributable mortality, morbidity and economic costs (SAMMEC). In its *Morbidity and Mortality Weekly Report* for 27 August 1993, the CDC states that 418,690 Americans died in 1990 of various diseases that they contracted because, according to the government, they smoked.

Diseases are categorized as smoking-related if the risk of death for smokers exceeds that for nonsmokers. In the jargon of epidemiology, a relative risk that is greater than 1 indicates a connection between exposure (smoking) and effect (death). Recall, however, the National Cancer Institute's guideline: "Relative risks of less than two are considered small. . . . Such increases may be due to chance, statistical bias, or effects of confounding factors that are sometimes not evident." And the *Federal Reference Manual on Scientific Evidence* confirms that the threshold test for legal significance is a relative risk of two or higher. At any ratio below two, the results are insufficiently reliable to conclude that a particular agent (e.g., tobacco) caused a particular disease.

What would happen if the SAMMEC data were to exclude deaths from those diseases that had a relative risk of less than two for current or former smokers? Table 1 (at the end of this article) shows that 163,071 deaths reported by CDC were from diseases that should not have been included in the report. Add to that another 1,362 deaths from burn injuries—unless one believes that Philip Morris is responsible when a smoker falls asleep with a lit cigarette. That is a total of 164,433 misreported deaths out of 418,690. When the report is properly limited to diseases that have a significant relationship with smoking, the death total declines to 254,257. Thus, on this count alone, SAMMEC overstates the number of deaths by 65 percent.

CALCULATING EXCESS DEATHS

But there is more. Writing on "Risk Attribution and Tobacco-Related Deaths" in the 1993 *American Journal of Epidemiology*, T. D. Sterling, W. L. Rosenbaum, and J. J. Weinkam expose another overstatement—exceeding 65 percent—that flows from using the American Cancer Society's Cancer Prevention Survey (CPS) as a baseline against which excess deaths are computed. Here is how one government agency, the Office of Technology Assessment (OTA), calculates the number of deaths caused by smoking:

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The OTA first determines the death rate for persons who were part of the CPS sample and never smoked. Next, that rate is applied to the total U.S. population in order to estimate the number of Americans who would have died if no one ever smoked. Finally, the hypothetical number of deaths for assumed never-smokers is subtracted from the actual number of U.S. deaths, and the difference is ascribed to smoking. That approach seems reasonable if one important condition is satisfied: The CPS sample must be roughly the same as the overall U.S. population with respect to those factors, other than smoking, that could be associated with the death rate. But as Sterling, Rosenbaum, and Weinkam point out, nothing could be further from the truth.

The American Cancer Society bases its CPS study on a million men and women volunteers, drawn from the ranks of the Society's members, friends, and acquaintances. The persons who participate are more affluent than average, overwhelmingly white, married, college graduates, who generally do not have hazardous jobs. Each of those characteristics tends to reduce the death rate of the CPS sample which, as a result, enjoys an average life expectancy that is substantially longer than the typical American enjoys.

Because OTA starts with an atypically low death rate for never-smokers in the CPS sample, then applies that rate to the whole population, its baseline for determining excess deaths is grossly underestimated. By comparing actual deaths with a baseline that is far too low, OTA creates the illusion that a large number of deaths are due to smoking.

That same illusion pervades the statistics released by the U.S. Surgeon General, who in his 1989 report estimated that 335,600 deaths were caused by smoking. When Sterling, Rosenbaum, and Weinkam recalculated the Surgeon General's numbers, replacing the distorted CPS sample with a more representative baseline from large surveys conducted by the National Center for Health Statistics, they found that the number of smoking-related deaths declined to 203,200. Thus, the Surgeon General's report overstated the number of deaths by more than 65 percent simply by choosing the wrong standard of comparison.

Sterling and his coauthors report that not only is the death rate considerably lower for the CPS sample than for the entire U.S. but, astonishingly, even smokers in the CPS sample have a lower death rate than the national average for both smokers and nonsmokers. As a result, if OTA were to have used the CPS death rate for smokers, applied that rate to the total population, then subtracted the actual number of deaths for all Americans, it would have found that smoking saves 277,621 lives each year. The authors caution, of course, that their calculation is sheer nonsense, not a medical miracle. Those "lives would be saved only if the U.S. population would die with the death rate of smokers in the affluent CPS sample."

Unhappily, the death rate for Americans is considerably higher than that for the CPS sample. Nearly as disturbing, researchers like Sterling, Rosenbaum, and Weinkam identified that statistical predicament many years ago; yet the government persists in publishing data on smoking-related deaths that are known to be greatly inflated.

CONTROLLING FOR CONFOUNDING VARIABLES

Even if actual deaths were compared against an appropriate baseline for nonsmokers, the excess deaths could not properly be attributed to smoking alone. It cannot be assumed that the only difference between smokers and nonsmokers is that the former smoke. The two groups are dissimilar in many other respects, some of which affect their propensity to contract diseases that have been identified as smoking-related. For instance, smokers have higher rates of alcoholism, exercise less on average, eat fewer green vegetables, are more likely to be exposed to workplace carcinogens, and are poorer than nonsmokers. Each of those factors can be a "cause" of death from a so-called smoking-related disease; and each must be statistically controlled for if the impact of a single factor, like smoking, is to be reliably determined.

Sterling, Rosenbaum, and Weinkam found that adjusting their calculations for just two lifestyle differences—in income and alcohol consumption—between smokers and nonsmokers had the effect of reducing the Surgeon General's smoking-related death count still further, from 203,200 to 150,000. That means the combined effect of using a proper standard of comparison coupled with controls for income and alcohol was to lower the Surgeon General's estimate 55 percent—from 335,600 to 150,000. Thus, the original estimate was a disquieting 124 percent too high, even without adjustments for important variables like occupation, exercise, and nutritional habits.

What if smokers got plenty of exercise and had healthy diets while nonsmokers were couch potatoes who consumed buckets of fast food? Naturally, there are some smokers and nonsmokers who satisfy those criteria. Dr. William E. Wecker, a consulting statistician who has testified for the tobacco industry, scanned the CPS database and found thousands of smokers with relatively low risk factors and thousands of never-smokers with high risk factors. Comparing the mortality rates of the two groups, Dr. Wecker discovered that the smokers were "healthier and die less often by a factor of three than the never-smokers." Obviously, other risk factors matter, and any study that ignores them is utterly worthless.

Yet, if a smoker who is obese; has a family history of high cholesterol, diabetes, and heart problems; and never exercises dies of a heart attack, the government attributes his death to smoking alone. That procedure, if applied to the other causal factors identified in the CPS study, would produce more than twice as many "attributed" deaths as there are actual deaths, according to Dr. Wecker. For example, the same calculations that yield 400,000 smoking-related deaths suggest that 504,000 people die each year because they engage in little or no exercise. Employing an identical formula, bad nutritional habits can be shown to account for 649,000 excess deaths annually. That is nearly 1.6 million deaths from only three causes—without considering alcoholism, accidents, poverty, etc.—out of 2.3 million deaths in 1995 from all causes combined. And on it goes—computer-generated phantom deaths, not real deaths—constrained

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neither by accepted statistical methods, by common sense, nor by the number of people who die each year.

ADJUSTING FOR AGE AT DEATH

Next and last, we turn to a different sort of deceit—one pertaining not to the number of smoking-related deaths but rather to the misperception that those deaths are somehow associated with kids and young adults. For purposes of this discussion, we will work with the far-fetched statistics published by CDC—an annual average from 1990 through 1994 of 427,743 deaths attributable to tobacco. Is the problem as serious as it sounds?

At first blush, it would seem that more than 400,000 annual deaths is an extremely serious problem. But suppose that all of the people died at age ninety-nine. Surely then, the seriousness of the problem would be tempered by the fact that the decedents would have died soon from some other cause in any event. That is not far from the truth: while tobacco does not kill people at an average age of ninety-nine, it does kill people at an average age of roughly seventy-two–far closer to ninety-nine than to childhood or even young adulthood. Indeed, according to a 1991 RAND study, smoking "reduces the life expectancy of a twenty-year-old by about 4.3 years"—not a trivial concern to be sure, but not the horror that is sometimes portrayed.

Consider Table 2, which shows the number of deaths and age at death for various causes of death: The three nonsmoking categories total nearly 97,000 deaths—probably not much different than the correctly calculated number of smoking-related deaths—but the average age at death is only thirty-nine. As contrasted with a seventy-two-year life expectancy for smokers, each of those nonsmoking deaths snuffs out thirty-three years of life—our most productive years, from both an economic and child-rearing perspective.

Perhaps that is why the Carter Center's "Closing the Gap" project at Emory University examined "years of potential life lost" (YPLL) for selected diseases, to identify those causes of death that were of greatest severity and consequence. The results were reported by R.W. Amler and D.L. Eddins, "Cross-Sectional Analysis: Precursors of Premature Death in the United States," in the 1987 *American Journal of Preventive Medicine*. First, the authors determined for each disease the annual number of deaths by age group. Second, they multiplied for each age group the number of deaths times the average number of years remaining before customary retirement at age sixty-five. Then they computed YPLL by summing the products for each disease across age groups.

Thus, if smoking were deemed to have killed, say, fifty thousand people from age sixty through sixty-four, a total of 150,000 years of life were lost in that age group—i.e., fifty thousand lives times an average of three years remaining to age sixty-five. YPLL for smoking would be the accumulation of lost years for all age groups up to sixty-five.

Amler and Eddins identified nine major precursors of preventable deaths. Measured by YPLL, tobacco was about halfway down the list—ranked four out of nine in terms of years lost—not "the number one killer in America" as alarmists have exclaimed. Table 3 shows the four most destructive causes of death, based on 1980 YPLL statistics. Bear in mind that the starting point for the YPLL calculation is the number of deaths, which for tobacco is grossly magnified for all of the reasons discussed above.

According to Amler and Eddins, even if we were to look at medical treatment—measured by days of hospital care—nonalcohol-related injuries impose a 58 percent greater burden than tobacco, and nutrition-related diseases are more burdensome as well.

Another statistic that more accurately reflects the real health repercussions of smoking is the age distribution of the 427,743 deaths that CDC mistakenly traces to tobacco. No doubt most readers will be surprised to learn that—aside from burn victims and pediatric diseases—*tobacco does not kill a single person below the age of 35.*

Each year from 1990 through 1994, as shown in Table 4, only 1,910 tobacco-related deaths—less than half of 1 percent of the total—were persons below age thirty-five. Of those, 319 were burn victims and the rest were infants whose parents smoked. But the relationship between parental smoking and pediatric diseases carries a risk ratio of less than 2, and thus is statistically insignificant. Unless better evidence is produced, those deaths should not be associated with smoking.

On the other hand, the National Center for Health Statistics reports that more than twenty-one thousand persons below age thirty-five died from motor vehicle accidents in 1992, more than eleven thousand died from suicide, and nearly seventeen thousand died from homicide. Over half of those deaths were connected with alcohol or drug abuse. That should put smoking-related deaths in a somewhat different light.

Most revealing of all, almost 255,000 of the smoking-related deaths—nearly 60 percent of the total—occurred at age seventy or above. More than 192,000 deaths—nearly 45 percent of the total—occurred at age seventy-five or higher. And roughly 72,000 deaths—almost 17 percent of the total—occurred at the age of 85 or above. Still, the public health community disingenuously refers to "premature" deaths from smoking, as if there is no upper age limit to the computation.

The vast overestimate of the dangers of smoking has had disastrous results for the health of young people. Risky behavior does not exist in a vacuum; people compare uncertainties and apportion their time, effort, and money according to the perceived severity of the risk. Each year, alcohol and drug abuse kills tens of thousands of people under the age of thirty-five. Yet according to a 1995 survey by the U.S. Department of Health and Human Services, high school seniors thought smoking a pack a day was more dangerous than daily consumption of four to five alcoholic beverages or using barbiturates. And the CDC reports that the number of pregnant women who drank frequently quadrupled between 1991 and 1995—notwithstanding that fetal alcohol syndrome is the largest cause of preventable mental retardation, occurring in one out of every one thousand births.

Can anyone doubt that the drumbeat of antismoking propaganda from the White House and the health establishment has

deluded Americans into thinking that tobacco is the real danger to our children? In truth, alcohol and drug abuse poses an immensely greater risk and antismoking zealots bear a heavy burden for their duplicity.

CONCLUSION

The unvarnished fact is that children do not die of tobacco-related diseases, correctly determined. If they smoke heavily during their teens, they may die of lung cancer in their old age, fifty or sixty years later, assuming lung cancer is still a threat then.

Meanwhile, do not expect consistency or even common sense from public officials. Alcoholism contributes to crime, violence, spousal abuse, and child neglect. Children are dying by the thousands in accidents, suicides, and homicides. But states go to war against nicotine—which is not an intoxicant, has no causal connection with crime, and poses little danger to young adults or family members.

The campaign against cigarettes is not entirely dishonest. After all, a seasoning of truth makes the lie more digestible. Evidence does suggest that cigarettes substantially increase the risk of lung cancer, bronchitis, and emphysema. The relationship between smoking and other diseases is not nearly so clear, however; and the scare-mongering that has passed for science is appalling. Not only is tobacco far less pernicious than Americans are led to believe, but its destructive effect is amplified by all manner of statistical legerdemain—counting diseases that should not be counted, using the wrong sample as a standard of comparison, and failing to control for obvious confounding variables.

To be blunt, there is no credible evidence that 400,000 deaths per year—or any number remotely close to 400,000—are caused by tobacco. Nor has that estimate been adjusted for the positive effects of smoking—less obesity, colitis, depression, Alzheimer's disease, Parkinson's disease and, for some women, a lower incidence of breast cancer. The actual damage from smoking is neither known nor knowable with precision. Responsible statisticians agree that it is impossible to attribute causation to a single variable, like tobacco, when there are multiple causal factors that are correlated with one another. The damage from cigarettes is far less than it is made out to be.

Most important, the government should stop lying and stop pretending that smoking-related deaths are anything but a statistical artifact. The unifying bond of all science is that truth is its aim. When that goal yields to politics, tainting science in order to advance predetermined ends, we are all at risk. Sadly, that is exactly what has transpired as our public officials fabricate evidence to promote their crusade against big tobacco.

Table 1

Disease Category	Relative Risk	Deaths from Smoking
Cancer of pancreas	1.1 - 1.8	2,931*
Cancer of cervix	1.9	647*
Cancer of bladder	1.9	2,348*
Cancer of kidney, other urinary	1.2 - 1.4	353
Hypertension	1.2 - 1.9	5,450
Ischemic heart disease (age 35-64)	1.4 - 1.8	15,535*
Ischemic heart disease (age 65+)	1.3 - 1.6	64,789
Other heart disease	1.2 - 1.9	35,314
Cerebrovascular disease (age 35-64)	1.4	2,681*
Cerebrovascular disease (age 65+)	1.0 - 1.9	14,610
Atherosclerosis	1.3	1,267*
Aortic aneurysm	1.3	448*
Other arterial disease	1.3	372*
Pneumonia and influenza	1.4 - 1.6	10,552*
Other respiratory diseases	1.4 - 1.6	1,063*
Pediatric diseases	1.5 - 1.8	1,711
Sub-total		160,071
Environmental tobacco smoke	1.2	3,000
Total		163,071

* Number of deaths for this category assumes population deaths distributed between current and former smokers in same proportion as in Cancer Prevention Survey CPS-II, provided by the American Cancer Society.

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Table 2

Cause of Death	Number of Deaths per Year	Mean Age at Death
Smoking-attributed	427,743	72
Motor vehicle accidents	40,982	39
Suicide	30,484	45
Homicide	25,488	32

Source: Centers for Disease Control and Prevention

Table 3

Cause	Deaths	YPLL
Alcohol-related	99,247	1,795,458
Gaps in primary care*	132,593	1,771,133
Injuries (excluding alcohol-related)	64,169	1,755,720
Tobacco-related	338,022	1,497,161

* Inadequate access, screening and preventive interventions.

Table 4

U.S. Smoking-Attributable Mortality by Cause and Age of Death
1990-1994 Annual Average

Age at Death	Pediatric Diseases	Burn Victims	All Other Diseases	Total
Under 1	1,591	19	0	1,610
1 - 34	0	300	0	300
35 - 49	0	221	21,773	21,994
50 - 69	0	286	148,936	149,222
70 - 74	0	96	62,154	62,250
75 - 84	0	133	120,537	120,670
85 +	0	45	71,652	71,697
Totals	1,591	1,100	425,052	427,743

Source: Private communication from the Centers for Disease Control and Prevention

Papers

Environmental tobacco smoke and tobacco related mortality in a prospective study of Californians, 1960-98

James E Enstrom, Geoffrey C Kabat

Abstract

Objective To measure the relation between environmental tobacco smoke, as estimated by smoking in spouses, and long term mortality from tobacco related disease.
Design Prospective cohort study covering 39 years.
Setting Adult population of California, United States.
Participants 118 094 adults enrolled in late 1959 in the American Cancer Society cancer prevention study (CPS I), who were followed until 1998. Particular focus is on the 35 561 never smokers who had a spouse in the study with known smoking habits.
Main outcome measures Relative risks and 95% confidence intervals for deaths from coronary heart disease, lung cancer, and chronic obstructive pulmonary disease related to smoking in spouses and active cigarette smoking.
Results For participants followed from 1960 until 1998 the age adjusted relative risk (95% confidence interval) for never smokers married to ever smokers compared with never smokers married to never smokers was 0.94 (0.85 to 1.05) for coronary heart disease, 0.75 (0.42 to 1.35) for lung cancer, and 1.27 (0.78 to 2.08) for chronic obstructive pulmonary disease among 9619 men, and 1.01 (0.94 to 1.08), 0.99 (0.72 to 1.37), and 1.13 (0.80 to 1.58), respectively, among 25 942 women. No significant associations were found for current or former exposure to environmental tobacco smoke before or after adjusting for seven confounders and before or after excluding participants with pre-existing disease. No significant associations were found during the shorter follow up periods of 1960-5, 1966-72, 1973-85, and 1973-98.
Conclusions The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect. The association between exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed.

Introduction

Several major reviews have determined that exposure to environmental tobacco smoke increases the relative risk of coronary heart disease, based primarily on comparing never smokers married to smokers with never smokers married to never smokers. The American Heart Association, the California Environmental Protection Agency, and the US surgeon general have concluded that the increase in coronary heart disease risk due to environmental tobacco smoke is 30% (relative risk 1.30).[1-3] Meta-analyses of epidemiological studies have reported summary relative risks (95% confidence intervals) of 1.30 (1.22 to 1.38), 1.25 (1.17 to 1.32), and 1.25 (1.17 to 1.33) for coronary heart disease[4-6] and 1.23 (1.13 to 1.35) and 1.23 (1.13 to 1.34) for lung cancer,[7 8] similar to the 1.20 found by the California Environmental Protection Agency and the US surgeon general.[2 3] The US Environmental Protection Agency has classified environmental tobacco smoke as a known human carcinogen.[7] Chronic obstructive pulmonary disease, primarily asthma, bronchitis, and emphysema, has been associated with exposure to environmental tobacco smoke, but the evidence for increased mortality is sparse.[2 3]

Although these reviews come to similar conclusions, the association between environmental tobacco smoke and tobacco related diseases is still controversial owing to several limitations in the epidemiological studies.[9-14] Exposure to environmental tobacco smoke is difficult to measure quantitatively and therefore has been approximated by self reported estimates, primarily smoking history in spouses. Confounding by active cigarette smoking is so strong that the association with environmental tobacco smoke can only be evaluated among never smokers. The relation between tobacco related diseases and environmental tobacco smoke may be influenced by misclassification of some smokers as never smokers, misclassification of exposure status to environmental tobacco smoke, and several potential confounders. It is also unclear how the reported increased risk of coronary heart disease due to environmental tobacco smoke could be so close to the increased risk due to active smoking (30% and 70%, respectively), since environmental tobacco smoke is much more dilute than actively inhaled smoke.

Most epidemiological studies have found that environmental tobacco smoke has a positive but not statistically significant relation to coronary heart disease and lung cancer. Meta-analyses have combined these inconclusive results to produce statistically significant summary relative risks.[4-8] However, there are problems inherent in using meta-analysis to establish a causal relation.[9-14] The epidemiological data are subject to the limitations described above. They have not been collected in a standardised way, and some relative risks

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Table 1 Follow up details of 51 343 men and 66 751 women in California cancer prevention study (CPS I) cohort

	Total cohort		Never smokers*	
Follow up category	**Men**	**Women**	**Men**	**Women**
1 Jan 1960:				
Dead, deleted from file (1 Oct to 31 Dec 1959)	22	14		
Alive, completed 1959 questionnaire (1 Oct 1959 to 31 Mar 1960)	51 321	66 737	9619	25 942
31 Dec 1965:				
Dead, ICD codes (1 Jan 1960 to 31 Dec 1965)	4 907	3 506	685	868
Dead, no ICD codes(1 Jan 1960 to 31 Dec 1965)	45	47	7	13
Withdrawn (1 Jan 1960 to 30 Sep 1965)†	718	974	79	257
Lost (1 Jan 1960 to 31 Dec 1965)‡	31	49	4	13
Alive, completed Sep 1965 questionnaire	44 757	61 079	8574	24 077
Alive, follow up to 31 Dec 1998	863	1 082	270	714
31 Dec 1972:				
Dead, ICD codes(1 Jan 1960 to 31 Dec 1972)	12 295	9 446	1865	2 634
Dead, no ICD codes (1 Jan 1960 to 31 Dec 1972)	146	160	19	41
Withdrawn (1 Jan 1960 to 30 Sep 1971)†	1 222	2 825	164	984
Lost (1 Jan 1960 to 31 Dec 1972)	1 525	3 367	269	1 103
Alive, completed Sep 1972 questionnaire	26 070	37 926	5455	16 171
Alive, follow up to 31 Jan 1998	10 063	13 013	1847	5 009
31 Dec 1998:				
Dead, ICD codes (1 Jan 1960 to 31 Dec 1998)	37 554	36 669	6673	13 160
Dead, no ICD codes (1 Jan 1960 to 31 Dec 1998)	2 456	2 722	464	1 130
Withdrawn (1 Jan 1960 to 30 Sep 1972)†	1 395	5 450	197	2 105
Lost (1 Jan 1960 to 31 Dec 1998)	2 962	6 953	560	2 579
Alive, correctly completed 1999 questionnaire	2 290	4 869	681	2 413
Alive, matched with California driver's licence and not known dead§	4 664	10 074	1044	4 555

*Never smokers who had spouse in cohort with known smoking habits.
†Further follow up not possible because of incomplete or missing name on 1972 master database.
‡Complete name on 1972 master file and no match with California driver's licence file, California death file, or social security death index until 1998.
§Based on 1990-9 match with California driver's licence file and no death match with California death file or social security death index during 1960-98.

have been inappropriately combined. Because it is more likely that positive associations get published, unpublished negative results could reduce the summary relative risks. Also, the meta-analyses of coronary heart disease omitted the published negative results from the large American Cancer Society cancer prevention study (CPS I).[10 11] We have extended the follow up for the California participants in this cohort, analysed the relation between environmental tobacco smoke and tobacco related diseases, and addressed concerns about this study.

Methods

CPS I is a prospective cohort study begun by the American Cancer Society in October 1959 and

Table 2 Personal and lifestyle characteristics of male 1959 never smokers in California cancer prevention study (CPS I) cohort by smoking status of spouse

	Smoking status of spouse, 1959						1999 respondents	
			Current (cigarettes/day)					
Characteristic	**Never**	**Former**	**1-19**	**20-39**	**≥40**	**Total**	**1959 value**	**1999 value**
No of participants in 1959	7458	624	905	587	45	9619		
No of participants in 1999	498	59	69	51	4	681	681	681
Withdrawn as of 1972 (%)	2.0 (146)	2.4 (15)	2.1 (19)	2.7 (16)	2.2 (1)	2.0 (197)		
Lost to follow up as of 1999 (%)	5.9 (441)	4.6 (29)	5.4 (49)	6.3 (37)	8.9 (4)	5.8 (560)		
Unknown cause of death (%)	6.6 (371)	6.1 (26)	6.6 (42)	5.4 (22)	8.8 (3)	6.5 (464)		
Widowed as of 1999	28.2 (1649)	25.1 (124)	31.9 (231)	38.1 (174)	39.4 (13)	29.0 (2191)		
Mean age (years) at enrolment	56.5 (7458)	51.9 (624)	52.8 (905)	51.7 (587)	52.6 (45)	55.5 (9619)	45.5 (681)	45.5 (681)
White people (%)	97.8 (7292)	98.6 (615)	98.0 (887)	98.1 (577)	100.0 (45)	97.9 (9416)	98.6 (672)	
Education ≥12 years (%)	67.3 (5017)	80.6 (403)	71.3 (645)	74.2 (436)	84.5 (38)	69.0 (6639)	89.0 (606)	92.9 (633)
Mean height (cm)	175.8 (7328)	176.3 (614)	176.3 (898)	176.5 (582)	176.8 (43)	175.8 (9465)	177.0 (681)	175.3 (681)
Mean weight (kg)	78.9 (7137)	79.7 (602)	79.6 (881)	80.9 (573)	82.2 (44)	79.1 (9237)	78.6 (681)	74.9 (681)
History of serious diseases (%):	13.8 (965)	10.0 (59)	11.9 (102)	11.9 (65)	12.5 (5)	13.3 (1196)	4.1 (28)	
Cancer	5.0 (369)	4.7 (29)	5.5 (50)	4.6 (27)	2.2 (1)	5.0 (476)	2.9 (20)	39.9 (272)
Heart disease	7.0 (471)	4.8 (27)	5.4 (44)	5.6 (29)	7.7 (3)	6.6 (574)	1.0 (7)	
Stroke	1.8 (125)	0.5 (3)	1.0 (8)	1.7 (9)	2.6 (1)	1.7 (146)	0.2 (1)	
Sick at present time (%)	6.4 (475)	4.8 (30)	6.3 (57)	5.6 (33)	4.4 (2)	6.2 (597)	4.2 (29)	22.2 (151)
Professional occupation (%)	14.3 (1068)	14.9 (93)	11.1 (100)	10.5 (62)	17.8 (8)	13.8 (1331)	17.8 (121)	
Urban residence (%)	85.9 (6404)	90.7 (566)	88.7 (803)	90.0 (529)	88.9 (40)	86.7 (8342)	86.0 (586)	
Moderate or heavy exercise (%)	76.2 (5683)	70.2 (438)	72.5 (656)	71.1 (418)	57.8 (26)	75.0 (7221)	70.7 (481)	70.9 (483)
Eat green salads (mean days/week)	4.8 (7201)	4.9 (617)	5.0 (887)	5.0 (573)	4.9 (45)	4.8 (9323)	5.1 (681)	4.5 (681)
Eat fruits or drink fruit juice (mean days/week)	6.0 (7226)	6.0 (614)	5.9 (886)	5.5 (574)	5.3 (43)	5.9 (9343)	5.9 (681)	5.6 (681)
Often use vitamin pills (%)	38.1 (2841)	39.7 (248)	33.2 (300)	28.7 (169)	42.2 (19)	37.2 (3577)	34.0 (232)	79.2 (539)

Some values do not agree with denominators due primarily to missing data.

described in detail elsewhere.[15–17] A total of 1 078 894 adults from 25 states were enrolled on the basis of a detailed four page questionnaire. In 1961, 1963, 1965, and 1972, surviving cohort members completed brief questionnaires. The American Cancer Society ascertained the vital status and current address for most of

Table 3 Personal and lifestyle characteristics of female 1959 never smokers in California cancer prevention study (CPS I) cohort by smoking status of spouse

	Smoking status of spouse, 1959							1999 respondents	
			Current status*						
Characteristic	Never	Former	Pipe or cigar	1-19	20-39	≥40	Total	1959 value	1999 value
No of participants in 1959	7399	6858	2691	3219	4934	841	25 942		
No of participants in 1999	788	573	252	233	479	87	2 412	2412	2412
Withdrawn as of 1972 (%)	8.1 (602)	8.1 (558)	8.1 (219)	8.2 (265)	7.7 (380)	9.6 (81)	8.1 (2 105)		
Lost to follow up as of 1999 (%)	9.8 (722)	9.8 (669)	9.7 (260)	10.1 (324)	10.4 (513)	10.8 (91)	9.9 (2 579)		
Unknown cause of death (%)	7.6 (304)	7.7 (305)	7.2 (111)	8.1 (149)	8.7 (218)	10.4 (43)	7.9 (1 130)		
Widowed as of 1999 (%)	59.7 (3464)	65.2 (3528)	64.2 (1368)	69.6 (1774)	73.4 (2859)	75.1 (480)	66.0 (13 473)		
Mean age (years) at enrolment	53.1 (7399)	54.5 (6858)	54.4 (2691)	53.7 (3219)	50.9 (4934)	49.8 (841)	53.1 (25 942)	44.5 (2412)	44.5 (2412)
White people (%)	97.6 (7225)	98.5 (6759)	97.8 (2631)	95.9 (3088)	97.9 (4828)	98.7 (831)	97.8 (25 362)	98.0 (2364)	
Education ≥12 years (%)	73.7 (5452)	68.2 (4685)	68.9 (1853)	65.6 (2109)	70.4 (3476)	77.2 (650)	70.2 (18 225)	87.9 (2120)	93.0 (2243)
Mean height (cm)	162.1 (7232)	161.8 (6706)	161.8 (2640)	161.5 (3168)	161.8 (4846)	162.3 (822)	161.8 (25 414)	162.6 (2412)	161.3 (2412)
Mean weight (kg)	63.9 (7085)	63.8 (6596)	64.0 (2581)	63.5 (3097)	63.7 (4777)	63.64 (824)	63.8 (24 960)	61.4 (2412)	62.3 (2412)
History of serious diseases (%):	11.8 (834)	12.8 (857)	11.3 (293)	10.2 (315)	10.1 (483)	10.2 (85)	11.4 (2 867)	5.8 (140)	
Cancer	5.8 (427)	6.7 (465)	5.8 (156)	5.2 (167)	5.9 (293)	7.2 (61)	6.0 (1 569)	4.1 (99)	36.4 (878)
Heart disease	5.1 (347)	5.1 (330)	4.7 (117)	4.2 (123)	3.4 (154)	2.9 (23)	4.5 (1 094)	1.5 (36)	
Stroke	0.9 (60)	1.0 (62)	0.8 (20)	0.8 (25)	0.8 (36)	0.1 (1)	0.9 (204)	0.2 (5)	
Sick at present time (%)	7.9 (586)	8.3 (572)	8.6 (231)	8.2 (264)	8.8 (436)	8.8 (74)	8.3 (2 163)	6.4 (154)	19.7 (475)
Professional occupation (%)	14.6 (1080)	12.8 (881)	13.0 (350)	12.9 (414)	10.6 (523)	10.9 (92)	12.9 (3 340)	17.4 (420)	
Urban residence (%)	85.8 (6349)	86.4 (5929)	85.4 (2296)	86.2 (2775)	85.7 (4229)	85.8 (722)	85.9 (22 302)	84.7 (2043)	
Moderate or heavy exercise (%)	82.5 (6097)	82.5 (5649)	83.3 (2242)	82.8 (2665)	82.3 (4058)	81.0 (681)	82.6 (21 392)	80.2 (1934)	65.5 (1580)
Eat green salads (mean days/week)	5.1 (7219)	5.0 (6701)	5.1 (2618)	4.9 (3122)	5.1 (4835)	5.1 (825)	5.0 (25 320)	5.4 (2412)	4.6 (2412)
Eat fruits or drink fruit juice (mean days/week)	6.4 (7227)	6.3 (6727)	6.3 (2621)	6.1 (3132)	6.0 (4846)	6.0 (826)	6.2 (25 379)	6.1 (2412)	5.6 (2412)
Often use vitamin pills (%)	40.4 (2985)	39.8 (2728)	38.2 (1028)	36.8 (1183)	35.3 (1739)	34.0 (286)	38.4 (9 949)	38.3 (924)	81.2 (1958)

*Cigar, pipe, or number of cigarettes consumed per day.
Some values do not agree with denominators due primarily to missing data.

Table 4 Percentage of cohort exposed to three measures of environmental tobacco smoke in 1999 by smoking status of spouse among 1959 never smokers who responded to 1999 follow up questionnaire. Subgroup of 1959 never smokers aged ≥50 years at entry (born before 1910) also shown. Values are percentage (number) exposed to environmental tobacco smoke in 1999, except for data on marital status

			Regular exposure to cigarette smoke from others in work or daily life			Married only once as of 1999	
Smoking status of spouse in 1959	Lived with smoker	Lived with smoking spouse	None	Light	Moderate or heavy	Current	Ever
1959 male never smokers							
Never (n=496)	24.0 (115)	3.8 (18)	43.5 (189)	34.5 (150)	22.1 (96)	66.2 (319)	82.2 (398)
Former (n=59)	53.5 (31)	27.6 (16)	20.8 (11)	43.4 (23)	35.9 (19)	62.5 (35)	78.6 (44)
Current (n=124)	89.5 (111)	75.0 (93)	23.1 (27)	38.5 (45)	38.5 (45)	45.1 (55)	70.5 (86)
1959 female never smokers							
Never (n=788)	32.5 (253)	3.7 (29)	61.7 (398)	24.3 (157)	14.0 (90)	39.5 (306)	89.2 (686)
Former (n=573)	73.6 (421)	55.2 (316)	41.3 (196)	26.5 (126)	32.2 (153)	32.6 (187)	84.0 (474)
Current:							
Pipe or cigar (n=252)	84.7 (211)	69.9 (174)	34.0 (73)	30.2 (65)	35.8 (77)	30.1 (75)	82.2 (198)
1-19 cigarettes/day (n=233)	93.0 (212)	83.3 (190)	25.5 (53)	28.8 (60)	45.7 (95)	22.0 (50)	80.4 (180)
20-39 cigarettes/day (n=479)	98.7 (467)	91.1 (431)	19.7 (84)	20.9 (89)	59.4 (253)	16.4 (78)	78.5 (365)
≥40 cigarettes/day (n=87)	98.8 (84)	83.5 (71)	16.2 (13)	12.5 (10)	71.3 (57)	14.8 (13)	73.9 (65)
Total of current smokers (1051)	94.1 (974)	83.7 (866)	24.0 (223)	24.1 (224)	51.9 (482)	20.8 (216)	79.4 (808)
1959 male never smokers aged ≥50 years at enrolment							
Never (n=94)	11.5 (10)	2.3 (2)	58.2 (46)	24.1 (19)	17.7 (12)	47.8 (43)	80.0 (72)
Former (n=11)	36.4 (4)	18.2 (2)	50.0 (5)	20.0 (2)	30.0 (3)	45.5 (5)	90.9 (10)
Current (n=17)	88.2 (15)	70.6 (12)	18.8 (3)	43.7 (7)	37.5 (6)	12.5 (2)	56.3 (9)
1959 female never smokers aged ≥50 years at enrolment							
Never (n=100)	26.0 (26)	4.0 (4)	71.2 (52)	21.9 (16)	6.9 (5)	16.3 (16)	92.7 (89)
Former (n=99)	83.0 (78)	68.1 (64)	40.7 (33)	24.7 (20)	34.6 (28)	17.2 (17)	80.4 (78)
Current:							
Pipe or cigar (n=43)	71.4 (30)	59.5 (25)	42.5 (14)	24.2 (8)	33.3 (11)	14.0 (6)	77.8 (28)
1-19 cigarettes/day (n=29)	96.3 (26)	85.2 (23)	20.0 (5)	28.0 (7)	52.0 (13)	6.9 (2)	84.6 (22)
20-39 cigarettes/day (n=75)	97.1 (72)	87.7 (67)	14.8 (7)	21.9 (13)	63.3 (43)	7.9 (6)	81.7 (58)
≥40 cigarettes/day (n=9)	100.0 (8)	75.0 (6)	0	0	100.0 (7)	11.1 (1)	88.9 (8)
Total of current smokers (n=156)	90.7 (136)	80.7 (121)	20.3 (26)	21.9 (28)	57.8 (74)	9.6 (15)	81.7 (116)

Some values do not agree with denominators due primarily to missing data.

Table 5 One measure of exposure to environmental tobacco smoke as of 1999 by smoking history of spouse in 1999 among 1959/1999 never smokers who responded to 1999 follow up questionnaire. Values are percentage (number) exposed to environmental tobacco smoke in 1999

	Regular exposure to cigarette smoke from others in work or daily life			
Ever lived with a smoking spouse	**None**	**Light**	**Moderate**	**Heavy**
1959/1999 male never smokers				
No (n=336)	50.0 (168)	33.9 (114)	14.9 (50)	1.2 (4)
Yes:				
No smoking nearby (n=23)	30.4 (7)	52.2 (12)	17.4 (4)	0
Exposed 1-19 years (n=17)	17.6 (3)	29.4 (5)	41.2 (7)	11.8 (2)
Exposed 20-39 years (n=35)	20.0 (7)	48.6 (17)	20.0 (7)	11.4 (4)
Exposed 40-80 years (n=33)	6.1 (2)	27.3 (9)	57.5 (19)	9.1 (3)
1959/1999 female never smokers				
No (n=570)	76.7 (437)	16.1 (92)	5.3 (30)	1.9 (11)
Yes:				
No smoking nearby (n=122)	36.9 (45)	36.9 (45)	23.7 (29)	2.5 (3)
Exposed 1-19 years (n=162)	29.0 (47)	38.9 (63)	27.2 (44)	4.9 (8)
Exposed 20-39 years (n=355)	19.7 (70)	24.5 (87)	44.5 (158)	11.3 (40)
Exposed 40-80 years (n=323)	14.1 (46)	20.5 (66)	44.3 (143)	21.1 (68)
1959/1999 male never smokers aged ≥50 years at enrolment				
No (n=70)	62.9 (44)	24.3 (17)	11.4 (8)	1.4 (1)
Yes:				
No smoking nearby (n=3)	33.3 (1)	33.3 (1)	33.3 (1)	0
Exposed 1-19 years (n=2)	0	50.0 (1)	0	50.0 (1)
Exposed 20-39 years (n=5)	20.0 (1)	60.0 (3)	20.0 (1)	0
Exposed 40-80 years (n=5)	20.0 (1)	0	60.0 (3)	20.0 (1)
1959/1999 female never smokers aged ≥50 years at enrolment				
No (n=73)	89.0 (65)	9.6 (7)	0	1.4 (1)
Yes:				
No smoking nearby (n=20)	25.0 (5)	60.0 (12)	10.0 (2)	5.0 (1)
Exposed 1-19 years (n=20)	55.0 (11)	40.0 (8)	5.0 (1)	0
Exposed 20-39 years (n=48)	8.3 (4)	16.7 (8)	62.5 (30)	12.5 (6)
Exposed 40-80 years (n=66)	15.2 (10)	18.2 (12)	45.4 (30)	21.2 (14)

Some values do not agree with denominators due primarily to missing data.

the adults up to September 1972 and obtained death certificates for most of those known dead.

Follow up

Long term follow up was undertaken at the University of California at Los Angeles on all 118 094 participants from California. This is described in detail elsewhere and summarised in table 1.[18] The participants were matched several times with the California death file and the social security death index on the basis of their name and other identifying variables.[18 19] Overall, 79 437 deaths were identified up to 31 December 1998, and the underlying cause was obtained from the California death file and death certificates for 93% (73 876) of these deaths.

Participants were also matched with information given on their California driver's licence, based primarily on name, date of birth, and height. We obtained the address given during the 1990s for 21 897 participants who were not known as dead as of 1999, and these participants were assumed to be alive in 1999. Of the remaining participants in the study's master database, 6845 were withdrawn from further follow up as of September 1972 because their complete name was not retained, and 9915 were lost to follow up as of 1999 because their vital status was unknown.

To assess the current status of surviving cohort members, in mid-1999 we sent out a two page questionnaire on smoking and lifestyle to those participants with an address for 1995 or later on their driver's licence. Overall, 2290 of 5275 men (43.4%) and 4869 of 10 738 women (45.3%) completed the questionnaire. Responses to name, date of birth, and height on the questionnaire confirmed that over 99% of the respondents had been accurately located.

The follow up period was from time of entry to the study (1 January to 31 March 1960) until death, withdrawal (date last known alive), or end of follow up (31 December 1998). The participants were aged 30-96 years at enrolment. We excluded the few person years of observation and the 36 deaths during 1959. The underlying cause of each death was assigned according to the international classification of diseases (seventh, eighth, or ninth revisions). Coronary heart disease was defined as 420 (ICD-7) during 1960-7, 410-4 (ICD-8) during 1968-78, and 410-4 (ICD-9) during 1979-98, lung cancer was defined as 162-3 (ICD-7), 162 (ICD-8), and 162 (ICD-9), and chronic obstructive pulmonary disease was defined as 241, 500-2, and 527.1 (ICD-7), 490-3 (ICD-8), and 490-6 (ICD-9). For the analysis of environmental tobacco smoke we selected the 35 561 participants who had never smoked as of 1959 and who had a spouse in the study with known smoking habits.

Statistical analysis

The independent variable used for analysis was exposure to environmental tobacco smoke based on smoking status of the spouse in 1959, 1965, and 1972. Never smokers married to current or former smokers were compared with never smokers married to never smokers. The 1959 never smokers were defined as those who had never smoked any form of tobacco as of 1959. The 1965 never smokers were defined as 1959 never smokers who did not smoke cigarettes as of 1965. The 1972 never smokers were defined as 1959

never smokers who did not smoke cigarettes as of 1965 and 1972. The 1959/1999 never smokers were defined as 1959 never smokers who had never smoked cigarettes as of 1999. Never smokers married to a current smoker were subdivided into categories according to the smoking status of their spouse: 1-9, 10-19, 20, 21-39, ≥40 cigarettes consumed per day for men and women, with the addition of pipe or cigar usage for women. Former smokers were considered as an additional category.

We calculated the age adjusted relative risk of death and 95% confidence interval as a function of smoking status of the spouse by using Cox proportional hazards regression.[19 20] A fully adjusted relative risk was calculated by using a model that included age and seven potential confounders at baseline: race (white, non-white), education level (< 12, 12, > 12 years), exercise (none or slight, moderate, heavy), body mass index (< 20, 20-22.99, 23-25.99, 26-29.99, ≥ 30), urbanisation (five population sizes), fruit or fruit juice intake (0-2, 3-4, 5-7 days a week), and health status (good, fair, poor, sick). Analyses were carried out for all participants and for healthy participants (those with no history of cancer, heart disease, or stroke at baseline). The relative risk was also calculated for current cigarette smokers (cigarettes only) as a function of number of cigarettes consumed per day for the entire cohort.[16] For reference, the age adjusted death rate has been calculated by cause of death for all never smokers.[18]

Results

The personal and lifestyle characteristics and follow up status for 1959 never smokers were relatively independent of their spouse's smoking status (tables 2 and 3). Also, the baseline characteristics of the 1999 respondents in 1959 were similar to those for all participants in 1959, except for a younger age at enrolment. Although heavily censored by age, the 1999 respondents seemed reasonably representative of survivors. Race, education, exercise, height, weight, and fruit intake had also remained largely unchanged among the 1999 respondents since 1959. The proportion of participants who had withdrawn as of 1972, were lost as of 1999, or had an unknown cause of death was not related to the smoking status of spouses. However, widowhood (widowed as of 1999) increased substantially with the level of smoking in the spouse.

The smoking status of spouses as of 1959 was related to three self reported measures of exposure to environmental tobacco smoke as of 1999 (table 4). Particularly for women, there was a clear relation between smoking status of spouses as of 1959 and self reported measures in 1999 of having lived with a smoker, having lived with a smoking spouse, and a positive answer to the question "In your work or daily life, are (were) you regularly exposed to cigarette smoke from others?" Also, the percentage of participants currently married as of 1999 declined substantially with the smoking status of the spouse, owing to increased widowhood.

Table 6 Percentage of current smokers by cigarettes consumed per day as of 1965, 1972, and 1999, and former smokers by year of cessation as of 1999 among 1959 never smokers by smoking status of spouse. Values are percentages (numbers) of cigarette smokers

	Current smoking as of 1965 (cigarettes/day)		Current smoking as of 1972 (cigarettes/day)		Cigarette smoking as of 1999		
1959 spousal smoking	**1-9**	**≥10**	**1-9**	**≥10**	**Current**	**Former (quit <1960)**	**Former (quit ≥1960)**
1959 male never smokers							
	(n=8 602)		(n=5 479)			(n=679)	
Never	0.3 (16)	0.8 (36)	0.2 (5)	0.2 (8)	0.2 (1)	5.2 (24)	0.7 (3)
Former	0.4 (2)	1.2 (6)	0	0.8 (2)	0	15.3 (6)	0
Current	0.7 (8)	2.0 (25)	0.3 (3)	0.5 (4)	0	6.5 (8)	1.6 (2)
1959 female never smokers							
	(n=24 112)		(n=16 237)			(n=2 412)	
Never	0.3 (16)	0.4 (19)	0.3 (9)	0.4 (12)	0.3 (2)	2.8 (16)	1.4 (8)
Former	0.5 (24)	0.4 (25)	0.2 (9)	0.3 (9)	0.2 (1)	5.0 (22)	0.9 (4)
Current:							
Pipe or cigar	0.6 (15)	0.4 (9)	0.6 (7)	0.4 (4)	0.4 (1)	1.8 (3)	1.8 (3)
1-19 cigarettes/day	0.8 (21)	0.9 (22)	0.6 (9)	0.5 (7)	0	1.7 (4)	2.2 (5)
20-39 cigarettes/day	1.0 (41)	1.2 (52)	0.5 (13)	0.6 (15)	0.2 (1)	1.4 (6)	1.7 (7)
≥40 cigarettes/day	1.4 (10)	1.6 (11)	0.6 (3)	0.2 (1)	1.1 (1)	6.4 (5)	3.9 (3)
Total of current smokers	0.9 (87)	0.9 (94)	0.6 (32)	0.5 (27)	0.3 (3)	2.0 (18)	2.0 (18)
1959 male never smokers aged ≥50 years at enrolment							
	(n=5 521)		(n=3 306)			(n=122)	
Never	0.3 (10)	0.8 (23)	0.1 (1)	0.2 (4)	0	5.3 (5)	0
Former	0	1.4 (4)	0	0	0	9.1 (1)	0
Current	0.6 (4)	2.5 (16)	0	0.6 (3)	0	11.8 (2)	0
1959 female never smokers aged ≥50 years at enrolment							
	(n=14 014)		(n=8 957)			(n=355)	
Never	0.2 (6)	0.3 (6)	0.3 (4)	0.3 (4)	0	1.0 (1)	0
Former	0.1 (4)	0.5 (13)	0.2 (4)	0	0	7.1 (7)	0
Current:							
Pipe or cigar	0.2 (3)	0.2 (4)	0.4 (1)	0	0	2.3 (1)	0
1-19 cigarettes/day	0.4 (5)	0.8 (12)	0.3 (2)	0.6 (4)	0	3.4 (1)	0
20-39 cigarettes/day	0.7 (14)	0.9 (20)	0.5 (5)	0.5 (4)	1.3 (1)	0	2.7 (2)
≥40 cigarettes/day	0.6 (2)	1.6 (5)	0.8 (2)	0	0	0	0
Total of current smokers	0.4 (24)	0.8 (41)	0.4 (10)	0.4 (8)	0.6 (1)	1.3 (2)	1.3 (2)

Some values do not agree with denominators due primarily to missing data.

Table 7 Level of smoking in spouse and deaths from selected causes among male never smokers in California cancer prevention study (CPS I) cohort, as of 1959 and 1972. Relative risk (95% confidence interval) comparing individuals with each level of exposure to those without exposure. Proportional hazards linear models adjusted for age and for age and seven confounders. For reference, 1960-98 death rate in deaths per 1000 person years adjusted to 1960 US population for attained ages 35-84 is given[18]

	All 1959 participants, followed 1960-98			1959 participants aged ≥50, followed 1960-98		Participants defined in 1972, followed 1973-98	
Smoking in spouse and cause of death	No of deaths/No of participants	Age adjusted relative risk (95% CI)	Fully adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)
Coronary heart disease (death rate 3.05/1000)							
Never (1)*	1860/7458	1.00	1.00	1534/5201	1.00	806/3404	1.00
Former (2)*	126/624	0.94 (0.78 to 1.12)	0.94 (0.77 to 1.14)	83/323	0.93 (0.74 to 1.16)	114/573	0.94 (0.77 to 1.14)
Current (cigarettes/day):							
1-9 (3)*	81/392	0.97 (0.78 to 1.21)	0.98 (0.78 to 1.24)	59/230	1.00 (0.77 to 1.30)	20/89	1.32 (0.84 to 2.06)
10-19 (4)*	99/513	0.86 (0.70 to 1.05)	0.82 (0.66 to 1.02)	73/282	0.91 (0.72 to 1.15)	33/153	1.02 (0.72 to 1.45)
20 (5)*	81/458	0.92 (0.74 to 1.15)	0.89 (0.70 to 1.13)	58/245	1.02 (0.78 to 1.32)	35/189	0.94 (0.67 to 1.32)
21-39 (6)*	27/129	1.16 (0.79 to 1.69)	1.13 (0.76 to 1.68)	19/62	1.30 (0.82 to 2.04)	14/58	1.20 (0.70 to 2.03)
≥40 (7)*	13/45	1.29 (0.75 to 2.22)	1.24 (0.70 to 2.19)	9/26	1.25 (0.65 to 2.41)	4/36	0.65 (0.24 to 1.73)
Total of current smokers	301/1537	0.94 (0.83 to 1.07)	0.92 (0.80 to 1.05)	218/845	1.00 (0.87 to 1.15)	106/525	1.04 (0.85 to 1.27)
Ever	427/2161	0.94 (0.85 to 1.05)	0.93 (0.83 to 1.04)	301/1168	0.98 (0.86 to 1.11)	220/1098	0.99 (0.85 to 1.15)
7 level index	2287/9619	0.99 (0.95 to 1.02)	0.98 (0.94 to 1.02)	1835/6369	1.00 (0.96 to 1.05)	1026/4502	1.00 (0.95 to 1.05)
Lung cancer (death rate 0.11/1000)							
Never	65	1.00	1.00	50	1.00	27	1.00
Former	5	0.92 (0.37 to 2.30)	0.82 (0.29 to 2.26)	3	0.89 (0.28 to 2.88)	3	0.63 (0.19 to 2.09)
Current	9	0.69 (0.34 to 1.39)	0.57 (0.26 to 1.26)	5	0.60 (0.24 to 1.52)	1	0.23 (0.03 to 1.68)
Ever	14	0.75 (0.42 to 1.35)	0.63 (0.33 to 1.22)	8	0.69 (0.32 to 1.46)	4	0.43 (0.15 to 1.24)
7 level index	79	0.94 (0.77 to 1.14)	0.88 (0.70 to 1.10)	58	0.91 (0.71 to 1.17)	31	0.68 (0.41 to 1.13)
Chronic obstructive pulmonary disease (death rate 0.12/1000)							
Never	69	1.00	1.00	59	1.00	30	1.00
Former	5	0.95 (0.38 to 2.37)	1.00 (0.40 to 2.50)	4	1.09 (0.40 to 3.02)	4	0.88 (0.31 to 2.50)
Current	17	1.40 (0.82 to 2.40)	1.28 (0.72 to 2.27)	13	1.51 (0.82 to 2.78)	7	1.80 (0.78 to 4.17)
Ever	22	1.27 (0.78 to 2.08)	1.20 (0.72 to 2.00)	17	1.39 (0.81 to 2.41)	11	1.29 (0.64 to 2.61)
7 level index	91	1.06 (0.91 to 1.25)	1.05 (0.88 to 1.24)	76	1.09 (0.91 to 1.30)	41	1.08 (0.86 to 1.38)

*Values in parentheses are index level of environmental tobacco smoke.

Smoking history of the spouse as assessed in 1999 was strongly related to exposure to environmental tobacco smoke as of 1999 for both men and women (table 5).

Misclassification of exposure and smoking status

Although there was substantial misclassification of environmental tobacco smoke exposure status from 1959 to 1999, it was less for those never smokers aged 50 or over at enrolment (see table 4), never smokers defined in 1972 (data not shown), and never smokers defined in 1999 (see table 5). Misclassification of exposure status produces a measured relative risk that is closer to 1.0 than the true relative risk.[8 13] The extent of misclassification from 1959 to 1999 could not obscure a true association with a relative risk of about 1.3, if it exists, among women, but it could largely obscure this association among men. However, this level of misclassification, which is based on the changes that occurred over 40 years among the younger than average 1999 respondents, exaggerates the true level of misclassification that occurred among the cohort as a whole, particularly during short follow up periods.

Essentially all 1959 never smokers remained never smokers on the basis of smoking status reported in 1965, 1972, and 1999 (table 6). Of those who reported a history of smoking in 1999, most had smoked no more than 10 cigarettes per day for a few years, and most had quit smoking before 1960. This indicates only a small degree of misclassification of smoking status. Some bias exists in the misclassification of smoking status among the 1959 never smokers, because the percentage who smoked in the 1965 and 1972 surveys was greatest among those with the highest levels of smoking in spouses. This bias produces a measured relative risk that is greater than the true relative risk, but by a negligible amount for this level of bias.[8 13]

Effect of exposure to environmental tobacco smoke

Exposure to environmental tobacco smoke was not significantly associated with the death rate for coronary heart disease, lung cancer, or chronic obstructive pulmonary disease in men or women (tables 7 and 8). This was true for all 1959 never smokers and 1959 never smokers aged 50 or over at enrolment followed during 1960-98 and for 1972 never smokers followed during 1973-98. The relative risks were slightly reduced after adjustment for seven confounders. Results were essentially unchanged among the healthy participants only (data not shown). The relative risks were consistent with 1.0 for virtually every level of exposure to environmental tobacco smoke, current or former. Only the relative risks for chronic obstructive pulmonary disease suggested an association. An environmental tobacco smoke index based on seven or eight levels of smoking in a spouse yielded a relative risk of about 1.0 for each level of change and no suggestion of a dose-response trend.

In addition, analyses for coronary heart disease were performed for three short follow up periods with presumably smaller misclassification errors. All relative risks for coronary heart disease were consistent with 1.0 for the follow up periods of 1960-5, 1966-72, and 1973-85 for never smokers defined as of 1959, 1965, and 1972 (table 9). In particular, the relative risk for current smoking in a spouse was not increased, and

there were no trends based on the environmental tobacco smoke index.

As expected, there was a strong, positive dose-response relation between active cigarette smoking and deaths from coronary heart disease, lung cancer, and chronic obstructive pulmonary disease during 1960-98 (table 10[10]). These relative risks were consistent with those for the full CPS I cohort until 1972.[15 17] As it is generally considered that exposure to environmental tobacco smoke is roughly equivalent to smoking one cigarette per day,[4] we extrapolated the relative risk due to exposure to environmental tobacco smoke from the relative risks for smoking 1-9 cigarettes per day. These extrapolated relative risks were about 1.03 for coronary heart disease and about 1.20 for lung cancer and chronic obstructive pulmonary disease. Based on these findings, exposure to environmental tobacco smoke could not plausibly cause a 30% increase in risk of coronary heart disease in this cohort, although a 20% increase in risk of lung cancer and chronic obstructive pulmonary disease could not be ruled out.

Discussion

On the basis of our findings from the long term follow up of the California cohort of the cancer prevention study (CPS I), the association between exposure to environmental tobacco smoke and coronary heart disease and lung cancer may be considerably weaker than generally believed. Although participants in CPS I are not a representative sample of the US population, never smokers in this cohort had a total death rate that was close to that of US white never smokers.[21] Furthermore, the relative risks were based on comparisons within the cohort and should be valid. Although the participants' total exposure to smoking in a spouse was affected by the substantial extent of smoking cessation since 1959,[18] this did not affect the relative comparisons. Also, the relative risks during short follow up periods, with limited cessation, were similar to the long term risks.

Strengths of study

CPS I has several important strengths: long established value as a prospective epidemiological study, large size, extensive baseline data on smoking and potential confounders, extensive follow up data, and excellent long term follow up. None of the other cohort studies on environmental tobacco smoke has more strengths, and none has presented as many detailed results. Considering these strengths as a whole, the CPS I cohort is one of the most valuable samples for studying the relation between environmental tobacco smoke and mortality.

Concern has been expressed that smoking status of the spouse as of 1959 does not accurately reflect total exposure to environmental tobacco smoke because there was so much exposure to non-residential

Table 8 Level of smoking in spouse and deaths from selected causes among female never smokers in California cancer prevention study (CPS I) cohort, as of 1959 and 1972. For reference, 1960-98 death rate in deaths per 1000 person years adjusted to 1960 US population for attained ages 35-84 is given[18]

	All 1959 participants, followed 1960-98			1959 participants aged ≥50, followed 1960-98		Participants defined in 1972, followed 1973-98	
Smoking in spouse and cause of death	No of deaths/No of participants	Age adjusted relative risk (95% CI)	Fully adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)
Coronary heart disease (death rate 1.65/1000)							
Never (1)*	1053/7399	1.00	1.00	891/4230	1.00	428/3090	1.00
Former (2)*	1059/6858	1.02 (0.93 to 1.11)	1.03 (0.94 to 1.13)	909/4424	0.98 (0.89 to 1.08)	772/5079	1.03 (0.92 to 1.16)
Current:							
Pipe or cigar (3)*	389/2691	0.99 (0.88 to 1.11)	0.97 (0.86 to 1.10)	162/1735	0.97 (0.86 to 1.10)	24/173	0.99 (0.66 to 1.49)
1-9 cigarettes/day (4)*	183/1102	1.13 (0.97 to 1.33)	1.03 (0.86 to 1.23)	162/719	1.15 (0.97 to 1.36)	24/200	0.89 (0.59 to 1.34)
10-19 cigarettes/day (5)*	310/2117	1.03 (0.91 to 1.17)	0.99 (0.86 to 1.14)	272/1301	1.03 (0.90 to 1.18)	42/344	0.90 (0.66 to 1.24)
20 cigarettes/day (6)*	412/3288	1.04 (0.92 to 1.16)	1.02 (0.90 to 1.16)	309/1735	0.96 (0.84 to 1.10)	89/616	1.30 (1.04 to 1.64)
21-39 cigarettes/day (7)*	167/1646	0.95 (0.80 to 1.12)	0.88 (0.74 to 1.06)	127/792	0.95 (0.79 to 1.15)	25/239	1.14 (0.76 to 1.71)
≥40 cigarettes/day (8)*	72/841	0.83 (0.65 to 1.06)	0.80 (0.62 to 1.03)	49/399	0.74 (0.55 to 0.98)	20/211	0.89 (0.57 to 1.40)
Total of current smokers	1533/11685	1.01 (0.93 to 1.09)	0.97 (0.89 to 1.06)	1258/6681	0.98 (0.90 to 1.07)	224/1783	1.06 (0.90 to 1.25)
Ever	2592/18543	1.01 (0.94 to 1.08)	0.99 (0.92 to 1.08)	2167/11105	0.98 (0.91 to 1.06)	996/6862	1.04 (0.93 to 1.16)
8 level index	3645/25942	1.00 (0.98 to 1.01)	0.99 (0.97 to 1.00)	3058/15335	0.99 (0.97 to 1.01)	1424/9952	1.02 (0.98 to 1.05)
Lung cancer (death rate 0.08/1000)							
Never	51	1.00	1.00	31	1.00	25	1.00
Former	51	1.08 (0.73 to 1.60)	1.04 (0.69 to 1.57)	33	1.02 (0.62 to 1.66)	39	0.92 (0.56 to 1.53)
Current	75	0.93 (0.65 to 1.33)	0.88 (0.60 to 1.28)	44	0.86 (0.54 to 1.36)	14	1.00 (0.52 to 1.92)
Ever	126	0.99 (0.72 to 1.37)	0.94 (0.66 to 1.33)	77	0.93 (0.61 to 1.41)	53	0.95 (0.59 to 1.53)
8 level index	177	0.97 (0.91 to 1.04)	0.97 (0.90 to 1.05)	108	0.98 (0.89 to 1.07)	78	0.99 (0.87 to 1.13)
Chronic obstructive pulmonary disease (death rate 0.08/1000)							
Never	45	1.00	1.00	35	1.00	21	1.00
Former	50	1.17 (0.78 to 1.75)	1.24 (0.80 to 1.93)	37	1.01 (0.64 to 1.60)	36	1.00 (0.59 to 1.72)
Current	78	1.11 (0.77 to 1.60)	1.12 (0.74 to 1.69)	54	0.94 (0.61 to 1.44)	18	1.57 (0.84 to 2.96)
Ever	128	1.13 (0.80 to 1.58)	1.16 (0.80 to 1.70)	91	0.97 (0.66 to 1.44)	54	1.14 (0.69 to 1.89)
8 level index	173	0.99 (0.92 to 1.06)	0.98 (0.91 to 1.06)	125	0.97 (0.89 to 1.06)	75	1.06 (0.94 to 1.20)

*Values in parentheses are index level of environmental tobacco smoke.

Table 9 Level of smoking in spouse and deaths from coronary heart disease among never smokers in California cancer prevention study (CPS I) cohort, as of 1959, 1965, and 1972

	1960-5		1966-72		1973-85	
Smoking in spouse	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)	No of deaths/No of participants	Age adjusted relative risk (95% CI)
Males*:						
Never	224/7458	1.00	304/6762	1.00	769/5300	1.00
Former	7/624	0.64 (0.30 to 1.35)	19/581	1.07 (0.67 to 1.71)	47/490	0.95 (0.71 to 1.28)
Current	30/1537	1.07 (0.72 to 1.57)	36/1429	0.85 (0.60 to 1.20)	120/1185	0.97 (0.80 to 1.18)
Ever	37/2161	0.94 (0.66 to 1.34)	55/2010	0.91 (0.68 to 1.21)	167/1675	0.97 (0.82 to 1.15)
7 level index†	261/9619	1.02 (0.91 to 1.15)	359/8772	0.95 (0.86 to 1.05)	936/6975	1.01 (0.95 to 1.06)
Females*:						
Never	49/7399	1.00	124/7008	1.00	408/5343	1.00
Former	63/6858	1.26 (0.87 to 1.84)	102/6432	0.83 (0.64 to 1.08)	410/4896	1.01 (0.88 to 1.15)
Current	61/11685	1.10 (0.75 to 1.62)	141/11002	0.87 (0.68 to 1.11)	565/8433	1.02 (0.90 to 1.16)
Ever	124/18543	1.16 (0.83 to 1.61)	243/17434	0.85 (0.68 to 1.06)	975/13323	1.02 (0.90 to 1.14)
8 level index†	173/25942	1.01 (0.93 to 1.10)	367/24442	0.98 (0.92 to 1.03)	1393/18666	1.00 (0.98 to 1.03)
Males‡:						
Never			271/6173	1.00	453/3404	1.00
Former			20/726	0.87 (0.55 to 1.37)	56/573	0.93 (0.70 to 1.23)
Current			26/1053	0.79 (0.53 to 1.19)	48/525	1.00 (0.74 to 1.35)
Ever			46/1779	0.82 (0.60 to 1.13)	104/1098	0.96 (0.77 to 1.20)
7 level index†			317/8265	0.97 (0.87 to 1.08)	557/4502	1.00 (0.93 to 1.09)
Undefined after 1959			42/726		379/2473	
Females‡:						
Never			92/6138	1.00	180/3090	1.00
Former			112/9042	0.81 (0.62 to 1.07)	287/5079	0.92 (0.76 to 1.11)
Current			62/5660	0.98 (0.70 to 1.36)	81/1783	1.02 (0.78 to 1.33)
Ever			174/14702	0.86 (0.67 to 1.11)	368/6862	0.94 (0.79 to 1.13)
8 level index†			266/20840	1.00 (0.94 to 1.07)	548/9952	1.03 (0.97 to 1.09)
Undefined after 1959			101/3602		845/8714	

*Smoking in spouse defined by 1959 questionnaire.
†Index of environmental tobacco smoke based on seven or eight levels of smoking in spouse.
‡Smoking in spouse defined by 1965 questionnaire for 1966-72 and by 1972 questionnaire for 1973-85.

environmental tobacco smoke at that time.[6] The 1999 questionnaire showed that the smoking status of spouses was directly related to a history of total exposure to environmental tobacco smoke. It also showed that the extent of misclassification of exposure was not sufficient to obscure a true association between environmental tobacco smoke and coronary heart disease among women (see tables 4 and 5).

Our methodology and results are fully described because of concern that the earlier analysis of coronary heart disease in CPS I [10] was flawed by author bias owing to funding by the tobacco industry.[4] Our results for coronary heart disease and lung cancer are consistent with those of most of the other individual studies on environmental tobacco smoke,[4-8] including the results for coronary heart disease and lung cancer in the full CPS I.[10 16] Moreover, when our results are included in a meta-analysis of all results for coronary heart disease, the summary relative risks for current and ever exposure to environmental tobacco smoke are reduced to about 1.05, indicating a weak relation.

Widowhood was strongly correlated with smoking status of spouses, owing to the reduced survival of smokers. Since widowers have higher death rates than married people,[22 23] controlling for widowhood would be expected to reduce the relative risks in this and other studies of smoking in spouses. The precise effect of widowhood due to smoking in spouses still needs to be determined, but it may partially explain the positive relative risks found in other cohorts.

Conclusion

The results of the California CPS I cohort do not support a causal relation between exposure to environ-

What is already known on this topic

Exposure to environmental tobacco smoke is generally believed to increase the risk of coronary heart disease and lung cancer among never smokers by about 25%

This increased risk, based primarily on meta-analysis, is still controversial due to methodological problems

What this study adds

In a large study of Californians followed for 40 years, environmental tobacco smoke was not associated with coronary heart disease or lung cancer mortality at any level of exposure

These findings suggest that the effects of environmental tobacco smoke, particularly for coronary heart disease, are considerably smaller than generally believed

Active cigarette smoking was confirmed as a strong, dose related risk factor for coronary heart disease, lung cancer, and chronic obstructive pulmonary disease

Table 10 1960-98 age adjusted relative risk (95% confidence interval) of death for coronary heart disease, lung cancer, and chronic obstructive pulmonary disease among cigarette smokers compared with never smokers as function of active smoking status (cigarettes per day) in 1959

	Men		Women	
Active smoking status	**No of deaths/No of participants**	**Age adjusted relative risk (95% CI)**	**No of deaths/No of participants**	**Age adjusted relative risk (95% CI)**
Coronary heart disease				
Never (1)*	2561/10862	1.00	6516/39216	1.00
Former (2)*	2579/10204	1.18 (1.12 to 1.25)	541/4838	0.98 (0.90 to 1.07)
Current (cigarettes/day):				
1-9 (3)*	376/1548	1.19 (1.07 to 1.33)	590/4687	1.13 (1.04 to 1.23)
10-19 (4)*	859/3740	1.42 (1.31 to 1.53)	855/6691	1.43 (1.33 to 1.54)
20 (5)*	1661/7186	1.57 (1.48 to 1.68)	912/6875	1.79 (1.66 to 1.92)
21-39 (6)*	1072/4789	1.75 (1.63 to 1.89)	254/2066	2.04 (1.80 to 2.32)
40-80 (7)*	573/2621	1.91 (1.74 to 2.10)	111/818	2.38 (1.97 to 2.87)
Total of current smokers	4541/19884	1.53 (1.45 to 1.61)	2722/21137	1.49 (1.42 to 1.56)
7 level index	9681/40950	1.11 (1.10 to 1.12)	9804/65191	1.14 (1.13 to 1.16)
Lung cancer				
Never (1)	92/10862	1.00	269/39216	1.00
Former (2)	281/10204	3.50 (2.77 to 4.43)	48/4838	1.45 (1.06 to 1.97)
Current (cigarettes/day):				
1-9 (3)	47/1548	4.08 (2.87 to 5.80)	62/4687	1.98 (1.50 to 2.62)
10-19 (4)	187/3740	7.86 (6.11 to 10.11)	205/6691	5.07 (4.19 to 6.12)
20 (5)	535/7186	12.50 (9.99 to 15.63)	355/6875	9.14 (7.73 to 10.81)
21-39 (6)	424/4789	16.43 (12.99 to 20.77)	162/2066	15.14 (12.26 to 18.69)
40-80 (7)	241/2621	18.65 (14.47 to 24.02)	62/818	15.77 (11.80 to 21.06)
Total of current smokers	1434/19884	11.91 (9.64 to 14.73)	846/21137	6.22 (5.39 to 7.16)
7 level index	1807/40950	1.54 (1.50 to 1.58)	1163/65191	1.69 (1.63 to 1.74)
Chronic obstructive pulmonary disease				
Never (1)	103/10862	1.00	296/39216	1.00
Former (2)	179/10204	2.06 (1.62 to 2.63)	48/4838	1.42 (1.05 to 1.94)
Current (cigarettes/day)				
1-9 (3)	35/1548	2.84 (1.94 to 4.17)	50/4687	1.64 (1.21 to 2.22)
10-19 (4)	125/3740	5.46 (4.19 to 7.11)	214/6691	5.69 (4.73 to 6.85)
20 (5)	326/7186	8.30 (6.62 to 10.40)	309/6875	9.32 (7.85 to 11.06)
21-39 (6)	258/4789	11.99 (9.39 to 15.31)	106/2066	12.87 (10.13 to 16.35)
40-80 (7)	148/2621	13.54 (10.33 to 17.75)	46/818	15.33 (11.06 to 21.23)
Total of current smokers	892/19884	8.08 (6.58 to 9.94)	725/21137	5.98 (5.19 to 6.89)
7 level index	1174/40950	1.55 (1.51 to 1.60)	1069/65191	1.67 (1.62 to 1.73)

*Values in parentheses are index level of active cigarette smoking.

mental tobacco smoke and tobacco related mortality, although they do not rule out a small effect. Given the limitations of the underlying data in this and the other studies of environmental tobacco smoke and the small size of the risk, it seems premature to conclude that environmental tobacco smoke causes death from coronary heart disease and lung cancer.

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Contributors: JEE conceived the study and obtained funding, conducted the extended follow up, analysed the data, and drafted the manuscript; he will act as guarantor for the paper. GCK contributed to the follow up questionnaire, advised on the data analysis and interpretation, and contributed extensively to the manuscript.

Funding: The American Cancer Society initiated CPS I in 1959, conducted follow up until 1972, and has maintained the original database. Extended follow up until 1997 was conducted at the University of California at Los Angeles with initial support from the Tobacco-Related Disease Research Program, a University of California research organisation funded by the Proposition 99 cigarette surtax (www.ucop.edu/srphome/trdrp). After continuing support from the Tobacco-Related Disease Research Program was denied, follow up through 1999 and data analysis were conducted at University of California at Los Angeles with support from the Center for Indoor Air Research, a 1988-99 research organisation that received funding primarily from US tobacco companies.[24]

Competing interests: In recent years JEE has received funds originating from the tobacco industry for his tobacco related epidemiological research because it has been impossible for him to obtain equivalent funds from other sources. GCK never received funds originating from the tobacco industry until last year, when he conducted an epidemiological review for a law firm which has several tobacco companies as clients. He has served as a consultant to the University of California at Los Angeles for this paper. JEE and GCK have no other competing interests. They are both lifelong non-smokers whose primary interest is an accurate determination of the health effects of tobacco.

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TOXIC TOXICOLOGY
PLACING SCIENTIFIC CREDIBILITY AT RISK

Review procedures and listing criteria used in the preparation of the DHHS Report on Carcinogens (RoC)

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Washington, D.C.

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Independent assessment and remarks

Littlewood & Fennell is an independent public and health policy research group, with no ties whatsoever to industry or any government agency. I am here today on my own time and at my own expense to address the clear possibility that the National Toxicology Program has actively undermined the process by which risk assessments should be conducted. NTP overlooked a substantial body of evidence showing uncertainty, vagueness, and lack of statistical support of what is and is not carcinogenic. In addition, NTP conducted its assessments in a manner reminiscent of a rubber stamp proceeding, which favored politics over science.

I have included a history of our involvement with the NTP carcinogen listing process as an addendum to this paper. Briefly, we became interested in the topic of environmental tobacco smoke (or ETS) during an ongoing study of increasing rates of asthma in the U.S. Because a review of the literature indicates a negative correlation between ETS and asthma, and because ETS is physically and chemically quite different from mainstream tobacco smoke, we were curious about NTP's decision to list ETS as a carcinogen. We requested background materials from them in order to review this listing process. It was during this review that we unearthed a number of gross scientific improprieties in both procedure and conduct by a number of federal agencies. NTP is simply the latest chapter in the same sad story.

Since the topic of today's meeting is NTP's review process and procedure for listing substances as human carcinogens, I will limit most of my discussion to these matters.

There are three areas I believe are critical to the process of listing human carcinogens, none of which are addressed by NTP:

One: Inclusion of a reasonably expected real-life exposure to toxic or carcinogenic substances. Exaggerated estimates of risk can themselves be toxic; inasmuch as these exaggerated estimates of risk create confusion, misunderstanding, anxiety and, inevitably, utter disdain by the general public. We call this phenomenon Toxic Toxicology for, in the end, it does far more harm than good.

Two: Assessment of scientific research and comments based on substance, merit and quality

irrespective of source -- industry, academia or government. I contend that if you are unable to determine what is sound science without knowing the antecedents of the researcher then you are not competent to assess risk.

Three: Animal studies must be put in perspective. Rats and mice are not little people. Nothing could make this more clear than Dr. Robert A. Weinberg's very recent success at finally inducing malignant transformation in human cells. As Gilbert L. Ross, M.D., Medical Director of the American Council on Science and Health, noted in a letter to the New York Times this past August: Scientists induced such cancerous changes in rodents 15 years ago. The fact that it took so many years to accomplish this feat in humans illustrates how differently carcinogens affect rodents than humans. Rodents are far more susceptible to cancer induction than humans. Merely because chemicals produce tumors in rodents does not mean that humans will be harmed.

Real-life exposure levels.

Each substance proposed for listing as a human carcinogen must be subjected to a careful and unbiased process of assessment for real-life exposures.

The compilers of reports from the National Academy of Sciences, the US Surgeon General, and the Environmental Protection Agency have simply inferred the presence of ETS components by proxy, based on the composition of the highly diffuse sidestream smoke from which ETS derives. Still, even CDC concludes, ETS contains higher amounts of some of the components of cigarette smoke in general only when it is obtained in its undiluted form under laboratory conditions (CDC/DHSS 1989).

I would propose that only those chemicals present in significant amounts - perhaps 10% of the maximum tolerated dose in rodents -- be considered. Independent laboratories could then conduct personal air monitoring for these chemicals under realistic conditions, rather than in laboratory conditions designed to exaggerate exposure risk.

CDC notes that ETS is diluted in the air before it is inhaled and thus is less concentrated than MS (mainstream smoke). Further, ... on the basis of urinary cotinine concentrations, the NRC [1986] concluded that non-smokers exposed to ETS absorb the equivalent of 0.1 to 1.0 cigarettes a day. On the basis of 1985 data, NIOSH estimates that each cigarette smoker in the US smokes an average of about 21 cigarettes a day. Blood and urine samples analyzed for vapor phase nicotine indicate that nonsmokers exposed to ETS absorb about 1% of the tobacco combustion products absorbed by active smokers [NRC 1986: DHHS 1986]. If these urine and blood samples were accurate, that would indicate that, at most, ETS would account for only 0.021 cigarette over exposure to 21 cigarettes on average.

In his RoC subcommittee testimony last December, Dr. Philips reported that, based on actual personal monitoring, average ETS exposure is as little as five to six cigarettes per year. I would tend to think these figures more accurate than our extrapolations from NIOSH data.

In the case of environmental tobacco smoke, such simple and rigorous personal air monitoring would have eliminated any possibility of listing ETS as a carcinogen. Most ETS components are

far below the sensitivity of current analytical capabilities.

Of those chemicals present in ETS, only a very few can even be classified as toxins or carcinogens. Some basic physics, a bit of chemistry and a series of rather simple mathematical calculations reveal that exposure to ETS is hardly a dangerous event. Indeed, the cancer risk of ETS to a non-smoker appears to be roughly equal to the risk of becoming addicted to heroin from eating poppy seed bagels.

Calculating the non-existent risks of ETS

We have taken the substances for which measurements have actually been obtained - very few, of course, because it is diffficult to even find these chemicals in diffuse and diluted ETS. We posit a 100m3 sealed and unventilated enclosure. For those of us who are metrically challenged, that is a room approximately 20-feet square with a 9-foot ceiling clearance. Taking the figures for ETS yields per cigarette directly from EPA, we calculated the number of cigarettes that would be required to reach the lowest published threshold for each of these substances. The results are actually quite amusing. In fact, it is difficult to imagine a situation where these threshold limits could be realized.

Our chart (see Table 1) illustrates each of these substances, but let me report some notable examples. For Benzo[a]pyrene, about which we heard so much last year, 222,000 cigarettes would be required to reach the lowest published danger threshold. For Acetone, 118,000 cigarettes would be required. At the lower end of the scale - in the case of Acetaldehyde or Hydrazine, more than 14,000 smokers would need to light up simultaneously in our little room to reach a threshold limit. Toluene would require 50,000 packs of smoldering cigarettes - given 20 cigarettes per pack.

For Hydroquinone only 1,250 cigarettes are required. Perhaps we could post a notice limiting this 20-foot square room to 300 rather tightly packed people smoking no more than 62 packs per hour?

Of course, the moment we introduce real world factors to the room - a door, an open window or two, or a healthy level of mechanical air exchange - achieving these levels becomes even more implausible.

It becomes increasingly clear to us that ETS, as well as other spurious indoor substances such as asbestos and radon, are political rather than scientific scapegoats for poorly ventilated, hermetically sealed, energy-efficient buildings, with endlessly re-circulated and poorly filtered air.

Table 1

CALCULATED NUMBER OF CIGARETTES REQUIRED TO REACH A THRESHOLD LIMIT FROM ETS IN A SEALED, UNVENTILATED 100m3 ENCLOSURE AT STP

ETS Component	CAS Number	Molecular Weight	ETS Output (mg/cigarette)(2)	Threshold Limit (ppm)	Threshold Limit (mg/m3)	Cigarettes Required
2-Toluidine	(3 isomers) (3)	107.15	0.003	2	8.7	**290,000**
Acetaldehyde	75-07-0	44.05	1.26	111	180 (4)	**14,285**
Acetic acid	64-19-7	60.05	1.5	10	25	**1,666**
Acetone	67-64-1	58.05	1	500	1187	**118,700**
Benzene	71-43-2	78.11	0.24	1	3.1 (5)	**1,290**
Benzo[a]Pyrene	50-32-8	252.30	0.00009	0.02	0.2 (6)	**222,000**
Cadmium	7440-43-9	112.40	0.0007	0.002	0.01	**1,430**
Catechol	120-80-9	110.11	0.14	5	22	**15,700**
Dimethylamine	124-40-3	45.08	0.036	10 (7)	9.2	**25,555**
Formic acid	64-18-6	46.02	0.525	5 (8)	9.4	**1,790**
Hydrazine	302-01-2	32.05	0.00009	0.01	0.013	**14,444**
Hydroquinone	123-31-9	110.11	0.16	0.4	2	**1,250**
Methylamine	74-89-5	31.09	0.1	5	13	**13,000**
Methylchloride	74-87-3	50.49	0.88	50	103.0	**11,170**
Nickel	7440-02-0	58.71	0.0025	0.4	1	**40,000**
Phenol	108-95-2	94.11	0.25	5	19	**7,600**
Polonium 210 (9)		210	0.4pCi	na	3pCi/liter (10)	**750,000**
Pyridine	110-86-1	70.01	0.39	5	16	**4,100**
Toluene	108-88-3	92.13	0.000035	50	375	**1,000,000**

These calculations are not complex. They assume a 100m3 enclosed and unventilated space at Standard Temperature and Pressure. STP assumes 24.45 = molar volume of air in liters at STP conditions (25oC. and 760 torr). Conversion equations are as follow:

(TLV in ppm)(gram mol wt of substance)
(TLV in mg/m3)(24.45)
TLV in mg/m3 = TLV in ppm =
24.45
gram mol wt of substance

Assessment of scientific research and comments based on substance, merit and quality irrespective of source -- industry, academia or government.

The tobacco industry's interest in the basic science and epidemiology of ETS may be a vested interest, but their research should be judged on its own merits -- not suppressed or ignored because thee results are politically inconvenient. When scrutiny of research -- both during peer review and post-publication -- is objective and scientific it is valuable. NTP's thinly veiled hostility toward presenters finding no convincing evidence of ETS carcinogenicity is unacceptable. We found the presentations last December of varying quality, but were generally impressed with their factual and substantive nature.

As we prepared our comments earlier this year on the 9th RoC subcommittee's decision regarding ETS, we dug into the original risk assessment proceedings of the EPA. It became abundantly clear that the so-called independent scientific bodies were not independent at all.

. . . the integrity of research sponsored by governmental or other private organizations is rarely questioned. Ignoring the possibility that the granting agencies may have specific agendas for the research they sponsor, there are substantial pressures on scientists to publish and a well-known bias against publication of negative data. (Letter, JAMA, 1998; 280:1141)

Rather, these groups - Scientific Advisory Boards -- were pressured by a wide variety of political and procedural forces to cast their weight (quite reluctantly in several cases) on the side of ETS as a carcinogen.

After reviewing the NTP materials forwarded to us, as well as what source documents we could acquire during the response period, we conclude that government and institutional bias far exceeds industry bias in the issue of ETS.

Biological gradient (exposure or dose-response consistently exhibited over the range of the studies) is a critical factor in establishing cause and effect. There is no clear pattern of dose/response in the majority of epidemiological studies tracking ETS and lung cancer where quantity of exposure is measured.

We had determined that only 16.6% of the papers used in the EPA report included the odds ratios necessary to conduct a trend analysis. There was no correlation between dose increase and odds or risk increase across the range of studies. Of the 24 trend tests reported by the EPA, only 11, or 41.6% showed any evidence of upward trend - thus 58.4% of the tests for trend were non-monotonic.

Time after time we encountered actual human measurements of ETS exposure. For example: Urine cotinine measurements between ETS exposed and non-exposed women, which showed no difference. Or, in the matter of DNA adducts, personal air monitoring of carcinogenic polycyclic aromatic hydrocarbons (PAH) showed no significant difference in DNA adduct levels between non-smokers and smokers for RSPs of <2.5 microns after controlling for exposure to ETS via urine cotinine. More recently we found that ETS- subjects had levels of carbon monoxide (CO) in expired air similar to that of the non-ETS nonsmokers, and significantly lower than the

smokers ($p <= 0.05$), their actual exposure (>=4 hr/day) to ETS may not have been sufficient enough to have the adverse effect. While CO is not a carcinogen, it is an absolute bell cow for indoor air quality.

The fact that NTP's RoC subcommittee overlooked so much information is disturbing. It suggests extreme bias. We strongly urge that NTP's supervisory agency insist that future subcommittees employ the services of independent specialists, untainted by bureaucratic pressure, past or potential grant seeking or advocacy/activist status.

Animal studies must be put in perspective.

All substances are toxic in quantity. Many therapeutic medications are acutely toxic, but beneficial when used at a therapeutic level. Water, oxygen, and table salt are toxic in large enough doses. The mere presence of a substance does not imply toxicity.

The National Research Council and the USEPA have both recommended improvements in the risk assessment process that involve incorporating consideration of dose to the target tissue, mechanism of action, and biologically based dose-response models, including a possible threshold of dose below which effects will not occur.

Testing for carcinogenicity at near-toxic doses in rodents does not provide enough information to predict the excess number of human cancers that might occur at low-dose exposures. Testing at the maximum tolerated dose (MTD) frequently can cause chronic cell killing and consequent cell replacement (a risk factor for cancer that can be limited to high doses), and ignoring this effect in risk assessment greatly exaggerates the risks.

...rodent bioassays provide little information about mechanisms of carcinogenesis and low-dose risk. (Gold LS et al)

Animal studies cited during the 9th RoC process (Witschi et al, various) used injectable concentrates of carcinogens, intense concentrations of tobacco smoke and skin application of tobacco smoke condensate. Not only do these studies bear no relation to inhalation of ETS; they also use concentrations that are so high that nearly any substance in these concentrations could be expected to cause deleterious effects.

In addition, Volume II of the NTP materials included animal study results (heavily relied upon according to the transcript) using concentrations of tobacco smoke that far exceed any exposure that humans could reasonably be expected to experience. Even then, despite the intense concentrations of condensates Exposure to tobacco smoke had no effect on pulmonary tumor incidence or tumor multiplicity. Vol. II (p 47).

Animal studies can be useful in identifying gross cause and effect relationships between substances and animal cancers. They should play only a minor role in the assessment of carcinogenicity in humans.

The dose makes the poison

A basic tenet in toxicology is the dose makes the poison. Cooking food generates thousands of chemicals. There are over 1,000 chemicals reported in a cup of coffee - 19 of them are rodent carcinogens, but this does not mean that coffee is dangerous. At some level, every chemical becomes toxic, but there are levels below which no adverse health effects are observed.

In addition, cancer is largely a disease of aging. Carcinogenic effects on a short-lived species such as rodents can hardly be expected to offer realistic estimates for effects in a long-lived species such as humans. High dose animal cancer testing and exaggerated risk assessment cannot be considered measures of true risks. Data on high doses in rodents simply cannot be extrapolated to low doses in humans without information on the precise mechanism of mutagenesis or carcinogenesis.

Indeed, the carcinogenic mechanisms of tobacco smoking are not well understood. What is more, cancers once associated with smoking are being quietly removed from the official lists. Stomach cancer - which is likely caused by undetected H. pylori, is one example; cervical cancer - in which fresh evidence suggests that human papillomavirus (HPV) may be the sole cause -- should soon meet the same fate.

How many hundreds of millions of dollars are being wasted on senseless, useless - and quite possibly harmful -- Blame ETTS research? How much time and talent is being diverted from useful research into chronic infections from bacteria and viruses that are major causes of cancer worldwide? How many more absurd risk alerts will it take before the public laughs scientists out of a position of trust altogether?

Even the best of epidemiological studies conclude only a very slight and easily confounded risk for lifetime exposure to ETS. Clearly NTP is not able to distinguish a cancer risk for the occasional exposures most of us experience during our daily activities. And just as clearly NTP must incorporate truly scientific standards and procedures before its reports deserve to be given even marginal credence.

A history of our involvement

Earlier this year, we undertook a review of the National Toxicology Program's 9th Report on Carcinogens regarding Environmental Tobacco Smoke (ETS). Our interest in ETS is an outgrowth of an ongoing project involving increased asthma rates in the U.S. What we have found is an negative correlation - smoking rates and exposure to ETS havee markedly decreased while the incidence of asthma has dramatically increased.

Unconvincing background materials.

We were somewhat surprised at the background materials we received from NTP. With the exception of one study, (Bofetta et al., which showed a tiny and easily confounded 1.14 RR for lung cancer and ETS and no consistent dose-response trend) there was nothing new. The two volumes we received consisted almost exclusively of the same inadequate and tortured data used by the USEPA and CalEPA to reach the conclusion that a lifetime of exposure to ETS confers

only a statistically marginal correlation with increased risk of lung cancer. As you know, such tiny expressions of risk are easily confounded by myriad variables.

The single animal study presented, in the words of its own authors (Witschi et al), may not serve as a valid model to assess
carcinogenicity in human.

RoC meeting transcript

After expressing our disappointment with these background materials, we received a verbatim transcript of the RoC subcommittee's discussion on ETS's possible listing as a human carcinogen. This was a most illuminating document. It included several informative and sound presentations from outside researchers who had made direct air measurements of ETS exposure, analyzed a variety of animal studies and assessed the raw data from several significant studies upon which the USEPA based its decision on ETS. This complete transcript is markedly different from the publicly posted, abridged version of the meeting available at the NTP website -- which completely conceals any of the twisted reasoning and political maneuvering that actually took place and ignores nearly every salient point made by the outside presenters. I will be happy to provide a copy of the entire transcript to anyone who is interested in the contorted and illogical process by which the subcommittee reached its questionable conclusions.

Unwarranted bias

During the actual meeting, subcommittee members routinely ignored convincing, well-documented presentations by outside sources - making it abundantly clear that current or past associations with industry rendered these presentations null. Several of these same subcommittee members then proceeded to base their final vote on a rather bizarre suggestion by a self-avowed anti-smoker activist (Repace) -- who proposed that a hypothetically pure control group be used to assess exposures. This is an appalling suggestion to those of us who understand quite clearly that case and control groups should be as alike as possible except for specific exposure to the substance being studied. One subcommittee member went so far as to say that she was comforted by this suggestion since it enabled her to vote on ETS as a carcinogen despite the fact that relative risks in this for ETS were (from her perspective) quite low.

Another sub-committee member stated that he hoped when we get to diesel we will get the same generous interpretation of epidemiology enjoyed by the ETS-as-carcinogen faction. This is science? No. It is politics. And it is insupportable, unacceptable - and, quite possibly, legally actionable.

Bias and *a priori* conclusions are not sound science

In our public comments, we raised several substantive issues and found serious faults not only in the data used to reach a decision, but also with the decision making process itself. I would be pleased to provide copies of this document as well. We concluded that:

It is the result of our careful review of the NTP process regarding ETS that brings me here today

- at my own time and expense. Dr. Michael R. Fox, a nationally recognized and highly respected chemist, generously assisted me in establishing and verifying real-life exposure levels for various components of ETS. I sincerely wish he were able to join us today to express his own concerns about NTP's risk assessment process.

Inadequate ventilation, not ETS, is the danger

We find continuing evidence that energy-efficient building techniques and increasingly limited ventilation - not ETS - adversely influence asthma and other upper respiratory problems - especially in children. There is an old maxim in the engineering world: The solution to pollution is dilution. Yet building ventilation rates continue to decrease -- replaced by inadequate filtering and re-filtering systems -- and exposing and re-exposing us via re-circulated, undiluted air to contaminants strongly associated with asthma and allergies: bacteria, fungi, viruses, algae, amoebae, dust mite and cockroach feces, pollen, etc.

Since ventilation rate recommendations for buildings accommodating smokers are high enough to promote dilution of all indoor air contaminants, we have been quite careful in our assessment of risks presented by ETS. Our conclusions may be politically incorrect, but they are scientifically sound: ETS poses little if any risk to non-smokers. Indeed, it is entirely possible that buildings ventilated to a level to comfortably accommodate smokers would promote higher indoor air quality overall.

Technology, not toxic toxicology, is the solution

Rather than desperately attempting to sustain the myth that ETS is some mysteriously lethal substance, we urge an honest, open-minded look at real risk factors and real solutions for respiratory health. Rather than accepting the incremental lowering of ventilation rates for indoor air, we would insist that energy efficiency take a back seat to respiratory health.

If buildings were designed to accommodate smokers, air quality would inevitably improve for us all, since fresh air exchanges would be increased and biocontaminants such as bacteria, fungi, viruses, algae, amoebae, dust mite and cockroach feces, pollen, would be diluted. Potential toxins -- from building materials, office chemicals, cleansers, cooking, etc. could be exhausted rather than inadequately filtered and re-circulated.

NTPs Investment in Toxic Toxicology

The National Toxicology Program has become a willing and enthusiastic participant in the disturbing trend toward Toxic Toxicology. In doing so, it undermines scientific credibility and contributes to the increasing skepticism and disdain with which the public views science in general and health warnings in particular.

NTP does so by implying that even the tiniest exposure to toxic or carcinogenic substances somehow constitutes a life threatening risk. This is patently absurd - contravening a basic tenet of toxicoloogy: the dose makes the poison.

Animal cancer tests and worst-case risk assessment should not be considered true risks. And regulatory policy aimed at reducing minute exposures to rodent carcinogens confuses and unnecessarily alarms the public about what factors are truly important for preventing cancers.

Data torturing of epidemiological studies concerns the many responsible scientists and public health researchers who work honestly, openly and willingly in the service of sound science. We are seeing an enormous diversion of funds and talent from truly important health risks - and the real possibility that anxiety raised by false health scares is in itself a risk factor. Thus the term Toxic Toxicology.

It is obvious that by straining at gnats, exaggerating risks and supporting researchers who are willing to do the same, the National Toxicology Program perpetuates its own existence. There is certainly nothing new about this type of metastasizing bureaucracy and empire building. What is new, is the public's increasing tendency to ignore this sound-bite science. Warning labels have become subjects of comics and late-night talk show hosts. Editorial cartoons poke fun at the Health Scare of the Day. Yet irresponsible risk assessment continues to be a growth industry. It is time to call a halt.

The financial and social costs of biased risk assessment are receiving increased scrutiny at both the Federal and State level. The Open Science Freedom of Information Act - making data from Federally funded studies available for independent review - is a good start. It should be followed immediately by GAO investigation of the shoddy standards and political pressures that have undermined the integrity of our nations health research programs.
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http://ehis.niehs.nih.gov/roc/toc9.html Ninth Report of Carcinogens

PH 5/5/11 RES SMOKING - COMMON AREAS

AM
CC

the Fairways
SOUTH AT LEISURE WORLD

RECEIVED
MONTGOMERY COUNTY
COUNCIL

2011 JUN -8 AM 9:51

June 3, 2011

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County Council for

Montgomery County, Maryland

Dear Council Members,

I am writing on behalf of the Board of Directors of Fairways South, a condominium located within Leisure World. The members of the board are not supportive of the proposed county regulation regarding smoking in common areas and outside of multi-unit residences and in playground areas. Their reasoning is that this can and should be handled at the residence itself by developing regulations and rules for residents in the building. Further there was some concern regarding the enforcement provision of the proposed regulation. It relies on witnesses reporting to infractions to the county in order for a citation to be issued. Members of the board felt that residents who might be reluctant to put their complaints in writing to the building management would be even more reluctant to notify the county of a violation.

Sincerely,

Barbara Cronin

Barbara Cronin, President

Fairways South Board of Directors

The Fairways South at Leisure World a condominium
3330 N. Leisure World Blvd. Silver Spring, MD 20906
301-598-1540

Harriet Hershman
2800 Clear Shot Dr., #11
Silver Spring, MD 20906
(301) 438-2219

May 5, 2011

The Honorable George Leventhal
100 Maryland Avenue
Rockville, MD 20850

RE: Enactment of a no smoking bill for multi-dwelling housing units, such as condominiums and rental apartments

Dear Councilman Leventhal,

I am writing to you in regard to an on going problem I have had with second hand smoke venting into my condo unit from another unit in our building. This was not an issue until the heavy chain smoker moved into his unit on the first floor on November 25, 2009. The smoke vents into my unit on the third floor, as well as my neighbors, on the second floor.

We have tried to resolve this matter with the unit owner through mediation, which he would not do, and he is now in violation of a cease and desist order with the homeowners association. Not only is it a nuisance, but it is a health hazard as well.

My neighbors have asthma, and have had to use inhalers on a regular basis to manage their asthma, as a result of the smoke. It has also impacted my health, as I have had bronchitis several times breathing this smoke on a constant basis.

A bio-physicist measured levels of smoke in our units with a Hammond passive nicotine monitor which was sent for analysis, and the report concluded that there were lethal levels of smoking venting into our units.

This situation has escalated to the point of having to hire legal counsel to represent us in court, as the unit owner will not comply with the rules and regulations of the homeowners association. I seriously doubt I would be able to sell or rent my condo as a result of the smoke. I've often thought of just packing up a truck and letting it go into foreclosure, rather than live here another day. I'm really at the end of my rope with this.

Since there are no laws in place for cigarette smoke in multi-dwelling units, I would like for you to enact a bill to change the law, as there are no safe levels of cigarette smoke for people to breathe.

Thank you for your help in this matter.

Sincerely Yours,

Harriet Hershman

Harriet Hershman

Testimony of Anne Marie Martinez

I thank you so much for introducing and sponsoring this Board of Health Regulation.

I would be there May 5, to testify if I could. However, I had major cancer surgery on April 7, and when I came back from my first post-op appointment, I walked into a lobby, hall and entrance to our unit filled with smoke. I have had bronchitis ever since, and ran a fever for several days after and there is a possibility I have pneumonia. This was not the first nor has it been the last experience with the entrance, hallways, and individual hall filled with smoke. It is an ongoing occurrence.

I have terrible allergies, asthma, chronic bronchitis, and COPD. The main trigger to my allergies is smoke. The main trigger to my asthma is smoke. For years I had been prohibited to go many places because of smoke. Montgomery County really took a courageous stand when they passed the smoke free legislation in public places. Finally, I was able to eat out! I was able to go somewhere and be entertained.

However, we moved to a retirement community, recently. We cannot open our windows, to let fresh air in. We cannot open our patio doors, and let fresh air in. We cannot use our patio. Our hallways/walkways in our building, are only about 5 feet wide. Our bedroom windows open to this walkway. These walkways are the only way we can enter and leave our unit. When smoking is taken place in our walkway, it goes into our small hall to our unit, and is trapped there. The walkway is open at both ends, and acts as a wind tunnel. Therefore the smoke just fills up the walkway, and the small enclosed entrances to each condo.

If the door is open to our entrance, which is our lobby, where we have to get our mail, use the elevator, and take out our trash and recycles, that too is smoke filled.

On a lovely crisp day,(before we knew of the secondhand smoke hazard), we opened our windows in our bedrooms to air them out. We closed the doors to both bedrooms. When we went back in, about an hour later, we were hit with smoke. Both of our bedrooms were smoke filled. Everything had to be washed down, cleaned, the bed had to be stripped, and pillows thrown away. We have a dressing room, and bathroom/shower off of our bedroom so on that lovely crisp day, our entire dressing area, and our bathroom were also filled with smoke. Our closets were closed but many pieces of clothing smelled of smoke, since the closet doors are not sealed tight nor should they be. Obviously we did this "airing out" before we knew that second hand smoke was even present anywhere near us.

We are now prisoners in our home. Not being able to open any windows. Not being able to open our patio doors, or use our patio. We cannot go outside of our patio and take a walk. And, we also have to be very careful to use the walkway ourselves, even to go visit a neighbor. All of us know what smoking does to us. Second hand smoke is toxic. This is common knowledge.

Again, thank you, Council Member Leventhal for doing all of this work to bring this regulation forward. Everyone in Montgomery County is entitled to be able to enjoy the common areas that each and everyone of us pay for. Our health should not be put into harms way, because of smoking. Common areas are for the common good.

Most sincerely,

Anne Marie Martinez
3510 Forest Edge Drive., 1-D
Silver Spring, MD 20906

Guthrie, Lynn

From: Rich Reis [rreis@verizon.net]
Sent: Thursday, April 28, 2011 9:48 AM
To: Montgomery County Council
Subject: Ban Smoking in common areas and grounds of Multifamily Units

Dear Council Members,

As the owner of small apartment houses, I totally support the move to ban smoking in all common areas and on the grounds of multi-family residences. Recently, a tenant contacted me about finding cigarette butts on the shared porch of my property. I found that a guest of another tenant smoked there and just left his butts on the porch, with burn marks.

Having a county law or regulation would bolster my efforts to prevent smoking anywhere on the property. This would benefit the health and safety of all residents and would contribute to a cleaner facility for me.

Sincerely yours,

Richard Reis
711 Copley Lane
Silver Spring, MD 20904-1312

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Montgomery County Council Public Hearing May 5, 2011

Dear Councilmembers,

Thank you for the opportunity to testify on Councilmember Leventhal's proposed resolution to adopt a Board of Health Regulation prohibiting smoking in certain common areas of certain multi-family residential dwellings and playground areas.

My name is Rita Turner, and I am Deputy Director of the Center for Tobacco Regulation at the University of Maryland School of Law. The Center provides technical legal assistance to state and local officials, and Maryland residents who are interested in protecting themselves or those they serve from the harmful effects of tobacco.

Jurisdictions around the country are implementing regulations such as this to protect the health of their constituents. This regulation is consistent with state and federal law. There is no constitutional right to smoke, and smokers are not a protected class. Therefore, regulations restricting where people may smoke are not discriminatory. If challenged in a court of law, a this regulation would be subjected to "rational basis review" which is the lowest judicial standard of review. Rational basis review only requires that the law or regulation be rationally related to a legitimate government interest. This regulation demonstrates a legitimate government interest in protecting the public health from the well-known harmful effects of second-hand smoke, with reduction in involuntary second-hand smoke exposure as the rational means to the end.

Additionally, this regulation is completely consistent with the Clean Indoor Air Act, which bans smoking in Maryland workplaces. In fact, the Clean Indoor Air Act has been enforced by the Maryland Department of Health and Mental Hygiene's Infectious Disease and Environmental Health Administration as inclusive of indoor common areas of multi-unit dwellings. While private homes and residences are exempted from the Clean Indoor Air Act, common areas are workplaces are are covered as such by the Act. However, this analysis does not render the proposed regulation unnecessary. The language of the Clean Indoor Air Act does not explicitly mention indoor common areas in residential buildings, and a regulation such as this will clarify and reinforce state law, while protecting Montgomery County residents from involuntary exposure to second hand smoke.

Thank you again for your time and attention.

Rita Turner
Deputy Director
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