Approved: March 30, 2005
Date

MINUTES OF THE HOUSE FEDERAL AND STATE AFFAIRS COMMITTEE

The meeting was called to order by Chairman John Edmonds at 1:30 P.M. on March 21, 2005 in Room 313-S of the Capitol.

All members were present except: Representative Kinzer
Representative Henderson
Representative Novascone
Representative Miller
Representative Huy

Committee staff present: Dennis Hodgins, Legislative Research Department
Athena Andaya, Legislative Research Department
Mary Torrence, Office of The Revisor
Carol Doel, Committee Secretary

Conferees:
Jack Walker, Vice Chairman of the Kansas Commission on Veteran Affairs

Others attending: See attached list

Chairman Edmonds opened the meeting for bill introductions. Hearing none, The Chair opened the public hearing on **SB 110** concerning the Kansas Commissions on veterans affairs; relating to memorials for Kansas veterans who served in the armed forces of the United States of America; prescribing certain guidelines and procedures; establishing the Kansas veterans memorial fund.

Jack Walker, Vice Chairman of the Kansas Commission on Veterans Affairs was recognized as a proponent of **SB 110**. This bill provides a means to honor those veterans with memorials that would be placed at the homes and cemeteries overseen by the Kansas Commission on Veterans Affairs. The bill contains the requisite controls for the management of funds, as they would be accumulated on deposit with the state. It also ensures equity among their facilities, the bill proposes that an advisory committee be established. The bill ensures that the requisite approvals by other state agencies are obtained as prescribed by state law. This bill is also significant in that it provides for the honoring of all veterans in a non-discriminatory and non-partisan manner. (Attachment 1)

There were no other proponents to address **SB 110** and no opponents. The Chair closed the hearing on **SB 110**.

Chairman Edmonds opened the floor for discussion on **SB 110**.

*Representative Ruff made a motion that **SB 110** be placed on the consent calender. Representative Dahl made a second to the motion. Vote was taken. Motion passed.*

Chairman Edmonds opened the floor for discussion on **SB 26**.

*Representative Dahl made a motion that **SB 26** be placed on the consent calender. Representative Oharah seconded the bill. Vote was taken. Motion passed.*

Chairman Edmonds asked for a motion to adopt the minutes from February 24th, March 2nd, March 3rd, March 7th, and March 8th.

Representative Siegfreid made a motion to adopt the minutes of February 24th, March 2nd, March 3rd, March 7th March 8th and March 9th. Representative Burroughs seconded the motion. Minutes were adopted.

Documentation of the Health Impact of Environmental Tobacco Smoke from Howard Rodenberg, M.D. M.P.H. Director of Health for the State of Kansas was presented to each member of the committee. (Attachment 2)

With no further business before the committee, Chairman Edmonds adjourned the meeting.

FEDERAL AND STATE AFFAIRS
GUEST LIST
Date 3-21-05

Kathy Johnson	Infant-Toddler Service, Harney Co.
Cathy Chapman	
Jack Fowler	KCVA
Jack E. Walker	KCVA
Senator Greta Goodwin	

TESTIMONY ON BEHALF OF
KANSAS SENATE BILL 110
A BILL TO ESTABLISH A MEMORIAL FUND WITHIN THE
KANSAS COMMISSION ON VETERANS AFFAIRS

HOUSE COMMITTEE ON FEDERAL AND STATE AFFAIRS

21 March 2005

Mr. Chairman, Representatives of the House Federal and State Affairs Committee, I am Jack Walker, Vice Chairman of the Kansas Commission on Veterans Affairs. It is my distinct honor and pleasure to appear before you today, representing the Kansas Commission on Veterans Affairs, on behalf of Senate Bill 110. We are grateful for the efforts of Senator Greta Goodwin for her support with this bill and her continuing efforts in support of the veterans of our state. As you know, the KCVA represents the nearly quarter million veterans in our State of Kansas. The bill before you today provides a means to honor those veterans with memorials that would be placed at the homes and cemeteries overseen by the Kansas Commission on Veterans Affairs.

Our commission has recognized that there are veterans, service organizations and individuals who would like to donate funds that would allow for memorials to be built honoring the veterans of our state. There is currently no mechanism by which such donated funds can be accepted and safeguarded until sufficient funds can be accumulated to build any large memorial. This bill is designed to facilitate such fund raising, security of the funds, management of the design effort and approval from other affected state agencies that would allow for large memorials, that are considered in the capital project range, to become reality, without resorting to a request for state funding. Things that come to mind would be flag malls, replacement of flags, gateways, outdoor picnic groves, carillons and statuary to name but a few ideas. These enhancements would not only beautify our facilities, but also provide comfort to the resident veterans and those who come to visit the facilities. All of this without funds from the state treasury.

The bill contains the requisite controls for the management of funds, as they would be accumulated on deposit with the state. All funds are raised from private sources and not requested from the state. The bill requires that all projects be fiscally responsible and able to be sustained by the KCVA Memorial Fund, without resorting to requests for assistance from the state. Further, that the memorials constructed would be maintained from the same funds. The bill specifically prohibits the use of these Memorial Funds to cover operational requirements. The bill also takes care to keep separate, the KCVA Memorial Funds and the Benefit Funds now authorized by law, which generally are designed to provide a more personal benefit to our veterans.

To ensure equity among our facilities, the bill proposes that an advisory committee be established that would include not only the KCVA commissioners, Executive Director, Superintendent of the Homes and Cemetery Director, but a member of the state legislature from the district in which each of our facilities are located. That advisory committee would have the charge to consider ideas and concepts presented along with ensuring that the funds were adequate to support the project under consideration.

The bill ensures that the requisite approvals by other state agencies are obtained as prescribed by state law. Since whatever construction may result becomes the property of the State of Kansas, the agencies charged with construction approval are included as a part of the memorial authorization process.

The bill is also significant in that it provides for the honoring of all veterans in a non-discriminatory and non-partisan manner. It provides a mechanism to do things properly with legislative and state oversight, at no cost to the state. Your support for this enabling legislation would be the next step in allowing the State to continue its efforts in caring for and honoring its veteran population.

We of the KCVA invite you to see the facilities you have already provided your veterans as you have done well in your efforts. We appreciate your past support and look forward to working with you now and in the future in support of the ever growing number of veterans from our state. I thank you for your courtesy and the opportunity to have been allowed to speak in behalf of Senate Bill 110. Subject to your questions, this concludes my presentation.

Jack E. Walker (LTC, USA, Ret)
Vice Chairman
Kansas Commission on Veterans Affairs
913-684-3732

Title: Health Harms from Secondhand Smoke

Publication Date: July 2004

Agency or Author: Campaign for Tobacco-Free Kids

Purpose: This document provides summary information on the harmful effects of Environmental Tobacco Smoke (ETS). Included in this document is a list of 69 known or probable human carcinogens that are found in ETS. Also in this document is a detailed list and summary of major findings concerning ETS, all of which point to the same conclusion; ETS has negative health consequences on non-smokers.

CAMPAIGN for TOBACCO-FREE Kids®

HEALTH HARMS FROM SECONDHAND SMOKE

The scientific evidence on the health risks associated with exposure to secondhand smoke is clear, convincing, and overwhelming. Secondhand smoke (also referred to as involuntary smoking, environmental tobacco smoke, and passive smoking) is a known cause of lung cancer, heart disease, chronic lung ailments such as bronchitis and asthma (particularly in children), and low birth-weight births. Exposure to secondhand smoke has been estimated to result in at least 38,000 annual deaths in the United States and over one million illnesses in children (see table below).

Annual Toll From Exposure to Secondhand Smoke in the United States[1]

Condition	Estimated Annual Deaths	Estimated Annual Diseases
Ischemic Heart Disease	35,000	--
Lung Cancer	3,000	--
Sudden Infant Death Syndrome	1,900	--
Low Birthweight Births	--	9,700
Asthma Exacerbation in Children	--	400,000
Acute Lower Respiratory Illness (Children < 18 mo.)	--	150,000
Otitis Media in Children	--	700,000

What is in a cigarette?[2]

To know what is in secondhand smoke, we first have to know what is in a cigarette. To that end, the following is a basic description of what is found in most cigarettes sold in the United States:

- Cigarette tobacco is blended from two main leaf varieties: Virginia tobacco that contains 2.5-3% nicotine; and 'burley' tobacco that has a higher nicotine content (3.5-4%). U.S. blends also contain up to 10% of imported 'oriental' tobacco that is aromatic but relatively low (less than 2%) in nicotine.

- In addition to the leaf blend, cigarettes contain 'fillers' which are made from the stems and other bits of tobacco that would otherwise be waste products. These are mixed with water and various flavorings and additives. The ratio of filler varies among brands.

- Additives are used to make tobacco products more acceptable to the consumer. They include humectants (moisturizers) to prolong shelf life; sugars to make the smoke seem milder and easier to inhale; and flavorings such as chocolate and vanilla.

- Additives are used to make cigarettes that provide high levels of 'free' nicotine that increases the addictive 'kick' of the nicotine. Ammonium compounds can fulfill this role by raising the alkalinity of smoke.

- Additives are used to enhance the taste of tobacco smoke, to make the product more desirable to consumers. Although seemingly innocuous, the addition of flavorings making the cigarette 'attractive' and 'palatable' is in itself cause for concern. Furthermore, sweeteners and chocolate may help to make cigarettes more palatable to children and first time users; eugenol and menthol numb the throat so the smoker cannot feel the smoke's aggravating effects. Also, additives such as cocoa may be used to dilate the airways allowing the smoke an easier and deeper passage into the lungs exposing the body to more nicotine and higher levels of tar.

What is in the smoke?

1400 I Street, NW Suite 1200 Washington, DC 20005
Phone (202) 296-5469 Fax (202) 296-5427 www.tobaccofreekids.org

Cigarette smoke is toxic soup of more than 4,000 known chemical compounds.[3] Cigarette smoke is made up of "sidestream" smoke from the burning tip of the cigarette and "mainstream" smoke from the filter or mouth end. Tobacco smoke contains thousands of different chemicals that are released into the air as particles and gases. The particulate phase of cigarette smoke includes nicotine, "tar" (itself composed of many chemicals), benzene and benzo(a)pyrene. The gas phase includes carbon monoxide, ammonia, dimethylnitrosamine, formaldehyde, hydrogen cyanide and acrolein. According to a November 2001 report issued by the National Cancer Institute[4], there are 69 known or probable carcinogens in cigarette smoke[5]. The complete list of these carcinogens appears in the table below.

LIST OF KNOWN, PROBABLE, AND POSSIBLE CANCER CAUSING CHEMICALS IN SECONDHAND SMOKE

Polycyclic Aromatic Hydrocarbons	Miscellaneous Organic Compounds
Benz(a)anthracene	Acetamide
Benzo(b)fluoranthene	Acrylamide
Benzo(j)fluoranthene	Acrylonitrile
Benzo(k)fluoranthene	Vinyl chloride
Benzo(a)pyrene	DDT
Dibenz(a,h)anthracene	DDE
Dibenzo(a,l)pyrene	Catechol
Dibenzo(a,e)pyrene	Caffeic acid
Indeno(1,2,3-cd)pyrene	1,1-Dimethylhydrazine
5-Methylchrysene	Nitromethane
	2-Nitropropane
	Nitrobenzene
	Ethyl carbamate
	Ethylene oxide
	Propylene oxide
	Methyleugenol
	MeAaC (2-amino-3-methyl-9-*H*-pyrido[2,3-*b*]indole
N-Nitrosamines	Inorganic Compounds
N-Nitrosodimethylamine	Hydrazine
N-Nitrosoethylmethylamine	Arsenic
N-Nitrosodiethylamine	Beryllium
N-Nitrosodi-n-propylamine	Nickel
N-Nitroso-di-n-butylamine	Chromium (only hexavalent)
N-Nitrosopyrrolidine	Cadmium
N-Nitrosopiperidine	Cobalt
N-Nitrosodiethanolamine	Lead
N-Nitrosonornicotine	Polonium-210
4-(Methylnitrosamino)-1-(3pyridyl)-1-butanone	
N-Heterocyblic Amines	Aldehydes
AaC	Formaldehyde
IQ	Acetaldhehyde
Trp-P-1	
Trp-P-2	
Glu-P-1	
Glu-P-2	
PhIP	
Volatile Hyrocarbons	Heterocyclic Compounds
1,3-Butadeine	Quinoline
Isoprene	Dibenz(a,h)acridine
Benzene	Dibenz(a,j)acridine
Styrene	Dibenzo(c,g)carbazole
	Benzo(b)furan
	Furan
Aromatic Amines	
2-Toluidine	2,6-Dimethylaniline
2-Naphthylamine	4-Aminobiphenyl

What are the health risks associated with exposure to secondhand smoke?

- *International Agency for Research on Cancer* (June 2002) – According to the IARC, "involuntary smoking (exposure to secondhand or 'environmental' tobacco smoke) is carcinogenic to humans (Group 1)."[6] Further, the IARC concluded that there is a "statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to secondhand tobacco smoke from the spouse who smokes. The excess risk is on the order of 20% for women and 30% for men."

In addition, the IARC found that "epidemiological studies have demonstrated that exposure to secondhand tobacco smoke is causally associated with coronary heart disease" and they estimated that "involuntary smoking increases the risk of an acute coronary heart disease event by 25-35%." Further, the IARC noted that, for adults, "the strongest evidence for a causal relation exists for chronic respiratory symptoms."

- *U.S. Environmental Protection Agency* (1992) – In its groundbreaking report, the EPA concluded that, for adults, "ETS [environmental tobacco smoke] is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. non-smokers" and the report found that secondhand smoke has a statistically significant effect on the respiratory health (e.g., reduced lung function) of non-smoking adults.[7]

  For children, the report concluded that, "ETS exposure is causally associated with an increased risk of lower respiratory tract infections (LRIs) such as bronchitis and pneumonia; increased prevalence of fluid in the middle ear, symptoms of upper respiratory tract irritation, and a small but significant reduction in lung function, and; additional episodes and increased severity of symptoms in children of asthma, with ETS exposure a risk factor for new cases of asthma in children who have not previously displayed symptoms."[8]

- In 1997, the National Cancer Institute (NCI) issued its 10th Monograph, *Health Effects of Exposure to Environmental Tobacco Smoke*, which evaluated the available scientific research and concluded that secondhand smoke exposure is causally associated with a number of negative health effects in adults and children.

  - For children, the NCI estimated that exposure to secondhand smoke resulted in more than 10,000 annual cases of low birthweight, more than 2,000 cases of SIDS (sudden infant death syndrome), more than 8,000 new cases of asthma, and as many as 1 million cases of exacerbated asthma.
  - For adults, the NCI estimated that each year secondhand smoke causes 3,000 deaths from lung cancer and 35,000 to 62,000 deaths associated with ischemic heart disease.[9]

- *U.S. Surgeon General* (1986) – In 1986, the Surgeon General concluded the following regarding exposure to secondhand smoke:

  - "Involuntary smoking is a cause of disease, including lung cancer, in healthy nonsmokers.
  - The children of parents who smoke, compared with the children of nonsmoking parents, have an increased frequency of respiratory infections, increased respiratory symptoms, and slightly smaller rates of increase in lung function as the lung matures.
  - Simple separation of smokers and nonsmokers within the same air space may reduce, but does not eliminate, exposure of nonsmokers to environmental tobacco smoke."[10]

- In 2000, the *American College of Occupational and Environmental Medicine* issued the following summary of current knowledge on health harms from workplace exposure to secondhand smoke:

  "Environmental tobacco smoke (ETS) contains numerous toxins. Robust epidemiologic evidence implicates ETS as a cause of lung cancer and as a primary cause and a source of exacerbation of excess respiratory disease. There is also increasing evidence that ETS may be associated with other outcomes, including heart disease. There is currently little doubt that ETS is an important and avoidable health hazard. Unfortunately, ETS is frequently encountered in the workplace - where it is no safer than in other environments and where it presents hazards to exposed workers and others."[11]

- In December 2002, the U.S. Public Health Service's National Toxicology Program issued its 10*th Report on Carcinogens*, which unambiguously states, based on a thorough review of the available scientific and medical evidence, that:

  "Environmental tobacco smoke (ETS) is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans that indicate a causal relationship between

passive exposure to tobacco smoke and human lung cancer (IARC 1986, EPA 1992, CEPA 1997). Studies also support an association of ETS with cancers of the nasal sinus (CEPA 1997). Evidence for an increased cancer risk from ETS stems from studies examining nonsmoking spouses living with individuals who smoke cigarettes, exposures of nonsmokers to ETS in occupational settings, and exposure to parents' smoking during childhood. Many studies, including recent large population-based case control studies, have demonstrated increased risks of approximately 20% for developing lung cancer following prolonged exposure to ETS, with some studies suggesting higher risks with higher exposures. Exposure to ETS from spousal smoking or exposure in an occupational setting appears most strongly related to increased risk."[12]

- A 2004 study published in the *British Medical Journal* found that exposure to secondhand smoke increases the risk of heart disease among non-smokers by as much as 60 percent.[13] This is the first study to show a direct physical link between secondhand smoke exposure and an increased risk of heart disease. The study, conducted over 20 years by researchers at St. George's Hospital Medical School in London, measured exposure to secondhand smoke from all sources – including in bars, restaurants, and other workplaces, as well as in the home – based on blood levels of a nicotine byproduct called cotinine. The study is one of the few that has sought to account for all sources of exposure to secondhand smoke, not just home exposure.
- A 2004 study published in the *British Medical Journal* examined whether there was a change in hospital admissions in Helena, Montana for acute myocardial infarction while a local law that prohibited smoking in most workplaces, including restaurants and bars, was in effect.[14] The study found that during the six months the law was enforced the number of admissions fell significantly - from an average of 40 admissions during the same months in the years before and after the law to a total of 24 admissions during the six months the law was in effect. In part, due to the Helena study, along with a "growing body of scientific data," a commentary was published in the same issue of the *British Medical Journal* (written by experts at the U.S. Centers for Disease Control and Prevention) that advised all clinician's with patients who have a history of coronary heart disease, that those patients "should be advised to avoid all indoor environments that permit smoking."[15]

- A 1997 analysis of 37 epidemiological studies of lung cancer and secondhand smoke, published in the *Journal of the National Cancer Institute,* found that lifelong nonsmokers living with smokers had, on average, a 24 percent higher chance of contracting lung cancer than those living with nonsmokers, and that those exposed to the heaviest smokers for the longest time had the highest risks.[16] Subsequent research studies have made similar findings.[17]

- A 1997 *British Medical Journal* meta-analysis of 19 published studies found that "Breathing other people's smoke is an important and avoidable cause of ischaemic heart disease, increasing a person's risk by a quarter."[18]

- A June 2001 study published in the journal *Pediatrics* found that exposure to secondhand smoke through the mother in utero was associated with increased rates of hospitalization in infants with non-smoking mothers, and that use of tobacco products by household members has an "enormous adverse impact" on the health of children.[19]

- A July 2001 study in the *Journal of the American Medical Association* concluded that exposure to secondhand smoke "substantially reduced" coronary circulation in healthy non-smokers, providing "direct evidence" that exposure to secondhand smoke causes coronary circulatory dysfunction in non-smokers.[20]

- A December 2001 study published in *The Lancet* found that exposure to secondhand smoke "increased the likelihood of experiencing [adverse] respiratory symptoms and was associated with increased [adverse] bronchial responsiveness." Specifically, the study found that exposure to secondhand smoke was "significantly associated" with nighttime chest tightness and breathlessness after physical activity, and that exposure to secondhand smoke in the workplace was significantly associated with all types of respiratory symptoms and current asthma.[21]

- A January 2002 study in the *British Medical Journal* found that maternal smoking during pregnancy represents a "true risk factor for early adult onset of diabetes." In addition, the study found that in utero exposures due to smoking during pregnancy "may increase the risk of both diabetes and obesity" possibly due to fetal malnutrition or toxicity.[22]

- Numerous research studies in the United States and overseas have found that smoking and exposure to secondhand smoke among pregnant women is a major cause of spontaneous abortions, stillbirths, and sudden infant death syndrome (SIDS) after birth.[23]

The Campaign for Tobacco-Free Kids, July 1, 2004, Matt Barry

Related Campaign Fact Sheets [All Campaign factsheets available at http://www.tobaccofreekids.org]

Clean Indoor Air Laws Encourage Smokers To Quit And Discourage Youth From Starting,
http://tobaccofreekids.org/research/factsheets

Smoke-Free Workplace Laws Reduce Smoking Rates – and the Cigarette Companies Know It,
http://www.tobaccofreekids.org/research/factsheets/pdf/0196.pdf

Smoke-free Restaurant & Bar Laws Do Not Harm Business,
http://tobaccofreekids.org/research/factsheets/pdf/0144.pdf

Ventilation Technology Does Not Protect People From Secondhand Smoke,
http://tobaccofreekids.org/research/factsheets/pdf/0145.pdf

---

[1] National Cancer Institute. *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency. Smoking and Tobacco Control Monograph no. 10.* Bethesda, MD. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 99-4645, 1999, http://cancercontrol.cancer.gov/tcrb/nci_monographs/MONO10/MONO10.HTM.

[2] This section is largely based from a document prepared by Action on Smoking and Health/United Kingdom entitled, *Fact Sheet No. 12, What's In A Cigarette?* (August 2001), http://www.ash.org.uk/html/factsheets/html/fact12.html.

[3] National Cancer Institute. *Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine.* Smoking and Tobacco Control Monograph No. 13. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 02-5074, October 2001. http://dccps.nci.nih.gov/tcrb/monographs/13/m13_5.pdf; personal communication, dated October 28, 2003, from Dietrich Hoffmann, Ph.D., Associate Director, Institute for Cancer Prevention, co-author of Chapter 5 of NCI Monograph 13, clarifying that Table 5.4 of the Monograph (that lists the 69 carcinogens) is missing a carcinogen, namely MeAaC (2-amino-3-methyl-*9-H*-pyrido[2,3-*b*]indole, and it should be inserted under "under "Miscellaneous Organic Compounds".

[4] National Cancer Institute. *Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine.* Smoking and Tobacco Control Monograph No. 13. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 02-5074, October 2001. http://dccps.nci.nih.gov/tcrb/monographs/13/m13_5.pdf.

[5] National Cancer Institute. *Risks Associated with Smoking Cigarettes with Low Machine-Measured Yields of Tar and Nicotine.* Smoking and Tobacco Control Monograph No. 13. Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 02-5074, October 2001. http://dccps.nci.nih.gov/tcrb/monographs/13/m13_5.pdf.

[6] International Agency for Research on Cancer, *Volume 83: Tobacco Smoke and Involuntary Smoking Summary of Data Reported and Evaluation, June 2002*, http://www.iarc.fr/.

[7] U.S. Environmental Protection Agency (EPA), Office of Research and Development & Office of Air and Radiation, *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*, EPA/600/6-90/006F, December 1992, http://www.epa.gov/nceawww1/ets/etsindex.htm.

[8] U.S. Environmental Protection Agency (EPA), Office of Research and Development & Office of Air and Radiation, *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders*, EPA/600/6-90/006F, December 1992, http://www.epa.gov/nceawww1/ets/etsindex.htm.

[9] National Cancer Institute. *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency. Smoking and Tobacco Control Monograph no. 10.* Bethesda, MD. U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute, NIH Pub. No. 99-4645, 1999, http://cancercontrol.cancer.gov/tcrb/nci_monographs/MONO10/MONO10.HTM.

[10] The Health Consequences of Involuntary Smoking: A Report of the Surgeon General (1986), U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control, Center for Health Promotion and Education, Office on Smoking and Health, Rockville, MD 20857, http://www.cdc.gov/tobacco/sgr/sgr_1986/SGR1986-PrefaceAndForward.PDF.

[11] American College of Occupational & Environmental Medicine, *Epidemiological Basis for an Occupational and Environmental Policy on Environmental Tobacco Smoke*, www.acoem.org/paprguid/papers/etspaper.htm, July 30, 2000.

[12] National Toxicology Program, Public Health Service, U.S. Department of Health and Human Services (HHS), 10*th Report on Carcinogens: Revised December 2002*, December 2002, http://ehp.niehs.nih.gov/roc/tenth/profiles/s176toba.pdf.

[13] Whincup, PH et al, "Passive smoking and risk of coronary heart disease and stroke: prospective study with cotinine measurement," *British Medical Journal*, BMJ, doi:10.1136/bmj.38146.427188.55, June 30, 2004, http://bmj.bmjjournals.com/cgi/reprint/bmj.38146.427188.55v1.

[14] Sargent, RP et al, "Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study," *British Medical Journal*, Volume 328, April 24, 2004.
[15] Pechacek TP, Babb S, "Commentary: How acute and reversible are the cardiovascular risks of secondhand smoke?," *British Medical Journal*, Volume 328, April 24, 2004.
[16] Hackshaw, AK et al, "The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke," *British Medical Journal* vol. 315 980-988, October 18, 1997.
[17] Boffetta, P, et al., "Multicenter Case-Control Study of Exposure to Environmental Tobacco Smoke and Lung Cancer in Europe," *Journal of the National Cancer Institute* 90: 1440-50, October 7, 1998. See, also, NCI, *Health Effects of Exposure to Environmental Tobacco Smoke: The Report of the California Environmental Protection Agency*, 1999, http://cancercontrol.cancer.gov/tcrb/nci_monographs/MONO10/MONO10.HTM.
[18] Law, M. R., et al., "Environmental Tobacco Smoke Exposure and Ischaemic Heart Disease: An Evaluation of the Evidence," *British Medical Journal* 315: 973-979, October 18, 1997. See, also, He, J., et al, "Passive Smoking and the Risk of Coronary Heart Disease -- A Meta-Analysis of Epidemiologic studies, *New England Journal of Medicine* 340(12): 920-26, March 25, 1999.
[19] Lam, Tai-Hing, et al, "The Effects of Environmental Tobacco Smoke on Health Services Utilization in the First Eighteen Months of Life," *Pediatrics* 107(6), June 2001. See, also, Anderson, HR & DG Cook, "Passive Smoking and Sudden Infant Death Syndrome: Review of the Epidemiological Evidence," *Thorax* 52: 1003-1009, November, 1997.
[20] Otsuka, Ryo, et al, "Acute Effects of Passive Smoking on the Coronary Circulation in Healthy Young Adults," *Journal of the American Medical Association* 286(4), July 25, 2001.
[21] Janson, Christer, et al, "Effect of passive smoking on respiratory symptoms, bronchial responsiveness, lung function, and total serum IgE in the European Community Respiratory Health Survey: a cross-sectional study," *The Lancet* v.358, December 22/29, 2001.
[22] Montgomery, S. & A. Ekbom, "Smoking during pregnancy and diabetes mellitus in a British longitudinal birth cohort," *British Medical Journal*, 324: 26-27, January 5, 2002.
[23] On spontaneous abortions, see, e.g., Mendola, P., et al., "Risk of Recurrent Spontaneous Abortion, Cigarette Smoking, and Genetic Polymorphisms in NAT2 and GSTM1," *Epidemiology* 9(6): 666-668 (November 1999); Shiverick, K.T. & C. Salafia, "Cigarette Smoking and Pregnancy I: Ovarian, Uterine and Placental Effects," *Placenta* 20(4): 265-272 (May 1999); Ness, R. B., et al., "Cocaine and Tobacco Use and the Risk of Spontaneous Abortion," *New England Journal of Medicine* 340(5): 333-339 (February 4, 1999); Chatenoud, L., et al., "Paternal and Maternal Smoking Habits Before Conception and During the First Trimester: Relation to Spontaneous Abortions," *Annals of Epidemiology* 8(8): 520-26 (November 1998); Dominquez-Rojas, V., et al., "Spontaneous Abortion in a Hospital Population: Are Tobacco and Coffee Intake Risk Factors?," *European Journal of Epidemiology* 10(6): 665-668 (December 1994); Walsh, R.A., "Effects of Maternal Smoking on Adverse Pregnancy Outcomes: Examination of the Criteria for Causation," *Human Biology* 66(6): 1059-1092 (December 1994); Windham, G.C., et al., "Parental Cigarette Smoking and the Risk of Spontaneous Abortion," *American Journal of Epidemiology* 135(12): 1394-403 (June 15, 1992); Armstrong, B.G. et al., "Cigarette, Alcohol, and Coffee Consumption and Spontaneous Abortion," *American Journal of Public Health* 82(1): 85-87 (January 1992); Pattinson, H.A. et al., "The Effect of Cigarette Smoking on Ovarian Function and Early Pregnancy Outcome Of In Vitro Fertilization Treatment," *Fertility and Sterility* 55(4): 780-783 (April 1991); Himmelberger, D. U., et al., "Cigarette Smoking During Pregnancy and the Occurrence of Spontanxous Abortion and Congenital Abnormality," *American Journal of Epidemiology* 108(6): 470-479 (December 1978); Kline, J., et al., "Smoking: A Risk Factor for Spontaneous Abortions," *New England Journal of Medicine* 291(15): 793-96 (October 1977). See, also, Kline, J. et al., "Cigarette Smoking and Spontaneous Abortion of Known Karyotype: Precise Data But Uncertain Inferences," *American Journal of Epidemiology* 141(5): 417-427 (March 1995); Economides, D. & J. Braithwaite, "Smoking, Pregnancy, and the Fetus," *Journal of the Royal Society of Health* 114(4): 198-201 (August 1994); Fredricsson, B. & H. Gilljam, "Smoking and Reproduction: Short and Long Term Effects and Benefits of Smoking Cessation," *Acta Obstetrica Gynecologica Scandinavica* 71(8): 580-592 (December 1992). But see, also, Windham, G.C. et al., "Exposure to Environmental and Mainstream Tobacco Smoke and Risk of Spontaneous Abortion," *American Journal of Epidemiology* 149(3): 243-247 (February 1, 1999); Sandahl, B. "Smoking Habits and Spontaneous Abortion," *European Journal of Obstetric Gynecology and Reproductive Biology* 31(1): 23-31 (April 1989).
On still births, see, e.g., Raymond, E.G. et al., "Effects of Maternal Age, Parity, and Smoking on the Risk of Stillbirth," *British Journal of Obstetric Gynaecology* 101(4): 301-306 (April 1994); Ahlborg, G. Jr. & L.. Bodin, "Tobacco Smoke Exposure and Pregnancy Outcome Among Working Women: A Prospective Study At Prenatal Care Centers In Orebro County, Sweden," *American Journal of Epidemiology* 133(4): 338-347 (February 1991).
On sudden infant death syndrome, see, e.g., Cooke, R.W., "Smoking, Intra-Uterine Growth Retardation and Sudden Infant Death Syndrome," *International Journal of Epidemiology* 27(2): 238-41 (April 1998).

Title: Secondhand Smoke Fact Sheet

Publication Date: February 2004

Agency or Author: Office of Smoking and Health, Centers for Disease Control and Prevention

Purpose: This document is the fact sheet found on the Centers for Disease Control and Prevention website concerning Environmental Tobacco Smoke (ETS). Included in this document are current estimates of health impacts, definitions of ETS, and references for primary literature on ETS.

Secondhand Smoke

Fact sheet

February 2004

Definition

- Secondhand smoke, also known as environmental tobacco smoke (ETS), is a mixture of the smoke given off by the burning end of tobacco products (sidestream smoke) and the smoke exhaled by smokers (mainstream smoke).[1,2]
- Secondhand smoke contains a complex mixture of more than 4,000 chemicals, more than 50 of which are cancer-causing agents (carcinogens).[1,2]
- People are exposed to secondhand smoke in the home, workplace, and in public venues such as bars, bowling alleys, and restaurants.[3]

Health Effects

- Secondhand smoke is associated with an increased risk for lung cancer and coronary heart disease in nonsmoking adults.[1,2,4] Secondhand smoke is a known human carcinogen (cancer-causing agent).[2,4]
- Because their lungs are not fully developed, young children are particularly susceptible to secondhand smoke. Exposure to secondhand smoke is associated with an increased risk for sudden infant death syndrome (SIDS), asthma, bronchitis, and pneumonia in young children.[1,5]

Current Estimates

- An estimated 3,000 lung cancer deaths and 35,000 coronary heart disease deaths occur annually among adult nonsmokers in the United States as a result of exposure to secondhand smoke.[6]
- Each year, secondhand smoke is associated with an estimated 8,000–26,000 new asthma cases in children.[4] Annually an estimated 150,000–300,000 new cases of bronchitis and pneumonia in children aged less than 18 months (7,500–15,000 of which will require hospitalization) are associated with secondhand smoke exposure in the United States.[4]
- Approximately 60% of people in the United States have biological evidence of secondhand smoke exposure.[7]
- Among children aged less than 18 years, an estimated 22% are exposed to secondhand smoke in their homes, with estimates ranging from 11.7% in

Utah to 34.2% in Kentucky.[8]

References

1 National Cancer Institute. *Health Effects of Exposure to Environment Tobacco Smoke.* Smoking and Tobacco Control Monograph No. 10 (PDF - 71k). Bethesda, MD: U.S. Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 1999. NIH Pub. No. 99-4645. Accessed: February 2004.

2 National Toxicology Program. *10th Report on Carcinogens.* Research Triangle Park, NC: U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, December 2002. Accessed: February 2004.

3 Pirkle JL, Flegal KM, Bernert JT, Brody DJ, Etzel RA, Maurer KR. Exposure of the U.S. population to environmental tobacco smoke: The Third National Health and Nutrition Examination Survey, 1988 to 1991. *Journal of the American Medical Association* 1996;275(16):1233–1240.

4 U.S. Environmental Protection Agency. *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders.* Washington, DC: U.S. Environmental Protection Agency;1992. Pub. No. EPA/600/6-90/006F. Accessed: February 2004.

5 U.S. Department of Health and Human Services. *Women and Smoking: A Report of the Surgeon General.* Rockville, MD: U.S. Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001. Accessed: February 2004.

6 CDC. Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999 (PDF - 225k). *Morbidity and Mortality Weekly Report* 2002;51(14):300–303. Accessed: February 2004.

7 CDC. *Second National Report on Human Exposure to Environmental Chemicals: Tobacco Smoke.* Atlanta, GA: U.S. Department of Health and Human Services, CDC, National Center for Environmental Health; 2003:80. NCEH Pub No. 03-0022. Accessed: February 2004.

8 CDC. State-specific prevalence of cigarette smoking among adults, and children's and adolescents' exposure to environmental tobacco smoke—United States, 1996. *Morbidity and Mortality Weekly Report* 1997;46(44):1038–1043. Accessed: February 2004.

Note: The next update of this fact sheet is scheduled for February 2005. More recent information may be available at the CDC'S Office on Smoking and Health Web site: http://www.cdc.gov/tobacco.

For Further Information

Office on Smoking and Health
National Center for Chronic Disease Prevention and Health Promotion
Centers for Disease Control and Prevention
Mailstop K-50
4770 Buford Hwy., N.E.
Atlanta, GA 30341-3717
770-488-5705
http://www.cdc.gov/tobacco
Media Inquiries: Contact the CDC's Office on Smoking and Health press line at 770-488-5493.

Title: The Effect of Smokefree Air Ordinance on Smoking Prevalence and Cessation

Publication Date: November 2004

Agency or Author: Americans for Nonsmokers' Rights

Purpose: This fact sheet provides summary information on the impact on current smoking rates following the introduction of smokefree air ordinances. Also included in this document are the sources of each study mentioned with a brief summary of the studies. This document concludes that following smokefree air ordinances the prevalence of smoking decreases and more individuals attempt to quit smoking. Utilization of quitlines increases following smokefree air ordinances.

ANR AMERICANS FOR NONSMOKERS' RIGHTS

Defending your right to breathe smokefree air since 1976

THE EFFECT OF SMOKEFREE AIR ORDINANCES ON SMOKING PREVALENCE AND CESSATION

November 2004

"Total prohibition of smoking in the workplace strongly affects industry volume. Smokers facing these restrictions consume 11-15% less than average and quit at a rate that is 84% higher than average... Milder workplace restrictions, such as smoking only in designated areas have much less impact on quitting rates and very little effect on consumption."
—Philip Morris (1992)[1]

- A study undertaken by researchers at the University of California, San Francisco investigated the effects of smokefree workplaces on smoking prevalence and cigarette consumption. Twenty-six studies on workplaces in the United States, Australia, Canada, and Germany were subjected to a process of systematic review and meta-analysis. Entirely smokefree workplaces were associated with a 3.8% reduction in smoking prevalence. Of those employees who continued to smoke, there was an average reduction in consumption of 3.1 fewer cigarettes per day. The combined effects of increased cessation and decreased consumption corresponded to a 29% relative reduction in tobacco use among all employees.[2]

- In 1993, the Joint Commission on Accreditation of Healthcare Organizations (JCAHO) mandated that all hospitals seeking accreditation go smokefree. A study conducted through the University of Missouri-Columbia investigated whether the rate of smoking cessation was higher among hospital employees than among other community employees not subject to a smokefree workplace policy. A total of 1,849 current or former smokers participated in the study over a period of three years. Hospital employees were found to be almost twice as likely as other community employees to quit smoking and tended to take a shorter time to quit.[3]

- Researchers at the University of California, Berkeley and the University of California, San Francisco investigated the effect of local workplace smoking laws in California on smoking cessation. Data from the 1990 California Tobacco Survey was used to collect information about 4,680 adult indoor smokers. The results of the study revealed that smokefree ordinances significantly increased the rate of smoking cessation and did so along a "dose-response" relationship – the stronger the ordinance, the higher the rate of cessation. While there was only a 19.1% cessation rate in areas with no ordinance, there was a 24.6% cessation rate in areas with weak ordinances, and a 26.4% cessation rate in areas with strong ordinances. Overall, researchers found that smokers who worked in communities with strong ordinances were 38% more likely to quit smoking than smokers in communities with no ordinance.[4]

- Massachusetts introduced a comprehensive tobacco control program in 1993 that brought together four elements of tobacco control: a cigarette tax increase; a mass media campaign; services for cessation and educational outreach; and the promotion of local smokefree ordinances. Prior to the program's implementation, the annual decline in cigarette consumption for Massachusetts adults was comparable to that for the rest of the nation (3-4% between 1988 and 1992). The year following the program's implementation (1992-3), consumption in Massachusetts dropped 12% while it remained steady for the rest of the nation at 4%.. After 1993, the annual decline in

2530 San Pablo Avenue, Suite J • Berkeley, California 94702 • (510) 841-3032 / FAX (510) 841-3071
www.no-smoke.org • anr@no-smoke.org

cigarette consumption leveled off in comparison states (declining less than 1% a year). In Massachusetts, however, consumption continued to decline by more than 4% a year.[5]

- Workplace smoking restrictions can significantly reduce smoking rates among young adults according to a study published in the *Journal of the American Medical Association* (JAMA). Researchers used data from the Current Population Surveys from 1992-1993 and 1995-1996 to question 17,185 adolescents between the ages of 15 and 17. Adolescents who worked in a smokefree workplace were found to be 32% less likely to smoke than adolescents who worked in a workplace with no smoking restrictions. Household smoking restrictions were also found to significantly reduce adolescent cigarette consumption and increase cessation rates.[6]

- Supplemental tobacco questionnaires were included in a series of national surveys conducted between September 1992 and May 1993. A total of 97,882 indoor workers were questioned regarding their smoking behavior and the smoking policies at their place of work. Researchers found that a 100% smokefree workplace was associated with a 6% reduction in smoking prevalence and a 14% decrease in the average daily cigarette consumption of smokers relative to workplaces with weak or no smoking restrictions. These results were found to be true for all demographic groups and in nearly all industries.[7]

- The Community Intervention Trial for Smoking Cessation (COMMIT) surveyed the behavior of 8,271 cigarette smokers in 22 North American communities between 1988 and 1993. Participants were questioned regarding their tobacco use behaviors, demographic characteristics, and workplace smoking policies. Employees in smokefree workplaces were found to be 25% more likely to make a serious attempt to quit smoking and 25% more likely to succeed than employees not subject to a smokefree workplace policy. Among continuing smokers, those in smokefree workplaces smoked an average of 2.75 fewer cigarettes a day.[8]

- A study published through the National Bureau of Economic Research investigated the effect of work area smoking bans on smoking behavior. Data from the 1991 and 1993 National Health Interview Surveys was used to obtain data for over 18,000 workers. Researchers found that workplace smoking bans are associated with a 5% to 6% decline in smoking prevalence and an average reduction in cigarette consumption of 2.3 cigarettes per day per smoker.[9]

What the Tobacco Industry thinks about workplace smoking restrictions…

- "Smoking bans are the biggest challenge we have ever faced. Quit rate goes frm [*sic*] 5% to 21% when smokers work in nonsmoking environments."[10]

- "The immediate implication for our business is clear: if our consumers have fewer opportunities to enjoy our products, they will use them less frequently and the result will be an adverse impact on our bottom line."[11]

- "Those who say they work under [smoking] restrictions smoked about one-and-one-quarter fewer cigarettes each day than those who don't. That may sound light but remember we're talking about light restrictions, too. Those 220 people in our survey who work under smoking restrictions represent some 15 million Americans. That one-and-one-quarter per day cigarette reduction, then,

means nearly 7 billion fewer cigarettes smoked each year because of workplace smoking restrictions… At a dollar a pack, even the lightest of workplace smoking restrictions is costing this industry 233 million dollars a year in revenue. How much more will it cost us with far more restrictive laws such as those in Suffolk County and Fort Collins now being enacted?"[12]

REFERENCES

[1] Heironimus, J., "Impact of Workplace Restrictions on Consumption and Incidence," *Philip Morris*, December 2, 1992, Bates No: 2023914280-4284. Download at http://legacy.library.ucsf.edu/tid/rvv24e00. Accessed on November 5, 2004.

[2] Fichtenberg, C.M.; Glantz, S.A., "Effect of smoke-free workplaces on smoking behavior: systematic review," *British Medical Journal 325: 188-191,* July 27, 2002.

[3] Longo, Johnson, Kruse, Brownson, and Hewett, "A Prospective Investigation of the Impact of Smoking Bans on Tobacco Cessation and Relapse," *Tobacco Control*, 2001.

[4] Moskowitz, Lin, and Hudes, "The Impact of Workplace Smoking Ordinances in California on Smoking Cessation," *American Journal of Public Health*, May 2000.

[5] Biener, Harris, and Hamilton, "Impact of the Massachusetts tobacco control programme: population based trend analysis," *British Medical Journal*, August 2000.

[6] Farkas, Gilpin, White, and Pierce, "Association Between Household and Workplace Smoking Restrictions and Adolescent Smoking," *Journal of the American Medical Association*, August 9, 2000.

[7] Farrelly, Evans, and Sfekas, "The impact of workplace smoking bans: results from a national survey," *Tobacco Control*, Autumn 1999.

[8] Glasgow, Cummings, Hyland, "Relationship of worksite smoking policy to changes in employee tobacco use: findings from COMMIT," *Tobacco Control*, 1997.

[9] Evans, Farrelly, and Montgomery, "Do Workplace Smoking Bans Reduce Smoking?" *NBER Working Paper Series 5567*, May 1996.

[10] [n.a.], "ETS World Conference," *Philip Morris*, April 1994, Bates No: 2054893642-3656. Download at http://legacy.library.ucsf.edu/tid/nyg12a00. Accessed on November 5, 2004.

[11] Walls, T. "CAC Presentation Number 4: Tina Walls – Introduction," *Philip Morris*, July 8, 1994, Bates No: 2041183751-3790. Download at http://legacy.library.ucsf.edu/tid/vnf77e00. Accessed on November 5, 2004.

[12] [n.a.], "Public Smoking: The Problem (SDC Introduction)," *Tobacco Institute*, [n.d.], Bates No: TIMN0014554-4565. Download at http://legacy.library.ucsf.edu/tid/mqo03f00. Accessed on November 5, 2004.

Title: Health Effects of Exposure to Environment Tobacco Smoke. Smoking and Tobacco Control Monograph No. 10

Publication Date: 1999

Agency or Author: National Cancer Institute

Purpose: This attached Executive Summary document summarizes the findings of many studies performed on the health impacts of Environmental Tobacco Smoke (ETS). The science presented in this document substantiates the indisputable fact that ETS causes illness and death. The full document can also be viewed their website (http://www.nci.nih.gov/) or in the included data CD.

Executive Summary

Exposure to environmental tobacco smoke (ETS) has been linked to a variety of adverse health outcomes. Many Californians are exposed at home, at work, and in public places. In the comprehensive reviews published as *Reports of the Surgeon General* and by the U.S. Environmental Protection Agency (U.S. EPA) and the National Research Council (NRC), ETS exposure has been found to be causally associated with respiratory illnesses—including lung cancer, childhood asthma, and lower respiratory tract infections. Scientific knowledge about ETS-related effects has expanded considerably since the release of the above-mentioned reviews. The state of California has therefore undertaken a broad review of ETS covering the major health endpoints potentially associated with ETS exposure: perinatal and postnatal manifestations of developmental toxicity, adverse impacts on male and female reproduction, respiratory disease, cancer, and cardiovascular disease. A "weight of evidence" approach has been used, in which the body of evidence is examined to determine whether or not it can be concluded that ETS exposure is causally associated with a particular effect. Because the epidemiological data are extensive, they serve as the primary basis for assessment of ETS-related effects in humans. The report also presents an overview on measurements of ETS exposure (particularly as they relate to characterizations of exposure in epidemiological investigations) and on the prevalence of ETS exposure in California and nationally.

ETS, or "secondhand smoke," is the complex mixture formed from the escaping smoke of a burning tobacco product and smoke exhaled by the smoker. The characteristics of ETS change as it ages and combines with other constituents in the ambient air. Exposure to ETS is also frequently referred to as "passive smoking," or "involuntary tobacco smoke" exposure. Although all exposures of the fetus are "passive" and "involuntary," for the purposes of this review, *in utero* exposure resulting from maternal smoking during pregnancy is not considered to be ETS exposure.

GENERAL FINDINGS

ETS is an important source of exposure to toxic air contaminants indoors. There is also some exposure outdoors in the vicinity of smokers. Despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures in the home, especially of infants and children, continue to be a public health concern. ETS exposure is causally associated with a number of health effects. Listed in Table ES.1 are the developmental, respiratory, carcinogenic, and cardiovascular effects for which there is sufficient evidence of a causal relationship—including fatal outcomes such as sudden infant death syndrome and heart disease

Table ES.1
Health Effects Associated with Exposure to Environmental Tobacco Smoke

Effects Causally Associated with ETS Exposure
Developmental Effects
Fetal Growth: Low birthweight or small for gestational age
Sudden Infant Death Syndrome (SIDS)
Respiratory Effects
Acute lower respiratory tract infections in children (*e.g.*, bronchitis and pneumonia)
Asthma induction and exacerbation in children
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children
Carcinogenic Effects
Lung Cancer
Nasal Sinus Cancer
Cardiovascular Effects
Heart disease mortality
Acute and chronic coronary heart disease morbidity
Effects with Suggestive Evidence of a Causal Association with ETS Exposure
Developmental Effects
Spontaneous abortion
Adverse impact on cognition and behavior
Respiratory Effects
Exacerbation of cystic fibrosis
Decreased pulmonary function
Carcinogenic Effects
Cervical cancer

mortality, as well as serious chronic diseases such as childhood asthma. There are, in addition, effects for which evidence is suggestive of an association, but further research is needed for confirmation. These include spontaneous abortion, cervical cancer, and exacerbation of asthma in adults (Table ES.1). Finally, it is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints including congenital malformations, changes in female fertility and fecundability, male reproductive effects, rare childhood cancers, and cancers of the bladder, breast, stomach, brain, hematopoietic system, and lymphatic system.

Many Californians are exposed to ETS, and the number of people adversely affected may be correspondingly large. Table ES.2 presents morbidity and mortality estimates for health effects causally associated with ETS exposure. For cancer, cardiovascular, and some respiratory endpoints, estimates are derived from figures published for the U.S. population, assuming that the number affected in California would be 12 percent of the total. The estimates for middle ear infection, sudden infant death syndrome, and low birthweight were derived using information on prevalence of ETS exposure in California and the U.S.

Relative risk estimates (RR) associated with some of these endpoints are small, but because the diseases are common, the overall impact can be quite large. A relative risk estimate of 1.3 for heart disease mortality in nonsmokers is supported by the collective evidence; this estimate corresponds to a lifetime risk of death of roughly 1 to 3 percent for exposed nonsmokers and approximately 4,000 deaths annually in California. The relative risk estimate of 1.2 to 1.4 associated with low birthweight implies that ETS may impact fetal growth of 1,200 to 2,200 newborns in California, roughly 1 to 2 percent of newborns of nonsmokers exposed at home or at work. ETS may exacerbate asthma (RR $\approx$ 1.6 to 2) in 48,000 to 120,000 children in California. Large impacts are associated with relative risks for respiratory effects in children such as middle ear infection (RR $\approx$ 1.62) and lower respiratory disease in young children (RR $\approx$ 1.5 to 2). Asthma induction (RR $\approx$ 1.75 to 2.25) may occur in as many as 0.5 to 2 percent of ETS-exposed children. ETS exposure may be implicated in 120 SIDS deaths per year in California (RR $\approx$ 3.5), with a risk of death approaching 0.1 percent for infants exposed to ETS in their homes. Lifetime risk of lung cancer death related to ETS-exposed nonsmokers may be about 0.7 percent (RR $\approx$ 1.2). For nasal sinus cancers, observed relative risks have ranged from 1.7 to 3.0, but future studies are needed to confirm the magnitude of ETS-related risks.

SPECIFIC FINDINGS AND CONCLUSIONS

ETS is a complex mixture of chemicals generated during the burning and smoking of tobacco products. Chemicals present in ETS include irritants and systemic toxicants such as hydrogen cyanide and sulfur dioxide; mutagens and carcinogens such as benzo[a]pyrene, formaldehyde, and 4-aminobiphenyl; and the reproductive toxicants nicotine, cadmium, and carbon monoxide. Many ETS constituents have been identified as hazardous by state, federal, and international agencies. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six identified as developmental or reproductive toxicants under California's Proposition 65 (California Health and Safety Code 25249.5 *et seq.*).

Exposure Measurement and Prevalence

Exposure assessment is critical in epidemiological investigations of the health impacts of ETS, and in evaluating the effectiveness of strategies to reduce exposure. Exposure can be assessed through the measurement of indoor air concentrations of ETS constituents, through surveys and ques-

Table ES.2
Estimated Annual Morbidity and Mortality in Nonsmokers Associated with ETS Exposure

Condition	Number of People or Cases[a]	
	in the U.S.	in California
Developmental Effects		
Low birthweight	9,700 - 18,600 cases[b]	1,200 - 2,200 cases[b]
Sudden Infant Death Syndrome (SIDS)	1,900 - 2,700 deaths[b]	120 deaths[b]
Respiratory Effects in Children		
Middle ear infection	0.7 to 1.6 million physician office visits[b]	78,600 to 188,700 physician office visits[b]
Asthma induction	8,000 to 26,000 new cases[c]	960 to 3,120 new cases[c]
Asthma exacerbation	400,000 to 1,000,000 children[c]	48,000 to 120,000 children[c]
Bronchitis or pneumonia in infants and toddlers (18 months and under)	150,000 to 300,000 cases[c] 7,500 to 15,000 hospitalizations[c] 136 - 212 deaths[c]	18,000 to 36,000 cases[c] 900 to 1,800 hospitalizations[c] 16 - 25 deaths[c]
Cancer		
Lung	3,000 deaths[c]	360 deaths[c]
Nasal sinus	N/A[d]	N/A[d]
Cardiovascular Effects		
Ischemic heart disease	35,000 - 62,000 deaths[c]	4,200 - 7,440 deaths[c]

[a] *The numbers in the table are based on maximum likelihood estimates of the relative risk. As discussed in the body of the report, there are uncertainties in these estimates, so actual impacts could be somewhat higher or lower than indicated in the table. The endpoints listed are those for which there is a causal association with ETS exposure based on observations of effects in exposed human populations.*

[b] *California estimates for low birthweight, SIDS, and middle ear infection (otitis media) are provided in Chapters 3, 4, and 6, respectively. U.S. estimates are obtained by dividing by 12 percent, the fraction of the U.S. population residing in California.*

[c] *Estimates of mortality in the U.S. for lung cancer and respiratory effects, with the exception of middle ear infection (otitis media), come from U.S. EPA (1992). U.S. range for heart disease mortality reflects estimates reported in Wells (1988 and 1994), Glantz and Parmley (1991), Steenland (1992). California predictions are made by multiplying the U.S. estimate by 12 percent, the fraction of the U.S. population residing in the State. Because of decreases in smoking prevalence in California in recent years, the number of cases for some endpoints may be somewhat overestimated, depending on the relative impacts of current versus past ETS exposures on the health endpoint.*

[d] *Estimates of the impact of ETS exposure on the occurrence of nasal sinus cancers are not available at this time.*

tionnaires, or more directly through the use of personal monitors and the measurement of biomarkers in saliva, urine, and blood. There are advantages and disadvantages associated with the various techniques, which must be weighed in interpreting study results. One important consideration in epidemiologic studies is misclassification of exposure. Studies on the reliability of questionnaire responses indicate that qualitative information obtained is generally reliable, but that quantitative information may not be. Also, individuals are often unaware of their ETS exposure, particularly outside the home. In studies using both self-reporting and biological markers, the exposure prevalence was higher when determined using biological markers.

Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the U.S. Among adults in California, the workplace, home, and other indoor locations all contribute significantly to ETS exposure. For children, the most important single location is the home. Over the past decade, ETS exposures in California have decreased significantly in the home, workplace, and in public places. Over the same period, restrictions on smoking in enclosed worksites and public places have increased (*e.g.*, Gov. Code, Section 19994.30 and California Labor Code, Section 6404.5), and the percentage of the adults who smoke has declined. Decreases in tobacco smoke exposure may not be experienced for some population subgroups, as patterns of smoking shift with age, race, sex, and socioeconomic status. For example, from 1975 to 1988, the overall smoking prevalence among 16 to 18 year olds declined, but after 1988 the trend reversed.

Perinatal Manifestations of Developmental Toxicity

ETS exposure adversely affects fetal growth, with elevated risks of low birth weight or "small for gestational age" observed in numerous epidemiological studies. The primary effect observed, reduction in mean birthweight, is small in magnitude. But if the distribution of birthweight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birthweight is associated with many well-recognized problems for infants and is strongly associated with perinatal mortality.

The impact of ETS on perinatal manifestations of development other than fetal growth is less clear. The few studies examining the association between ETS and perinatal death are relatively non-informative, with only two early studies showing increased risk associated with parental smoking, and with the sparse data on stillbirth not indicative of an effect. Studies on spontaneous abortion are suggestive of a role for ETS, but further work is needed, particularly as a recent report did not confirm the findings of four earlier studies. Although epidemiological studies suggest a moderate association of severe congenital malformations with paternal smoking, the findings are complicated by the use of paternal smoking status as a surrogate for ETS exposure, since a direct effect of active smoking on sperm cannot be ruled out. In general, the defects implicated differed across the stud-

ies, with the most consistent association seen for neural tube defects. At this time, it is not possible to determine whether there is a causal association between ETS exposure and this or other birth defects.

Postnatal Manifestations of Developmental Toxicity Numerous studies have demonstrated an increased risk of sudden infant death syndrome, or "SIDS," in infants of mothers who smoke. Until recently it has not been possible to separate the effects of postnatal ETS exposure from those of prenatal exposure to maternal active smoking. Recent epidemiological studies now have demonstrated that postnatal ETS exposure is an independent risk factor for SIDS.

Although definitive conclusions regarding causality cannot yet be made on the basis of available epidemiological studies of cognition and behavior, there is suggestive evidence that ETS exposure may pose a hazard for neuropsychological development. With respect to physical development, while small but consistent effects of active maternal smoking during pregnancy have been observed on height growth, there is no evidence that postnatal ETS exposure has a significant impact in otherwise healthy children. As discussed in greater detail below, developmental effects of ETS exposure on the respiratory system include lung growth and development, childhood asthma exacerbation, and, in children, acute lower respiratory tract illness, middle ear infection, and chronic respiratory symptoms.

Female and Male Reproductive Toxicity Though active smoking by women has been found to be associated with decreased fertility in a number of studies, and tobacco smoke appears to be anti-estrogenic, the epidemiological data on ETS exposure and fertility are not extensive and show mixed results, and it is not possible to determine whether ETS affects fecundability or fertility. Regarding other female reproductive effects, while studies indicate a possible association of ETS exposure with early menopause, the analytic methods of these studies could not be thoroughly evaluated, and therefore at present, there is not firm evidence that ETS exposure affects age at menopause. Although associations have been seen epidemiologically between active smoking and sperm parameters, conclusions cannot be made regarding ETS exposure and male reproduction, as there is very limited information available on this topic.

Respiratory Effects ETS exposure produces a variety of acute effects involving the upper and lower respiratory tract. In children, ETS exposure can exacerbate asthma, and increases the risk of lower respiratory tract illness and acute and chronic middle ear infection. Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS. Odor annoyance has been demonstrated in several studies.

Regarding chronic health effects, there is compelling evidence that ETS is a risk factor for induction of new cases of asthma as well as for increasing the severity of disease among children with established asthma. In addition, chronic respiratory symptoms in children—such as cough,

2-27

phlegm, and wheezing—are associated with parental smoking. While the results from all studies are not wholly consistent, there is evidence that childhood exposure to ETS affects lung growth and development, as measured by small but statistically significant decrements in pulmonary function tests; associated reductions may persist into adulthood. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small and is unlikely by itself to result in clinically significant chronic disease. However, in combination with other insults (*e.g.*, prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment in adults. In addition, regular ETS exposure in adults has been reported to increase the risk of occurrence of a variety of lower respiratory symptoms.

Children are especially sensitive to the respiratory effects of ETS exposure. Children with cystic fibrosis are likely to be more sensitive than healthy individuals. Several studies of patients with cystic fibrosis, a disease characterized by recurrent and chronic pulmonary infections, suggest that ETS can exacerbate the condition. Several studies have shown an increased risk of atopy (a predisposition to develop IgE antibodies against common allergens, which can then be manifested as a variety of allergic conditions) in children of smoking mothers, though the evidence regarding this issue is mixed.

Carcinogenic Effects The role of ETS in the etiology of cancers in nonsmokers was explored, as smoking is an established cause of a number of cancers (lung, larynx, oral cavity, esophagus, and bladder), and a probable cause of several others (cervical, kidney, pancreas, and stomach). Also, ETS contains a number of constituents which have been identified as carcinogens.

Reviews published in the 1986 *Report of the Surgeon General,* by the National Research Council in 1986, and by the U.S. EPA in 1992 concluded that ETS exposure causes lung cancer. Three large U.S. population-based studies and a smaller hospital-based, case-control study have been published since the completion of the U.S. EPA review. The population-based studies were designed to, and have successfully, addressed many of the weaknesses for which the previous studies on ETS and lung cancer have been criticized. Results from these studies are compatible with the causal association between ETS exposure and lung cancer already reported by the U.S. EPA, Surgeon General, and National Research Council. Of the studies examining the effect of ETS exposure on nasal sinus cancers, all three show consistent associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in nonsmoking adults. Further study is needed to characterize the magnitude of the risk of nasal sinus cancer from ETS exposure.

The epidemiological and biochemical evidence suggests that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in two of three case-control studies, and a statistically non-significant positive association was observed in the only cohort study con-

ducted. Findings of DNA adducts in the cervical epithelium as well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers provides biological plausibility.

For other cancer sites in adults, there has been limited ETS-related epidemiological research in general; there is currently insufficient evidence to draw any conclusion regarding the relationship between ETS exposure and the risk of occurrence. A review of the available literature clearly indicates the need for more research. For example, although compounds established as important in the etiology of stomach cancer are present in tobacco smoke, only a single cohort study has been performed for this site. Precursors of endogenously formed N-nitroso compounds suspected of causing brain tumors are present in high concentrations in ETS, and the one cohort and two case-control studies available suggest a positive association, but the results are based on small numbers and may be confounded by active smoking. In biochemical studies of nonsmokers, higher levels of hemoglobin adducts of the established bladder carcinogen, 4-aminobiphenyl, have been found in those exposed to ETS. However, no significant increases in bladder cancer were seen in the two epidemiological studies (case-control) conducted to date, although both studies were limited in their ability to detect an effect. Several compounds in tobacco smoke are associated with increased risk of leukemia, but only one small case-control study in adults, reporting an increased risk with ETS exposure during childhood, has been performed. Finally, all four studies on ETS exposure and breast cancer suggest an association, but in two of the studies the associations were present only in select groups, and in three studies there is either no association between active smoking and the risk of breast cancer, or the association for active smoking is weaker than for passive smoking. Moreover, there is no indication of increasing risk with increasing intensity of ETS exposure. Still, results from a recent study suggest that tobacco smoke may influence the risk of breast cancer in certain susceptible groups of women, an association which requires further investigation.

Regarding childhood cancers, it is unclear whether parental smoking increases risk, either overall or for specific cancers such as acute lymphoblastic leukemia or brain tumors, the two most common cancers in children. The lack of clarity is due to the conflicting results reported and the limitations of studies finding no association. The epidemiological data on ETS exposure and rare childhood cancers also provide an inadequate foundation for making conclusions regarding causality. Some studies in children found small increased risks in relation to parental smoking for neuroblastoma, Wilm's tumor, bone and soft-tissue sarcomas, but not for germ cell tumors. Studies to date on these rare cancers have been limited in their power to detect effects. The impact of ETS exposure on childhood cancer would benefit from far greater attention than it has received to date.

Cardiovascular Effects The epidemiological data from prospective and case-control studies conducted in diverse populations, in males and females and in western and eastern countries, are supportive of a causal association

between ETS exposure from spousal smoking and coronary heart disease (CHD) mortality in nonsmokers. To the extent possible, estimates of risk were determined with adjustment for demographic factors and often for other factors related to heart disease—factors such as blood pressure, serum cholesterol level, and obesity index. Risks associated with ETS exposure were almost always strengthened by adjustment for other cofactors. For nonsmokers exposed to spousal ETS compared to nonsmokers not exposed, the risk of CHD mortality is increased by a factor of 1.3. The association between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina.

Data from clinical studies suggest various mechanisms by which ETS causes heart disease. In a number of studies wherein nonsmokers were exposed to ETS, carotid wall thickening and compromise of endothelial function were similar to, but less extensive than those experienced by active smokers. Other effects observed include impaired exercise performance, altered lipoprotein profiles, enhanced platelet aggregation, and increased endothelial cell counts. These findings may account for both the short- and long-term effects of ETS exposure on the heart.

ATTACHMENT I

Review of the OEHHA Assessment of Environmental Tobacco Smoke by the Scientific Review Panel (SRP)

Interest in the health effects of second-hand tobacco smoke on the part of members of the Scientific Review Panel (SRP) on Toxic Air Contaminants led to a request by the SRP for a health assessment of environmental tobacco smoke and a collaborative agreement between the Office of Environmental Health Hazard Assessment (OEHHA) and the Air Resources Board (ARB) to initiate such an assessment. SRP members reviewed the drafts as they were developed and participated in each of the workshops held as the document underwent public review. The final draft reflected the input of SRP members, as well as that of other reviewers.

Specific changes made at the request of the SRP following its review of the final draft include the addition of new studies (*e.g.*, the results of Kawachi *et al.*'s analysis of cardiovascular disease risk in the Nurse's Health study, published after the release of the final draft, in which it was reported as an abstract), a discussion of issues related to misclassification of smoking status and cancer risk, and clarifying language in the presentation of attributable risk estimates; minor editorial changes were also requested and made. The SRP discussed the assessment and made findings on the health effects of exposure to environmental tobacco smoke as a result of its review; these findings are included in this attachment.

UNIVERSITY OF CALIFORNIA, IRVINE

BERKELEY • DAVIS • IRVINE • LOS ANGELES • RIVERSIDE • SAN DIEGO • SAN FRANCISCO SANTA BARBARA • SANTA CRUZ

DEPARTMENT OF CHEMISTRY IRVINE, CALIFORNIA 92717-2025

July 18, 1997

Richard Becker, Ph.D.
Director
Office of Environmental
Health Hazard Assessment
301 Capitol Mall, Second Floor
Sacramento, California 95814

Dear Dr. Becker:

On behalf of the Scientific Review Panel (SRP/Panel) I am pleased to transmit to you our Findings as a result of our review of the Office of Environmental Health Hazard Assessment (OEHHA) final report "Health Effects of Exposure to Environmental Tobacco Smoke" (ETS).

As you will see in a review of the SRP meeting transcript, the Panel is very impressed with the quality of the report and view it as the most current and definitive statement of the science applicable to ETS. As we noted OEHHA staff scientists are to be highly commended for this successful completion.

We are also pleased that the Air Resources Board (ARB) is considering holding an "informational hearing" on the report. As you will see in the enclosed Findings, the Panel views ETS as a toxic air contaminant, and it has a major impact on public health.

If the Panel may be of further help as this health risk is addressed in California, we would be pleased to do so.

We trust our Findings and this transmittal letter will be made a part of the final report

Sincerely,

James N. Pitts, Jr. Ph.D.
Chairman
Scientific Review Panel

Enclosure

cc: John D. Dunlap, Chairman, ARB
Scientific Review Panel Members
Bill Lockett, ARB

Findings of the Scientific Review Panel on
HEALTH EFFECTS OF EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE
as Adopted at the Panel's June 19, 1997 Meeting

The Scientific Review Panel (SRP/Panel) has reviewed the report "Health Effects of Exposure to Environmental Tobacco Smoke" prepared by the Office of Environmental Health Hazard Assessment (OEHHA). The Panel members also reviewed the public comments received on this report. Based on this review, the SRP makes the following findings:

1. Environmental Tobacco Smoke (ETS) is an important source of exposure to toxic air contaminants. Thus, despite an increasing number of restrictions on smoking and increased awareness of health impacts, exposures continue to be a major public health concern.

2. A causal association exists between ETS exposure from spousal smoking and coronary heart disease (CHD) mortality in nonsmokers. Risks associated with ETS exposure were almost always strengthened by adjustment for other cofactors. For nonsmokers exposed to spousal ETS compared to nonsmokers not exposed, the risk of CHD mortality is increased by a factor of 1.3. The association between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina. Heart disease is the primary fatal endpoint from ETS exposure.

3. ETS is a complex mixture of chemicals generated during the burning and smoking of tobacco products. Chemicals present in ETS include irritants and systemic toxicants, mutagens and carcinogens, and reproductive and developmental toxicants. To date, over 50 compounds in tobacco smoke have been identified as carcinogens and six as developmental or reproductive toxicants under California's Proposition 65 (California Health and Safety Code 25249.5 *et seq.*) and twelve have been identified as a toxic air contaminant under AB 1807.

4. The 1986 *Report of the Surgeon General,* the 1986 National Research Council report *Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects,* and the 1992 U.S. EPA report *Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders* have established that ETS exposure causes lung cancer. Results from recent epidemiological studies are compatible with the causal association already established.

5. Available data suggest that the prevalence of ETS exposure in California is lower than elsewhere in the U.S. Nevertheless, among adults in California, the workplace, home and other indoor locations all contribute significantly to ETS exposure. For children the most important single location is the home.

6. ETS exposure adversely affects fetal growth, with elevated risks of low birth weight or "small for gestational age" observed in numerous epidemiological studies. The primary effect observed, reduction in mean birth weight, is small in magnitude. If the distribution of birth weight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birth weight is associated with many well-recognized problems for infants and is strongly associated with perinatal mortality.

7. Numerous studies have demonstrated an increased risk of sudden infant death syndrome, or "SIDS," in infants of mothers who smoke. Until recently it has not been possible to separate the effects of postnatal ETS exposure from those of prenatal exposure to maternal active smoking. Recent epidemiological studies now have demonstrated that postnatal ETS exposure is an independent cause of SIDS.

8. ETS exposure produces a variety of acute effects involving the upper and lower respiratory tract. In children, ETS exposure can exacerbate asthma, and increases the risk of lower respiratory tract illness, and acute and chronic middle ear infection. Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS. Odor annoyance has been demonstrated in several studies.

9. Regarding chronic health effects, there is compelling evidence that ETS is a risk factor for induction of new cases of asthma as well as for increasing the severity of disease among children with established asthma. In addition, chronic respiratory symptoms in children, such as cough, phlegm, and wheezing, are associated with parental smoking. While the results from all studies are not wholly consistent, there is evidence that childhood exposure to ETS affects lung growth and development, as measured by small, but statistically significant decrements in pulmonary function tests; associated reductions may persist into adulthood.

10. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small. However, in combination with other insults (*e.g.*, prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment in adults. In addition, regular ETS exposure in adults has been reported to increase the risk of occurrence of a variety of lower respiratory symptoms (e.g. bronchitis and wheezing apart from colds).

11. Children are especially sensitive to the respiratory effects of ETS exposure. Children with cystic fibrosis are likely to be more sensitive than healthy individuals. Several studies of patients with cystic fibrosis, a disease characterized by recurrent and chronic pulmonary infections, suggest that ETS can exacerbate the condition. Several studies have shown an increased risk of atopy (a predisposition to develop IgE antibodies against common allergens, which can then be manifested as a variety of allergic conditions) in children of smoking mothers, though the evidence regarding this issue is mixed.

12. Of the studies examining the effect of ETS exposure on nasal sinus cancers, all three show consistent associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in nonsmoking adults. Further study is needed to characterize the magnitude of the risk of nasal sinus cancer from ETS exposure.

13. The epidemiological and biochemical evidence suggest that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in two of three case-control studies and a statistically nonsignificant positive association was observed in the only cohort study conducted. Findings of DNA adducts in the cervical epithelium as well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers provides biological plausibility.

14. Studies on ETS exposure and breast cancer suggest an association, but the associations were present only in select groups, or there is either no association between active smoking and the risk of breast cancer or the association for active smoking is weaker than for passive smoking. However, there is no indication of increasing risk with increasing intensity of ETS exposure. Still, results from a recent study suggest that tobacco smoke may influence the risk of breast cancer in certain susceptible groups of women, and this requires further investigation.

15. In summary, ETS exposure is causally associated with a number of fatal and non-fatal health effects. Heart disease mortality, sudden infant death syndrome, and lung and nasal sinus cancer have been causally linked to ETS exposure. Serious impacts of ETS on the young include childhood asthma induction and exacerbation, bronchitis and pneumonia, middle ear infection, chronic respiratory symptoms, and low birth weight. In adults acute and chronic heart disease morbidity is causally associated with ETS exposure. ETS also causes eye and nasal irritation and odor annoyance.

16. Effects for which evidence is suggestive of an association, but further research is needed for confirmation, include: spontaneous abortion, adverse neuropsychological development, cervical cancer, exacerbation of cystic fibrosis, and decreased pulmonary function.

17. It is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints, including congenital malformations, changes in female fertility and fecundability, male reproductive effects, rare childhood cancers and cancers of the bladder, breast, stomach, brain, hematopoietic system, and lymphatic system.

18. Many Californians are exposed to ETS, and the number of people adversely affected is correspondingly large. Each year ETS contributes to asthma exacerbation in 48,000 to 120,000 children, 960 to 3120 new cases of asthma in children, 78,600 to 188,700 physicians office visits due to middle ear infections in children, 18,000 to 36,000 cases and 900 to 1800 hospitalizations from bronchitis or pneumonia in toddlers and infants, and 1,200 to 2,200 cases of low birth weight. Annual mortality estimates associated with ETS

exposure in California are: Approximately 120 deaths from SIDS, 16-25 deaths in toddlers and infants from bronchitis and pneumonia, approximately 360 deaths from lung cancer, and 4,200 - 7,440 deaths from ischemic heart disease. Thus, ETS has a major public health impact.

After careful review of the February 1997 draft of the OEHHA report, "Health Effects of Exposure to Environmental Tobacco Smoke," we find the draft, with the changes specified by OEHHA in our June 19, 1997 meeting, as representing a complete and balanced assessment of current scientific understanding. Based on the available evidence we conclude ETS is a toxic air contaminant.

I certify that the above is a true and correct copy of the findings adopted by the Scientific Review Panel on June 19, 1997

James N. Pitts, Jr., Ph.D.
Chairman, Scientific Review Panel

4

Title: Fact Sheet: Respiratory Health Effects of Passive Smoking

Publication Date: January 1993

Agency or Author: Office of Research and Development, Office of Air and Radiation, Environmental Protection Agency

Purpose: This Executive Summary document is a summary of the major health assessment on Environmental Tobacco Smoke (ETS) by the EPA. Included is a summary of findings, methodology, and scope of the larger document. This document concludes that ETS has known negative impacts on the health of non-smokers. The full document can also be viewed at the website (www.epa.gov) or on the included data CD

"Fact Sheet: Respiratory Health Effects of Passive Smoking"

Office of Research and Development, and Office of Air and Radiation
EPA Document Number 43-F-93-003, January 1993

Summary

The U.S. Environmental Protection Agency (EPA) has published a major assessment of the respiratory health risks of passive smoking (Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders EPA/600/6-90/006F). The report concludes that exposure to environmental tobacco smoke (ETS) -- commonly known as secondhand smoke -- is responsible for approximately 3,000 lung cancer deaths each year in nonsmoking adults and impairs the respiratory health of hundreds of thousands of children.

Background

EPA studies of human exposure to air pollutants indicate that indoor levels of many pollutants often are significantly higher than outdoor levels. These levels of indoor air pollutants are of particular concern because it is estimated that most people spend approximately 90 percent of their time indoors.

In recent years, comparative risk studies performed by EPA and its Science Advisory Board have consistently ranked indoor air pollution among the top five environmental risks to public health. EPA, in close cooperation with other federal agencies and the private sector, has begun a concerted effort to better understand indoor air pollution and to reduce peoples' exposure to air pollutants in offices, homes, schools and other indoor environments where people live, work and play.

Tobacco smoking has long been recognized as a major cause of death and disease, responsible for an estimated 434,000 deaths per year in the United States. Tobacco use is known to cause lung cancer in humans, and is a major risk factor for heart disease.

In recent years, there has been concern that non-smokers may also be at risk for some of these health effects as a result of their exposure ("passive smoking") to the smoke exhaled by smokers and smoke given off by the

burning end of cigarettes. As part of its effort to address all types of indoor air pollution, in 1988, EPA's Indoor Air Division (now the Indoor Environments Division) requested that EPA's Office of Research and Development (ORD) undertake an assessment of the respiratory health effects of passive smoking. The report was prepared by ORD's Office of Health and Environmental Assessment.

The document has been prepared under the authority of Title IV of Superfund (The Radon Gas and Indoor Air Quality Research Act of 1986), which directs EPA to conduct research and disseminate information on all aspects of indoor air quality.

Go to top

Public and Scientific Reviews

A draft of this assessment was released for public review in June 1990. In December 1990, EPA's Science Advisory Board (SAB), a committee of independent scientists, conducted a review of the draft report and submitted its comments to the EPA Administrator in April 1991. In its comments, the SAB's Indoor Air Quality/Total Human Exposure Committee concurred with the primary findings of the report, but made a number of recommendations for strengthening it.

Incorporating these recommendations, the Agency again transmitted a new draft to the SAB in May of 1992 for a second review. Following a July 1992 meeting, the SAB panel endorsed the major conclusions of the report, including its unanimous endorsement of the classification of environmental tobacco smoke (ETS) as a Group A (known human) carcinogen.

EPA also received and reviewed more than 100 comments from the public, and integrated appropriate revisions into the final risk assessment.

Go to top

Major Conclusions

Based on the weight of the available scientific evidence, EPA has concluded that the widespread exposure to environmental tobacco smoke in the U.S. presents a serious and substantial public health risk.

In adults:

ETS is a human lung carcinogen, responsible for approximately 3,000 lung cancer deaths annually in U.S. nonsmokers. ETS has been classified as a Group A carcinogen under EPA's carcinogen assessment guidelines. This classification is reserved for those compounds or mixtures which have been shown to cause cancer in humans, based on studies in human populations.

. . .. .

In children:

ETS exposure increases the risk of lower respiratory tract infections such as bronchitis and pneumonia. EPA estimates that between 150,000 and 300,000 of these cases annually in infants and young children up to 18 months of age are attributable to exposure to ETS. Of these, between 7,500 and 15,000 will result in hospitalization.

ETS exposure increases the prevalence of fluid in the middle ear, a sign of chronic middle ear disease.

ETS exposure in children irritates the upper respiratory tract and is associated with a small but significant reduction in lung function.

ETS exposure increases the frequency of episodes and severity of symptoms in asthmatic children. The report estimates that 200,000 to 1,000,000 asthmatic children have their condition worsened by exposure to environmental tobacco smoke.

ETS exposure is a risk factor for new cases of asthma in children who have not previously displayed symptoms.

Go to top

Scope of the Report

In 1986, the National Research Council (NRC) and the U.S. Surgeon General independently assessed the health effects of exposure to ETS. Both of these reports concluded that ETS can cause lung cancer in adult non-smokers and that children of parents who smoke have increased frequency of respiratory symptoms and lower respiratory tract infections. The EPA scientific assessment builds on these reports and is based on a thorough review of all of the studies in the available literature.

Since 1986, the number of studies which examine these issues in human populations has more than doubled, resulting in a larger database with which to conduct a comprehensive assessment of the potential effects which passive smoking may have on the respiratory health of adults as well as children.

Because only a very small number of studies on the possible association between exposure to secondhand smoke and heart disease and other cancers existed in the scientific literature at the time this assessment was first undertaken, EPA has not conducted an assessment of the possible association of heart disease and passive smoking. EPA is considering whether such an assessment should be undertaken in the future, but has no plans to do so at this time.

Go to top

Scientific Approach

EPA reached its conclusions concerning the potential for ETS to act as a human carcinogen based on an analysis of all of the available data, including more than 30 epidemiologic (human) studies looking specifically at passive smoking as well as information on active or direct smoking. In addition, EPA considered animal data, biological measurements of human uptake of tobacco smoke components and other available data. The conclusions were based on what is commonly known as the total weight-of-evidence" rather than on any one study or type of study.

The finding that ETS should be classified as a Group A carcinogen is based on the conclusive evidence of the dose-related lung carcinogenicity of mainstream smoke in active smokers and the similarities of mainstream and sidestream smoke given off by the burning end of the cigarette. The finding is bolstered by the statistically significant exposure-related increase in lung cancer in nonsmoking spouses of smokers which is found in an analysis of more than 30 epidemiology studies that examined the association between secondhand smoke and lung cancer.

The weight-of-evidence analysis for the non-cancer respiratory effects in children is based primarily on a review of more than 100 studies, including 50 recent epidemiology studies of children whose parents smoke.

Go to top

Beyond the Risk Assessment

Although EPA does not have any regulatory authority for controlling ETS, the Agency expects this report to be of value to other health professionals and policymakers in taking appropriate steps to minimize peoples' exposure to tobacco smoke in indoor environments.

In cooperation with other government agencies, EPA will continue its education and outreach program to inform the public and policy makers on what to do to reduce the health risks of ETS as well as other indoor air pollutants.

Go to top

For Further Information

A limited number of copies of the complete report can be obtained free of charge from:

Center for Environmental Research Information (CERI)
U.S. EPA
26 W. Martin Luther King Drive
Cincinnati, OH 45268
Telephone: 513-569-7562
Fax: 513-569-7566
Ordering Number: EPA/600/6-90/006F

The report Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders is available via a series of Adobe Acrobat PDF files from EPA's Office of Research and Development (ORD)

You can also get a copy of the report from:

Indoor Air Quality Information Clearinghouse (IAQ INFO)
P.O. Box 37133,
Washington D.C. 20013-7133
Telephone: 1-800-438-4318 or (703) 356-4020
Fax: (703) 356-5386 or
e-mail: iaqinfo@aol.com

A number of government agencies can provide additional information addressing the health risks of environmental tobacco smoke. These include:

Office on Smoking and Health/Centers for Disease Control
Center for Chronic Disease Prevention and Health Promotion
Mail Stop K-50, 4770 Buford Highway
Atlanta, GA 30341
1-800-CDC-1311

National Cancer Institute
Building 31, Room 10A24
Bethesda, MD 20892
1-800-4-CANCER

The National Heart, Lung, and Blood Institute
Information Center
4733 Bethesda Avenue, Suite 530
Bethesda, MD 20814

National Institute for Occupational Safety and Health
4676 Columbia Parkway
Cincinnati, Ohio 45226-1998
1-800-35-NIOSH

How to Order EPA Publications

EPA publications are available through the IAQ INFO Clearinghouse.

IAQ INFO
P.O. Box 37133, Washington, DC 20013-7133
1-800-438-4318/703-356-4020
(fax) 703-356-5386
iaqinfo@aol.com

or, you can order these publications directly via EPA's **National Service Center for Environmental Publications (NSCEP)** (http://www.epa.gov/ncepihom/). web site. Your publication requests can also be mailed, called or faxed directly to:

U.S. Environmental Protection Agency
National Center for Environmental Publications (NSCEP)
P.O. Box 42419
Cincinnati, OH 42419
1-800-490-9198/(513) 489-8695 (fax)

Please use the EPA Document Number, which is usually bolded or highlighted, when ordering from **NSCEP** or from **IAQ INFO**.

Go to top

Title: Setting the Record Straight: Secondhand Smoke is a Preventable Health Risk

Publication Date: June 1994

Agency or Author: Office of Air and Radiation, Office of Radiation and Indoor Environments Division, Environmental Protection Agency

Purpose: This document provides clarifications and responses on previous EPA documents concerning Environmental Tobacco Smoke (ETS). Also included in this document is more information concerning the epidemiological methodology used by the EPA in their assessment of ETS. This document resolves all challenges brought against the body of evidence and maintains that ETS is known health concern.

"Setting the Record Straight: Secondhand Smoke is a Preventable Health Risk"

Office of Air and Radiation
Office of Radiation and Indoor Air
Indoor Environments Division (6609J)
EPA Document Number 402-F-94-005, June 1994

Contents

Background/Statistics/Conclusions

In early 1993, EPA released a report (Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders; EPA/600/6-90/006 F) that evaluated the respiratory health effects from breathing secondhand smoke (also called environmental tobacco smoke (ETS)). In that report, EPA concluded that secondhand smoke causes lung cancer in adult nonsmokers and impairs the respiratory health of children. These findings are very similar to ones made previously by the National Academy of Sciences and the U.S. Surgeon General.

The EPA report classified secondhand smoke as a Group A carcinogen, a designation which means that there is sufficient evidence that the substance causes cancer in humans. The Group A designation has been used by EPA for only 15 other pollutants, including asbestos, radon, and benzene. Only secondhand smoke has actually been shown in studies to cause cancer at typical environmental levels. EPA estimates that approximately 3,000 American nonsmokers die each year from lung cancer caused by secondhand smoke.

Every year, an estimated 150,000 to 300,000 children under 18 months of age get pneumonia or bronchitis from breathing secondhand tobacco smoke. Secondhand smoke is a risk factor for the development of asthma in children and

worsens the condition of up to one million asthmatic children.

EPA has clear authority to inform the public about indoor air pollution health risks and what can be done to reduce those risks. EPA has a particular responsibility to do everything possible to warn of risks to the health of children.

A recent high profile advertising and public relations campaign by the tobacco industry may confuse the American public about the risks of secondhand smoke. EPA believes it's time to set the record straight about an indisputable fact: secondhand smoke is a real and preventable health risk.

EPA absolutely stands by its scientific and well documented report. The report was the subject of an extensive open review both by the public and by EPA's Science Advisory Board (SAB), a panel of independent scientific experts. Virtually every one of the arguments about lung cancer advanced by the tobacco industry and its consultants was addressed by the SAB. The panel concurred in the methodology and unanimously endorsed the conclusions of the final report.

The report has also been endorsed by the U.S. Department of Health and Human Services, the National Cancer Institute, the Surgeon General, and many major health organizations.

Go to top

Classification of Secondhand Smoke as a Known Human (Group A) Carcinogen

The finding that secondhand smoke causes lung cancer in nonsmoking adults is based on the total weight of the available evidence and is not dependent on any single analysis. This evidence includes several important facts.

First, it is indisputable that smoking tobacco causes lung cancer in humans, and there is no evidence that there is a threshold below which smoking will not cause cancer.

Second, although secondhand smoke is a dilute mixture of mainstream" smoke exhaled by smokers and sidestream" smoke from the burning end of a cigarette or other tobacco product, it is chemically similar to the smoke inhaled by smokers, and contains a number of carcinogenic compounds.

Third, there is considerable evidence that large numbers of people who do not smoke are exposed to, absorb, and metabolize significant amounts of secondhand smoke.

Fourth, there is supporting evidence from laboratory studies of the ability of secondhand smoke both to cause cancer in animals and to damage DNA, which is recognized by scientists as being an instrumental mechanism in cancer development.

Finally, EPA conducted multiple analyses on the then-available 30 epidemiology studies from eight different countries which

examined the association between secondhand smoke and lung cancer in women who never smoked themselves but were exposed to their husband's smoke. Since the epidemiology studies are the major thrust of the tobacco industry arguments against the EPA report, these studies are examined in more detail below.

Go to top

The Epidemiology Studies

The most important aspect of the review of the epidemiology studies is the remarkable consistency of results across studies that support a causal association between secondhand smoke and lung cancer.

In assessing the studies several different ways, it becomes clear that the extent of the consistency defies attribution to chance. When looking only at the simple measure of exposure of whether the husband ever smoked, 24 of 30 studies reported an increase in risk for nonsmoking women with smoking husbands. Since many of these studies were small, the chance of declaring these increases statistically significant was small. Still, nine of these were statistically significant, and the probability that this many of the studies would be statistically significant merely by chance is less than *1 in 10 thousand*.

The simple overall comparison of risks in ever vs. never exposed to spousal smoking tends to hide true increases in risk in two ways. First, it categorizes many women as never exposed who actually received exposure from sources other than spousal smoking. It also includes some women as exposed who actually received little exposure from their husband's smoking. One way to correct for this latter case is to look at the women whose husbands smoked the most. When one looks at the 17 studies that examined cancer effects based on the level of exposure of the subjects, every study found an increased lung cancer risk among those subjects who were most exposed. Nine were statistically significant. The probability of 9 out of 17 studies showing statistically significant results occurring by chance is less than *1 in ten million.*

Probably the most important finding for a causal relationship is one of increasing response with increasing exposure, since such associations cannot usually be explained by other factors. Such exposure-response trends were seen in all 14 studies that examined the relationship between level of exposure and effect. In 10 of the studies the trends were statistically significant. The probability of this happening by chance is less than *1 in a billion*.

It is unprecedented for such a consistency of results to be seen in epidemiology studies of cancer from environmental levels of a pollutant. One reason is that it is extremely difficult to detect an effect when virtually everyone is exposed, as is the case with secondhand smoke. However, consistent increased risks for those most exposed and consistent trends of increasing exposure showing an increasing effect provide strong evidence that secondhand smoke increases the risk of lung cancer in nonsmokers.

Go to top

How Big a Lung Cancer Risk for Adults?

The evidence is clear and consistent: secondhand smoke is a cause of lung cancer in adults who don't smoke. EPA has never claimed that minimal exposure to secondhand smoke poses a huge individual cancer risk. Even though the lung cancer risk from secondhand smoke is relatively small compared to the risk from direct smoking, unlike a smoker who chooses to smoke, the nonsmoker's risk is often involuntary. In addition, exposure to secondhand smoke varies tremendously among exposed individuals. For those who must live or work in close proximity to one or more smokers, the risk would certainly be greater than for those less exposed.

EPA estimates that secondhand smoke is responsible for about 3,000 lung cancer deaths each year among nonsmokers in the U.S.; of these, the estimate is 800 from exposure to secondhand smoke at home and 2,200 from exposure in work or social situations.

Go to top

The Risks to Children are Widely Acknowledged

The conclusion that secondhand smoke causes respiratory effects in children is widely shared and virtually undisputed. Even the tobacco industry does not contest these effects in its media and public relations campaign.

EPA estimates that every year, between 150,000 and 300,000 children under 1-1/2 years of age get bronchitis or pneumonia from breathing secondhand tobacco smoke, resulting in thousands of hospitalizations. In children under 18 years of age, secondhand smoke exposure also results in more coughing and wheezing, a small but significant decrease in lung function, and an increase in fluid in the middle ear. Children with asthma have more frequent and more severe asthma attacks because of exposure to secondhand smoke, which is also a risk factor for the onset of asthma in children who did not previously have symptoms.

Go to top

Other Risks

Secondhand smoke contains strong irritants and sensitizers and many adults, as well as children, suffer irritation and other acute effects whenever they are exposed to secondhand smoke. In addition, there is mounting evidence that exposure to secondhand smoke can have an effect on the cardiovascular system, although the EPA report does not address this issue.

Go to top

Tobacco Industry Media Campaign

The tobacco industry is raising numerous issues which may distract the public from the fact that secondhand smoke poses a real and preventable health risk.

The tobacco industry neither acknowledges nor disputes EPA's conclusions of respiratory effects in children. It focuses instead on EPA's findings on lung cancer.

The overall thrusts of the tobacco industry's arguments are that EPA manipulated the lung cancer data to come to a predetermined conclusion. The industry also argues that a nonsmoker's exposure to secondhand smoke is so small as to be insignificant. The argument on minimal exposure is belied both by the acute irritation and respiratory effects and the fallacy of the cigarette equivalents" approach discussed below. Responses to the specific criticisms of EPA's assessment of the lung cancer data follow.

Go to top

The 11 U.S. Lung Cancer Studies

Critics of the EPA report argue that by normal statistical standards, none of the 11 U.S. studies included in the EPA report showed a statistically significant increase in the simple overall risk measure, and that EPA should therefore have been unable to conclude that secondhand smoke causes lung cancer in nonsmokers. These critics are misrepresenting a small part of the total evidence on secondhand smoke and lung cancer.

The consistency of study results in the highest exposure category and exposure-response trends discussed above also apply to the U.S. studies. For example, seven of the 11 U.S. studies had fewer than 45 cases, making statistical comparisons difficult. Nonetheless, eight of the 11 had increased overall risks, and for the seven studies which reported on risks by amount of exposure, the highest exposure groups in all seven had increased risks. While the 11 U.S. studies are not, by themselves, conclusive, they do support the conclusion that secondhand smoke is causally associated with lung cancer.

Go to top

Studies Completed Since Release of the EPA Report

Critics claim that had EPA not excluded" the recent Brownson study, the Agency could not have concluded that secondhand smoke causes cancer. In fact, four new lung cancer epidemiology studies, including the Brownson study, have been published since the literature review cutoff date for the 1993 EPA report, and all support EPA's conclusions. Three of these are large U.S. studies funded, at least in part, by the National Cancer Institute. A 1992 study of Florida women by Stockwell et al. found a 60% overall increased risk of lung cancer from exposure to their husband's smoke, with significant results for both the highest exposure group and the exposure-response trend. The 1992 study of Missouri women by Brownson et al. found no overall increased risk, but did demonstrate a significant increase in risk in the highest spousal smoking exposure group and a positive exposure-response trend.

The 1994 study by Fontham et al. of women in two California and three Southern cities is the largest case-control study on the subject ever conducted and is considered by EPA to be the best designed study on secondhand smoke and lung cancer conducted to date. This study found significantly increased risks for

overall exposure and in the highest exposure group and a strong positive exposure-response relationship. These findings were significant not only for exposure from spouses, but also for exposure in the workplace and in social situations.

Go to top

90% vs. 95% Confidence Intervals

Critics of the EPA report have charged that EPA changed the confidence interval in order to come to a predetermined conclusion. However, the conclusion that secondhand smoke is a known human carcinogen simply does not hinge on whether or not a 95% or 90% confidence interval" was used. A confidence interval is used to display variability in relative risk estimates in the epidemiology studies. As discussed above, the Group A designation is based on the total weight of the available evidence. The consistency of results that are seen in the numerous studies examined lead to a certainty of greater than 99.9% that secondhand smoke increases the risk of lung cancer in nonsmokers.

Use of what is called in statistics a one-tailed test of significance," which often corresponds to a 90% confidence interval, is a standard and appropriate statistical procedure in certain circumstances. The one-tailed test" is used when there is prior evidence that if there is an effect from a substance, it is highly likely to be an adverse rather than a protective effect, or vice versa. In the case of secondhand smoke, an extensive database exists for direct smoking indicating that if chemically similar secondhand smoke also has a lung cancer effect, this effect is likely to be similarly adverse. EPA used one-tailed significance tests for lung cancer in both external drafts of the risk assessment document as well as the final report. Ninety percent confidence intervals were also used in other EPA cancer risk assessments, including methylene chloride, coke oven emissions, radon, nickel, and dioxin.

In the non-cancer respiratory effects portions of the report, two-tailed tests" and 95% confidence intervals were used, since there was less prior evidence from smokers to suggest that secondhand smoke would cause bronchitis, pneumonia, and ear infections in children.

Go to top

The Meta-analysis

Meta-analysis was used for the lung cancer data as an objective method of combining results from many studies and was specifically endorsed by the SAB for use with this database. Some critics argue both that the meta-analysis was not an appropriate technique, and that had EPA included the Brownson study (addressed above) in the meta-analysis of overall spousal exposure, EPA could not possibly have classified secondhand smoke as a known human carcinogen. This just isn't true.

The finding that secondhand smoke is a known cause of lung cancer in humans is based on all the evidence and is not dependent on the meta-analysis of the simple ever- vs. never-exposed comparisons, as the critics suggest. If the meta-analysis were removed from the report entirely, the findings would be precisely

the same. The meta-analysis was used primarily for estimating and quantifying the population risks from exposure to secondhand smoke, and an alternative approach also used in the report gave very similar results.

Go to top

Confounders

In the secondhand smoke report, a confounder would be a specific factor that could be responsible for the lung cancer increases observed in nonsmokers instead of secondhand smoke. The tobacco industry and its consultants have suggested, for example, that nonsmoking wives might share in the same poor dietary habits as their smoking husbands, increasing their risk.

The consistency of results across different countries where lifestyle factors, including diet, vary, argues against confounding. For example, while the tobacco industry theorizes that a high fat diet is a confounding factor, the studies from Japan, where dietary fat intake is among the lowest in the world, show a strong dose-response relationship for secondhand smoke and lung cancer.

The EPA report did examine the available data for six potential confounders such as occupation, dietary factors, and history of lung disease, and concluded that none was likely to explain the lung cancer increases seen in the studies.

The 1994 Fontham et al. study controlled for diet and other potential confounders, and concluded, These observations indicate that the strong association in this study between adult secondhand smoke exposure and lung cancer risk cannot be attributed to any likely confounder.

Go to top

"The Threshold Theory"

Although some have argued that tobacco smoke cannot cause cancer below a certain level, there is no evidence that this threshold exists. In the absence of such evidence, carcinogens at any level are considered by EPA to increase risk somewhat, although the degree of risk certainly is reduced as exposure decreases. The increased risks observed in the secondhand smoke epidemiology studies are further evidence that any threshold for secondhand smoke would have to be at very low levels.

Go to top

"Cigarette Equivalents"

The tobacco industry uses the cigarette equivalent" method of comparing smokers' and nonsmokers' exposures to a single component of tobacco smoke to infer that a nonsmoker's exposure to tobacco smoke is insignificant. However, the cigarette equivalent method has no scientific support, and was rejected by the SAB panel that reviewed the EPA report. Among the many problems with this method is the fact that while secondhand smoke and mainstream smoke contain the same approximately 4,000 compounds, their ratios of individual compounds

differ by factors in the thousands. Thus, there is no single compound in tobacco smoke that is an adequate indicator for drawing such comparisons. An RJ Reynolds newspaper ad, while utilizing the method, acknowledges it may not be relevant for assessing risk from secondhand smoke.

Go to top

Residential Exposures Translated to the Workplace

The tobacco industry frequently argues that because most studies were based on residential exposures, secondhand smoke has not been shown to be a hazard in the workplace. A substance capable of causing cancer in one environment is certainly capable of causing it in any other environment where exposures are comparable, as is the case with residential and workplace exposure to secondhand smoke. In fact, the 1994 Fontham study found a slightly higher risk for workplace exposure than for residential exposures.

Go to top

The Congressional Research Service (CRS) Report

The RJ Reynolds' media campaign cites a report prepared by the Congressional Research Service (CRS) on cigarette taxes to fund health care reform to argue that CRS believes that the epidemiological evidence on secondhand smoke and health effects is weak and uncertain." However, CRS has not taken a position on either EPA's risk assessment or the health effects of passive smoking.

Two economists from CRS, citing material largely prepared by the tobacco industry, included a discussion of EPA's risk assessment in an economic analysis of a cigarette excise tax proposal to fund health care reform. In EPA's view, the CRS economists' cursory look at the issues is not comparable to the exhaustive analyses and rigorous review process which EPA undertook when examining the extensive database on secondhand smoke and respiratory health. EPA is confident that a comprehensive analysis of the secondhand smoke database by expert scientists from CRS, with adequate peer review, will come to conclusions about the risks of secondhand smoke similar to those of EPA and many other organizations.

Go to top

Cigarette Prohibition

The claim that the government is attempting to bring back prohibition -- this time for cigarettes -- is a complete fabrication and utter nonsense. EPA's interest is to provide information to protect the nonsmoker from involuntary exposure to a hazardous substance. Having a choice to take a risk for themselves should not permit smokers to impose a risk on others.

Go to top

For Further Information

For additional information on secondhand smoke and other indoor air pollutants, contact EPA's:

Indoor Air Quality Information Clearinghouse [IAQ INFO]
at 1-800-438-4318 or
(703) 356-4020 in Washington Metro area
PO Box 37133
Washington DC 20013-7133
(703) 356-5386 (fax)
iaqinfo@aol.com

How to Order EPA Publications

EPA publications are available through the IAQ INFO Clearinghouse.

IAQ INFO
P.O. Box 37133, Washington, DC 20013-7133
1-800-438-4318/703-356-4020
(fax) 703-356-5386
iaqinfo@aol.com

or, you can order these publications directly via EPA's **National Service Center for Environmental Publications (NSCEP)** (http://www.epa.gov/ncepihom/). web site. Your publication requests can also be mailed, called or faxed directly to:

U.S. Environmental Protection Agency
National Center for Environmental Publications (NSCEP)
P.O. Box 42419
Cincinnati, OH 42419
1-800-490-9198/(513) 489-8695 (fax)

Please use the EPA Document Number, which is usually bolded or highlighted, when ordering from **NSCEP** or from **IAQ INFO**.

Go to top

Title: Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant

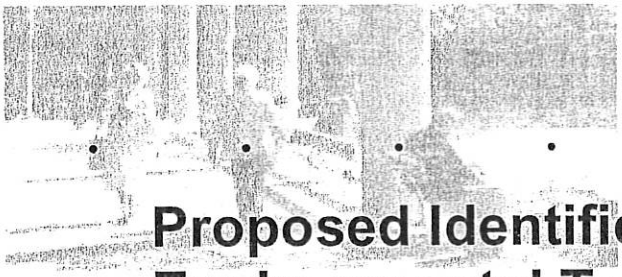
Publication Date: March 2005

Agency or Author: Air Resources Board, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency

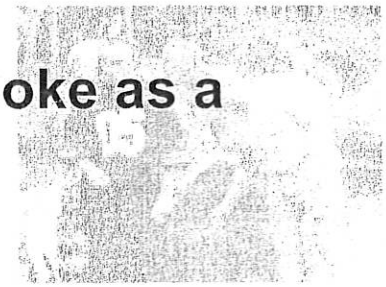
Purpose: Executive summary of proposal to identify Environmental Tobacco Smoke (ETS) as a toxic air contaminant. Document address exposure information for California specific population, potential health impacts of ETS in California, and provides evidence to identify ETS as a toxic air contaminant as defined in California's Assembly Bill 1807. The full document is also available to view at their website (http://www.arb.ca.gov/toxics/ets/dreport/dreport.htm) or on the attached data CD.

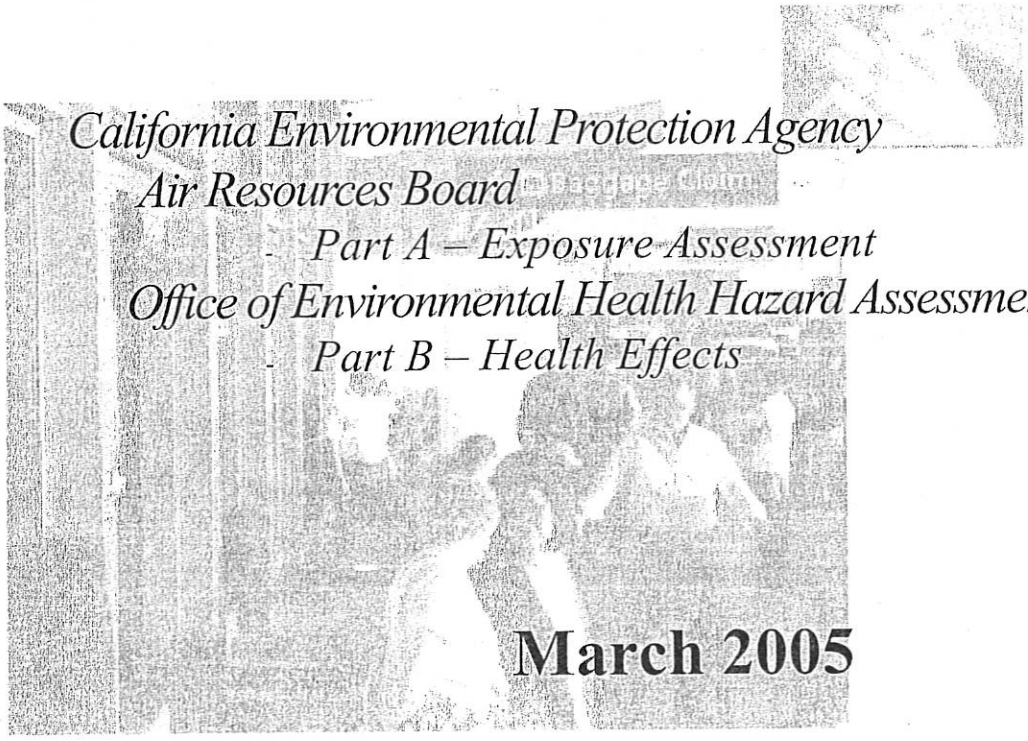
State of California

Executive Summary



Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant





California Environmental Protection Agency

Air Resources Board

- *Part A – Exposure Assessment*

Office of Environmental Health Hazard Assessment

- *Part B – Health Effects*

March 2005

EXECUTIVE SUMMARY

For the "Proposed Identification of Environmental Tobacco Smoke as a Toxic Air Contaminant"

California Environmental Protection Agency
Air Resources Board
Office of Environmental Health Hazard Assessment

Introduction

In 1983, the State of California established a program to identify the health effects of toxic air contaminants (TACs) and to reduce exposure to these contaminants to protect the public health (Assembly Bill 1807: Health and Safety Code sections 39650-39674). The program includes a two-step process to address the potential health effects from TACs. The first step involves the evaluation of a substance, by the Air Resources Board (ARB) and the Office of Environmental Health Hazard Assessment (OEHHA), to determine if it is toxic and to estimate public exposure. This step is the risk assessment (or identification) phase. Under state law, the ARB is authorized to identify a substance as a TAC if it determines the substance is "an air pollutant which may cause or contribute to an increase in mortality, in serious illness, or which may pose a present or potential hazard to human health (Health and Safety Code section 39655)."

The second step, determining the need for and appropriate degree of control measures, occurs only if the ARB identifies the substance as a toxic air contaminant. This step is the risk management (or control) phase of the process (Health and Safety Code sections 39665 and 39666). This report does not address the need for control measures to reduce ETS exposure, nor contain any recommendations in that regard.

The ARB and the OEHHA are evaluating environmental tobacco smoke (ETS) as a candidate toxic air contaminant under the State's air toxics identification program. This report presents the information upon which this assessment is based.

What is Contained in This Report?

This report, prepared by the staff of the Air Resources Board (ARB) and the Office of Environmental Health Hazard Assessment (OEHHA), presents an evaluation of exposures to environmental tobacco smoke and the potential health effects associated with these exposures.

Part A of the report, prepared by the staff of the ARB, addresses the exposures to ETS in California. Some of the information in this document is based on data presented in the OEHHA's 1997 report: "Health Effects of Exposure to Environmental Tobacco Smoke." Specifically, Chapter 2 (Exposure Measurement and Prevalence) of the

OEHHA report was updated to include ETS exposure information developed subsequent to the data presented in the report.

Part B of the report, prepared by the staff of the OEHHA, evaluates the potential health impacts from exposures to ETS. In this document, information from their 1997 report, which was later published by the U.S. National Cancer Institute in 1999, has been updated to include more recent literature. OEHHA's evaluation includes numerous published papers on ETS-related health effects since their initial 1997 ETS review.

Part C of the report, prepared by both ARB and OEHHA staff, addresses the comments received on the first public version of the report. The Part C document contains the staff responses to comments and the comment letters.

The Part A and B of this report will serve as the basis for the identification of ETS as a toxic air contaminant (TAC) under the authority of California's TAC Program (Assembly Bill 1807: Health and Safety Code Sections 39660-39662).

How Does the ARB Identify a Substance as a TAC?

With input from the public, industry, and the scientific community, the ARB and the OEHHA gather all of the relevant scientific information on a substance. Under the requirements of law (Health and Safety Code sections 39660-39662), the ARB and OEHHA must answer the following questions:

1. Is the substance used in California?
2. Who is exposed to the substance?
3. How many people are exposed?
4. How much is emitted into the air?
5. How long does the substance stay in the air?
6. How much of a substance can be measured in the air?
6. Does exposure to the substance cause increased health impacts in children?
7. Does the substance pose a potential health risk to Californians?

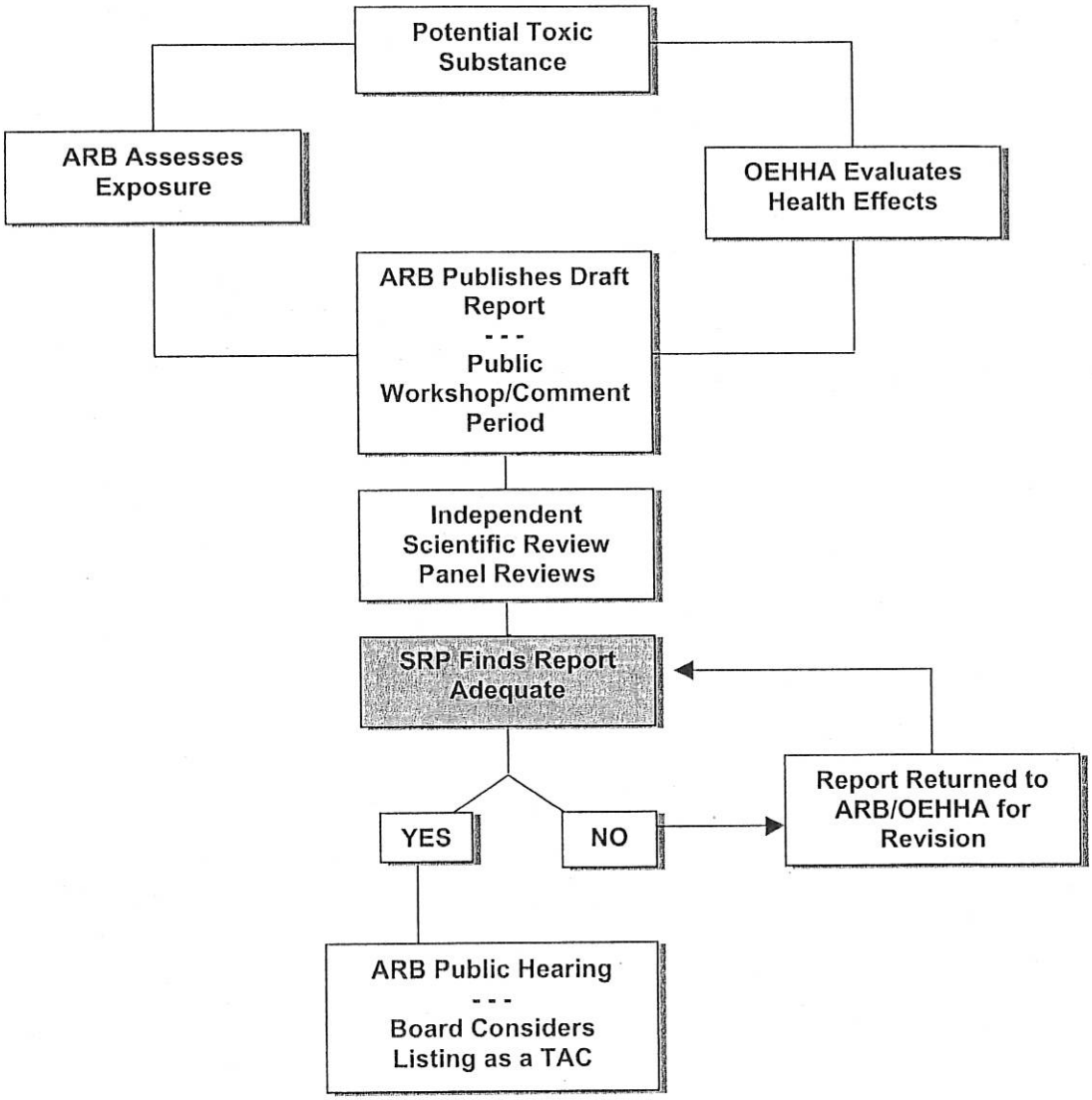
The ARB staff determines the public's potential exposure to the substance while the OEHHA must determine if exposure to the substance poses a potential health risk. Both agencies then prepare a draft report which serves as the basis for identifying a substance.

Once the draft report is released, the public review process begins. The public review is a critical step in identifying a substance. After the release of the report, a workshop is held to discuss the report during a formal comment period. After receiving public comments, both verbal and written, we carefully review all comments, incorporate new information, and revise the report where appropriate.

After the comment period and public workshop, the report is then submitted to the Scientific Review Panel (SRP) on Toxic Air Contaminants. The SRP is an independent group of scientists, who review the report for its scientific accuracy. If the SRP

determines that the report is not based on sound scientific information, it is sent back to the staff for revisions. If the SRP approves the revised report, the SRP prepares its "findings" which are submitted, along with the staff report, to the ARB for consideration at a public hearing. The Board then decides whether to identify a substance as a TAC (see illustration below). If the substance is identified as a TAC, it is listed in Title 17 of the California Code of Regulations under section 93000.

The Identification Process



What Happens When a Substance is Identified as a TAC by the Air Resources Board?

After a substance is identified as a TAC, the ARB is required to conduct a needs analysis to determine if any regulatory action is warranted. Specifically, the law requires the ARB to prepare a report which assesses the need and appropriate degree of control of a TAC, in consultation with the local districts, affected industry, and the public.

Where is Environmental Tobacco Smoke in the Toxic Air Contaminant Process?

Environmental Tobacco Smoke has undergone a thorough and extensive evaluation since it entered the identification program in June 2001. In December 2003, the draft report, which included the Executive Summary, Part A (exposure assessment), and Part B (health assessment), was released to the public for a three month comment period. In March 2002, a public workshop was held to discuss the report. On November 30, 2004, the SRP held a meeting to discuss the report and the comments received on the draft report (Part C - responses to public comments). The meeting was continued on January 6, 2005. On March 14, 2005, the SRP held a third meeting to discuss the draft report and to develop their findings.

What is Environmental Tobacco Smoke?

Environmental Tobacco Smoke is a complex mixture of thousands of gases and fine particles emitted by the burning of tobacco products and from smoke exhaled by the smoker. Other minor contributors are from the smoke that escapes while the smoker inhales and some vapor-phase related compounds that diffuse from the tobacco product. The composition will vary depending on the heat of combustion, the tobacco content, additives present, and the type of filter material used.

Many of the substances found in ETS have known adverse health effects. The table below lists some of these compounds.

Some Substances in Environmental Tobacco Smoke with Known Adverse Health Effects

1,3-butadiene	Chromium VI
2-Naphthylamine	Ethyl benzene
4-Aminobiphenyl	Formaldehyde
4-nitrobiphenyl	Hydrazine
Acetaldehyde	Methyl chloride
Acrolein	N-Nitrosonornicotine
Aniline	Nickel
Arsenic (inorganic)	Nicotine
Benzene	NNK
Benz[a]anthracene	Phenol

Benzo[a]pyrene	Styrene
Cadmium	Toluene

The size of ETS particles range from 0.01 to about 1 *u*m. Freshly produced ETS undergoes complex atmospheric changes such as coagulation, evaporation, dilution and condensation. However, ETS fine particles essentially remain below 1 *u*m in size.

What are the Total ETS Emissions in California?

ETS emissions were characterized using the most widely measured components of ETS: nicotine, respirable particulate matter (RSP), and carbon monoxide (CO). Total emissions, as a result of combustion of tobacco products, were estimated using data from the California Tobacco Surveys, emission rates from the scientific literature, and cigarette sales data from the State Board of Equalization.

2002 California Statewide ETS Emissions (tons/year)

	Cigarettes	Cigars	Total
Nicotine	36	4	40
RSP	335	30	365
CO	1475	432	1907

How much ETS is Emitted Outdoors in California?

The amount of ETS emitted into the outdoor environment depends in large part on the smoking public's behavior. Outdoor ETS emissions would include direct emissions from outdoor smoking, plus ETS emissions generated indoors which eventually ventilate outside. Apportioning ETS emissions as either outdoor or indoor emissions is difficult to determine due to limited information. However, existing information shows that most smoking in California occurs outdoors. This is demonstrated by the fact that most workplaces (including bars and restaurants) in California, through the enactment of Assembly Bill 13 (AB13) in 1998, are now smoke-free. In addition, data from the 2002 California Adult Tobacco Survey (CATS), shows that over 80% of all California homes with children are now smokefree and that about 50% of California smokers report that they do not smoke in their own homes. For ETS generated indoors, building ventilation studies show that 50 – 80 percent of indoor air (including ETS constituents) gets exchanged with outdoor air. From this information, the ARB staff estimates that at least 80% of total ETS emissions are emitted to the outdoor environment.

What is the Prevalence of Smokers in California?

The California Tobacco Survey (CTS), developed by the California Department of Health Services (CDHS), indicates that during the past decade, smoking prevalence among adults (over age 18) and adolescents (12 to 17 years) has gradually decreased.

Starting in 2001, CDHS began measuring adolescent prevalence through their California Student Tobacco Survey (CSTS). The CSTS was incorporated by CDHS since it samples school populations and provides better statistical accuracy. The most recent CTS and CSTS surveys show that both the adult (2002 data) and adolescent (2001 data) smoking prevalence is about 16%. The CSTS data also shows that the range of adolescent smokers varies from 10% in 9th grade to 23% in 12th grade.

How does California Compare to the Rest of the Nation?

Since the passage of Proposition 99 in 1988, the annual adult per capita cigarette consumption has declined by over 60% in California. Adult smoking prevalence in California has dropped at a faster rate relative to the rest of the nation.

Comparison of Reduction in Cigarette Consumption: California versus U.S.

Fiscal Year	1987/1988 (packs per adult)	2001/2002 (packs per adult)	% Decline
California	126.6 packs	47.7 packs	62.3
United States	154.8 packs	99.2 packs	35.9

What is the Prevalence of ETS Exposure in California?

Smoking behavior and other factors that change smoking patterns such as smoking regulations, affect present and future exposure patterns. Information from several smoking behavior related surveys indicate that California's adults, adolescents, and children are exposed to ETS during some time of the day.

According to studies from the late 1980s and the early 1990s, on a given day, 56% of adults (over age 18), 64% of adolescents (12-17 years), and 38% of children (0-11 years), may be exposed to ETS during their daily activity. Actual incidence may be lower today due to decreases in workplace smoking and in public locations such as restaurants, bars, and gaming clubs due to California smoking restrictions.

How do we Measure ETS Exposure in the Environment?

Exposure to ETS is difficult to characterize because it is a complex mixture of substances and the difficulty in determining an appropriate marker that is representative of ETS as a whole. Given its complex nature, it is necessary to select a surrogate measure of exposure that is representative of ETS as a whole.

Several components of ETS have been studied as surrogates or markers for ETS. Nicotine has been most widely studied as a potential marker because its only major source is tobacco smoke. Other ETS markers that have been studied include: solanesol, 3-ethenylpyridine (3-EP), carbon monoxide, iso- and anteisoalkanes (C_{29}-C_{34}), PAHs, fluorescing particulate matter, respirable suspended particles, and ultraviolet particulate matter.

Are there Studies that have Determined Outdoor Air Concentrations of ETS?

Yes. There are studies that have either measured or modeled outdoor air concentrations of ETS constituents. One study estimated concentrations of fine smoke particles in the Los Angeles air using tobacco-specific iso- and anteisoalkanes. Using the measurements from these marker compounds, the annual average ambient fine (less than 2.5 microns) ETS particles in the Los Angeles air was estimated to range from 0.28 to 0.36 microgram of ETS particle per cubic meter of air ($\mu g/m^3$). The levels were based on annual measurement data from 1982. Another study used personal badge monitors to directly measure ambient nicotine levels. This study reported a 7-day median nicotine concentration in the outdoor environment of 0.025 $\mu g/m^3$.

One study used a chemical mass balance receptor model based on organic compounds to estimate source contributions to fine particle mass concentrations in the Los Angeles air. The modeled annual average concentration for the Los Angeles air was estimated to be 0.21 µg/m3 fine ETS particulate matter in 1982.

Has the ARB Measured Outdoor Concentrations of ETS?

Yes. To obtain data on current levels of ETS in ambient air where people spend part of their day, the ARB monitored nicotine concentrations at several outdoor smoking areas in California. The study gathered two 8-hour samples and six 1-hour samples per site tested. Depending on the site location and number of smokers present, the results showed a range of nicotine concentrations from 0.013-3.1 $\mu g/m^3$ for the 8-hour samples and 0.016-4.6 $\mu g/m^3$ for the 1-hour measurements. Overall, the results indicate that concentrations of nicotine corresponded mainly to the number of smokers in the smoking areas, the size of the smoking area and meteorological conditions.

What are the Outdoor Air Levels of ETS that Most Californians Breathe?

Although a scenario-based approach was used to characterize the range of the public's exposure to ETS in this report, Californians who neither smoke nor associate with many smokers will have limited ETS exposure. In this case, individuals will likely experience the majority of their lifetime ETS exposure from background levels of ETS which result from occasional or steady state near-source emissions. Since most Californians live and work in urban areas, the ARB staff has estimated an outdoor annual average ambient ETS particle concentration for the Los Angeles air for 2003. The staff used the two Los Angeles studies discussed above as a basis for this estimate. The staff applied an adjustment factor to the 1982 fine particle estimates presented in the two Los Angeles studies to reflect reductions in cigarette sales and cigarette emission rates that have occurred since 1982. The results show that estimated annual average fine ETS particle concentrations in Los Angeles in 2003 likely decreased to between 0.06 to

0.10 µg/m^3. The table below summarizes the outdoor air concentration data for ETS nicotine and fine particles from all outdoor estimates.

Estimates of ETS Outdoor Ambient Concentrations

Method/Reference	Data Year	Concentrations (µg/m^3)	
		Fine $PM_{2.5}$	Nicotine
Fine PM – Source Apportionment Schauer *et al.*, 1996	1982	0.21 µg/m^3 annual average	*0.026 µg/m^3 annual average
Iso- and anteisoalkanes – measurement Rogge *et al.*, 1994	1982	0.28 – 0.36 µg/m^3 annual average	*0.035 – 0.044 µg/m^3 annual average
Nicotine – measurement Eisner *et al.*, 2001	2001	*0.20 µg/m^3 7-day median conc.	0.025 µg/m^3 7-day median conc.
Nicotine – measurement ARB, 2003	2003	*0.11 – 25 µg/m^3 8-hour range *0.073 – 0.97 µg/m^3 8-hour background	0.013 – 3.1 µg/m^3 8-hour range 0.009 – 0.12 µg/m^3 8-hour background
Los Angeles background - Estimate ARB, 2004	2003	0.06 – 0.10 µg/m^3 annual average	*0.008 - 0.013 µg/m^3 annual average

* Calculated value using: PM2.5/Nicotine concentration = 8

Are There Estimates of Indoor Air Exposure to ETS?

Yes. Several studies have estimated ETS levels in different indoor environments using nicotine and respirable particulate matter (RSP) as markers for ETS exposure. Current indoor concentrations of nicotine in California are estimated to range from 0.5 to 6.0 µg/m^3 in the home environment, 2-8 µg/m^3 in offices or public buildings where smoking is permitted, and less than 1 µg/m^3 in public buildings where smoking is prohibited. However, certain workplaces, such as the documented 20% of free-standing bars that do not comply with California's workplace smoking ban, would likely have higher levels of ETS. Based on measurements from several studies, levels could range from 9.8 µg/m^3 in betting establishments to 76.0 µg/m^3 for bingo parlours. RSP concentrations are estimated to range from less than 15 µg/m^3 where smoking is prohibited to about 300 µg/m^3 in the home environment where one cigarette is being smoked.

How do we Estimate the California Public's Exposure to ETS?

An individual's exposure depends on the air concentration of a pollutant in a given environment, and the time they spend in that environment. An individual's total daily exposure is the sum of all the exposures they experience across their 24-hour day, including both indoor and outdoor environments.

A scenario-based approach was used to characterize the range of the public's exposure to ETS during a 24-hour period. The scenario-based exposure method uses the results from ARB's ETS air monitoring study, available indoor ETS concentration data, and scenario-based activity patterns to estimate exposures under different situations. The results show a wide range of possible population subgroup daily exposures. For individuals living in non-smoking homes and having only brief encounters with ETS, their 24-hour exposures are low, about 1 μg-hr/m^3. For those living in homes with indoor smokers and experiencing in-vehicle exposures, their integrated 24-hour exposure estimate can range up to 81 μg-hr/m^3. Such exposures are especially of concern for young children because they are likely to recur daily and may adversely affect the physiological sensitivity of developing children.

This approach differs from previous TAC exposure assessments, which were based on California population-weighted exposures to outdoor average ambient concentrations. That approach was appropriate for TACs emitted from area-wide or region-wide sources such as motor vehicles and industrial plants. However, cigars and cigarettes, the primary source of ETS, are smaller sources that emit pollutants near people and thereby exposures to ETS are very localized. Therefore, since exposures are localized and ETS is not monitored at ambient monitoring stations, we believe the scenario-based approach provides better and more informative estimates of public exposure to ETS.

The primary and often the only exposure for individuals that do not spend time near smokers, exposure occurs outdoors in locations over which the individual typically has little control. For non-smokers whose work or other activities bring them into contact with outdoor smokers regularly, 100% of their exposure can be attributable to proximity to outdoor smoking.

Are There Other Methods for Estimating Human Exposure to ETS?

One of the most accurate methods for estimating ETS exposure in a person is through the use of biological markers. Biological markers of ETS exposure are metabolites of tobacco smoke ingredients found in physiological fluids or attached to DNA or proteins. The ability to quantify exposure objectively is an important step in linking exposure to relative risk of adverse outcomes.

Cotinine, a metabolite of nicotine, is the biological marker of choice in most epidemiological studies. Physiological fluid levels correlate very well with ETS exposure documented both by questionnaire and by personal exposure monitoring. Cotinine levels differ between smokers and ETS-exposed non-smokers by 2 to 3 orders of magnitude. From an epidemiological perspective, this difference is useful to determine when people misrepresent their smoking status. Cotinine assays are sensitive enough that individuals without ETS exposure can be distinguished from those persons with low exposure.

The nicotine concentration in hair is emerging as another viable biological marker of ETS exposure. In some instances, hair nicotine has been shown to better correlate with exposure than cotinine, especially where exposure is highly episodic.

What is the Persistence of ETS in the Atmosphere?

Gaseous chemicals that are present in ETS can react in the atmosphere with other pollutants and sunlight to form new chemical species. The ETS particles and particle-associated chemicals (those with low vapor pressure that deposit or chemically bind onto the particles) are subject to wet and dry deposition and atmospheric transformation of species adsorbed to the particles.

Nicotine, the principal alkaloid in tobacco, is most commonly found in the gas phase in the environment. In the ambient air, nicotine may react with hydroxyl radicals to have a half-life of approximately one day.

What are the Health Effects Associated with Exposure to ETS?

ETS exposure is causally associated with a number of health effects, including effects on infants and children. ETS has a number of serious impacts on children's health including sudden infant death syndrome (SIDS), exacerbation of asthma, increased respiratory tract infections, increased middle ear infections, and causes developmental toxicity resulting in low birth weight, and impaired lung function growth, predisposition to SIDS (to the extent that this is a developmental effect), and other developmental impacts.

Listed in Table ES.1 are the developmental, respiratory, carcinogenic and cardiovascular effects for which there is sufficient evidence of a causal relationship, including fatal outcomes such as sudden infant death syndrome and heart disease mortality, cancers of the lung and other organs, as well as serious chronic diseases such as childhood asthma. There are, in addition, effects for which evidence is suggestive of an association but further research is needed for confirmation. These include spontaneous abortion, cervical cancer, decreased female fertility, and chronic respiratory symptoms in adults (Table ES.1). Finally, it is not possible to judge on the basis of the current evidence the impact of ETS on a number of endpoints, including congenital malformations, male reproductive effects, and rare childhood cancers.

Many Californians are exposed to ETS, and the number of people adversely affected may be correspondingly large. Table ES.2 presents morbidity and mortality estimates for health effects causally associated with ETS exposure. Derivation of these estimates is described further in Part B.

Relative risk estimates associated with some of these endpoints are small, but because the diseases are common the overall impact can be quite large. A relative risk estimate of 1.2-1.7 for heart disease mortality in nonsmokers is supported by the collective evidence; this corresponds to approximately 1,700-5,500 deaths annually in California. The relative risk estimate of 1.38 associated with low birthweight implies that ETS may impact fetal growth of 1,600 newborns in California. At least 31,000 children in California experience one or more ETS-related asthma episodes (new onset or exacerbation) each year. Large impacts are also associated with relative risks for

respiratory effects in children such as middle ear infection (RR ≈ 1.62) (about 52,000 children annually), and lower respiratory infection in young children (RR ≈ 1.5 to 2) (18,000 to 36,000 children annually). ETS exposure is implicated in 21 SIDS deaths per year in California (RR ≈ 3.5). About 400 to 1100 lung cancer deaths in California are ETS-related. For nasal sinus cancers, observed relative risks have ranged from 1.7 to 3.0. This is as high or higher than the relative risks observed for lung cancer. Finally, for breast cancer, a relative risk estimate of 1.26 is derived from our meta-analysis, and when restricted to studies with better exposure assessment, an overall relative risk estimate of 1.90 is obtained. This represents a significant number of cases as this is a relatively common disease in women.

TABLE ES.1
HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

Effects Causally Associated with ETS Exposure

Developmental Effects
Fetal Growth: Low birthweight and decrease in birthweight, and pre-term Delivery
Sudden Infant Death Syndrome (SIDS)

Respiratory Effects
Acute lower respiratory tract infections in children (*e.g.*, bronchitis and pneumonia)
Asthma induction and exacerbation in children and adults
Chronic respiratory symptoms in children
Eye and nasal irritation in adults
Middle ear infections in children

Carcinogenic Effects
Lung Cancer
Nasal Sinus Cancer
Breast Cancer

Cardiovascular Effects
Heart disease mortality
Acute and chronic coronary heart disease morbidity
Altered vascular properties

Effects with Suggestive Evidence of a Causal Association with ETS Exposure

Reproductive and Developmental Effects
Spontaneous abortion, IUGR
Adverse impact on cognition and behavior
Allergic sensitization
Decreased pulmonary function growth
Adverse effects on fertility or fecundability
Menstrual cycle disorders

Cardiovascular and Hematological Effects
Elevated risk of stroke in adults

Respiratory Effects
Exacerbation of cystic fibrosis

Chronic respiratory symptoms in adults

Table ES.2 Attributable Risks Associated with ETS

	Conclusion OEHHA 1997	Conclusion OEHHA 1997	Conclusion Update	Conclusion Update
Outcome	Excess # in CA	Excess # in US	Excess # in CA	Excess # in US
Pregnancy: **Low Birth Weight** **Pre-term Delivery**	1,200-2,200	9,700-18,600	1,600 4,700	24,300[1] 71,900
Cardiac death (Ischemic heart disease death)	4,200-7,440	35,000-62,000	1,700-5,500[2]	22,700-[3] 69,600
Lung Cancer Death	360	3,000	400 [4]	3400
Asthma (children)				
Episodes			31,000 [5]	202,300 [6]
New cases	960-3120	8,000-26,000		
Exacerbation	48,000-120,000	400,000-1,000,000		
Lower respiratory illness	18,000-36,000	150,000-300,000	N/A	N/A
Otitis media visits	78,600-188,700	700,000-1,600,000	51,700 [7]	789,700 [8]
SIDS	120	1,900-2,700	21[9]	431[10]
Breast cancer			All studies: OR 1.26 (95% CI 1.10-1.45)[11] Best studies: OR 1.90 (95% CI 1.53-2.37) Approximate 26 - 90% increased risk	

1 Based on adult females reporting exposure to ETS in NHANES III for 1995 (Pirkle et al., 1996)

2 Based on California Dept Health Services. www.dhs.cs.gov/hisp/chs/OHIR/vssdata/2000data/OOCh5pdf/5_9_Reorg.PDF. Table 5-9 for yr 2000

3 Based on Anderson and Arias (2003). National Vital Statistics Report. Vol 51(9) Table 2 for yr 2000 Ischemic heart diseases including AMI.

4 Assuming California exposure and death rates are similar to national rates and California population is 12% of national population.

5 Based on number asthma attacks or episodes in previous 12 months for 0-17 year olds. Calculated from California Health Interview Survey for 2001

6 Based on number asthma attacks or episodes in previous 12 months for 0-14 year olds. CDC-MMWR 2002 51(SS01)

7 Calculated by applying national value (H6) and assuming 12% of US population lives in California

8 Based on National Center for Health Statistics Series 13 No. 137. Ambulatory Health Care Visits by Children: Principal Diagnosis and Place of Visit for yrs 1993-1995.

9 Based on California Dept Health Services. www.dhs.ca.gov/hisp/chs/ohir/vssdata/2000data/00ch4pdf/8reorg.pdf. Table 4-8 for yr 2000

10 Based on National Center for Health Statistics. www.cdc.gov/nchs/fastats/infort.htm for yr 2000
LBW = low birth weight; N/A = data not available.

11 OEHHA is unable at this time to calculate an attributable risk as it is not possible to account accurately for the portion attributable to other known risk factors. The OR for all studies is based on our meta-analysis of all studies overall risk estimates. The OR for best studies is based on the OR for all studies which did a better job of ascertaining exposure.

What Perinatal Health Effects have been Observed?

ETS exposure adversely affects fetal growth, with elevated risks of low birth weight or "small for gestational age" observed in numerous epidemiological studies. The primary

effect observed, reduction in mean birthweight, is small in magnitude. But if the distribution of birthweight is shifted lower with ETS exposure, as it appears to be with active smoking, infants who are already compromised may be pushed into even higher risk categories. Low birthweight is associated with many well-recognized problems for infants, and is strongly associated with perinatal mortality. ETS is also associated with pre-term delivery. Premature babies are at higher risk for a number of health problems.

The adverse effects of ETS exposure on the mother-fetus unit also manifest as premature births. Preterm delivery has been found to be causally related to ETS exposure with relative risks in the range of 1.5 to 1.8.

The impact of ETS on perinatal manifestations of development other than fetal growth is less clear. The few studies examining the association between ETS and perinatal death are relatively non-informative. Studies on spontaneous abortion are suggestive of a role for ETS, but further work is needed. Although epidemiological studies suggest a moderate association of severe congenital malformations with paternal smoking, the findings are complicated by the use of paternal smoking status as a surrogate for ETS exposure, since a direct effect of active smoking on sperm cannot be ruled out. In general, the defects implicated differed across the studies, with the most consistent association seen for neural tube defects.

What Postnatal Developmental Effects of ETS Exposure have been Observed?

Numerous studies have demonstrated an increased risk of sudden infant death syndrome, or "SIDS," in infants of mothers who smoke. Until recently it has not been possible to separate the effects of postnatal ETS exposure from those of prenatal exposure to maternal active smoking. Recent epidemiological studies now have demonstrated that postnatal ETS exposure is an independent risk factor for SIDS.

Although definitive conclusions regarding causality cannot yet be made on the basis of available epidemiological studies of cognition and behavior, there is suggestive evidence that ETS exposure may pose a hazard for neuropsychological development. With respect to physical development, while small but consistent effects of active maternal smoking during pregnancy have been observed on height growth, there is no evidence that postnatal ETS exposure has a significant impact on growth in otherwise healthy children. Developmental effects of ETS exposure on the respiratory system include decreased lung growth and development, and childhood asthma induction.

What are the Effects of ETS Exposure on Female and Male Reproductive Systems?

Active smoking by women has been found to be associated with decreased fertility in a number of studies, and tobacco smoke appears to be anti-estrogenic. The epidemiological data on ETS exposure, though not conclusive are suggestive of adverse effects on fecundability and fertility. Newer studies reviewed in the update suggest adverse effects of ETS exposure on menstrual cycle disorders. Regarding

other female reproductive effects, while studies indicate a possible association of ETS exposure with early menopause, the analytic methods of these studies could not be thoroughly evaluated, and therefore at present, there is not firm evidence that ETS exposure affects age at menopause. Although associations have been seen epidemiologically between active smoking and sperm parameters, conclusions cannot be made regarding ETS exposure and male reproduction, as there is very limited information available on this topic.

What are the Effects on the Respiratory System?

ETS exposure produces a variety of acute effects involving the upper and lower respiratory tract. In children, ETS exposure can exacerbate asthma, and increases the risk of lower respiratory tract illness, and acute and chronic middle ear infection. Eye and nasal irritation are the most commonly reported symptoms among adult nonsmokers exposed to ETS. Odor annoyance has been demonstrated in several studies.

Regarding chronic health effects, there is compelling evidence that ETS is a risk factor for induction of new cases of asthma (in children and adults) as well as for increasing the severity of disease among children and adults with established asthma. In addition, chronic respiratory symptoms in children, such as cough, phlegm, and wheezing, are associated with parental smoking. While the results from all studies are not wholly consistent, there is evidence that childhood exposure to ETS affects lung growth and development, as measured by small, but statistically significant decrements in pulmonary function tests; associated reductions may persist into adulthood. The effect of chronic ETS exposure on pulmonary function in otherwise healthy adults is likely to be small, and unlikely by itself to result in clinically significant chronic disease. However, in combination with other insults (*e.g.*, prior smoking history, exposure to occupational irritants or ambient air pollutants), ETS exposure could contribute to chronic respiratory impairment in adults. In addition, regular ETS exposure in adults has been reported to increase the risk of occurrence of a variety of lower respiratory symptoms.

Children are especially sensitive to the respiratory effects of ETS exposure. Children with cystic fibrosis are likely to be more sensitive than healthy individuals. Several studies of patients with cystic fibrosis, a disease characterized by recurrent and chronic pulmonary infections, suggest that ETS can exacerbate the condition. Several studies have shown an increased risk of atopy (a predisposition to become allergic to common allergens, which can then be manifested as a variety of allergic conditions) in children of smoking mothers, though the evidence regarding this issue is mixed.

What Carcinogenic Effects does ETS have?

The role of ETS in the etiology of cancers in nonsmokers was explored, as smoking has been recognized as an established cause of a number of cancers (lung, larynx, oral cavity, esophagus and bladder), and a probable cause of several others (cervical, kidney, pancreas, and stomach). Also, ETS contains a number of constituents that have been identified as carcinogens.

Reviews published in the 1986 *Report of the Surgeon General,* by the National Research Council in 1986, and by the U.S. EPA in 1992, as well as the original OEHHA report (CalEPA 1997) concluded that ETS exposure causes lung cancer. Since the previous OEHHA review (Cal/EPA, 1997), numerous epidemiological studies and several meta-analyses have examined the association between passive smoking and lung cancer. The population-based studies were designed to and have successfully addressed many of the weaknesses for which the previous studies on ETS and lung cancer have been criticized. Results from these studies are compatible with the causal association between ETS exposure and lung cancer already reported by the U.S. EPA, Surgeon General, and National Research Council. The studies examining the effect of ETS exposure on nasal sinus cancers consistently (though not uniformly) show statistically significant associations, presenting strong evidence that ETS exposure increases the risk of nasal sinus cancers in nonsmoking adults. Finally, studies of the association between nasopharyngeal cancer and ETS suggest elevated risks.

Epidemiological studies, supported by animal data, provide evidence consistent with a causal association between ETS exposure and breast cancer in humans, which appears stronger for pre-menopausal breast cancer. Studies assessing the association between passive smoking and breast cancer have generally reported a positive, and often statistically significant association. This risk appears to vary by several factors including menopausal status and timing of exposure; factors not always controlled or analyzed for in studies, including the large U.S. cohort studies. Perhaps for these reasons, in addition to concerns of potential ETS exposure misclassification due to limited or excluded occupational, childhood or total lifetime exposure, most of the large cohort studies available have not identified significantly elevated increases in breast cancer risk. However, the more recent primary, population-based case-control studies (as well as three cohort studies), controlling for several important reproductive, dietary and other potential confounding factors, have consistently identified elevated estimates for residential and occupational exposure overall or in individual strata. Higher risks were noted for breast cancer diagnosed in women under age fifty (pre-menopausal) and women exposed per-pubertally and prior to first pregnancy. The toxicological data on tobacco smoke constituents continues to strongly support that the cancer risk associated with active smoking and with ETS exposure alone remains highly plausible. In comparison to studies reviewed in the previous OEHHA report (Cal/EPA, 1997), current epidemiological and toxicological data are substantially more indicative of a positive association between ETS exposure and breast cancer risk, particularly in subgroups of women defined by early age of exposure onset, menopausal status, or underlying genetic susceptibility (e.g. for metabolic enzymes). Future studies need to account for these other factors to establish the extent of this exposure-disease relationship. Overall, the weight of evidence (including biomarker, animal and epidemiological studies) is consistent with a causal association between ETS and breast cancer. Our conclusion is primarily based on the strength of the evidence in younger women (< age 50) diagnosed prior to menopause.

The epidemiological and biochemical evidence suggest that exposure to ETS may increase the risk of cervical cancer. Positive associations were observed in three of four case-control studies and a statistically nonsignificant positive association was observed in the only cohort study conducted. Findings of DNA adducts in the cervical

epithelium as well as nicotine and cotinine in the cervical mucus of ETS-exposed nonsmokers provides biological plausibility.

Precursors of endogenously formed N-nitroso compounds suspected of causing brain tumors are present in high concentrations in ETS. In adults, the epidemiological evidence for an association between ETS exposure and risk of brain tumor remains weak and inadequately researched. More recent studies have focused on the potential association between ETS and childhood brain tumors. In children, recent studies or others not previously reviewed by OEHHA, provide no substantial evidence for an association between maternal smoking and childhood brain tumors, with risk estimates generally near the null. Several studies indicated a slightly stronger association with paternal smoking and brain cancer, although the association is still somewhat weak. The most recent and largest individual study (Filippini et al., 2002) did not consistently observe statistically elevated brain cancer risk. However, the pooled estimate of risk from the Oxford Survey of Childhood Cancers studies (together the largest sample size of the studies reviewed), comparing paternal smokers versus paternal nonsmokers, did find a significantly elevated risk of deaths in offspring of smokers from tumors of the central nervous system (Sorahan et al., 1997b). Overall, the generally positive, but inconsistent, associations reported between paternal smoking and childhood brain tumors, in combination with biologically plausible hypothesis, provide suggestive evidence of an association between ETS and brain cancer in children. Similarly, suggestive evidence of an association between exposure to ETS and childhood cancer is noted for lymphomas and acute lymphocytic leukemia (children of paternal smokers).

For other cancer sites in adults, there has been limited ETS-related epidemiological research in general: there is currently insufficient evidence to draw any conclusion regarding the relationship between ETS exposure and the risk of occurrence of cancer in sites other than lung, nasal cavity, breast, and possibly brain and lymphoma and leukemia. A review of the available literature clearly indicates the need for more research. For example, although compounds established as important in the etiology of stomach cancer are present in tobacco smoke, only a single well designed population based study has been performed for this site. In biochemical studies of nonsmokers, higher levels of hemoglobin adducts of the established bladder carcinogen, 4-aminobiphenyl, have been found in those exposed to ETS. However, no significant increases in bladder cancer were seen in the two epidemiological studies (case-control) conducted to date, although both studies were limited in their ability to detect an effect.

The epidemiological data on ETS exposure and rare childhood cancers provide an inadequate foundation for making conclusions regarding causality. Some studies found small increased risks in children in relation to parental smoking for neuroblastoma, Wilm's tumor, bone and soft-tissue sarcomas, but not for germ cell tumors. Studies to date on these rare cancers have been limited in their power to detect effects. The impact of ETS exposure on childhood cancer would benefit from far greater attention than it has received to date.

What are the Effects on the Cardiovascular System?

The epidemiological data, from prospective and case-control studies conducted in diverse populations, in males and females and in western and eastern countries, support a conclusion that there is a causal association between ETS exposure from spousal smoking and death from coronary heart disease (CHD) in nonsmokers. To the extent possible, estimates of risk were determined with adjustment for demographic factors, and often for other factors related to heart disease, such as blood pressure, serum cholesterol level and obesity index. Risks associated with ETS exposure were almost always strengthened by adjustment for other confounders. For nonsmokers exposed to spousal ETS compared to nonsmokers not exposed, the risk of CHD mortality is increased by a factor of 1.3. The association between CHD and risk is stronger for mortality than for non-fatal outcomes, including angina. It is also evident that these effects exacerbate or are exacerbated by underlying conditions, and individuals with other chronic conditions such as diabetes, vascular disease or hypertension comprise a susceptible population at even greater risk from ETS exposure.

Data from clinical and animal studies suggest various mechanisms by which ETS causes heart disease. In a number of studies in which nonsmokers were exposed to ETS, carotid wall thickening, lesion formation, aortic distensibility and reactivity, and compromise of endothelial function were similar to, but less extensive than those experienced by active smokers. Other effects observed include impaired exercise performance, altered lipoprotein profiles, enhanced platelet aggregation, and increased endothelial cell counts. These findings may account for both the short- and long-term effects of ETS exposure on the heart. The data reviewed also suggests that the effects of ETS may also contribute to stroke, the etiology of which includes atherosclerosis of the carotid and large arteries of the brain, and degeneration of intracerebral arteries.

Title: Preventing Tobacco Use Among Young People: A Report of the Surgeon General

Publication Date: March 1994

Agency or Author: Surgeon General's Report

Purpose: This Executive Summary document highlights the youth tobacco experience and how prevention is a key aspect. Included topics are parental influence on smoking habits, peer influence on smoking habits, and health consequences among youth tobacco users. The full document is also available at the website (www.surgeongeneral.gov) or on the included data CD.

CDC
CENTERS FOR DISEASE CONTROL AND PREVENTION

March 11, 1994 / Vol. 43 / No. RR-4

MMWR

Recommendations and Reports

MORBIDITY AND MORTALITY WEEKLY REPORT

Preventing Tobacco Use Among Young People

A Report of the Surgeon General

Executive Summary

U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
Public Health Service
Centers for Disease Control
and Prevention (CDC)
Atlanta, Georgia 30333

Z-70

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), Public Health Service, U.S. Department of Health and Human Services, Atlanta, Georgia 30333.

SUGGESTED CITATION

Centers for Disease Control and Prevention. Preventing tobacco use among young people: a report of the Surgeon General (Executive Summary). MMWR 1994;43(No. RR-4):[inclusive page numbers].

Copies can be purchased from Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402-9325. Telephone: (202) 783-3238.

NOTICE

The issue of *MMWR Recommendations and Reports* (Vol. 43, No. RR-4) is a reprint of the Executive Summary of the Surgeon General's report entitled *Preventing Tobacco Use Among Young People*, released February 1994. The report is included in the *MMWR* series of publications so that the material may be readily accessible to the public health community.

THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

The Honorable Thomas S. Foley
Speaker of the House of Representatives
Washington, D.C. 20515

Dear Mr. Speaker:

It is my pleasure to transmit to the Congress the Surgeon General's report on the health consequences of smoking entitled Preventing Tobacco Use Among Young People. This report is mandated by section 8(a) of the Public Health Cigarette Smoking Act of 1969 (Public Law 91-222) and includes the health effects of smokeless tobacco products as mandated by section 8(a) of the Comprehensive Smokeless Tobacco Health Education Act of 1986 (Public Law 99-252). The report was prepared by the Centers for Disease Control and Prevention's Office on Smoking and Health.

This report focuses on the vulnerable adolescent ages of 10 through 18 when most users start smoking, chewing, or dipping and become addicted to tobacco. It examines the health effects of early smoking and smokeless tobacco use, the reasons that young men and women begin using tobacco, the extent to which they use it, and efforts to prevent tobacco use by young people.

Smoking kills 434,000 Americans each year. Adolescent smoking and smokeless tobacco use are the first steps in this totally preventable public health tragedy. The facts are simple: one out of three adolescents in the United States is using tobacco by age 18, adolescent users become adult users, and few people begin to use tobacco after age 18. Preventing young people from starting to use tobacco is the key to reducing the death and disease caused by tobacco use. This report documents that intervention programs targeting the broad social environment of adolescents are both effective and warranted.

A great opportunity lies before us to prevent millions of premature deaths and improve the quality of lives. This report points out the overwhelming need in public health for efforts directed toward stopping young people before they start using tobacco.

Sincerely,

Donna E. Shalala

Enclosure

2-73

THE SECRETARY OF HEALTH AND HUMAN SERVICES
WASHINGTON, D.C. 20201

The Honorable Albert Gore, Jr.
President of the Senate
Washington, D.C. 20510

Dear Mr. President:

It is my pleasure to transmit to the Congress the Surgeon General's report on the health consequences of smoking entitled Preventing Tobacco Use Among Young People. This report is mandated by section 8(a) of the Public Health Cigarette Smoking Act of 1969 (Public Law 91-222) and includes the health effects of smokeless tobacco products as mandated by section 8(a) of the Comprehensive Smokeless Tobacco Health Education Act of 1986 (Public Law 99-252). The report was prepared by the Centers for Disease Control and Prevention's Office on Smoking and Health.

This report focuses on the vulnerable adolescent ages of 10 through 18 when most users start smoking, chewing, or dipping and become addicted to tobacco. It examines the health effects of early smoking and smokeless tobacco use, the reasons that young men and women begin using tobacco, the extent to which they use it, and efforts to prevent tobacco use by young people.

Smoking kills 434,000 Americans each year. Adolescent smoking and smokeless tobacco use are the first steps in this totally preventable public health tragedy. The facts are simple: one out of three adolescents in the United States is using tobacco by age 18, adolescent users become adult users, and few people begin to use tobacco after age 18. Preventing young people from starting to use tobacco is the key to reducing the death and disease caused by tobacco use. This report documents that intervention programs targeting the broad social environment of adolescents are both effective and warranted.

A great opportunity lies before us to prevent millions of premature deaths and improve the quality of lives. This report points out the overwhelming need in public health for efforts directed toward stopping young people before they start using tobacco.

Sincerely,

Donna E. Shalala

Enclosure

Contents

2-75

Foreword

This Surgeon General's report on smoking and health is the twenty-third in a series that was begun in 1964 and mandated by federal law in 1969. This report is the first in this series to focus on young people. It underscores the seriousness of tobacco use, its relationship to other adolescent problem behaviors, and the responsibility of all citizens to protect the health of our children.

Since 1964, substantial changes have occurred in scientific knowledge of the health consequences of smoking and smokeless tobacco use. Much more is also known about programs and policies that encourage nonsmoking behavior among adults and protect nonsmokers from exposure to environmental tobacco smoke. Although considerable gains have been made against smoking among U.S. adults, this progress has not been realized with young people. Onset rates of cigarette smoking among our youth have not declined over the past decade, and 28 percent of the nation's high school seniors are currently cigarette smokers.

The onset of tobacco use occurs primarily in early adolescence, a developmental stage that is several decades removed from the death and disability that are associated with smoking and smokeless tobacco use in adulthood. Currently, very few people begin to use tobacco as adults; almost all first use has occurred by the time people graduate from high school. The earlier young people begin using tobacco, the more heavily they are likely to use it as adults, and the longer potential time they have to be users. Both the duration and the amount of tobacco use are related to eventual chronic health problems. The processes of nicotine addiction further ensure that many of today's adolescent smokers will regularly use tobacco when they are adults.

Preventing smoking and smokeless tobacco use among young people is critical to ending the epidemic of tobacco use in the United States. This report examines the past few decades' extensive scientific literature on the factors that influence the onset of use among young people and on strategies to prevent this onset. To better understand adolescent tobacco use, this report draws not only on medical and epidemiologic research but also on behavioral and social investigations. The resulting examination of the advertising and promotional activities of the tobacco industry, as well as the review of research on the effects of these activities on young people, marks an important contribution to our understanding of the epidemic of tobacco use in the United States and elsewhere. In particular, this research on the social environment of young people identifies key risk factors that encourage tobacco use. The careful targeting of these risk factors—on a communitywide basis—has proven successful in preventing the onset and development of tobacco use among young people.

Philip R. Lee, M.D.
Assistant Secretary for Health
Public Health Service

David Satcher, M.D., Ph.D.
Director
Centers for Disease
Control and Prevention

2-76

Preface
from the Surgeon General
U.S. Department of Health and Human Services

The public health movement against tobacco use will be successful when young people no longer want to smoke. We are not there yet. Despite 30 years of decline in overall smoking prevalence, despite widespread dissemination of information about smoking, despite a continuing decline in the social acceptability of smoking, substantial numbers of young men and women begin to smoke and become addicted. These current and future smokers are new recruits in the continuing epidemic of disease, disability, and death attributable to tobacco use. When young people no longer want to smoke, the epidemic itself will die.

This report of the Surgeon General, *Preventing Tobacco Use Among Young People*, delineates the problem in no uncertain terms. The direct effects of tobacco use on the health of young people have been greatly underestimated. The long-term effects are, of course, well established. The addictive nature of tobacco use is also well known, but it is perhaps less appreciated that early addiction is the chief mechanism for renewing the pool of smokers. Most people who are going to smoke are hooked by the time they are 20 years old.

Young people face enormous pressures to smoke. The tobacco industry devotes an annual budget of nearly $4 billion to advertising and promoting cigarettes. As this report so well describes, there has been a continuing shift from advertising to promotion, largely because of banning cigarette ads from broadcast media. The effect of the ban is dubious, however, since the use of promotional materials, the sponsoring of sports events, and the use of logos in nontraditional venues may actually be more effective in reaching target audiences. Clearly, young people are being indoctrinated with tobacco promotion at a susceptible time in their lives.

A misguided debate has arisen about whether tobacco promotion "causes" young people to smoke—misguided because single-source causation is probably too simple an explanation for any social phenomenon. The more important issue is what effect tobacco promotion might have. Current research suggests that pervasive tobacco promotion has two major effects: it creates the perception that more people smoke than actually do, and it provides a conduit between actual self-image and ideal self-image—in other words, smoking is made to look cool. Whether causal or not, these effects foster the uptake of smoking, initiating for many a dismal and relentless chain of events.

On the brighter side, a large portion of this report is devoted to countervailing influences. We have the justification: there is a substantial scientific basis for primary prevention of cigarette smoking and smokeless tobacco use. A number of successful prevention programs, based on the psychological and behavioral factors that create susceptibility to smoking, are available. We have the means: the report defines a coordinated, effective, nonsmoking public health program for young people. And we have the will: schools, communities, legislatures, and public opinion all testify to the growing support for encouraging young people to avoid tobacco use.

The task is by no means easy. This report underscores the commitment all of us must have to the health of young people in the United States. Substantial work will be required to translate the justification, the means, and the will into a world in which young people no longer want to smoke. I, for one, relish the task.

M. Joycelyn Elders, M.D.
Surgeon General

Acknowledgments

This report was prepared by the Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, under the general editorship of the Office on Smoking and Health, Michael P. Eriksen, Sc.D., Director. The Managing Editor was Gayle Lloyd, M.A.

The scientific editor of the Report was:

Cheryl L. Perry, Ph.D., Professor, Division of Epidemiology, School of Public Health, University of Minnesota, Minneapolis, MN.

More than 100 persons from government, academic, and private institutions contributed to the preparation of *Preventing Tobacco Use Among Young People*. Space constraints prevent acknowledging these authors, reviewers, and staff in this Executive Summary. Their names are listed, however, in the "Acknowledgments" section of the full Report.

2-79

Blank

CHAPTER 1

INTRODUCTION, SUMMARY, AND CHAPTER CONCLUSIONS

INTRODUCTION

Previous Surgeon General's reports on tobacco use and health have largely focused on the epidemiologic, clinical, biologic, and pharmacologic aspects of adult use of tobacco products. This report on *Preventing Tobacco Use Among Young People* provides a more detailed look at adolescence, the time of life when most tobacco users begin, develop, and establish their behavior. Because regular use soon results in addiction to nicotine, this behavior may persist through adulthood, significantly increasing, through the extended years of use, the risk of long-term, severe health consequences.

Despite three decades of explicit health warnings, large numbers of young people continue to take up tobacco; currently, over three million adolescents smoke cigarettes, and over one million adolescent males currently use smokeless tobacco. Clearly, effective interventions are needed to prevent more young people from trying tobacco. To achieve significant long-term reductions in tobacco use and tobacco-related deaths in the United States, we must examine the nature and scope of adolescent tobacco use, consider the social, psychological, and marketing factors that influence young people in their decision to use tobacco products, and evaluate current efforts to prevent young people from becoming users. This report addresses the crucial problems of adolescent tobacco use.

Development of the Report

This report of the Surgeon General was prepared by the Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC and Prevention, Public Health Service, U.S. Department of Health and Human Services, as part of the department's responsibility, under Public Law 91-222 and Public Law 99-252, to report current information on the health effects of cigarette smoking and smokeless tobacco use to the United States Congress. This report is the first to focus on the problem of tobacco use among young people. Given the continuing onset of use in adolescence and the growing evidence of health consequences associated with early use, the report was seen as both needed and timely.

The current report has been produced through the efforts of experts in the medical, pharmacologic, epidemiologic, developmental, economic, behavioral, legal, and public health aspects of smoking and smokeless tobacco use among young people. Initial manuscripts for the report were prepared by 28 scientists who were selected for their expertise in specific content areas. This material was consolidated into chapters, each of which underwent peer review. The entire document was reviewed by a number of experts in the field, as well as by institutes and agencies within the U.S. Public Health Service. The final draft of the report was reviewed by the Assistant Secretary for Health and by the Secretary, Department of Health and Human Services.

Several concerns guided the development of this report. The first, which is addressed in Chapter 2, is whether tobacco use is associated with health consequences during the period of adolescence (broadly defined as ages 10 through 18, although research cited in this report varies somewhat in the ages considered adolescent). The long-term health consequences—that is, those that emerge in adulthood—have been the subject of extensive review and are widely acknowledged in the scientific and public literature. The chapter thus focuses on the serious health consequences, as well as

the increased risk factors for subsequent health consequences, that are evident early in life among young smokers and smokeless tobacco users. Chapter 3 examines the epidemiologic patterns of tobacco use among the young. National data on trends in adolescent use are analyzed to determine the extent of the current problem, as well as to note changes in patterns of initiation and use. The factors that influence adolescents in their decision to use tobacco are examined in Chapter 4, which considers psychosocial risk factors, and Chapter 5, which examines the influence of tobacco advertising and promotion. The final concern, the focus of Chapter 6, was to assess what has been done—from the individual level to the legislative level—to prevent tobacco use among young people.

Major Conclusions

1. Nearly all first use of tobacco occurs before high school graduation; this finding suggests that if adolescents can be kept tobacco-free, most will never start using tobacco.
2. Most adolescent smokers are addicted to nicotine and report that they want to quit but are unable to do so; they experience relapse rates and withdrawal symptoms similar to those reported by adults.
3. Tobacco is often the first drug used by those young people who use alcohol, marijuana, and other drugs.
4. Adolescents with lower levels of school achievement, with fewer skills to resist pervasive influences to use tobacco, with friends who use tobacco, and with lower self-images are more likely than their peers to use tobacco.
5. Cigarette advertising appears to increase young people's risk of smoking by affecting their perceptions of the pervasiveness, image, and function of smoking.
6. Communitywide efforts that include tobacco tax increases, enforcement of minors' access laws, youth-oriented mass media campaigns, and school-based tobacco-use prevention programs are successful in reducing adolescent use of tobacco.

SUMMARY

Introduction

The health effects of cigarette smoking have been the subject of intensive investigation since the 1950s. Cigarette smoking is still considered the chief preventable cause of premature disease and death in the United States. As was documented extensively in previous Surgeon General's reports, cigarette smoking has been causally linked to lung cancer and other fatal malignancies, atherosclerosis and coronary heart disease, chronic obstructive pulmonary disease, and other conditions that constitute a wide array of serious health consequences (USDHHS 1989). More recent studies have concluded that passive (or involuntary) smoking can cause disease, including lung cancer, in healthy nonsmokers. In 1986, an advisory committee appointed by the Surgeon General released a special report on the health consequences of smokeless tobacco, concluding that smokeless tobacco use can cause cancer and can lead to nicotine addiction (USDHHS 1986). In the 1988 report, nicotine was designated a

highly addictive substance, comparable in its physiological and psychological properties to other addictive substances of abuse (USDHHS 1988). Considerable evidence indicates that the health problems associated with smoking are a function of the duration (years) and the intensity (amount) of use. The younger one begins to smoke, the more likely one is to be a current smoker as an adult. Earlier onset of cigarette smoking and smokeless tobacco use provides more life-years to use tobacco and thereby increases the potential duration of use and the risk of a range of more serious health consequences. Earlier onset is also associated with heavier use; those who begin to use tobacco as younger adolescents are among the heaviest users in adolescence and adulthood. Heavier users are more likely to experience tobacco-related health problems and are the least likely to quit smoking cigarettes or using smokeless tobacco. Preventing tobacco use among young people is therefore likely to affect both duration and intensity of total use of tobacco, potentially reducing long-term health consequences significantly.

Health Consequences of Tobacco Use Among Young People

Active smoking by young people is associated with significant health problems during childhood and adolescence and with increased risk factors for health problems in adulthood. Cigarette smoking during adolescence appears to reduce the rate of lung growth and the level of maximum lung function that can be achieved. Young smokers are likely to be less physically fit than young nonsmokers; fitness levels are inversely related to the duration and the intensity of smoking. Adolescent smokers report that they are significantly more likely than their nonsmoking peers to experience shortness of breath, coughing spells, phlegm production, wheezing, and overall diminished physical health. Cigarette smoking during childhood and adolescence poses a clear risk for respiratory symptoms and problems during adolescence; these health problems are risk factors for other chronic conditions in adulthood, including chronic obstructive pulmonary disease.

Cardiovascular disease is the leading cause of death among adults in the United States Athero-sclerosis, however, may begin in childhood and become clinically significant by young adulthood. Cigarette smoking has been shown to be a primary risk factor for coronary heart disease, arteriosclerotic peripheral vascular disease, and stroke. Smoking by children and adolescents is associated with an increased risk of early atherosclerotic lesions and increased risk factors for cardiovascular diseases. These risk factors include increased levels of low-density lipoprotein cholesterol, increased very-low-density lipoprotein cholesterol, increased triglycerides, and reduced levels of high-density lipoprotein cholesterol. If sustained into adulthood, these patterns significantly increase the risk for early development of cardiovascular disease.

Smokeless tobacco use is associated with health consequences that range from halitosis to severe health problems such as various forms of oral cancer. Use of smokeless tobacco by young people is associated with early indicators of adult health consequences, including periodontal degeneration, soft tissue lesions, and general systemic alterations. Previous reports have documented that smokeless tobacco use is as addictive for young people as it is for adults. Another concern is that smokeless tobacco users are more likely than nonusers to become cigarette smokers.

Among addictive behaviors such as the use of alcohol and other drugs, cigarette smoking is most likely to become established during adolescence. Young people who

begin to smoke at an earlier age are more likely than later starters to develop long-term nicotine addiction. Most young people who smoke regularly are already addicted to nicotine, and they experience this addiction in a manner and severity similar to what adult smokers experience. Most adolescent smokers report that they would like to quit smoking and that they have made numerous, usually unsuccessful attempts to quit. Many adolescents say that they intend to quit in the future and yet prove unable to do so. Those who try to quit smoking report withdrawal symptoms similar to those reported by adults. Adolescents are difficult to recruit for formal cessation programs, and when enrolled, are difficult to retain in the programs. Success rates in adolescent cessation programs tend to be quite low, both in absolute terms and relative to control conditions.

Tobacco use is associated with a range of problem behaviors during adolescence. Smokeless tobacco or cigarettes are generally the first drug used by young people in a sequence that can include tobacco, alcohol, marijuana, and hard drugs. This pattern does not imply that tobacco use causes other drug use, but rather that other drug use rarely occurs before the use of tobacco. Still, there are a number of biological, behavioral, and social mechanisms by which the use of one drug may facilitate the use of other drugs, and adolescent tobacco users are substantially more likely to use alcohol and illegal drugs than are nonusers. Cigarette smokers are also more likely to get into fights, carry weapons, attempt suicide, and engage in high-risk sexual behaviors. These problem behaviors can be considered a syndrome, since involvement in one behavior increases the risk for involvement in others. Delaying or preventing the use of tobacco may have implications for delaying or preventing these other behaviors as well.

The Epidemiology of Tobacco Use Among Young People

Overall, about one-third of high-school-aged adolescents in the United States smoke or use smokeless tobacco. Smoking prevalence among U.S. adolescents declined sharply in the 1970s, but this decline slowed significantly in the 1980s, particularly among white males. Although female adolescents during the 1980s were more likely than male adolescents to smoke, female and male adolescents are now equally likely to smoke. Male adolescents are substantially more likely than females to use smokeless tobacco products; about 20 percent of high school males report current use, whereas only about 1 percent of females do. White adolescents are more likely to smoke and to use smokeless tobacco than are black and Hispanic adolescents.

Sociodemographic, environmental, behavioral, and personal factors can encourage the onset of tobacco use among adolescents. Young people from families with lower socioeconomic status, including those adolescents living in single-parent homes, are at increased risk of initiating smoking. Among environmental factors, peer influence seems to be particularly potent in the early stages of tobacco use; the first tries of cigarettes and smokeless tobacco occur most often with peers, and the peer group may subsequently provide expectations, reinforcement, and cues for experimentation. Parental tobacco use does not appear to be as compelling a risk factor as peer use; on the other hand, parents may exert a positive influence by disapproving of smoking, being involved in children's free time, discussing health matters with children, and encouraging children's academic achievement and school involvement.

How adolescents perceive their social environment may be a stronger influence on behavior than the actual environment. For example, adolescents consistently overestimate the number of young people and adults who smoke. Those with the highest overestimates are more likely to become smokers than are those with more accurate perceptions. Similarly, those who perceive that cigarettes are easily accessible and generally available are more likely to begin smoking than are those who perceive more difficulty in obtaining cigarettes.

Behavioral factors figure heavily during adolescence, a period of multiple transitions to physical maturation, to a coherent sense of self, and to emotional independence. Adolescents are thus particularly vulnerable to a range of hazardous behaviors and activities, including tobacco use, that may seem to assist in these transitions. Young people who report that smoking serves positive functions or is potentially useful are at increased risk for smoking. These functions are associated with bonding with peers, being independent and mature, and having a positive social image. Since reports from adolescents who begin to smoke indicate that they have lower self-esteem and lower self-images than their nonsmoking peers, smoking can become a self-enhancement mechanism. Similarly, not having the confidence to be able to resist peer offers of tobacco seems to be an important risk factor for initiation. Intentions to use tobacco and actual experimentation also strongly predict subsequent regular use.

The positive functions that many young people attribute to smoking are the same functions advanced in most cigarette advertising. Young people are a strategically important market for the tobacco industry. Since most smokers try their first cigarette before age 18, young people are the chief source of new consumers for the tobacco industry, which each year must replace the many consumers who quit smoking and the many who die from smoking-related diseases. Despite restrictions on tobacco marketing, children and adolescents continue to be exposed to cigarette advertising and promotional activities, and young people report considerable familiarity with many cigarette advertisements. In the past, this exposure was accomplished by radio and television programs sponsored by the cigarette industry. Barred since 1971 from using broadcast media, the tobacco industry increasingly relies on promotional activities, including sponsorship of sports events and public entertainment, outdoor billboards, point-of-purchase displays, and the distribution of specialty items that appeal to the young. Cigarette advertisements in the print media persist; these messages have become increasingly less informational, replacing words with images to portray the attractiveness and function of smoking. Cigarette advertising frequently uses human models or human-like cartoon characters to display images of youthful activities, independence, healthfulness, and adventure-seeking. In presenting attractive images of smokers, cigarette advertisements appear to stimulate some adolescents who have relatively low self-images to adopt smoking as a way to improve their own self-image. Cigarette advertising also appears to affect adolescents' perceptions of the pervasiveness of smoking, images of smokers, and the function of smoking. Since these perceptions are psychosocial risk factors for the initiation of smoking, cigarette advertising appears to increase young people's risk of smoking.

2-86

Efforts to Prevent the Onset of Tobacco Use

Most of the U.S. public strongly favors policies that might prevent tobacco use among young people. These policies include mandated tobacco education in schools, a complete ban on smoking by anyone on school grounds, further restrictions on tobacco advertising and promotional activities, stronger prohibitions on the sale of tobacco products to minors, and increases in earmarked taxes on tobacco products. Interventions to prevent initiation among young people—even actions that involve restrictions on adult smoking or increased taxes—have received strong support among smoking and nonsmoking adults.

Numerous research studies over the past 15 years suggest that organized interventions can help prevent the onset of smoking and smokeless tobacco use. School-based smoking-prevention programs, based on a model of identifying social influences on smoking and providing skills to resist those influences, have demonstrated consistent and significant reductions in adolescent smoking prevalence; these program effects have lasted one to three years. Programs to prevent smokeless tobacco use have used a similar model to achieve modest reductions in initiation of use. The effectiveness of these school-based programs appears to be enhanced and sustained, at least until high school graduation, by adding coordinated communitywide programs that involve parents, youth-oriented mass media and counteradvertising, community organizations, or other elements of adolescents' social environments.

A crucial element of prevention is access: adolescents should not be able to purchase tobacco products in their communities. Active enforcement of age-at-sale policies by public officials and community members appears necessary to prevent minors' access to tobacco. Communities that have adopted tighter restrictions have achieved reductions in purchases by minors. At the state and national levels, price increases have significantly reduced cigarette smoking; the young have been at least as responsive as adults to these price changes. Maintaining higher real prices of cigarettes provides a barrier to adolescent tobacco use but depends on further tax increases to offset the effects of inflation. The results of this review thus suggest that a coordinated, multicomponent campaign involving policy changes, taxation, mass media, and behavioral education can effectively reduce the onset of tobacco use among adolescents.

Summary

Smoking and smokeless tobacco use are almost always initiated and established in adolescence. Besides its long-term effects on adults, tobacco use produces specific health problems for adolescents. Since nicotine addiction also occurs during adolescence, adolescent tobacco users are likely to become adult tobacco users. Smoking and smokeless tobacco use are associated with other problem behaviors and occur early in the sequence of these behaviors. The outcomes of adolescent smoking and smokeless tobacco use continue to be of great public health importance, since one out of three U.S. adolescents uses tobacco by age 18. The social environment of adolescents, including the functions, meanings, and images of smoking that are conveyed through cigarette advertising, sets the stage for adolescents to begin using tobacco. As tobacco products are available and as peers begin to try them, these factors become personalized and relevant, and tobacco use may begin. This process most affects adolescents who, compared with their peers, have lower self-esteem and

2-87

self-images, are less involved with school and academic achievement, have fewer skills to resist the offers of peers, and come from homes with lower socioeconomic status. Tobacco-use prevention programs that target the larger social environment of adolescents are both efficacious and warranted.

CHAPTER CONCLUSIONS

Following are the specific conclusions for each chapter of this report:

Chapter 2. The Health Consequences of Tobacco Use by Young People

1. Cigarette smoking during childhood and adolescence produces significant health problems among young people, including cough and phlegm production, an increased number and severity of respiratory illnesses, decreased physical fitness, an unfavorable lipid profile, and potential retardation in the rate of lung growth and the level of maximum lung function.
2. Among addictive behaviors, cigarette smoking is the one most likely to become established during adolescence. People who begin to smoke at an early age are more likely to develop severe levels of nicotine addiction than those who start at a later age.
3. Tobacco use is associated with alcohol and illicit drug use and is generally the first drug used by young people who enter a sequence of drug use that can include tobacco, alcohol, marijuana, and harder drugs.
4. Smokeless tobacco use by adolescents is associated with early indicators of periodontal degeneration and with lesions in the oral soft tissue. Adolescent smokeless tobacco users are more likely than nonusers to become cigarette smokers.

Chapter 3. Epidemiology of Tobacco Use Among Young People in the United States

1. Tobacco use primarily begins in early adolescence, typically by age 16; almost all first use occurs before the time of high school graduation.
2. Smoking prevalence among adolescents declined sharply in the 1970s, but the decline slowed significantly in the 1980s. At least 3.1 million adolescents and 25 percent of 17- and 18-year-olds are current smokers.
3. Although current smoking prevalence among female adolescents began exceeding that among males by the mid- to late-1970s, both sexes are now equally likely to smoke. Males are significantly more likely than females to use smokeless tobacco. Nationally, white adolescents are more likely to use all forms of tobacco than are blacks and Hispanics. The decline in the prevalence of cigarette smoking among black adolescents is noteworthy.
4. Many adolescent smokers are addicted to cigarettes; these young smokers report withdrawal symptoms similar to those reported by adults.
5. Tobacco use in adolescence is associated with a range of health-compromising behaviors, including being involved in fights, carrying weapons, engaging in higher-risk sexual behavior, and using alcohol and other drugs.

Chapter 4. Psychosocial Risk Factors for Initiating Tobacco Use

1. The initiation and development of tobacco use among children and adolescents progresses in five stages: from forming attitudes and beliefs about

tobacco, to trying, experimenting with, and regularly using tobacco, to being addicted. This process generally takes about three years.

2. Sociodemographic factors associated with the onset of tobacco use include being an adolescent from a family with low socioeconomic status.
3. Environmental risk factors for tobacco use include accessibility and availability of tobacco products, perceptions by adolescents that tobacco use is normative, peers' and siblings' use and approval of tobacco use, and lack of parental support and involvement as adolescents face the challenges of growing up.
4. Behavioral risk factors for tobacco use include low levels of academic achievement and school involvement, lack of skills required to resist influences to use tobacco, and experimentation with any tobacco product.
5. Personal risk factors for tobacco use include a lower self-image and lower self-esteem than peers, the belief that tobacco use is functional, and lack of self-efficacy in the ability to refuse offers to use tobacco. For smokeless tobacco use, insufficient knowledge of the health con-sequences is also a factor.

Chapter 5. Tobacco Advertising and Promotional Activities

1. Young people continue to be a strategically important market for the tobacco industry.
2. Young people are currently exposed to cigarette messages through print media (including outdoor billboards) and through promotional activities, such as sponsorship of sporting events and public entertainment, point-of-sale displays, and distribution of specialty items.
3. Cigarette advertising uses images rather than information to portray the attractiveness and function of smoking. Human models and cartoon characters in cigarette advertising convey independence, healthfulness, adventure-seeking, and youthful activities—themes correlated with psychosocial factors that appeal to young people.
4. Cigarette advertisements capitalize on the disparity between an ideal and actual self-image and imply that smoking may close the gap.
5. Cigarette advertising appears to affect young people's perceptions of the pervasiveness, image, and function of smoking. Since misperceptions in these areas constitute psychosocial risk factors for the initiation of smoking, cigarette advertising appears to increase young people's risk of smoking.

Chapter 6. Efforts to Prevent Tobacco Use Among Young People

1. Most of the American public strongly favor policies that might prevent tobacco use among young people. These policies include tobacco education in the schools, restrictions on tobacco advertising and promotions, a complete ban on smoking by anyone on school grounds, prohibition of the sale of tobacco products to minors, and earmarked tax increases on tobacco products.
2. School-based smoking-prevention programs that identify social influences to smoke and teach skills to resist those influences have demonstrated consistent and significant reductions in adolescent smoking prevalence, and program effects have lasted one to three years. Programs to prevent smokeless tobacco use that are based on the same model have also demonstrated modest reductions in the initiation of smokeless tobacco use.

2-89

3. The effectiveness of school-based smoking-prevention programs appears to be enhanced and sustained by comprehensive school health education and by communitywide programs that involve parents, mass media, community organizations, or other elements of an adolescent's social environment.
4. Smoking-cessation programs tend to have low success rates. Recruiting and retaining adolescents in formal cessation programs are difficult.
5. Illegal sales of tobacco products are common. Active enforcement of age-at-sale policies by public officials and community members appears necessary to prevent minors' access to tobacco.
6. Econometric and other studies indicate that increases in the real price of cigarettes significantly reduce cigarette smoking; young people are at least as responsive as adults to such price changes. Maintaining higher real prices of cigarettes depends on further tax increases to offset the effects of inflation.

Bibliography

CDC. The health consequences of smoking: nicotine addiction—a report of the Surgeon General. Rockville, MD: US Department of Health and Human Services, Public Health Service, 1988; DHHS publication no. (CDC)88-8406.

CDC. Reducing the health consequences of smoking: 25 years of progress—a report of the Surgeon General. Rockville, MD: US Department of Health and Human Services, Public Health Service, 1989; DHHS publication no. (CDC)89-8411.

Public Health Service. The health consequences of using smokeless tobacco: a report of the advisory committee to the Surgeon General. Rockville, MD: US Department of Health and Human Services, Public Health Services, National Institutes of Health, 1986; DHHS publication no. (NIH)86-2874.

2-90

BLANK

The *Morbidity and Mortality Weekly Report (MMWR)* Series is prepared by the Centers for Disease Control and Prevention (CDC) and is available on a paid subscription basis from the Superintendent of Documents, U.S. Government Printing Office, Washington, DC 20402; telephone (202) 783-3238.

The data in the weekly *MMWR* are provisional, based on weekly reports to CDC by state health departments. The reporting week concludes at close of business on Friday; compiled data on a national basis are officially released to the public on the succeeding Friday. Inquiries about the *MMWR* Series, including material to be considered for publication, should be directed to: Editor, *MMWR* Series, Mailstop C-08, Centers for Disease Control and Prevention, Atlanta, GA 30333; telephone (404) 332-4555.

☆U.S. Government Printing Office: 1994-733-131/830— Region IV

2-92

Title: Report on Carcinogens, Tobacco Related Exposures, Eleventh Edition

Publication Date: 2004

Agency or Author: National Toxicological Program, Department of Health and Human Services

Purpose: This document establishs Environmental Tobacco Smoke (ETS) as a known carcinogen based upon summary information from primary literature. Provides information on carcinogenicity, properties of ETS, exposure, and current federal regulations concerning ETS.

Tobacco Related Exposures

Introduction

Tobacco contains more than 2,500 chemical constituents, many of which are known human carcinogens. Chewing tobacco and snuff are the two main forms of smokeless tobacco used in the United States. Tobacco smoking produces both mainstream smoke, which is drawn through the tobacco column and exits through the mouthpiece during puffing, and sidestream smoke, which is emitted from the smoldering tobacco between puffs.

Environmental tobacco smoke, smokeless tobacco, and tobacco smoking were first listed (separately) in the *Ninth Report on Carcinogens* (2000). The profiles for these compounds, which are listed (separately) as *known to be a human carcinogen*, follow this introduction.

Environmental Tobacco Smoke*

Known to be a human carcinogen
First Listed in the *Ninth Report on Carcinogens* (2000)

Carcinogenicity

Environmental tobacco smoke is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans that indicate a causal relationship between passive exposure to tobacco smoke and lung cancer. Some studies also support an association of environmental tobacco smoke with cancers of the nasal sinus (CEPA 1997).

Evidence for an increased cancer risk from environmental tobacco smoke stems from studies examining nonsmoking spouses living with individuals who smoke cigarettes, exposures of nonsmokers to environmental tobacco smoke in occupational settings, and exposure to parents' smoking during childhood (IARC 1986, EPA 1992, CEPA 1997). Many epidemiological studies, including large population-based case-control studies, have demonstrated increased risks for developing lung cancer following prolonged exposure to environmental tobacco smoke. A meta-analysis found an overall increase in risk of 20% for exposure to environmental tobacco smoke from a spouse who smokes. Exposure to environmental tobacco smoke from spousal smoking or exposure in an occupational setting appears most strongly related to increased risk.

Exposure of nonsmokers to environmental tobacco smoke has been demonstrated by detecting nicotine, respirable smoke particulates, tobacco specific nitrosamines, and other smoke constituents in the breathing zone, and by measurements of a nicotine metabolite (cotinine) in the urine. However, there is no good biomarker of cumulative past exposure to tobacco smoke, and all of the information collected in epidemiology studies determining past exposure to environmental tobacco smoke relies on estimates that may vary in their accuracy (recall bias). Other suggestions of systematic bias have been made concerning the epidemiological information published on the association of environmental tobacco smoke with cancer. These include misclassification of smokers as nonsmokers, factors related to lifestyle, diet, and other exposures that may be common to couples living together and that may influence lung cancer incidence, misdiagnosis of cancers that metastasized from other organs to the lung, and the possibility that epidemiology studies examining small populations and showing no effects of environmental tobacco smoke would not be published (publication bias).

Three population-based (Brownson *et al.* 1992, Stockwell *et al.* 1992, Fontham *et al.* 1994) and one hospital-based (Kabat *et al.* 1995) case-control studies addressed potential systematic biases. Each of the three population-based studies showed an increased risk from prolonged environmental tobacco smoke exposure of a magnitude consistent with prior estimates. The hospital-based study gave similarly increased risk estimates, but the results were not statistically significant. The potential for publication bias has been examined and dismissed (CEPA 1997), and the reported absence of increased risk for lung cancer for nonsmokers exposed only in occupational settings has been found not to be the case when the analysis is restricted to higher quality studies (Wells 1998). Thus, factors related to chance, bias, and/or confounding have been adequately excluded, and exposure to environmental tobacco smoke is established as causally related to human lung cancer.

Since environmental tobacco smoke was listed in the *Ninth Report on Carcinogens*, the International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence that involuntary smoking (exposure to secondhand or environmental tobacco smoke) causes lung cancer in humans (IARC 2002).

Witschi *et al.* (1997a,b) found a significant increase in lung tumor incidence and multiplicity in groups of mice exposed for five months to filtered and unfiltered environmental tobacco smoke (defined as a mixture of 89% sidestream and 11% mainstream smoke [sidestream and mainstream smoke are defined under "Properties"]) and allowed to recover for another four months in filtered air; however, no significant increase in tumor incidence was observed in mice exposed for five months without a recovery period (Witschi *et al.* 1997a,b). Other studies indicate that inhaled cigarette smoke and topically applied cigarette-smoke condensate can induce cancer in experimental animals. There is evidence from animal studies that the condensate of sidestream smoke is more carcinogenic to the skin of mice than equivalent weight amounts of mainstream smoke. Since environmental tobacco smoke was listed in the *Ninth Report on Carcinogens*, IARC (2002) concluded that there is sufficient evidence in experimental animals for the carcinogenicity of sidestream smoke condensates and limited evidence in experimental animals for the carcinogenicity of mixtures of mainstream and sidestream tobacco smoke.

Additional Information Relevant to Carcinogenicity

Sidestream smoke and mainstream smoke contain many of the same chemical constituents, including at least 250 chemicals known to be toxic or carcinogenic. Exposure to primarily mainstream smoke through active tobacco smoking has been determined to cause cancer of the lung, urinary bladder, renal pelvis, oral cavity, pharynx, larynx, esophagus, lip, and pancreas in humans. Between 80% and 90% of all human lung cancers are attributed to tobacco smoking (see profile for Tobacco Smoking below). Environmental tobacco smoke, sidestream smoke, sidestream smoke condensate, and a mixture of sidestream and mainstream smoke condensate cause genetic damage. Increased concentrations of mutagens have been found in the urine of humans exposed to environmental tobacco smoke. Lung tumors from nonsmokers exposed to tobacco smoke have similar mutations in *p53* and K-*ras* as those found in smokers (IARC 2002).

Properties

Environmental tobacco smoke is a complex mixture of thousands of chemicals that are emitted from burning tobacco. Tobacco smoking produces both mainstream smoke, which is drawn through the tobacco column and exits through the mouthpiece during puffing, and sidestream smoke, which is emitted from the smoldering tobacco between puffs. Approximately 4,000 chemicals have been identified in mainstream tobacco smoke, and some have estimated that the actual number of compounds may be more than 100,000; however, the current identified compounds make up more than 95% of the total mass. Environmental tobacco smoke is the sum of sidestream smoke, mainstream smoke, compounds that diffuse through the wrapper, and exhaled mainstream smoke. Sidestream smoke contributes at least half

2-94

of the smoke generated. The composition of tobacco smoke is affected by many factors, including the tobacco product, properties of the tobacco blend, chemical additives, smoking pattern, pH, type of paper and filter, and ventilation (IARC 1986, NRC 1986, EPA 1992, Vineis and Caporaso 1995, CEPA 1997).

Although many of the same compounds are present in both mainstream and sidestream smoke, important differences exist. The ratios of compounds in sidestream and mainstream smoke are highly variable; however, there is less variability in emissions from sidestream smoke compared to mainstream smoke because smoking patterns and cigarette design have more of an impact on mainstream smoke (CEPA 1997). Sidestream smoke is generated at lower temperatures than is mainstream smoke (600°C versus 900°C), is produced in an oxygen-deficient environment, and is rapidly diluted and cooled after leaving the burning tobacco. Mainstream smoke is generated at higher temperatures in the presence of oxygen and is drawn through the tobacco column. These conditions favor formation of smaller particulates in sidestream smoke (0.01 to 0.1 μm) compared to mainstream smoke (0.1 to 1 μm). Sidestream smoke also typically contains higher concentrations of ammonia (40 to 170 fold), nitrogen oxides (4 to 10 fold), and chemical carcinogens (e.g., benzene, 10 fold; *N*-nitrosoamines, 6 to 100 fold; and aniline, 30 fold) than mainsteam smoke (IARC 1986).

Tobacco pyrolysis products are formed both during smoke inhalation and during the interval between inhalations (NRC 1986). A number of chemicals present in environmental tobacco smoke are known or suspected toxicants/irritants with various acute health effects. Prominent among them are the respiratory irritants ammonia, formaldehyde, and sulfur dioxide. Acrolein, hydrogen cyanide, and formaldehyde affect mucociliary function and at higher concentrations can inhibit smoke clearance from lungs (Battista 1976). Nicotine is addictive and has several pharmacological and toxicological actions. Nitrogen oxides and phenol are additional toxicants present in environmental tobacco smoke. Over 50 compounds in environmental tobacco smoke have been identified as *known or reasonably anticipated human carcinogens*, including some naturally occurring radionuclides. Most of these compounds are present in the particulate phase (IARC 1986, CEPA 1997).

Use

Environmental tobacco smoke is a by-product of smoking and has no industrial or commercial uses. It is used in scientific research to study its composition and health effects. See the profile on "Tobacco Smoking" for a brief description of the history and uses of tobacco products.

Production

Environmental tobacco smoke is produced by smoking the various forms of tobacco products. Information on tobacco production is provided below in the profile for tobacco smoking.

Exposure

Smoking prevalence in the United States has declined by approximately 40% since reaching a peak in the mid 1960s. Since then, public policies have restricted smoking in buildings and other indoor public places. Nevertheless, environmental tobacco smoke remains as an important source of exposure to indoor air contaminants. Based on data from the Third National Health and Nutrition Examination Survey (NHANES III) conducted from 1988 to 1991, approximately 43% of U.S. children aged 2 months to 11 years lived in a home with at least one smoker. In addition, 37% of non-smoking adults reported exposure to environmental tobacco smoke at home or at work (Pirkle *et al.* 1996). It is estimated that more than half of U.S. youth are still exposed to environmental tobacco smoke (CDC 2001) and approximately 9 to 12 million children, aged six and younger, are exposed to environmental tobacco smoke in their homes (EPA 2002).

Because environmental tobacco smoke is a complex mixture, exposure is difficult to measure. Various monitoring methods typically focus on nicotine levels or respirable suspended particulates in indoor air, or cotinine levels (the primary metabolite of nicotine) in blood, saliva, or urine.

Mean nicotine levels in a variety of indoor environments range from 0.3 to 30 $\mu g/m^3$. Typical average concentrations in homes with at least one smoker range from 2 to 14 $\mu g/m^3$. Nicotine concentrations measured at work from the mid 1970s to 1991 were similar to those measured in homes; however, maximum values were much higher at work (CEPA 1997). Levels of environmental tobacco smoke in restaurants were found to be approximately 1.6 to 2.0 times higher than in office workplaces and 1.5 times higher than in residences with at least one smoker. Isolating smokers to a specific section of restaurants was found to afford some protection for nonsmokers, but the best protection resulted from seating arrangements that segregated smokers by a wall or partition. However, nonsmokers are still exposed to nicotine and respirable particles. Food-servers, who spend more time in restaurants, are exposed even more to environmental tobacco smoke, though they may work in nonsmoking sections (Lambert *et al.* 1993).

Levels of environmental tobacco smoke in bars were found to be approximately 3.9 to 6.1 times higher than in office workplaces and 4.4 to 4.5 times higher than in residences (Siegel 1993). Nicotine levels as high as 50 to 75 $\mu g/m^3$ were measured in bars and on airplanes (before smoking was banned). The highest measured nicotine concentration (1,010 $\mu g/m^3$) was measured in a car with the ventilation system shut off (CEPA 1997).

Environmental tobacco smoke exposure levels have been estimated in many studies by measuring respirable suspended particles (particles less than 2.5 μm in diameter). The average respirable suspended particles values reported in these studies generally ranged from 5 to 500 $\mu g/m^3$. Respirable suspended particles values in homes with one or more smokers had concentrations that were 20 to 100 $\mu g/m^3$ higher than in comparable homes with no smokers (CEPA 1997).

The NHANES III survey indicated that approximately 90% of the U.S. population aged 4 years and older had detectable levels of cotinine (Pirkle *et al.* 1996). The median serum cotinine level among nonsmokers was 0.20 nanograms per milliliter (ng/mL) in 1991, but decreased by more than 75% to 0.05 ng/mL by 1999 (CDC 2001). An independent, nonfederal Task Force on Community Preventive Services, in collaboration with the U.S. Department of Health and Human Services and various public and private partners, recommended various strategies for reducing cigarette smoking and exposure to environmental tobacco smoke. The baseline level for cigarette smoking (1997) was 24%, that for nonsmokers exposed to environmental tobacco smoke (1994) was 65%, and that for children exposed to environmental tobacco smoke (1994) was 27%. The objective is to reduce cigarette smoking to 12% and environmental tobacco smoke exposure to 45% for nonsmoking adults and to 10% for children by 2010 (CDC 2000).

Regulations

Executive Order 13058

It is the policy of the executive branch to establish a smoke-free environment for Federal employees and members of the public visiting or using Federal facilities and, therefore, the smoking of tobacco products is prohibited in all interior space owned, rented, or leased by the executive branch of the Federal Government, and in any outdoor areas under executive branch control in front of air intake ducts

FAA

Smoking of tobacco products is banned on air carrier and foreign air carrier flights in schedule intrastate, interstate and foreign air transportation

OSHA

OSHA has developed regulations that prohibit cigarette smoking in certain hazardous environments

Guidelines

NIOSH

Environmental tobacco smoke is a potential occupational carcinogen; exposure should be reduced to the lowest feasible concentration

*No separate CAS registry number is assigned to environmental tobacco smoke.

REFERENCES

Battista, S. P. 1976. Ciliatoxic Components of Cigarette Smoke. In Smoking and Health I Measurement in the Analysis and Treatment of Smoking Behavior. E. L. Wynder, D. Hoffman and G. B. Gori, eds. Washington, D.C.: U.S. Government Printing Office.

Brownson, R. C., M. C. Alavanja, E. T. Hock and T. S. Loy. 1992. Passive smoking and lung cancer in nonsmoking women. Am J Public Health 82(11): 1525-30.

CDC. 2000. Strategies for reducing exposure to environmental tobacco smoke, increasing tobacco-use cessation, and reducing initiation in communities and health-care systems. A report on recommendations of the Task Force on Community Preventative Services. Morbid Mort Weekly Report 49(RR-12).

CDC. 2001. National Report on Human Exposure to Environmental Chemicals. Reduced Exposure of the U.S. Population to Environmental Tobacco Smoke. Centers for Disease Control. http://www.cdc.gov/nceh/dls/report/highlights.htm#ReducedExposure.

CEPA. 1997. Health Effects of Exposure to Environmental Tobacco Smoke. California Environmental Protection Agency, Office of Environmental Health Hazard Assessment.

EPA. 1992. Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders. EPA/600/6-90/006F. Washington, D.C.: U.S. Environmental Protection Agency, Office of Research and Development.

EPA. 2002. Indoor Air - Secondhand Smoke. Secondhand Smoke/Smoke-free Homes. U.S. Environmental Protection Agency. http://www.epa.gov/iaq/ets.

Fontham, E. T., P. Correa, P. Reynolds, A. Wu-Williams, P. A. Buffler, R. S. Greenberg, *et al.* 1994. Environmental tobacco smoke and lung cancer in nonsmoking women. A multicenter study. Jama 271(22): 1752-9.

IARC. 1986. Tobacco Smoking. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 38. Lyon, France: International Agency for Research on Cancer. 421 pp.

IARC. 2002. Tobacco Smoking and Involuntary Smoking. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 83. Lyon, France: International Agency for Research on Cancer.

Kabat, G. C., S. D. Stellman and E. L. Wynder. 1995. Relation between exposure to environmental tobacco smoke and lung cancer in lifetime nonsmokers. Am J Epidemiol 142(2): 141-8.

Lambert, W. E., J. M. Samet and J. D. Spengler. 1993. Environmental tobacco smoke concentrations in no-smoking and smoking sections of restaurants. Am J Public Health 83(9): 1339-41.

NRC. 1986. Environmental Tobacco Smoke. Measuring exposures and assessing effects. Washington, D.C.: National Academy Press.

Pirkle, J. L., K. M. Flegal, J. T. Bernert, D. J. Brody, R. A. Etzel and K. R. Maurer. 1996. Exposure of the US population to environmental tobacco smoke: the Third National Health and Nutrition Examination Survey, 1988 to 1991. Jama 275(16): 1233-40.

Siegel, M. 1993. Involuntary smoking in the restaurant workplace. A review of employee exposure and health effects. Jama 270(4): 490-3.

Stockwell, H. G., A. L. Goldman, G. H. Lyman, C. I. Noss, A. W. Armstrong, P. A. Pinkham, E. C. Candelora and M. R. Brusa. 1992. Environmental tobacco smoke and lung cancer risk in nonsmoking women. J Natl Cancer Inst 84(18): 1417-22.

Vineis, P. and N. Caporaso. 1995. Tobacco and cancer: epidemiology and the laboratory. Environ Health Perspect 103(2): 156-60.

Wells, A. J. 1998. Lung cancer from passive smoking at work. Am J Public Health 88(7): 1025-9.

Witschi, H., I. Espiritu, R. R. Maronpot, K. E. Pinkerton and A. D. Jones. 1997b. The carcinogenic potential of the gas phase of environmental tobacco smoke. Carcinogenesis 18(11): 2035-42.

Witschi, H., I. Espiritu, J. L. Peake, K. Wu, R. R. Maronpot and K. E. Pinkerton. 1997a. The carcinogenicity of environmental tobacco smoke. Carcinogenesis 18(3): 575-86.

Smokeless Tobacco*

Known to be a human carcinogen

First Listed in the *Ninth Report on Carcinogens* (2000)

Carcinogenicity

The oral use of smokeless tobacco is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans that indicate a causal relationship between exposure to smokeless tobacco and human cancer. Smokeless tobacco has been determined to cause cancers of the oral cavity (IARC 1985, 1987, Gross *et al.* 1995). Cancers of the oral cavity have been associated with the use of chewing tobacco as well as snuff, which are the two main forms of smokeless tobacco used in the United States. Tumors often arise at the site of placement of the tobacco.

The International Agency for Research on Cancer (IARC) (IARC 1985, 1987) determined that there was inadequate evidence for the carcinogenicity of smokeless tobacco in experimental animals. Most reported studies had deficiencies in design. Subsequent studies provided some evidence that snuff or extracts of snuff produced tumors of the oral cavity in rats (Johansson *et al.* 1989).

Additional Information Relevant to Carcinogenicity

Smokeless tobacco products contain a variety of nitrosamines that are carcinogenic to animals and are *reasonably anticipated to be human carcinogens.* The oral use of smokeless tobacco is estimated to be the greatest exogenous source of human exposure to these compounds. Nitrosamines are metabolically hydroxylated to form unstable compounds that bind to DNA. Extracts of smokeless tobacco have been shown to induce mutations in bacteria and mutations and chromosomal aberrations in mammalian cells. Furthermore, cells in oral cavity tissue from smokeless tobacco users have been shown to contain more chromosomal damage than those from nonusers (IARC 1985).

Properties

Chewing tobacco and snuff are the two main forms of smokeless tobacco used in the United States. Chewing tobacco consists of the tobacco leaf with the stem removed and various sweeteners and flavorings such as honey, licorice, and rum. Snuff consists of the entire tobacco leaf (dried and powdered or finely cut), menthol, peppermint oil, camphor, and/or aromatic additives such as attar of roses and oil of cloves (IARC 1985).

Tobacco contains more than 2,500 chemical constituents. Some of these chemicals are applied to tobacco during cultivation, harvesting, and processing. The major chemical groups include aliphatic and aromatic hydrocarbons, aldehydes, ketones, alcohols, phenols, ethers, alkaloids, carboxylic acids, esters, anhydrides, lactones, carbohydrates, amines, amides, imides, nitrites, *N*- and *O*-heterocyclic compounds, chlorinated organic compounds, and at least 35 metal compounds. Smokeless tobacco products contain known carcinogens such as volatile and nonvolatile nitrosamines, tobacco-specific *N*-nitrosamines (TSNAs), polynuclear aromatic hydrocarbons, and polonium-210 (^{210}Po). The carcinogenic TSNAs are present at concentrations that are at least two-fold higher than the concentration found in other consumer products (Brunnemann *et al.* 1986).

TSNAs, including 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone (NNK) and *N*-nitrosonornicotine (NNN), present in tobacco are formed from nicotine and other tobacco alkaloids. The concentrations of NNK and NNN, the most carcinogenic of the TSNAs, are high enough in tobacco that their total estimated doses to long-term snuff users are similar in magnitude to the total doses required to produce cancer in laboratory animals (Hecht and Hoffman 1989).

Use

Tobacco was widely used by native populations throughout both North and South America by the time the first European explorers arrived in the late 1400s and early 1500s. Over the next few centuries, tobacco use spread to Europe, Africa, China, and Japan. Snuff use was introduced to North American colonists at Jamestown, Virginia in 1611. Tobacco chewing among American colonists began in the early 1700s, but was not widely accepted until the 1850s (IARC 1985).

Snuff was the most popular form of tobacco in both Europe and the United States prior to the 1800s. At that time, the finely ground tobacco was primarily sniffed through the nose. The current practice in the United States is to place a small pinch between the lip and gum or cheek and gum (IARC 1985). Moist snuff is the only smokeless tobacco product that has shown increased sales in the United States in recent years. This product is considered the most dangerous form of smokeless tobacco (NCI 1991, USDA 2001). In the three leading brands of snuff

2-96

that account for 92% of the U.S. market, concentrations of nicotine and TSNAs were significantly higher than in the fourth and fifth most popular brands (Hoffman *et al.* 1995). The highest per-capita consumption of snuff in the United States occurred from 1910 to 1920 at 0.5 lb, but had decreased to 0.15 lb by 1979. After the USDA reclassified several chewing tobacco products as snuff in 1982, the male per-capita consumption of snuff increased to 0.26 lb and remained at 0.2 to 0.3 lb through 2000 (IARC 1985, USDA 2001).

Peak consumption of chewing tobacco in the United States for persons aged 15 years and over occurred in 1900 at 4.1 lb and gradually declined to 0.5 lb by 1962. However, per-capita consumption for males aged 18 and over ranged from 1.05 to 1.34 lb between 1966 and 1983 (IARC 1985). Per-capita consumption for males declined to 0.8 lb in 1991, increased to 1.04 lb in 1992, and then declined gradually to 0.9 lb by 2000 (USDA 2001).

Production

There are five major manufacturers of smokeless tobacco products in the United States. These five companies control approximately 99% of the market. The largest of these companies controls more than 40% of the total smokeless tobacco market and approximately 75% of the moist snuff market (FTC 2001).

U.S. production of snuff increased from approximately 1.8 million kilograms (4 million pounds) in 1880 to more than 18 million kilograms (40 million pounds) in 1930. Production remained steady through 1950 at approximately 16.4 to 19.9 million kilograms/yr (36 to 44 million pounds/yr) and then declined to approximately 10.9 million kilograms (24 million pounds) by 1980 (IARC 1985). Since 1986, U.S. sales of moist snuff have steadily increased from approximately 36 million pounds (16.4 million kilograms) to more than 58 million pounds (26.5 million kilograms) in 1999. Sales of Scotch snuff or dry snuff products declined from approximately 8.1 million pounds (3.7 million kilograms) in 1986 to 3.6 million pounds (1.6 million kilograms) in 1999 (FTC 2001). The United States imported approximately 7,900 kg (17,400 lb) of snuff and snuff flours in 2000 and 4,500 kg (9,900 lb) in 2002. Exports were approximately 620,800 kg (1.4 million pounds) in 2000 and 560,000 kg (1.2 million pounds) in 2002 (ITA 2003).

Chewing tobacco products include plug, moist plug, twist/roll, and loose leaf. Total U.S. production declined from approximately 67.4 million kilograms (148.6 million pounds) in 1931 to 29.4 million kilograms (64.8 million pounds) in 1962. Production then rose to 48.1 million kilograms (106.0 million ponds) by 1980, but has shown steady declines since then. Plug tobacco accounted for approximately 51% of production in 1931, but only approximately 16% by 1980. During this time, loose-leaf tobacco increased its share of the market from approximately 41% to 68% (IARC 1985). Sales of loose-leaf chewing tobacco were approximately 65.7 million pounds (29.8 million kilograms) in 1986, but declined to approximately 44.5 million pounds (20.2 million kilograms) in 1999. Sales of plug and twist chewing tobacco combined were 8.8 million pounds (4 million kilograms) in 1986 and 2.8 million pounds (1.3 million kilograms) in 1999 (FTC 2001). U.S. imports of chewing tobacco were approximately 38,200 kg (84,200 lb) in 2000 and 97,900 kg (215,900 lb) in 2002. Exports were 116,500 kg (256,800 lb) in 2000 and 59,700 kg (131,600 lb) in 2002 (ITA 2003).

Exposure

Individuals that use smokeless tobacco are primarily exposed by absorption through the oral or nasal mucosa and ingestion. Occupational exposure to tobacco may occur from skin contact, inhalation of dust, and ingestion of dust during processing and manufacturing. Many smokeless tobacco users are exposed during most of their working hours, and some use these products 24 hours/day (IARC 1985).

Consumption of smokeless tobacco products showed resurgence in the late 1970s after decades of decline. Increased use of these products was particularly dramatic among adolescent boys, increasing by 250% or more between 1970 and 1985 (NCI 1991). The percentage of current users, aged 18 and up, in the United States population ranges from approximately 1.4% to 8.8% across the states. Use was much higher among men (2.6% to 18.4%) than women (0 to 1.7%) in 17 states surveyed in 1997 (CDC 1998). The estimated number of smokeless tobacco users in the early 1980s ranged from 7 to 22 million (IARC 1985). In 1991, 2.9% of adults aged 18 and over were current users of smokeless tobacco. This value included an estimated 4.8 million men and 0.53 million women. Approximately 67% of snuff users and 45% of chewing tobacco users reported daily use. The prevalence of use was highest (8.2%) in men aged 18 to 24 (CDC 1993). More recent data indicate that there are approximately 10 million users of smokeless tobacco in the United States and approximately 3 million of these are under 21 years of age (UoM 2001).

Regulations

FTC

All smokeless tobacco products and advertisements for smokeless tobacco must contain a label statement on the risks of smokeless tobacco

*No separate CAS registry number is assigned to smokeless tobacco.

REFERENCES

Brunnemann, K. D., B. Prokopczyk, J. Nair, H. Ohshima and H. Bartsch. 1986. Laboratory studies on oral cancer and smokeless tobacco. Banbury Rep 23: 197-213.

CDC. 1993. Use of smokeless tobacco among adults - United States, 1991. Morbid Mort Weekly Report 42(14): 263-266.

CDC. 1998. Cigarette smoking, smokeless tobacco use, and per capita tax-paid sales of cigarettes. Morbid Mort Weekly Report 47(43): 922-926.

FTC. 2001. Smokeless Tobacco Report. Report to Congress for the Years 1998 and 1999. Federal Trade Commission. http://www.ftc.gov/reports/tobacco/smokeless98_99.htm.

Gross, A. J., D. T. Lackland and D. S. Tu. 1995. Oral cancer and smokeless tobacco: Literature review and meta-analysis. Environ Int 21(4): 381-394.

Hecht, S. S. and D. Hoffmann. 1989. The relevance of tobacco-specific nitrosamines to human cancer. Cancer Surv 8(2): 273-94.

Hoffman, D., M. Djordjevic, J. Fan, E. Zang, T. Glynn and G. Connolly. 1995. Five leading U.S. commercial brands of moist snuff in 1994: Assessment of carcinogenic *N*-nitrosamines. J Natl Cancer Inst 87: 1862-1869.

IARC. 1985. Tobacco Habits Other than Smoking: Betel-Quid and Areca-Nut Chewing; Some related Nitrosamines. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 37. Lyon, France: International Agency for Research on Cancer. 291 pp.

IARC. 1987. Overall Evaluations of Carcinogenicity. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, Supplement 7. Lyon, France: International Agency for Research on Cancer. 440 pp.

ITA. 2003. Subheading 240399: Manufactured Tobacco and its Substitutes, Tobacco Extracts and Essences. International Trade Administration. U.S. Department of Commerce. http://www.ita.doc.gov/td/industry/otea/Trade-Detail/.

Johansson, S. L., J. M. Hirsch, P. A. Larsson, J. Saidi and B. G. Osterdahl. 1989. Snuff-induced carcinogenesis: effect of snuff in rats initiated with 4-nitroquinoline *N*-oxide. Cancer Res 49(11): 3063-9.

NCI. 1991. Smokeless Tobacco or Health, Monograph 2. National Institutes of Health. http://rex.nci.nih.gov/NCI_MONOGRAPHS/MONO2/MONO2.htm.

UoM. 2001. Tobacco and Periodontal Diseases. Smokeless Tobacco Facts. University of Michigan. http://www1.umn.edu/perio/tobacco/smokeless.html.

USDA. 2001. Tobacco Situation and Outlook (TBS-249). Economic Research Service. 9 pp.

Tobacco Smoking*

Known to be a human carcinogen

First Listed in the *Ninth Report on Carcinogens* (2000)

Carcinogenicity

Tobacco smoking is *known to be a human carcinogen* based on sufficient evidence of carcinogenicity from studies in humans, which indicate a causal relationship between tobacco smoking and human cancer. Tobacco smoking has been determined to cause cancer of the lung, urinary bladder, renal pelvis, oral cavity, pharynx, larynx, esophagus, lip, and pancreas in humans (IARC 1986). Lung cancer deaths are associated with certain tobacco smoking patterns; these patterns increase with increasing consumption of tobacco products and decrease in certain

2-97

groups as the amount of tobacco smoked declines. Smoking cessation is associated with a decreased risk of developing cancer. The carcinogenic effects of tobacco smoke are increased in individuals with certain predisposing genetic polymorphisms. Since tobacco smoking was first listed in the *Ninth Report on Carcinogens,* the International Agency for Research on Cancer (IARC) reviewed tobacco smoking and tobacco smoke again. They concluded that there was sufficient evidence for the carcinogenicity of cigarette smoking and cancers of the nasal cavities and nasal sinus, stomach, liver, kidney (renal cell carcinoma), uterine cervix, and myeloid leukemia in addition to the tissue sites mentioned above.

Tobacco smoke has been demonstrated to be carcinogenic in several species of experimental animals. The carcinogenicity of cigarette smoke has been tested by inhalation in mice, rats, hamsters, and dogs. The evidence is most clearly established for the larynx in the hamster following inhalation of tobacco smoke. Inhalation exposure to tobacco smoke also resulted in malignant respiratory tract tumors in rats and lung tumors in mice and dogs; however, the incidence of lung tumors was not statistically significant in mice, and the data were insufficient for evaluation in dogs. Concomitant exposure to tobacco smoke and other carcinogens (polycyclic aromatic hydrocarbons or radon daughters) increased tumor incidence compared to either substance alone. Tobacco-smoke condensate has been tested by topical application in mice, rats, and rabbits. The strongest evidence is for skin tumors in mice receiving dermal applications of tobacco smoke condensates. Dermal application of cigarette-smoke condensate also caused skin tumors in rabbits, and topical application to the oral mucosa caused lung tumors and lymphomas in mice. Intrapulmonary injection of cigarette-smoke condensate caused lung tumors in rats (IARC 1986, 1987).

Additional Information Relevant to Carcinogenicity

Individual chemical components of tobacco smoke have been shown to be carcinogenic to humans and/or experimental animals. Tobacco smoke or tobacco smoke condensates cause cell transformation and mutations or other genetic alterations in a variety of *in vitro* and *in vivo* assays. The urine of smokers has been found to be mutagenic, and there is evidence that the somatic cells of smokers contain more chromosomal damage than those of nonsmokers (IARC 1986). Lung tumors from smokers contained a higher frequency of mutations in *p53* and K-*ras* than tumors from non-smokers. Most of the mutations are G to T transversions (Vineis and Caporaso 1995, IARC 2002).

Properties

Tobacco smoking produces both mainstream smoke (drawn through the tobacco column and exiting through the mouthpiece during puffing) and sidestream smoke (emitted from the smoldering tobacco between puffs). The composition of tobacco smoke is affected by many factors, including the tobacco product, properties of the tobacco blend, chemical additives, smoking pattern, pH, type of paper, filter, and ventilation. Mainstream tobacco smoke contains 4,000 or more chemicals. These include carbon oxides, nitrogen oxides, ammonia, hydrogen cyanide, volatile aldehydes and ketones, nonvolatile alkanes and alkenes, benzene, hydrazine, vinyl chloride, isoprenoids, phytosterols, polynuclear aromatic compounds, alcohols, nonvolatile aldehydes and ketones, phenols, quinones, carboxylic acids, esters, lactones, amines and amides, alkaloids, pyridines, pyrroles, pyrazines, *N*-nitrosamines, metals, radioactive elements, agricultural chemicals, and chemical additives. Mainstream smoke includes more than 400 individual gaseous components with nitrogen (58%), carbon dioxide (13%), oxygen (12%), carbon monoxide (3.5%) and hydrogen (0.5%) dominating. Particulate phase components account for approximately 8% and other vapor phase components for approximately 5% of mainstream smoke (IARC 1986, Vineis and Caporaso 1995).

Although many of the same compounds are present in both mainstream and sidestream smoke, there are important differences. Sidestream smoke is generated at lower temperatures than is mainstream smoke (600°C versus 900°C), is produced in an oxygen-deficient environment, and is rapidly diluted and cooled after leaving the burning tobacco. Mainstream smoke is generated at higher temperatures in the presence of oxygen and is drawn through the tobacco column. These conditions favor formation of smaller particulates in sidestream smoke (0.01 to 0.1 μm) compared to mainstream smoke (0.1 to 1 μm). Sidestream smoke also typically contains higher concentrations of ammonia (40 to 170 fold), nitrogen oxides (4 to 10 fold), and chemical carcinogens (e.g., benzene, 10 fold; *N*-nitrosoamines, 6 to 100 fold; and aniline, 30 fold) than mainstream smoke (IARC 1986).

Use

Smoking was introduced to Europe from the Americas in the middle of the sixteenth century and then spread throughout the world. Currently, the primary source for tobacco smoke is cigarettes. Pipes, cigars, bidis, and other forms are used less frequently (IARC 1986). The use of pipes and cigars was more prevalent in the 18th and 19th centuries, but there was a shift from these products to cigarettes after 1910. Per-capita consumption of cigarettes in the United States was 54 in 1900, peaked at 4,345 in 1963, and declined to fewer than 2,000 by 2002 (ALA 2003). Data from the 2002 National Survey on Drug Use and Health (NSDUH) for past month tobacco use indicated that 30.4% of persons in the United States aged 12 or older reported any tobacco use, while 26.0% reported use of cigarettes, 5.4% cigars, and 0.8% pipes (SAMHSA 2003).

The use of tobacco products varies with gender, age, education, and culture. The percentage of adults who smoke cigarettes has declined steadily from 42.4% in 1965 to 22.6% in 2001. Prevalence of smoking has always been higher in men than women. More than half (51.9%) of adult men smoked in 1965, compared to 33.9% of women. Smoking prevalence peaked at 67% for men in the 1940s and 1950s and at 44% for women in the 1960s. By 2001, the percentages declined to 24.9% for men and 20.6% for women. Smoking prevalence was highest in the 25 to 44 age group between 1965 and the mid 1990s. However, smoking increased in the 18 to 24 age group during the 1990s reaching a peak in 1997, while prevalence continued to decrease in the 25 to 44 age group. Since 1997, smoking prevalence has been highest in the 18 to 24 age group. Smoking prevalences as of 2001 by ethnic group are as follows: Native Americans (31.5%), non-Hispanic whites (24%), non-Hispanic blacks (22%), Hispanics (16.5%), and Asians (12.5%). Overall, smoking declined by approximately 47% in the United States from 1965 to 2001 (ALA 2003).

Although the percentage of adults that smoke has shown a steady decline since the mid 1960s, the total number of smokers has remained about the same since the early 1990s. Smoking among high school students has declined after increasing during the first half of the 1990s. Per capita consumption of cigarettes also declined. The percentage of adult smokers who smoke fewer than 15 cigarettes per day increased by 48% between 1974 and 2001, while the percentage of heavy smokers (more than 24 cigarettes/day) declined by 42%. The prevalence of smoking cessation increased by more than 70% between 1965 and 2001, with approximately 44.8 million adults identified as former smokers (ALA 2003).

Production

Tobacco has been an important economic agricultural crop since the 1600s. North and Central America produce the highest quantity. *Nicotiana tabacum* is the most common species of tobacco used in cigarettes, but *N. rustica* is also used in some areas. For smoking

tobacco, the tobacco leaf material is manipulated by physical and chemical methods during the manufacturing process, some of which are intended to reduce the yields of toxic agents and tars in smoke. The tobacco is fine cut and wrapped in paper for consumption. Generally, cigarettes are a blend of different flue-cured grades, burley, Maryland, and oriental tobaccos (IARC 1986). The total tobacco harvest in the United States ranged from approximately 1.19 to 1.79 billion pounds/yr (540 to 812 million kilograms/yr) between 1987 and 1997 (USDA 1993, 1998). The Unites States imported more than 11 billion cigarettes in 2000 and more than 20 billion in 2002. Exports greatly exceed imports with more than 148 billion cigarettes in 2000 and 127 billion in 2002 (ITA 2003).

Exposure

Smokers are primarily exposed by inhalation; however, some exposure may occur by absorption of chemicals present in the tobacco or tobacco smoke directly through the lining of the mouth and gums. In addition, nonsmokers may be exposed by inhalation of tobacco smoke any time they are near smokers (see the profile for Environmental Tobacco Smoke above). In 1991, for the first time in more than 25 years of observation, the percentage of the adult U.S. population who had not smoked or had smoked fewer than 100 cigarettes was more than 50%. Cigarette consumption levels in the United States increased from 2.5 billion in 1900 to 640 billion in 1981 but have declined since then to 420 billion by 2002. There were an estimated 46.2 million adult smokers in the United States in 2001, which is a 7.8% decrease since 1965 (ALA 2003).

Current strategies in the United States for reducing exposure to tobacco smoke include goals for increasing tobacco-use cessation and reducing the number of new smokers. The objectives include reducing smoking prevalence among U.S. adults to 12%, and increasing smoking cessation attempts to 75% for adult smokers and 84% for adolescent smokers by 2010 (CDC 2000).

Regulations

Executive Order 13058

It is the policy of the executive branch to establish a smoke-free environment for Federal employees and members of the public visiting or using Federal facilities and, therefore, the smoking of tobacco products is prohibited in all interior space owned, rented, or leased by the executive branch of the Federal Government, and in any outdoor areas under executive branch control in front of air intake ducts

FDA

Oral contraceptives must contain a package insert concerning the increased risks associated with tobacco smoking and oral contraceptive use

FTC

All cigarette packages and advertisements for cigarettes must contain a label statement on the risks of smoking

OSHA

OSHA has developed regulations that prohibit cigarette smoking in certain hazardous environments

*No separate CAS registry number is assigned to tobacco smoking.

REFERENCES

ALA. 2003. Trends in Tobacco Use. Epidemiology and Statistics Unit. American Lung Association. http://www.lungusa.org/data (Smoking (Narrative and Tables)).

CDC. 2000. Strategies for reducing exposure to environmental tobacco smoke, increasing tobacco-use cessation, and reducing initiation in communities and health-care systems. A report on recommendations of the Task Force on Community Preventative Services. Morbid Mort Weekly Report 49(RR-12).

IARC. 1986. Tobacco Smoking. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, vol. 38. Lyon, France: International Agency for Research on Cancer. 421 pp.

IARC. 1987. Overall Evaluations of Carcinogenicity. IARC Monographs on the Evaluation of Carcinogenic Risk of Chemicals to Humans, Supplement 7. Lyon, France: International Agency for Research on Cancer. 440 pp.

IARC. 2002. Tobacco Smoking and Involuntary Smoking. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, vol. 83. Lyon, France: International Agency for Research on Cancer.

ITA. 2003. Subheading 240220: Cigarettes Containing Tobacco. International Trade Administration. U.S. Department of Commerce. http://www.ita.doc.gov/td/industry/otea/Trade-Detail/.

SAMHSA. 2003. Results From the 2002 National Survey on Drug Use and Health. U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration. Last updated: 9/23/03. http://www.samhsa.gov/oas/nhsda/2k2nsduh/Results/2k2Results.htm. Last accessed: 3/4/04.

USDA. 1993. Field Crops. Final estimates 1987-1992. Statistical Bulletin No. 896. National Agriculture Statistics Service. http://usda.mannlib.cornell.edu/.

USDA. 1998. Field Crops. Final estimates 1992-1997. Statistical Bulletin No. 947. National Agriculture Statistics Service. http://www.usda.gov/nass/pubs/histdata.htm.

Vineis, P. and N. Caporaso. 1995. Tobacco and cancer: epidemiology and the laboratory. Environ Health Perspect 103(2): 156-60.

Title: Involuntary Smoking

Publication Date: 2002

Agency or Author: International Agency for Research on Cancer (IARC)

Purpose: This document provides evidence on exposure, human carcinogenicity and animal models to establish Environmental Tobacco Smoke (ETS) as a group 1 carcinogen. Group 1 carcinogens are those substances that are known human carcinogens.

INVOLUNTARY SMOKING
(Group 1)

For definition of groups, see Preamble.

VOL.: 83 (2002)

5. Summary of Data Reported and Evaluation

5.1 Exposure data

Involuntary (or passive) smoking is exposure to secondhand tobacco smoke, which is a mixture of exhaled mainstream smoke and sidestream smoke released from the smouldering cigarette or other smoking device (cigar, pipe, bidi, etc.) and diluted with ambient air. Involuntary smoking involves inhaling carcinogens, as well as other toxic components, that are present in secondhand tobacco smoke. Secondhand tobacco smoke is sometimes referred to as 'environmental' tobacco smoke. Carcinogens that occur in secondhand tobacco smoke include benzene, 1,3-butadiene, benzo[*a*]pyrene, 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone and many others.

Secondhand tobacco smoke consists of a gas phase and a particulate phase; it changes during its dilution and distribution in the environment and upon ageing. The concentrations of respirable particles may be elevated substantially in enclosed spaces containing secondhand tobacco smoke. The composition of tobacco smoke inhaled involuntarily is variable quantitatively and depends on the smoking patterns of the smokers who are producing the smoke as well as the composition and design of the cigarettes or other smoking devices. The secondhand tobacco smoke produced by smoking cigarettes has been most intensively studied.

Secondhand tobacco smoke contains nicotine as well as carcinogens and toxins. Nicotine concentrations in the air in homes of smokers and in workplaces where smoking is permitted typically range on average from 2 to 10 micrograms/m^3.

5.2 Human carcinogenicity data

Lung cancer

Involuntary smoking involves exposure to the same numerous carcinogens and toxic substances that are present in tobacco smoke produced by active smoking, which is the principal cause of lung cancer. As noted in the previous *IARC Monograph* on tobacco smoking, this implies that there will be some risk of lung cancer from exposure to secondhand tobacco smoke.

More than 50 studies of involuntary smoking and lung cancer risk in never-smokers, especially spouses of smokers, have been published during the last 25 years. These studies have been carried out in many countries. Most showed an increased risk, especially for persons with higher exposures. To evaluate the information collectively, in particular from those studies with a limited number of cases, meta-analyses have been conducted in which the relative risk estimates from the individual studies are pooled together. These meta-analyses show that there is a statistically significant and consistent association between lung cancer risk in spouses of smokers and exposure to secondhand tobacco smoke from the spouse who smokes. The excess risk is of the order of 20% for women and 30% for men and remains after controlling for some potential sources of bias and confounding. The excess risk increases with increasing exposure. Furthermore, other published meta-analyses of lung cancer in never-smokers

2-101

exposed to secondhand tobacco smoke at the workplace have found a statistically significant increase in risk of 12–19%. This evidence is sufficient to conclude that involuntary smoking is a cause of lung cancer in never-smokers. The magnitudes of the observed risks are reasonably consistent with predictions based on studies of active smoking in many populations.

Breast cancer

The collective evidence on breast cancer risk associated with involuntary exposure of never-smokers to tobacco smoke is inconsistent. Although four of the 10 case–control studies found statistically significant increases in risks, prospective cohort studies as a whole and, particularly, the two large cohort studies in the USA of nurses and of volunteers in the Cancer Prevention Study II provided no support for a causal relation between involuntary exposure to tobacco smoke and breast cancer in never-smokers. The lack of a positive dose–response also argues against a causal interpretation of these findings. Finally, the lack of an association of breast cancer with active smoking weighs heavily against the possibility that involuntary smoking increases the risk for breast cancer, as no data are available to establish that different mechanisms of carcinogenic action operate at the different dose levels of active and of involuntary smoking.

Childhood cancer

Overall, the findings from studies of childhood cancer and exposure to parental smoking are inconsistent and are likely to be affected by bias. There is a suggestion of a modest association between exposure to maternal tobacco smoke during pregnancy and childhood cancer for all cancer sites combined; however, this is in contrast with the null findings for individual sites. Studies on paternal tobacco smoking suggest a small increased risk for lymphomas, but bias and confounding cannot be ruled out.

Other cancer sites

Data are conflicting and sparse for associations between involuntary smoking and cancers of the nasopharynx, nasal cavity, paranasal sinuses, cervix, gastrointestinal tract and cancers at all sites combined. It is unlikely that any effects are produced in passive smokers that are not produced to a greater extent in active smokers or that types of effects that are not seen in active smokers will be seen in passive smokers.

5.3 Animal carcinogenicity data

Secondhand tobacco smoke for carcinogenicity studies in animals is produced by machines that simulate human active smoking patterns and combine mainstream and sidestream smoke in various proportions. Such mixtures have been tested for carcinogenicity by inhalation studies in rodents. The experimental model systems for exposure to secondhand tobacco smoke do not fully simulate human exposures, and the tumours that develop in animals are not completely representative of human cancer. Nevertheless, the animal data provide valuable insights regarding the carcinogenic potential of secondhand tobacco smoke.

A mixture of 89% sidestream smoke and 11% mainstream smoke has been tested for carcinogenic activity in mouse strains that are highly susceptible to lung tumours (strains A/J and Swiss). In strain A/J mice, this mixture consistently produces a significant, modest increase in lung tumour incidence and lung tumour multiplicity when the mice are exposed for 5 months followed by a 4-month recovery period. These lung tumours are predominantly adenomas. Continuous exposure of strain A/J mice to the above mixture of mainstream and sidestream tobacco smoke for 9 months with no

2-102

recovery period did not increase the incidence of lung tumours. In Swiss strain mice, the same mixture induced lung tumours by both protocols, i.e. when the animals were exposed for 5 months followed by a 4-month recovery period and when they were exposed continuously for 9 months with no recovery period. In addition, exposure of Swiss mice to the tobacco smoke mixture for a shorter period was sufficient to induce lung tumours.

Condensates of sidestream and of mainstream cigarette smoke have been tested for carcinogenicity. Both kinds of condensates produced a spectrum of benign and malignant skin tumours in mice following topical application, and the sidestream condensate exhibited higher carcinogenic activity. Sidestream smoke condensate was shown to produce a dose-dependent increase in lung tumours in rats following implantation into the lungs.

Increased relative risks for lung and sinonasal cancer have been reported in companion animals (dogs) exposed to secondhand tobacco smoke in homes.

5.4 Other relevant data

Involuntary smoking has been associated with a number of non-neoplastic diseases and adverse effects in never-smokers, including both children and adults. Epidemiological studies have demonstrated that exposure to secondhand tobacco smoke is causally associated with coronary heart disease. From the available meta-analyses, it has been estimated that involuntary smoking increases the risk of an acute coronary heart disease event by 25–35%. Adverse effects of involuntary smoking on the respiratory system have also been detected. In adults, the strongest evidence for a causal relation exists for chronic respiratory symptoms. Some effects on lung function have been detected, but their medical relevance is uncertain.

Data on the hormonal and metabolic effects of involuntary smoking are sparse. However, female involuntary smokers do not appear to weigh less than women who are not exposed to secondhand tobacco smoke, a pattern that contrasts with the findings for active smoking. No consistent association of maternal exposure to secondhand smoke with fertility or fecundity has been identified. There is no clear association of passive smoking with age at menopause.

Maternal cigarette smoking has repeatedly been associated with adverse effects on fetal growth; full-term infants born to women who smoke weigh about 200 g less than those born to nonsmokers. A smaller adverse effect has been attributed to maternal passive smoking.

Cotinine, and its parent compound nicotine, are highly specific for exposure to secondhand smoke. Because of its favourable biological half-life and the sensitivity of techniques for quantifying it, cotinine is currently the most suitable biomarker for assessing recent exposure to secondhand tobacco smoke uptake and metabolism in adults, children and newborns.

Several studies in humans have shown that concentrations of adducts of carcinogens to biological macromolecules, including haemoglobin adducts of aromatic amines and albumin adducts of polycyclic aromatic hydrocarbons, are higher in adult involuntary smokers and in the children of smoking mothers than in individuals not exposed to secondhand tobacco smoke. Protein adduct concentrations in fetal cord blood correlate with those in maternal blood but are lower. Fewer studies have investigated DNA adduct levels in white blood cells of exposed and unexposed nonsmokers, and most studies have not shown clear differences.

In studies of urinary biomarkers, metabolites of the tobacco-specific carcinogen, 4-

2-103

(methylnitrosamino)-1-(3-pyridyl)-1-butanone, have been found to be consistently elevated in involuntary smokers. Levels of these metabolites are 1–5% as great as those found in smokers. The data demonstrating uptake of 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone, a lung carcinogen in rodents, by nonsmokers are supportive of a causal link between exposure to secondhand tobacco smoke and development of lung cancer.

The exposure of experimental animals, primarily rodents, to secondhand tobacco smoke has several biological effects that include (i) increases or decreases in the activity of phase I enzymes involved in carcinogen metabolism; (ii) increased expression of nitric oxide synthase, xanthine oxidase and various protein kinases; (iii) the formation of smoke-related DNA adducts in several tissues; and (iv) the presence of urinary biomarkers of exposure to tobacco smoke.

In adult experimental animals, sidestream tobacco smoke has been found to produce changes that are similar to those observed with exposure of humans to secondhand tobacco smoke. These include inflammatory changes in the airways and accelerated formation of arteriosclerotic plaques. Although the changes are often comparatively minor and require exposure to rather elevated concentrations of sidestream smoke, they support the results of human epidemiological studies. During pre- and postnatal exposure, sidestream smoke produces intrauterine growth retardation, changes the pattern of metabolic enzymes in the developing lung, and gives rise to hyperplasia of the pulmonary neuroendocrine cell population. In addition, it adversely affects pulmonary compliance and airway responsiveness to pharmacological challenges.

In humans, involuntary smoking is associated with increased concentrations of mutagens in urine. Some studies have shown a correlation of urinary mutagenicity with concentrations of urinary cotinine. Increased levels of sister chromatid exchanges have not been observed in involuntary smokers; however, there is some indication of elevated levels in exposed children. Lung tumours from nonsmokers exposed to tobacco smoke contain *TP53* and *KRAS* mutations that are similar to those found in tumours from smokers. The genotoxicity of sidestream smoke, 'environmental' tobacco smoke, sidestream smoke condensate or a mixture of sidestream and mainstream smoke condensates has been demonstrated in experimental systems *in vitro* and *in vivo*.

5.5 Evaluation

There is *sufficient evidence* that involuntary smoking (exposure to secondhand or 'environmental' tobacco smoke) causes lung cancer in humans.

There is *limited evidence* in experimental animals for the carcinogenicity of mixtures of mainstream and sidestream tobacco smoke.

There is *sufficient evidence* in experimental animals for the carcinogenicity of sidestream smoke condensates.

In addition, the Working Group noted that there are published reports on possible carcinogenic effects of secondhand tobacco smoke in household pet dogs.

Overall evaluation

Involuntary smoking (exposure to secondhand or 'environmental' tobacco smoke) is *carcinogenic to humans (Group 1)*.

2-104

Title: Indoor Air Quality of Hospitality Venues Before and After Implementation of a Clean Indoor Air Law – Western New York, 2003

Publication Date: November 2004

Agency or Author: Morbidity and Mortality Weekly Report

Purpose: To establish the impact on air quality of Clean Indoor Air (CIA) laws. This document measures particulate matter levels in the air of hospitality venues before and after the implementation of CIA laws. Conclusions show that particulate matter was decreased in all types of venues (bar, restaurant, bowling alleys) by an average of 84%.

MMWR™
Morbidity and Mortality Weekly Report
Weekly November 12, 2004 / Vol. 53 / No. 44

Great American Smokeout — November 18, 2004

In 2002, a total of 45.8 million U.S. adults (22.5%) were current smokers, a decrease from 24.1% in 1998, and an estimated 46 million adults were former smokers (*1*). For the first time, more adults had quit smoking than were still smoking (*1*). To assist in continuing this trend, the American Cancer Society (ACS) is sponsoring the 28th Great American Smokeout on November 18, 2004. Cigarette smokers are encouraged to quit smoking for at least 24 hours in the hope they might stop smoking.

The likelihood of permanently quitting smoking is increased when effective therapies are used, such as physician assistance, pharmacologic treatment, and behavioral counseling (*2*). In addition to individual methods, an environmental approach to reducing tobacco use involves increasing the excise tax for tobacco products, developing multicomponent mass media campaigns, fostering provider reminder systems, using telephone quitlines, reducing patient out-of-pocket costs for effective cessation therapies, and reducing exposure to secondhand smoke through smoking bans and restrictions (*3*). Additional information about the Great American Smokeout is available at http://www.cancer.org or by telephone, 800-227-2345.

References

1. CDC. Cigarette smoking among adults—United States, 2002. MMWR 2004;53:427–31.
2. Fiore MC, Bailey WC, Cohen SJ, et al. Treating tobacco use and dependence: clinical practice guidelines. Rockville, MD: US Department of Health and Human Services, Public Health Service; 2000. AHQR publication 00-0032.
3. CDC. Strategies for reducing exposure to environmental tobacco smoke, increasing tobacco-use cessation, and reducing initiation in communities and health-care systems: a report on recommendations of the Task Force on Community Preventive Services. MMWR 2000;49(No. RR-12):2–9.

State-Specific Prevalence of Current Cigarette Smoking Among Adults — United States, 2003

Cigarette smoking causes approximately 440,000 deaths annually in the United States (*1*). To assess the prevalence of current cigarette smoking among adults, CDC analyzed data from the 2003 Behavioral Risk Factor Surveillance System (BRFSS) survey. This report summarizes the results of that analysis, which indicated substantial variation in cigarette smoking prevalence in the 50 states, the District of Columbia (DC), Guam, Puerto Rico, and the U.S. Virgin Islands (USVI) (range: 10.0%–34.0%). To further reduce the prevalence of smoking, states/areas should implement comprehensive tobacco-control programs.

BRFSS is a state-based, random-digit–dialed, telephone survey of the U.S. civilian, noninstitutionalized population aged ≥18 years. In 2003, the median state/area response rate was 53.2% (range: 34.4%–80.5%). Estimates were weighted by age and sex distributions for each state's population, and 95% confidence intervals were calculated. BRFSS respondents were asked, "Have you smoked at least 100 cigarettes in your entire life?" and "Do you now smoke cigarettes every day, some

INSIDE

DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

Z-107

The *MMWR* series of publications is published by the Coordinating Center for Health Information and Service (Proposed), Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION
Centers for Disease Control and Prevention. [Article Title]. MMWR 2004;53:[inclusive page numbers].

days, or not at all?" Current smokers were defined as those who reported having smoked ≥100 cigarettes during their lifetimes and who currently smoke every day or some days.

In 2003, the median prevalence of current cigarette smoking among adults was 22.1% in the 50 states and DC (range: 12.0% [Utah]–30.8% [Kentucky]) (Table). Smoking prevalence was higher among men (median: 24.8%; range: 14.0%–33.8%) than women (median: 20.3%; range: 9.9%–28.1%) in the 50 states and DC. Smoking prevalence for both men and women was highest in Kentucky (men: 33.8%; women: 28.1%) and lowest in Utah (men: 14.0%; women: 9.9%). In areas other than the 50 states and DC, the median prevalence of current cigarette smoking among adults was 13.6% (range: 10.0% [USVI]–34.0% [Guam]).

Reported by: *J Bombard, MSPH, A Malarcher, PhD, M Schooley, MPH, A MacNeil, MPH, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.*

Editorial Note: Although the prevalence of current cigarette smoking among U.S. adults has declined, the rate of decline has not been rapid enough for the nation to achieve the 2010 national health objective of ≤12% of adults smoking cigarettes (objective 27-1) (*2,3*). The median prevalence of adult smoking decreased 1 percentage point from 2002 to 2003, and the national objective for 2010 was achieved in Utah and the USVI. The high prevalence of current cigarette smoking in most of the remaining states/areas underscores the need for increased efforts to reduce tobacco use.

The findings in this report are subject to at least three limitations. First, the BRFSS survey does not sample persons in households without telephones, a population that might be more likely to smoke (*4*). Second, data for cigarette smoking are based on self-reports and are not validated with biochemical tests. However, self-reported data on current smoking status have high validity (*4*). Third, the median response rate was 53.2% (range: 34.4%–80.5%); lower response rates indicate a potential for response bias. However, BRFSS estimates for cigarette smoking are comparable with current smoking estimates from other surveys with higher response rates (*5*).

Comprehensive tobacco control is effective in preventing and reducing tobacco use (*6*). CDC recommends the following evidence-based interventions as strategies within comprehensive tobacco-control programs: clean indoor air laws, telephone support quitlines, media campaigns, increased excise taxes on tobacco products, insurance coverage for cessation counseling and pharmaceuticals, and health-care system changes that support cessation (*7*). Substantial variation exists across states in their use of these strategies. For example, in 2002, two states offered Medicaid coverage for all recommended medication and counseling treatments for tobacco dependence, whereas 11 states covered no tobacco-dependence

2-108

TABLE. Prevalence of current cigarette smoking among adults*, by state/area and sex — Behavioral Risk Factor Surveillance System, 50 states, District of Columbia, Guam, Puerto Rico, and U.S. Virgin Islands, 2003

State/Area	Men		Women		Total	
	%	(95% CI†)	%	(95% CI)	%	(95% CI)
Alabama	28.5	(±3.1)	22.4	(±2.0)	**25.3**	**(±1.8)**
Alaska	30.3	(±3.6)	21.9	(±3.0)	**26.3**	**(±2.4)**
Arizona	23.8	(±3.9)	18.2	(±2.7)	**21.0**	**(±2.4)**
Arkansas	27.6	(±2.5)	22.3	(±1.8)	**24.8**	**(±1.5)**
California	20.5	(±2.3)	13.2	(±1.5)	**16.8**	**(±1.4)**
Colorado	19.6	(±2.2)	17.5	(±1.7)	**18.5**	**(±1.4)**
Connecticut	19.7	(±1.9)	17.9	(±1.6)	**18.7**	**(±1.2)**
Delaware	26.0	(±3.0)	18.2	(±2.0)	**21.9**	**(±1.8)**
District of Columbia	26.2	(±4.2)	19.0	(±2.9)	**22.3**	**(±2.5)**
Florida	26.0	(±3.1)	22.1	(±2.3)	**23.9**	**(±1.9)**
Georgia	25.8	(±2.3)	20.0	(±1.5)	**22.8**	**(±1.4)**
Hawaii	20.1	(±2.5)	14.4	(±1.7)	**17.3**	**(±1.5)**
Idaho	19.5	(±2.1)	18.5	(±1.7)	**19.0**	**(±1.3)**
Illinois	28.3	(±2.8)	20.5	(±1.9)	**24.3**	**(±1.7)**
Indiana	28.6	(±2.2)	23.8	(±1.6)	**26.1**	**(±1.3)**
Iowa	22.8	(±2.2)	20.7	(±1.9)	**21.7**	**(±1.5)**
Kansas	21.0	(±2.3)	19.7	(±1.7)	**20.4**	**(±1.4)**
Kentucky	33.8	(±2.7)	28.1	(±1.9)	**30.8**	**(±1.7)**
Louisiana	30.3	(±2.5)	23.2	(±1.7)	**26.6**	**(±1.5)**
Maine	23.1	(±3.1)	24.0	(±2.5)	**23.6**	**(±2.0)**
Maryland	23.0	(±2.6)	17.7	(±1.8)	**20.2**	**(±1.6)**
Massachusetts	20.0	(±1.8)	18.4	(±1.4)	**19.2**	**(±1.2)**
Michigan	30.2	(±3.0)	22.3	(±2.1)	**26.2**	**(±1.8)**
Minnesota	22.4	(±2.4)	19.9	(±1.9)	**21.1**	**(±1.5)**
Mississippi	31.1	(±2.7)	20.7	(±1.7)	**25.6**	**(±1.6)**
Missouri	31.2	(±3.1)	23.8	(±2.5)	**27.3**	**(±2.0)**
Montana	19.5	(±2.5)	20.3	(±2.2)	**19.9**	**(±1.7)**
Nebraska	23.6	(±2.2)	19.0	(±1.6)	**21.3**	**(±1.4)**
Nevada	29.0	(±3.5)	21.3	(±2.9)	**25.2**	**(±2.3)**
New Hampshire	22.4	(±2.2)	20.2	(±1.8)	**21.2**	**(±1.4)**
New Jersey	21.2	(±1.5)	17.9	(±1.1)	**19.5**	**(±0.9)**
New Mexico	23.6	(±2.2)	20.5	(±1.7)	**22.0**	**(±1.4)**
New York	24.8	(±2.2)	18.8	(±1.6)	**21.6**	**(±1.3)**
North Carolina	28.0	(±2.4)	21.9	(±1.7)	**24.8**	**(±1.5)**
North Dakota	22.0	(±2.5)	19.0	(±2.2)	**20.5**	**(±1.7)**
Ohio	26.9	(±2.8)	24.0	(±2.2)	**25.4**	**(±1.8)**
Oklahoma	27.8	(±2.0)	22.7	(±1.4)	**25.2**	**(±1.2)**
Oregon	23.1	(±2.4)	18.9	(±1.8)	**21.0**	**(±1.5)**
Pennsylvania	27.1	(±2.7)	24.1	(±2.1)	**25.5**	**(±1.7)**
Rhode Island	23.8	(±2.7)	21.1	(±2.0)	**22.4**	**(±1.6)**
South Carolina	28.5	(±2.3)	22.8	(±1.6)	**25.5**	**(±1.4)**
South Dakota	24.7	(±2.3)	20.7	(±1.8)	**22.7**	**(±1.4)**
Tennessee	27.3	(±3.3)	24.2	(±2.4)	**25.7**	**(±2.0)**
Texas	26.7	(±2.2)	17.6	(±1.4)	**22.1**	**(±1.3)**
Utah	14.0	(±2.2)	9.9	(±1.6)	**12.0**	**(±1.4)**
Vermont	19.8	(±2.3)	19.4	(±1.9)	**19.6**	**(±1.5)**
Virginia	26.4	(±2.5)	18.0	(±1.6)	**22.1**	**(±1.5)**
Washington	20.9	(±1.2)	18.2	(±0.9)	**19.5**	**(±0.7)**
West Virginia	27.6	(±2.8)	27.2	(±2.3)	**27.4**	**(±1.8)**
Wisconsin	24.0	(±2.6)	20.3	(±2.0)	**22.1**	**(±1.6)**
Wyoming	25.2	(±2.4)	24.1	(±2.0)	**24.6**	**(±1.6)**
Median	*24.8*		*20.3*		*22.1*	
Guam	42.0	(±5.9)	25.8	(±4.6)	**34.0**	**(±3.8)**
Puerto Rico	19.3	(±2.6)	8.5	(±1.3)	**13.6**	**(±1.5)**
U.S. Virgin Islands	14.2	(±3.2)	6.6	(±1.6)	**10.0**	**(±1.7)**
Median	*19.3*		*8.5*		*13.6*	

* Persons aged ≥18 years who reported having smoked ≥100 cigarettes during their lifetimes and who currently smoke every day or some days.
† Confidence interval.

treatments (*8*). In addition, the average cost of a single pack of cigarettes (which includes state-based excise taxes) ranged from $3.10 in Kentucky to $5.54 in New York in 2003 (*9*). The majority of states offer telephone support quitlines, and residents of all states soon will have access to a nationwide network of quitlines. Finally, only six states (California, Connecticut, Delaware, Maine, Massachusetts, and New York) have comprehensive statewide bans in effect on smoking in indoor workplaces and public places.

The more funds that states spend on comprehensive tobacco-control programs, the greater the reduction in smoking (*6*). However, the amount of money that states spend for tobacco control decreased 28% during the preceding 2 years to $541.1 million, which is less than 3% of the estimated $19 billion states expected to receive from tobacco excise taxes and tobacco settlement money in 2003 (*10*). For fiscal year 2004 (i.e., July 1, 2003–June 31, 2004), only four states (Arkansas, Delaware, Maine, and Mississippi) were investing at least the minimum per capita amount that CDC recommends for tobacco-control programs (*10*). Efforts and resources must be expanded if more states are to reduce smoking prevalence to ≤12% by 2010.

References

1. CDC. Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States 1995–1999. MMWR 2002;51:300–3.
2. CDC. Cigarette smoking among adults—United States, 2002. MMWR 2004;53:427–31.
3. US Department of Health and Human Services. Healthy people 2010 (conference ed, in 2 vols). Washington, DC: US Department of Health and Human Services; 2000. Available at http://www.health.gov/healthypeople.
4. Nelson DE, Holtzman D, Bolen J, Stanwyck CA, Mack KA. Reliability and validity of measures from the Behavioral Risk Factor Surveillance System (BRFSS). Social Prev Med 2001;46:S3–S42.
5. US Department of Health and Human Services. Women and smoking: a report of the Surgeon General. Rockville, MD: US Department of Health and Human Services, Public Health Service, Office of the Surgeon General; 2001:24–25.
6. Farrelly MC, Pechacek TP, Chaloupka FJ. The impact of tobacco control program expenditures on aggregate cigarette sales: 1981–2000. Health Econ 2003;22:843–59.
7. Task Force on Community Preventive Services. Guide to community preventive services: tobacco use prevention and control. Am J Prev Med 2001;20(2 Suppl 1):1–87.
8. CDC. State Medicaid coverage for tobacco-dependence treatments—United States, 1994–2002. MMWR 2004;53:54–7.
9. Orzechowski W, Walker RC. The tax burden on tobacco, volume 38. Arlington, VA: Orzechowski and Walker; 2003.
10. Campaign for Tobacco-Free Kids, American Heart Association, American Cancer Society, American Lung Association. A broken promise to our children: the 1998 state tobacco settlement five years later. Washington, DC: Campaign for Tobacco-Free Kids; 2003. Available at http://www.tobaccofreekids.org/reports/settlements/2004/fullreport.pdf.

2-109

Indoor Air Quality in Hospitality Venues Before and After Implementation of a Clean Indoor Air Law — Western New York, 2003

Secondhand smoke (SHS) contains more than 50 carcinogens (*1*). SHS exposure is responsible for an estimated 3,000 lung cancer deaths and more than 35,000 coronary heart disease deaths among never smokers in the United States each year (*2*), and for lower respiratory infections, asthma, sudden infant death syndrome, and chronic ear infections among children (*3*). Even short-term exposures to SHS, such as those that might be experienced by a patron in a restaurant or bar that allows smoking, can increase the risk of experiencing an acute cardiovascular event (*4*). Although population-based data indicate declining SHS exposure in the United States over time (*5*), SHS exposure remains a common but preventable public health hazard. Policies requiring smoke-free environments are the most effective method of reducing SHS exposure (*6*). Effective July 24, 2003, New York implemented a comprehensive state law requiring almost all indoor workplaces and public places (e.g., restaurants, bars, and other hospitality venues) to be smoke-free. This report describes an assessment of changes in indoor air quality that occurred in 20 hospitality venues in western New York where smoking or indirect SHS exposure from an adjoining room was observed at baseline. The findings indicate that, on average, levels of respirable suspended particles (RSPs), an accepted marker for SHS levels, decreased 84% in these venues after the law took effect. Comprehensive clean indoor air policies can rapidly and effectively reduce SHS exposure in hospitality venues.

The specific class of RSP monitored was $PM_{2.5}$ (i.e., particulate matter that is <2.5 microns in diameter). Particles of this size are released in substantial amounts from burning cigarettes and are easily inhaled deep into the lungs. Baseline measurements were made during July 11–23 in a purposeful sample of 22 hospitality venues in three counties in western New York. Sites were selected to provide a range of venue types, sizes, and locations. The sample consisted of seven bars, six bar/restaurants, five restaurants, two bowling alleys, a pool hall, and a bingo hall. The venues were located in popular downtown entertainment districts and suburban areas and ranged from small neighborhood bars to large bar/restaurant chains.

At baseline, smoking was occurring in 14 bars and restaurants and four large recreation venues. Two bar/restaurant combinations allowed smoking in the bar section but not in the adjoining restaurant section. In these two venues, air quality was monitored separately in the restaurant and bar areas. In two restaurants, no smoking was occurring at baseline because restaurants were already required to be smoke-free by local clean indoor air ordinances. Follow-up measurements of air quality were made in all 22 venues during September 9–November 1. The follow-up measurements were taken on the same day of the week and at approximately the same time of day as the measurements taken before the smoke-free law was implemented.

The median time spent in each venue for all 44 baseline and follow-up observations combined was 38 minutes (range: 22–140 minutes). Measurements were taken at 1-second intervals. The number of persons and the number of burning cigarettes in each venue were recorded every 10 minutes during sampling, and the average number of persons and the average number of burning cigarettes in each venue were calculated. The volume of each venue also was measured*, and the cigarette density was calculated by dividing the average number of burning cigarettes by the room volume.

An air monitor† was used to sample and record RSP levels. The monitor was placed in a central location on a table or bar near the height at which a person breathes air. The monitor recorded continuous measurements, which were averaged over time. The first and last minute of logged data were removed, and the remaining data points were averaged to provide an average concentration of $PM_{2.5}$ within the venue. The percentage change in $PM_{2.5}$ levels was then determined by comparing average $PM_{2.5}$ levels in each venue before the law went into effect with levels after the law was implemented. The Wilcoxon signed-rank test was used to assess changes between pre-law and post-law $PM_{2.5}$ levels, stratified by type of venue.

The average $PM_{2.5}$ concentration was substantially lower after the law went into effect in every venue where smoking or indirect SHS exposure had been observed at baseline, with a grand mean reduction in $PM_{2.5}$ concentration of 84% (324 $\mu g/m^3$ to 25 $\mu g/m^3$; $p<0.001$) (Table). When stratified by the type of venue sampled, the average $PM_{2.5}$ concentration decreased 90% (412 $\mu g/m^3$ to 27 $\mu g/m^3$; $p<0.001$) in the 14 bars and restaurants in which smoking was occurring at baseline (including bar/restaurant J, which was the only venue where smoking was observed during the post-law sampling). The restaurant portions of the two bar/restaurants that allowed smoking in the bar section but not in the restaurant section experienced an average 58% decrease in $PM_{2.5}$

*The Zircon DM S50 Sonic Measure® (Zircon Corporation, Campbell, California) was used to perform this measurement.

†The air monitor used was a TSI SidePak AM510 Personal Aerosol Monitor® (TSI, Inc., St. Paul, Minnesota). The SidePak uses a built-in sampling pump to draw air through the device, which then measures the real-time concentration in milligrams per cubic meter of $PM_{2.5}$. The SidePak was calibrated against a SHS-calibrated nephelometer, which had been previously calibrated and used in similar studies. The SidePak was zero-calibrated before each use according to the manufacturer's specifications.

2-110

TABLE. Change in concentrations of respirable suspended particles after the implementation of a clean indoor air law, by venue — western New York, 2003

Venue	Size (m^3)	Cigarette density* Before July 24, 2003	Cigarette density* After July 24, 2003	Average $PM_{2.5}$† level ($\mu g/m^3$) Before July 24, 2003	Average $PM_{2.5}$† level ($\mu g/m^3$) After July 24, 2003	% reduction in $PM_{2.5}$
Bars and restaurants in which smoking was occurring						
Bar A	349	0.86	0	353	56	84.1
Bar B	453	1.32	0	375	20	94.7
Bar C	225	1.34	0	1,375	52	96.2
Bar D	319	0.94	0	386	35	90.9
Bar E	245	0.86	0	104	28	73.1
Bar F	339	3.25	0	569	26	95.4
Bar G	335	1.79	0	681	13	98.1
Bar/Restaurant H	299	1.34	0	425	10	97.6
Bar/Restaurant I	321	1.56	0	198	21	89.3
Bar/Restaurant J	551	1.45	0.09	597	83	86.1
Bar/Restaurant K	479	0.42	0	62	10	83.9
Bar/Restaurant L	318	0.52	0	352	6	98.0
Bar/Restaurant M	786	0.25	0	54	11	79.6
Restaurant N	95	3.15	0	233	6	97.4
Mean§	*365*	*1.36*	*0.01*	*412*	*27*	*90.3*
Restaurant portions of bar/restaurant combinations with indirect secondhand smoke (SHS) exposure¶						
Restaurant O	438	0	0	273	34	87.5
Restaurant P	381	0	0	38	27	28.9
Mean§	*410*	*0*	*0*	*156*	*31*	*58.2*
Other venues in which smoking was occurring						
Bowling alley Q	5,930	0.03	0	35	13	62.9
Bowling alley R	2,916	0.17	0	87	26	70.1
Pool hall S	1,570	0.26	0	176	6	96.6
Bingo hall T	3,704	0.40	0	105	26	75.2
Mean§	*3,530*	*0.22*	*0*	*101*	*18*	*76.2*
Grand mean**	**1,003**	**1.01**	**0.01**	**324**	**25**	**84.3**
Restaurants in which no smoking and no indirect SHS exposure was occurring						
Restaurant U	446	0	0	6	6	0.0
Restaurant V	337	0	0	41	40	2.4
Mean§	*392*	*0*	*0*	*24*	*23*	*1.2*

* Average number of burning cigarettes per 100 m^3.
† Particulate matter <2.5 microns in diameter.
§ Results represent the average of the values for the venues listed in each category.
¶ Restaurant O is attached to Bar A with little physical separation between the two spaces; Restaurant P is attached to Bar B but with substantial physical separation between the two spaces.
** For all venues where any smoking or indirect SHS exposure was occurring at baseline (i.e., venues A–T).

concentrations (156 $\mu g/m^3$ to 31 $\mu g/m^3$; $p<0.001$) after the law was implemented, even though they had only indirect SHS exposure at baseline. In the four other large recreation venues, which had larger volumes and lower smoker densities, the average $PM_{2.5}$ concentration decreased 76% (101 $\mu g/m^3$ to 18 $\mu g/m^3$). In contrast, the $PM_{2.5}$ concentration remained low and virtually constant in the two restaurants that were already smoke-free at baseline; these venues were not included in the grand mean calculation.

Reported by: *MJ Travers, KM Cummings, PhD, A Hyland, PhD, Dept of Health Behavior, Roswell Park Cancer Institute, Buffalo, New York. J Repace, MSc, Repace Associates, Bowie, Maryland. S Babb, MPH, T Pechacek, PhD, R Caraballo, PhD, Office on Smoking and Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.*

Editorial Note: The findings in this report indicate that a statewide law to eliminate smoking in enclosed workplaces and public places substantially reduced RSP levels in western New York hospitality venues. RSP levels were reduced in

2-111

every venue that permitted smoking before the law was implemented, including venues in which only SHS from an adjacent room was observed at baseline.

These findings are consistent with those of previous studies. In Delaware, a similar decline in RSP levels was observed in eight hospitality venues after smoking was prohibited there by state law (*7*). Previous studies also have assessed the health effects of smoke-free laws. One study indicated that respiratory health improved rapidly among a sample of bartenders after a state smoke-free workplace law was implemented in California (*8*), and another study reported a 40% reduction in acute myocardial infarction admissions to a regional hospital during the 6 months that a local smoke-free ordinance was in effect in Helena, Montana (*9*). The results of these studies (both those assessing changes in indoor air quality and those assessing changes in health) suggest that improvements can occur within months of policy implementation.

The findings in this report are subject to at least two limitations. First, the venues sampled were not necessarily representative of venues in western New York. However, they did provide a range of venue types, sizes, and locations. Second, SHS is not the only source of indoor particulate matter. However, although ambient particle concentrations and cooking are additional sources of indoor particle levels, secondhand smoke is the largest contributor to indoor RSP pollution (*3*).

Eliminating nonsmoker exposure to SHS is one of the four goals of comprehensive state tobacco-control programs, as set forth in CDC's *Best Practices for Comprehensive Tobacco Control Programs* (*10*). The results of the study described in this report indicate that a comprehensive statewide ban on smoking in indoor workplaces and public places can substantially reduce SHS exposure in these settings. Six states (California, Connecticut, Delaware, Maine, Massachusetts, and New York) currently meet the national health objective for 2010 calling for implementation of such laws. These six states account for approximately 23% of the U.S. population. Rhode Island also has adopted such a law, but the law does not take full effect until 2006. To further reduce the nearly 40,000 deaths among never smokers caused by SHS exposure each year, similar comprehensive laws are needed in the other 43 states and the District of Columbia.

References

1. National Toxicology Program. 9th report on carcinogens. Research Triangle Park, NC: US Department of Health and Human Services, National Institute of Environmental Health Sciences; 2000.
2. CDC. Annual smoking-attributable mortality, years of potential life lost, and economic costs—United States, 1995–1999. MMWR 2002;51:300–3.
3. National Cancer Institute. Health effects of exposure to environmental tobacco smoke: the report of the California Environmental Protection Agency. Bethesda, MD: US Department of Health and Human Services, National Institutes of Health, National Cancer Institute; 1999.
4. Pechacek TF, Babb S. Commentary: how acute and reversible are the cardiovascular risks of secondhand smoke? BMJ 2004;328:980–3.
5. CDC. Second national report on human exposure to environmental chemicals. Atlanta, GA: US Department of Health and Human Services, CDC; 2003.
6. CDC. Reducing tobacco use: a report of the Surgeon General. Atlanta, GA: US Department of Health and Human Services, CDC; 2000.
7. Repace J. Respirable particles and carcinogens in the air of Delaware hospitality venues before and after a smoking ban. J Occup Environ Med 2004;46:887–905.
8. Eisner MD, Smith AK, Blanc PD. Bartenders' respiratory health after establishment of smoke-free bars and taverns. JAMA 1998;280:1909–14.
9. Sargent RP, Shepard RM, Glantz SA. Reduced incidence of admissions for myocardial infarction associated with public smoking ban: before and after study. BMJ 2004;328:977–80.
10. CDC. Best practices for comprehensive tobacco control programs—August 1999. Atlanta, GA: US Department of Health and Human Services, CDC; 1999. Available at http://www.cdc.gov/tobacco/bestprac.htm.

Vaccination Coverage Among Children Entering School — United States, 2003–04 School Year

One of the national health objectives for 2010 is to sustain ≥95% vaccination coverage among children in kindergarten through first grade (objective 14-23) (*1*). To determine the percentage of vaccination coverage among children entering kindergarten, data on vaccination coverage were analyzed from reports submitted to the National Immunization Program by states, the District of Columbia (DC)*, and eight current or former U.S. territories for the 2003–04 school year. This report summarizes the results of that analysis, which determined that coverage for all vaccines except hepatitis B (HepB) and varicella was reported at >90% in 45 areas. However, the vaccines required in each reporting area and the methods for surveying kindergarten-aged children vary substantially; in seven states, <20% of eligible children were surveyed. The wide variations in survey populations underscore the need for CDC to continue working with immunization programs in states, DC, and current or former territories to improve survey methods and automate reporting of data.

For the 2003–04 school year, all states except one submitted reports of vaccination coverage levels for children entering kindergarten. Fifty reports included coverage for poliovirus vaccine, diphtheria and tetanus toxoids and pertussis vaccine, diphtheria and tetanus toxoids and acellular pertussis vaccine,

* For this report, DC is included in state totals.

2-112

Title: Ventilation and Air Filtration: The Science

Publication Date: December 2004

Agency or Author: Americans for Nonsmokers' Rights

Purpose: This fact sheet provides summary information on various studies conducted on the impact of ventilation systems and individual exposure to Environmental Tobacco Smoke (ETS). Also included in this document are the sources of each study mentioned. The document concludes that following implementation of ventilation systems measurable levels of particulate matter are still found in all areas.

ANR AMERICANS FOR NONSMOKERS' RIGHTS

Defending your right to breathe smokefree air since 1976

VENTILATION AND AIR FILTRATION: THE SCIENCE

December 2004

- A study published in the September 2004 edition of the *Journal of Occupational and Environmental Medicine* compared the indoor air quality of a casino, six bars, and a pool hall in Wilmington, Delaware, before and after the implementation of a smokefree law. The study found that the ventilation technology installed in these establishments did not protect the workers and the public, as secondhand smoke contributed 85-95% of the carcinogen PPAH, and 90-95% of the respirable particulate air pollution into the air. These contamination levels greatly exceed those encountered on major truck highways and polluted city streets.[1]

- In less than two hours after New York's smokefree law went into effect and smoking stopped, the level of respirable particulate matter (PM) dropped to 15 percent of the level on a smoking night in restaurants and bars. Three months after the law became effective, the level of PM dropped by 90 percent in these venues. Prior to the smokefree law's implementation, New York hospitality employees working an eight hour shift, 250 days a year, were exposed to particulate matter levels seven times greater than the maximum level deemed as acceptable by the U.S. Environmental Protection Agency. In addition, PM dropped an average of 77 percent after the law went into effect in bowling alleys, pool halls, and bingo halls.[2]

- The 2002 Environmental Health Information Service's *10th Report on Carcinogens* classifies SHS as a Group A (Human) Carcinogen--a substance known to cause cancer in humans. There is no safe level of exposure for Group A toxins.[3]

- The 1986 Surgeon General's report on involuntary smoking concluded that, "the simple separation of smokers and nonsmokers within the same airspace may reduce, but does not eliminate, the exposure of nonsmokers to ETS [environmental tobacco smoke]."[4]

- Using current indoor air quality standards, ventilation rates would have to be increased more than a thousand-fold to reduce cancer risk associated with ETS to a level considered acceptable to federal regulatory agencies. Such a ventilation rate is impractical since it would result in a virtual windstorm indoors.[5,6]

- "Separation of smoking areas does not protect the workers and occupants within the smoking area. When separation is properly done (and this is not common), it can reduce the exposure of occupants in the nonsmoking areas, but there is no quantitative assurance that the remaining exposure meets any current health standard or goal."[7]

- "[T]o be at all effective in reducing the concentration of smoke in a space, any air cleaner must process many room air volumes per hour.... [E]ven large, expensive air cleaners with efficiencies for captured particles are capable of reducing, but not eliminating the environmental tobacco smoke tar particles in room air, and are not at all effective for gases, which contain most of the irritants.... [E]ven expensive particulate air cleaners cannot remove enough tar particles in room air to eliminate the cancer risk from environmental tobacco smoke.

2530 San Pablo Avenue, Suite J • Berkeley, California 94702 • (510) 841-3032 / FAX (510) 841-3071
www.no-smoke.org • anr@no-smoke.org

In general, filtration of indoor air to remove environmental tobacco smoke contaminants is futile – like trying to filter a lake to control water pollution."[8]

- Ventilated smoking rooms leak smoke into the rest of the building, harming everyone in the building. A recent research study conducted by and published for the American Society of Heating, Refrigerating and Air-Conditioning Engineers (ASHRAE) "showed that up to 10 percent of smoking room air enters non-smoking areas just by opening and closing of a swing type entry door.... With supply and exhaust air flow that are practical for small smoking rooms, leaving the smoking room door open results in a large flow of air to adjoining non-smoking areas. To prevent this, smoking room doors should be equipped with an automatic closure mechanism."[9]

- "Changes in ventilation rates during smoking do not have a significant influence on the air concentrations of tobacco components. This means, in effect, that efforts to reduce indoor air pollution through higher ventilation rates in buildings and homes would hardly lead to a measurable improvement of indoor air quality."[10]

- "[I]t is noted that the specific amount of additional ventilation cannot be determined until cognizant health authorities have determined an acceptable level of environmental tobacco smoke (ETS).... An appendix ... provides a method to allow designers to determine additional ventilation over what would be provided in a similar non-smoking area. However, this additional ventilation is for the purpose of odor control only."[11]

- "In managing workplace ETS risks, smoking policies such as separating smokers from nonsmokers in the same space or on the same ventilation system expose nonsmokers to unacceptable risk."[12]

REFERENCES

[1] Repace, J. "Respirable Particles and Carcinogens in the Air of Delaware Hospitality Venues Before and After a Smoking Ban." *Journal of Occupational and Educational Medicine.* September 10, 2004.
[2] RTI International, "First Annual Independent Evaluation of New York's Tobacco Control Program," *New York State Department of Health*, November 2004. Accessed on November 29, 2004. Download at http://www.health.state.ny.us/nysdoh/tobacco/reports/docs/nytcp_eval_report_final_11-19-04.pdf.
[3] Report on Carcinogens, Tenth Edition; U.S. Department of Health and Human Services, Public Health Service, National Toxicology Program, December 2002.
[4] U.S. Surgeon General. *The Health Consequences of Involuntary Smoking.* Washington, DC: U.S. Department of Health and Human Services, 1986.
[5] Repace, J., "Smoking in the workplace: ventilation. In: Smoking Policy: Questions and Answers, no. 5.," *Seattle: Smoking Policy Institute,* [n.d.].
[6] Repace, J., "An air quality survey of respirable particles and particulate carcinogens in Delaware hospitality venues before and after a smoking ban," *Bowie, MD: Repace Associates, Inc.,* February 7, 2003.
[7] Schoen, Lawrence J. Principal Engineer of Schoen Engineering, Inc. [Letter to M. J. Nicchio re: ventilation.] October 7, 2003.
[8] Repace, J.,"Smoking in the workplace: ventilation. In: Smoking Policy: Questions and Answers, no. 5.," *Seattle: Smoking Policy Institute,* [n.d.].
[9] "ASHRAE Journal: Shutting the Door on ETS Leakage," *ashrae.org*, July 2003.
[10] Joint Research Centre, Indoor air pollution: new EU research reveals higher risks than previously thought. Brussels: European Commission. September 22, 2003.
[11] "ANSI Upholds Approval of ASHRAE Smoking Addendum," *csemag.com*, September 29, 2003.
[12] Repace, J.L., "Risk management of passive smoking at work and at home," *St. Louis University Public Law Review 8(2); 763-785*, 1994.

Title: Designated "no smoking" areas provide from partial to no protection from Environmental Tobacco Smoke

Publication Date: 2004

Agency or Author: T Cains, S Cannaat, R Poulos, M J Ferson, B W Stewart

Purpose: Primary literature study on the impacts and exposure to Environmental Tobacco Smoke (ETS) for persons in no smoking sections of restaurants. Provides information on methodology, objectives, as well as the final results of the study. The study concludes that individuals in non-smoking sections are still exposed to ETS.

RESEARCH PAPER

Designated "no smoking" areas provide from partial to no protection from environmental tobacco smoke

T Cains, S Cannata, R Poulos, M J Ferson, B W Stewart

Tobacco Control 2004;**13**:17–22. doi: 10.1136/tc.2003.005488

See end of article for authors' affiliations

Correspondence to: Professor Bernard W Stewart, South East Sydney Public Health Unit, Locked Bag 88, Randwick NSW 2031 Australia; stewartb@sesahs.nsw.gov.au

Received 29 July 2003
Accepted 21 October 2003

Objective: To determine the efficacy of designated "no smoking" areas in the hospitality industry as a means of providing protection from environmental tobacco smoke (ETS), and whether certain design features assist in achieving this end.

Methodology: In the greater metropolitan region of Sydney, a representative group of 17 social and gaming clubs, licensed to serve alcoholic beverages and in which, apart from designated areas, smoking occurs, agreed to participate. In each establishment, simultaneous single measurements of atmospheric nicotine, particulate matter (10 μm; PM10) and carbon dioxide (CO_2) levels were measured in a general use area and in a designated "no smoking" area during times of normal operation, together with the levels in outdoor air (PM10 and CO_2 only). Analyses were made of these data to assess the extent to which persons using the "no smoking" areas were protected from exposure to ETS.

Results: By comparison with levels in general use areas, nicotine and particulate matter levels were significantly less in the "no smoking" areas, but were still readily detectable at higher than ambient levels. For nicotine, mean (SD) levels were 100.5 (45.3) $\mu g/m^3$ in the areas where smoking occurred and 41.3 (16.1) $\mu g/m^3$ in the "no smoking" areas. Corresponding PM10 levels were 460 (196) $\mu g/m^3$ and 210 (210) $\mu g/m^3$, while outdoor levels were 61 (23) $\mu g/m^3$. The reduction in pollutants achieved through a separate room being designated "no smoking" was only marginally better than the reduction achieved when a "no smoking" area was contiguous with a smoking area. CO_2 levels were relatively uninformative.

Conclusion: Provision of designated "no smoking" areas in licensed (gaming) clubs in New South Wales, Australia, provides, at best, partial protection from ETS—typically about a 50% reduction in exposure. The protection afforded is less than users might reasonably have understood and is not comparable with protection afforded by prohibiting smoking on the premises.

The source, composition, and hazardous nature of environmental tobacco smoke (ETS) are well recognised.[1–4] In respect of cancer causation, the International Agency for Research on Cancer recently determined that ETS is appropriately categorised as a group 1 carcinogen—that is, ETS is established as causing cancer in humans. It is an accepted principle of public health that humans should not knowingly be exposed to recognised carcinogens in circumstances where such exposure is effectively preventable. Eliminating, or minimising, exposure is generally achieved by changes in practice and behaviour and, where practicable, encouraging or mandating the requisite change by legislation or similar procedure.[5] In respect of exposure to ETS in the workplace, change has been motivated by the award of damages to employees against employers in relation to disease attributable to ETS. Specifically, in Australia, damages were so awarded in respect of laryngeal cancer suffered by a barmaid.[6] For the hospitality industry in particular, where there is a perception that a prohibition on smoking might reduce patronage, an attractive option has been the designation of "no smoking" areas. The practice is widespread, and common experience indicates great variation in the manner in which such areas are configured. At one extreme, specific rooms may be identified as "no smoking". At the other extreme, space defined only with reference to arbitrary and often approximate boundaries, in an otherwise continuous expanse, may be designated a "no smoking" area.

The extent to which designated "no smoking" areas provide protection from tobacco may be anticipated to vary according to their configuration.[7] That said, modelling studies indicate that such areas will not provide complete protection from ETS.[1] In respect of monitoring levels of ETS, an inference of reduced exposure may be made from some studies. A brief Japanese report of a "practical and low cost" non-smoking area established in an office using screens resulted in improved air quality (by survey) and decreased suspended particulates.[8] A study based on 25 diverse worksites (principally manufacturing facilities) recorded nicotine concentrations which fell from a median of 8.6 $\mu g/m^3$ in open office where smoking was allowed, to 1.3 $\mu g/m^3$ at sites that restricted smoking to 0.3 $\mu g/m^3$ in worksites where smoking was banned.[9] Likewise, inference may be made from two studies in restaurants that some reduction in exposure is achieved as a consequence of being located in the "no smoking" section.[10 11]

While the studies summarised above provide some inference that designated "no smoking" areas may provide protection from ETS, the relatively scant data do not allow a clear understanding of the extent of such protection, and any design limitations on achieving it. There are some indications that in bars, nightclubs, and gaming venues, levels of ETS are among the highest recorded.[12] We report here a study undertaken in "licensed clubs" in Sydney, New South Wales, Australia: premises licensed to sell alcoholic beverages for consumption on the premises and which provide gaming machines as a principal recreation for patrons. In the premises studied, the "no smoking" areas involved either designation of specific rooms as "no smoking" or areas

Abbreviations: ETS, environmental tobacco smoke; OS, one space; SR, separate room

2-117

within an otherwise single space were designated as "no smoking". Levels of nicotine, particulates, and carbon dioxide (CO_2) were determined in such areas and contrasted with those levels in other indoor spaces where smoking occurred and also with the outside air. Though the present investigation was limited in some respects, the data now available indicate that designated "no smoking" areas, regardless of their configuration, provide only partial protection from tobacco smoke at best. At worst, the data suggest that no protection whatsoever is afforded by the measures in question.

METHODS

A list of potential participants in the study, covering "clubs" catering to diverse interests (including football, lawn bowling, golf, veterans, social and community improvement clubs) was generated from the telephone directory. Club managers were then contacted and asked to participate in an interview in which the principles and mechanics of the project were delineated. None of the clubs so approached subsequently declined an invitation to participate in the study, and the process of contacting clubs ceased when the number of clubs agreeing to participate reached 17. All managers expressed the wish that their clubs' identity and participation in the study not be made public.

Following agreement to participate, a questionnaire was sent to each club. Details were requested concerning the configuration of designated "no smoking" areas in relation to similar spaces in which smoking occurred. The designated "no smoking" area of each club was categorised according to whether such facilities involved provision of a room in which smoking was not permitted, or the identification of a subsection of a room as a "no smoking" area. In general, smoking occurred in all interior spaces of each club apart from the designated "no smoking" areas. In respect of those clubs in which a separate room was designated "no smoking", sufficient data were obtained to allow the ventilation system in such a room to be characterised as independent of that responsible for ventilating other space, or a common ventilation system was involved. Of those clubs having separate rooms, only three had separate ventilation systems. Preliminary evaluation of the data showed no clear difference between results according to the type of ventilation system. Accordingly, data for the "separate rooms" configuration were treated as a single group irrespective of the type of ventilation system.

For each facility, in the designated "no smoking" area and in an area where there was no such restriction, determinations of atmospheric levels of nicotine, particulate matter (10 μm) (PM10), and CO_2 were made; PM10 and CO_2 levels were also measured outdoors. Measurements were undertaken at a time of maximal occupancy as advised by management of each facility. Typically, this was a Friday evening. In all instances, analytical equipment was located centrally and remote from ventilation related fixtures in the ceiling. In the "no smoking" areas, equipment was positioned equi-distant from doorways. Measurements in a space where smoking occurred were undertaken in a high traffic area, namely in the room in which gaming machines were located. Outdoor determinations at each facility were undertaken in an area adjacent to the club building, but remote from human or vehicular traffic and from ventilation fixtures.

Nicotine levels were measured using a passive sampler (CSIRO Division of Atmospheric Research, Aspendale, Victoria, Australia) based on the principle of molecular diffusion of a gas onto a filter with a sorbent species, integrated over the time of exposure.[13] The samplers require an optimal collection period of six hours. Two samplers were employed at each facility: one located in the smoking and the other in the "no smoking" area of the club as previously described, and each positioned at least 1.5 m above floor level.

PM 10 levels were determined using a DUSTRAK Aerosol Monitor (Model 8520, TSI, Minneapolis, Minnesota, USA) with a cut-point for particles less than 10 μm programmed to log every 30 seconds. Ten minute samples were taken in each of the smoking, "no smoking", and outdoor areas.

Carbon dioxide levels were measured simultaneously using a TSI Q–Trak Indoor Air Monitor (Model 8551), programmed to log every 30 seconds. Samples were taken in the same manner as indicated for the PM10 determinations.

All data collected from the TSI Dustrak and the TSI Q–Trak were downloaded each day into the Trak Pro software program. The nicotine samplers were analysed blind by the CSIRO Atmospheric Research Branch.

Data were expressed as mean (SD). Missing data on nicotine levels occurred for the smoking area of one club, and the non-smoking area of another club. These clubs have been excluded from comparative analysis of nicotine levels. Differences between determinations for particular room configurations were tested using the independent samples *t* test; differences between smoking and "no smoking" areas and between "no smoking" and outside areas were tested using the paired samples *t* test.

RESULTS

Information concerning each participating club is summarised in table 1. The estimated number of patrons in the areas where smoking occurred and which were designated "no smoking" were made on the basis of observations during the sampling period. No person was observed to be smoking in any of the designated "no smoking" areas during the observation period.

Nicotine

Pronounced variation in levels of nicotine recorded was evident in both the smoking and designated "no smoking" areas of the respective clubs. In all clubs, the concentration of nicotine in the "no smoking" area was less than in the corresponding smoking area (fig 1).

The mean (SD) concentration of nicotine in the designated "no smoking" areas was 41.4 (16.1) $\mu g/m^3$ and the mean level in the areas where smoking occurred was 100.5 (45.3) $\mu g/m^3$ ($n = 16$).

In clubs where the "no smoking" area was a separate room (such facilities being designated "SR"), the mean nicotine level in the "no smoking" area was 35.8 (10.7) $\mu g/m^3$, compared to a level of 83.2 (24.2) $\mu g/m^3$ in the smoking areas, the difference being significant ($p < 0.0005$). In those clubs where the designated "no smoking" area was a subsection of a room where smoking otherwise occurred (one space facilities, designated "OS"), the mean level of nicotine in the "no smoking" area was 54.9 (19.4) $\mu g/m^3$ compared with 143.1 (54.3) $\mu g/m^3$ in the area where smoking took place ($p < 0.05$). Thus the atmospheric concentration of nicotine in both the smoking and non-smoking areas of facilities in the OS category were slightly, but not significantly, greater than levels in the corresponding areas of the SR facilities.

To evaluate the protection from tobacco smoke that could be achieved by moving from a smoking area to a "no smoking" area, the per cent reduction in nicotine level was calculated as follows: % reduction = [nicotine level (smoking) − nicotine level (no smoking)]/nicotine level (smoking).

Taking results for all facilities into account, an individual could expect, on average, to achieve a 53% (median 63%,

2-118

Table 1 Particulars concerning individual "clubs", and their respective premises

Club number	Club type	Smoking room or area (m^2)	"No-smoking" room or area (m^2)	Club membership	Estimated number of patrons during sampling in smoking room or area	Estimated number of patrons during sampling in "no smoking" room or area
1	Social	150	60	28000	120	15
2	Football	800	250	46100	80	80
3	RSL	300	100	6000	50	10
4	Social	400	100	28000	150	60
5	Social	100	200	35000	35	30
6	Social	250	60	6300	80	10
7	Golf	120	300	4800	25	65
8	Football	350	180	33000	80	60
9	Football	450	200	48500	100	10
10	Veterans	350	200	2000	60	40
11	Veterans	250	300	7900	70	50
12	Social	350	200	9200	90	15
13	Veterans	250	120	2000	40	10
14	Football	200	100	10000	120	60
15	Veterans	450	200	8200	300	40
16	Veterans	450	130	8000	120	80
17	Bowling	90	100	4000	15	10

range 12–86%) reduction in nicotine level by relocating from the smoking to the "no smoking" area (table 2). The mean per cent reduction of 53% was similar for SR facilities (median 63%, range 15%–75%) as for OS facilities where the mean reduction was 55% (median 60%, range 12%–86%).

Particulate matter (PM10)

In all clubs but two (clubs 15 and 17), the PM10 levels in designated "no smoking" areas were less than those which prevailed in smoking areas. However, except for one club (club 1), PM10 levels recorded inside were invariably greater than the "outdoors" determination (fig 2).

The mean PM10 level in outdoors—that is, in an open area remote from vehicle or human traffic or any ventilation plant—was 61 (23) $\mu g/m^3$. The mean indoor levels were higher and subject to wide variation as are readily evident from the mean of 210 (210) $\mu g/m^3$ for "no smoking" areas and 460 (197) $\mu g/m^3$ for smoking areas. The difference in PM10 levels between smoking and "no smoking" areas was significant ($p < 0.01$) as was the difference between PM10 levels in "no smoking" and outdoors ($p < 0.05$).

A distinction was evident between PM10 levels in the "no smoking" areas of SR compared to OS facilities. In SR premises, the mean PM10 level was 129 (76) $\mu g/m^3$ in "no smoking" compared to 421 (191) $\mu g/m^3$ in smoking areas ($p < 0.0005$). By contrast, in OS premises, the mean PM10 level of 404 (307) $\mu g/m^3$ for "no smoking" areas was not significantly different from the level of 555 (197) $\mu g/m^3$ in smoking areas.

With the exception of one facility, PM10 levels in designated "no smoking" areas were greater than PM10 levels outdoors. The difference was significant in the case of SR facilities ($p < 0.05$) and approached significance in the case of OR clubs ($p = 0.068$).

Using the same formula as specified for per cent reduction in nicotine levels (see above), in respect of all determinations taken together, an individual could expect, on average, to achieve a 52% reduction (median 59%, range –40% to 87%) in PM10 levels by relocating from the smoking to the "no smoking" area of the clubs. The mean reduction was 66% (median 67%, range 37–87%) for SR facilities but only 17% (median 4%, range –40% to 82%) for OS facilities. Moving outside achieved a mean 85% reduction (median 85%, range 73–95%) in PM10 levels compared to the PM10 levels of smoking areas of clubs.

Carbon dioxide

Differences were modest between CO_2 determinations variously made within smoking and "no smoking" areas, and at outdoor locations (fig 3).

The mean (SD) CO_2 levels across all clubs were 600 (94) parts per million (ppm) outdoors, 872 (159) ppm in the "no smoking" areas and 849 (135) ppm in the smoking areas. There was no significant difference between levels in the smoking and "no smoking" areas. CO_2 levels indoors were significantly higher than outdoor levels ($p < 0.0005$).

DISCUSSION

As indicated earlier, the present study was undertaken as an initial approach to the evaluation of protection from ETS afforded by designated "no smoking" areas, particularly in the hospitality industry. While certain conclusions may be

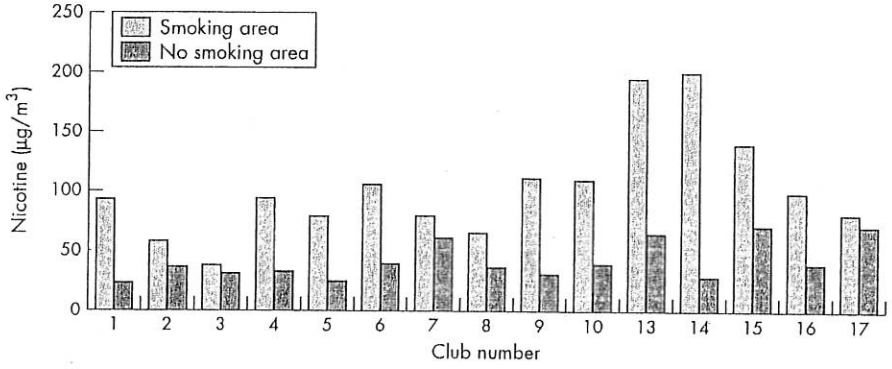


Figure 1 Concentration of atmospheric nicotine in clubs: smoking versus "no smoking" areas*. *Clubs 11 and 12 excluded because of missing data.

2-119

Table 2 Atmospheric nicotine levels (μg/m³) in clubs for smoking and non-smoking areas, and per cent reduction in nicotine levels gained by moving from the smoking to the no-smoking area

Club number	Type of space	Smoking area nicotine level (μg/m³)	No-smoking area nicotine level (μg/m³)	% reduction in atmospheric nicotine level from smoking to no-smoking area
1	SR	93.1	23.3	75.0
2	SR	57.4	36.2	36.9
3	SR	37.5	31.8	15.2
4	SR	94.2	33.6	64.3
5	SR	79.6	25.0	68.6
6	SR	106.4	39.4	63.0
7	SR	79.0	61.8	21.8
8	SR	65.2	35.6	45.4
9	SR	110.6	31.0	72.0
10	SR	109.1	39.8	63.5
11	SR	59.9	–	–
12	SR	–	29.6	–
13	OS	195.7	63.8	67.4
14	OS	199.7	28.7	85.6
15	OS	140.3	71.1	49.3
16	OS	98.7	39.9	59.6
17	OS	81.0	71.0	12.4

OS, one space; SR, single room.

drawn from the analyses undertaken, there are clear limitations by dint of study design. The study involved single measurements at each location. The one similar study (involving nicotine and particulate determinations at seven restaurants) involved two determinations in each location.[10] Single measurements are insufficient to definitively establish the situation at a specific location: the present study was relatively more expansive concerning the number of locations. That said, the study involved "licensed clubs" (a term previously explained) drawing upon different sectors of the community. The clubs involved included sporting, cultural, general entertainment, and community service backgrounds. However, no inferences may be drawn in respect of differences attributable to or correlated with particular types of clubs. No attempt has been made in this study to relate levels of smoking derived contaminants to the size of occupied spaces, or the number of people present. In relation to the latter, it was evident that available technology required analysis of nicotine in particular to be conducted over a period of several hours during which change in number of occupants was inevitable.

In the study now reported, differentiation was made between "no smoking" facilities that involved separate rooms being nominally smoke-free (the SR scenario) and those in which an area in an otherwise single space was designated "no smoking" (the OS scenario). In respect of the SR locations, initial data recorded by us included whether the respective "smoking" and "no smoking" rooms had separate or common ventilation systems. It was determined that only three of the facilities had separate ventilation systems. Preliminary assessment indicated that the data from these three locations were not notably different from the other SR data, and in consequence no attempt was made to make inferences in relation to ventilation systems.

In respect of the limitations identified, it is evident that a more comprehensive study might involve multiple analyses being undertaken at each location and further sub-categorisation of the facilities. Possible relations between numbers of persons present, the size of the space occupied, and the level of tobacco smoke derived pollution might be addressed.

Determination of the extent of occupational and related exposure to ETS have typically involved contrast between situations in which smoking is either permitted or prohibited.[14] Despite such extremes, attempts to prevent exposure to ETS in public places and/or places of employment have specifically included the designation of "no smoking areas" within premises where smoking is otherwise permitted. Few, if any, data are available to determine the efficacy of this measure to reduce or prevent exposure to tobacco smoke.

In the present context, ETS is virtually the only source of atmospheric nicotine. The relevant measurements indicate some reduction in the level of exposure to tobacco smoke is achieved by being in a designated "no smoking area" rather than where smoking is permitted: there is significant

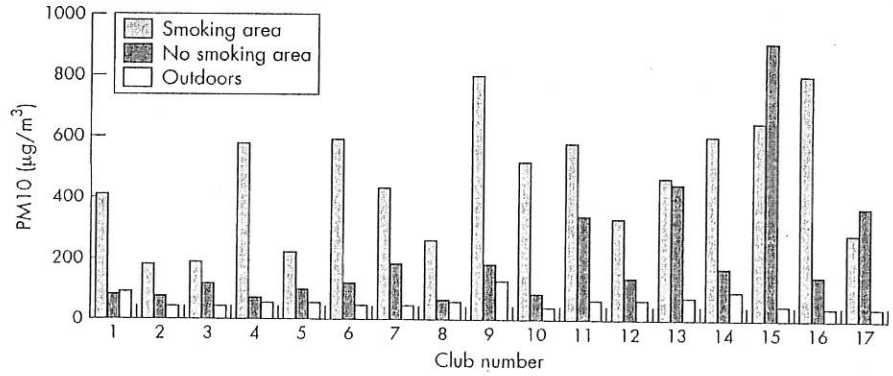


Figure 2 Concentration of atmospheric particulate matter (PM10) in the smoking and "no smoking" areas of clubs, and outdoor areas.

2120

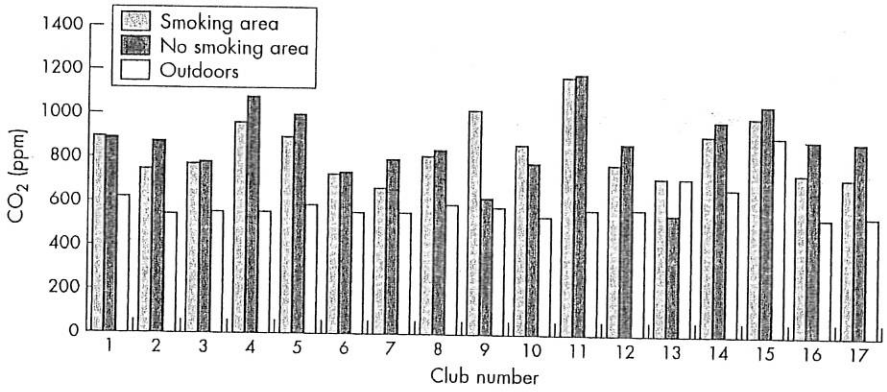


Figure 3 Concentration of atmospheric CO_2 in clubs: smoking versus "no smoking" and outdoor areas.

difference between the mean values. Reduced levels of atmospheric nicotine were apparent in both the configurations examined in this study: the provision of a separate room or the simple designation of an area as "no smoking" within a larger space where smoking occurs. Although data are limited, the hazard presented by passive smoking appears to be dose dependent,[15] and therefore any reduction in exposure is arguably a positive development. That said, and irrespective of the particular configuration of "no smoking" area under consideration, the per cent reduction achieved by relocating from a smoking area to a non-smoking area should not be presumed to be anything better than a 50% reduction on average. Moreover, the wide extent of variation, including individual site per cent reduction values of less than 20% for both SR and OS configurations (table 2), caution against a presumption of any biologically significant outcome from such relocation.

Particulate matter of the type measured in this investigation is not uniquely sourced to tobacco smoke. As it occurs outdoors, such particulate matter is an indicator of air pollution and, among other sources, is derived from engine emissions.[16] On the other hand, cigarette smoke is the preeminent source of such particulate matter as it pollutes the indoor atmosphere. Moreover, in respect of being generated as a result of burning tobacco, particulate matter may be directly related to the carcinogenic activity of tobacco smoke as a major vehicle of inhaled polycyclic aromatic hydrocarbons.[3 12] In the first instance, our determination of particulates confirmed the inference arising from atmospheric nicotine measurements—namely, that designated "no smoking" areas fail to provide protection from ETS. The data are more complicated, insofar as nicotine does not occur in outdoor air, but particulates do. With a single exception, the outdoor PM10 levels were less than either of the values areas measured indoors—that is, in the "smoking" and "no-smoking" areas (fig 2). The differences were significant; in particular, the mean PM10 levels outdoors were significantly less than those in the designated "no smoking" areas, indicating that occupancy of the latter resulted in greater exposure than would have been achieved by going outside.

Determination of PM10 levels provided distinction between the two configurations of "no smoking" areas under consideration—SR and OS. Provision of a separate "no smoking" room resulted in significantly lower particulate levels by comparison to those in the corresponding smoking areas. However, the identification of a "no smoking" area within a larger room or space where smoking was otherwise permitted cannot be presumed to result in a significant reduction in exposure to particulate matter than that occurring were an individual to remain in that area where smoking is allowed (table 3). Such a consequence of the different configurations is also reflected in the corresponding "per cent reduction". Thus, in contrast to an average 66% reduction achieved from relocating from smoking to "no smoking" areas in an SR situation, simply moving from that

Table 3 Atmospheric PM10 in clubs for smoking and no-smoking areas, and per cent reduction in PM10 levels achieved by moving from the smoking to the no-smoking area, and from the smoking area to outdoors

Club number	Type of space	Smoking area PM10 ($\mu g/m^3$)	No-smoking area PM10 ($\mu g/m^3$)	Outdoor area PM10 ($\mu g/m^3$)	% reduction in PM10 from smoking to non-smoking area	% reduction in PM10 from smoking to outdoor area
1	SR	409	83	93	80	77
2	SR	185	76	44	59	76
3	SR	187	117	48	37	74
4	SR	569	72	58	87	90
5	SR	223	99	61	56	73
6	SR	586	115	45	80	92
7	SR	430	179	48	58	89
8	SR	260	67	61	74	77
9	SR	790	179	126	77	84
10	SR	512	87	41	83	92
11	SR	569	337	64	41	89
12	SR	332	135	64	59	81
13	OS	458	439	73	4	84
14	OS	595	169	88	72	85
15	OS	646	904	47	−40	93
16	OS	798	141	39	82	95
17	OS	278	368	38	−32	86

2-121

What this paper adds

Within the hospitality industry in particular, designation of rooms or parts of rooms as "no smoking", in premises where smoking otherwise occurs, is common. Thus, for example, in clubs licensed to sell alcohol for consumption on the premises and providing gaming machines as a principal recreation, and which are located in metropolitan Sydney, Australia, such "no smoking" facilities are offered. Data regarding the protection from environmental tobacco smoke (ETS), which is afforded to club patrons taking advantage of these facilities, are extremely limited. We have sought to evaluate the extent of protection by monitoring levels of ETS related pollutants in smoking and "no smoking" areas of such licensed clubs.

Levels of atmospheric nicotine and tobacco related particulates determined in smoking, "no smoking", and outdoor areas of 17 licensed clubs around Sydney indicate that designated "no smoking" areas do not provide complete protection from ETS. Some reduction in exposure is evident, and separate rooms are in some respects an improvement upon simply delineating a "no smoking" area in a smoking room. However, club patrons might typically expect no more than a 50% reduction in exposure by having recourse to these "no smoking" areas. In some instances, no reduction was evident. Accordingly, such areas cannot be characterised as "smoke-free" and patrons occupying these areas do not achieve the protection from ETS they would experience were smoking not to occur on the premises.

part of the room to another identified with "no smoking" signs resulted in an almost trivial average reduction (17%) in exposure. Indeed, in two individual situations, the PM10 levels were actually higher in such "no smoking" spaces, a scenario not observed in the SR clubs (table 3).

The measurement of CO_2 levels, while indicating differences between outdoor and indoor levels (fig 3), did not allow for further inferences concerning the efficacy of designated "no smoking" areas than could be drawn from nicotine and particulate measurements.

Walsh and Tzelepis[17] have recently reviewed support for smoking restrictions evident from relevant studies involving bars and gaming areas in Australia. In their review, what are described as designated "no smoking" areas in the present report have been variously described in other studies as "separate areas", "restricted areas", "special areas", and "smoke-free areas". The respective terms were employed in studies, but any of them might be employed more widely. All of these epithets are reasonable, except the last one. It is clear from the present studies that the use in any context of the term "smoke-free area" to identify where, as distinct from practice elsewhere in that same building, smoking is not permitted, is incorrect. On the basis of atmospheric contamination as recorded by us, the use of the term "smoke-free area" is inappropriate, perhaps to the point of such usage now being misleading and deceptive.

The present results indicate that designated "no smoking" areas may provide some reduction in the level of exposure of individuals to ETS. However, such areas clearly do not eliminate exposure to ETS, and the reduction achieved may be marginal or trivial. Occupying a separate room designated "no smoking" offers, at best, a marginal improvement in the protection afforded from ETS achieved by being in a "no smoking" area which is part of a room where smoking occurs everywhere else. At least, however, by having recourse to a separate "no smoking" room, an individual would not be worse off. In respect of "no smoking" areas in rooms where smoking occurs, an individual might actually be more heavily exposed to ETS (in terms of particulate matter, specifically) by moving from the smoking to the "no smoking" area. Our results suggest that regulations to permit licensed clubs, taverns, and casinos to permit patrons to smoke in certain parts of the premises if smoking is barred in other parts, must be regarded as ineffective in protecting individuals at risk of passive smoking. The evidence presently available indicates that banning smoking on the premises is the only viable option to prevent exposure to ETS and this option is being adopted in a progressively increasing number of centres. Finally, recovery of damages as a result of a failure to prevent exposure to ETS is now recognised. The present report would suggest that designation of "no smoking" in certain arbitrary areas cannot be raised as an adequate response to the hazard presented by passive smoking.

ACKNOWLEDGEMENTS

This study was supported in part by a grant from Cancer Council NSW. The authors are grateful for the assistance provided by management in the various clubs whose facilities provided the basis of our investigation. The authors are grateful to Dr Valerie Delpech (now Medical Epidemiologist, NSW Health) for discussion of the design of the study.

Authors' affiliations

T Cains, S Cannata, R Poulos*, M J Ferson*, B W Stewart†, South East Sydney Public Health Unit, Randwick, NSW, Australia

*Also School of Public Health and Community Medicine, University of New South Wales, Sydney, NSW, Australia

†Also School of Women's and Children's Health, University of New South Wales

REFERENCES

1 **Repace JL**, Lowrey AH. Indoor air pollution, tobacco smoke, and public health. *Science* 1980;**208**:464–72.
2 **Taylor R**, Cumming R, Woodward A, *et al.* Passive smoking and lung cancer: a cumulative meta-analysis. *Aust N Z J Public Health* 2001;**25**:203–11.
3 **Hoffmann D**, Wynder EL. Active and passive smoking. In: Marquardt H, Schafer SG, McClellan R, Welsch F, eds. *Toxicology*. San Diego: Academic Press, 1999:879–98.
4 **Law MR**, Hackshaw AK. Environmental tobacco smoke. *Br Med Bull* 1996;**52**:22–34.
5 **Daynard R**. Why tobacco litigation? *Tobacco Control* 2003;**12**:1–2.
6 **Stewart BW**, Semmler PCB. *Sharp v Port Kembla RSL Club*: establishing causation of laryngeal cancer by environmental tobacco smoke. *Med J Aust* 2002;**176**:113–6.
7 **Maskarinec MP**, Jenkins RA, Counts RW, *et al.* Determination of exposure to environmental tobacco smoke in restaurant and tavern workers in one US city. *J Expo Anal Environ Epidemiol* 2000;**10**:36–49.
8 **Yamato H**, Seto T, Hori H, Higashi T, *et al.* [The effective smoking corner in an office]. *Sangyo Eiseigaku Zasshi* 2000;**42**:1–5.
9 **Hammond SK**, Sorensen G, Youngstrom R, *et al.* Occupational exposure to environmental tobacco smoke. *JAMA* 1995;**274**:956–60.
10 **Lambert WE**, Samet JM, Spengler JD. Environmental tobacco smoke concentrations in no-smoking and smoking sections of restaurants. *Am J Public Health* 1993;**83**:1339–41.
11 **Brauer M**, Mannetje A. Restaurant smoking restrictions and environmental tobacco smoke exposure. *Am J Public Health* 1998;**88**:1834–6.
12 **International Agency for Research on Cancer**. Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 38: Tobacco smoking. Lyon: International Agency for Research on Cancer, 1986.
13 **Ayers GP**, Selleck PW, Gillett RW, *et al.* Determination of nicotine in water by gradient ion chromatography. *J Chromatogr A* 1998;**824**:241–5.
14 **Bates MN**, Fawcett J, Dickson S, *et al.* Exposure of hospitality workers to environmental tobacco smoke. *Tobacco Control* 2002;**11**:125–9.
15 **International Agency for Research on Cancer**. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Volume 83. Tobacco smoke and involuntary smoking. Lyon: International Agency for Reserch on Cancer (in press).
16 **Groten P**, Cassee FR, Van Bladeren PJ, *et al.* Mixtures. In: Marquardt H, Schafer SG, McClellan R, Welsch F, eds. *Toxicology*. San Diego: Academic Press, 1999:257–70.
17 **Walsh RA**, Tzelepis F. Support for smoking restrictions in bars and gaming areas: review of Australian studies. *Aust N Z J Public Health* 2003;**27**:310–22.

2/22

Title: CDC's Second National Report on Human Exposure to Environmental Chemicals

Publication Date: January 2003

Agency or Author: National Center for Environmental Health, Centers for Disease Control and Prevention

Purpose: This document provides a definition of what cotinine is and why it is used to measure exposure to Environmental Tobacco Smoke (ETS). Cotinine is a substance that can be measured in blood to indicate the degree of exposure to ETS. Also included in this document is limited information on national cotinine levels and that a reduction in cotinine levels is beginning to emerge as more states and communities enact clean indoor air laws.

CDC's *Second National Report on Human Exposure to Environmental Chemicals*

Spotlight on Cotinine

What Cotinine Is

- Cotinine is a "metabolite"—or breakdown product—of nicotine. It forms in the body when a person is exposed to nicotine.
- Nicotine is one of the chemicals found in tobacco smoke and chewing tobacco.
- Nicotine gets into people's bodies if they smoke or chew tobacco, if they are involved in tobacco production and must handle tobacco, or if they are exposed to secondhand tobacco smoke (also called environmental tobacco smoke, or ETS).
- Levels of cotinine in the body track the amount of exposure a person has to tobacco smoke.

Levels of Cotinine Found in the U.S. Population

- For the *Second Report*, scientists tested blood samples for levels of cotinine people selected at random to be representative of nonsmokers in the United States.
- The *Second Report* shows that cotinine levels in nonsmoking Americans have gone down by more than 70% since the early 1990s. Levels have decreased by 58% in children, by 55% in adolescents, and by 75%. These results are encouraging.
- The *Second Report* also includes results showing differences in cotinine levels between different groups of people. Levels in adolescents and children were more than twice the levels measured in adults. Non-Hispanic blacks had higher levels of cotinine than did either non-Hispanic whites or Mexican Americans.

For More Information

- Centers for Disease Control and Prevention, Tobacco Web site: www.cdc.gov/tobacco/index
- U.S. Environmental Protection Agency (EPA), Office of Air and Radiation Secondhand Smoke/Smoke-Free Homes: http://www.epa.gov/smokefree/

Title: Exposure to Environmental Tobacco Smoke and Cotinine Levels – Fact Sheet

Publication Date:

Agency or Author: Centers for Disease Control and Prevention

Purpose: This document provides background information on Environmental Tobacco Smoke (ETS) as well as cotinine, a substance that can be measured in blood to indicate the degree of exposure to ETS. Also included in this document is current trend information concerning cotinine levels nationally and that a reduction in cotinine levels is beginning to emerge as more states and communities enact clean indoor air laws.

Exposure to Environmental Tobacco Smoke and Cotinine Levels — Fact Sheet

---

- The National Institutes of Health's National Toxicology Program's 9th issue of the Report on Carcinogens listed ETS as a "known" human carcinogen, which indicates that there is a cause and effect relationship between exposure and human cancer incidence.

- ETS is classified as a Group A carcinogen (known to cause cancer in humans) under the EPA's carcinogen assessment guidelines. Exposure to ETS causes lung cancer and has been linked to an increased risk for heart disease in nonsmokers.

- ETS causes about 3,000 lung cancer deaths annually among adult nonsmokers. Scientific studies have also estimated that ETS accounts for as many as 35,000 deaths from ischemic heart disease annually in the United States. More research is needed to know exactly how recent changes in ETS exposure may affect lung cancer rates among adult nonsmokers.

- ETS causes serious respiratory problems in children, such as greater number and severity of asthma attacks and lower respiratory tract infections. ETS exposure increases the risk for sudden infant death syndrome (SIDS) and middle ear infections for children.

- Cotinine is a major metabolite of nicotine. Exposure to nicotine can be measured by analyzing the cotinine levels in the blood, saliva, or urine. Since nicotine is highly specific for tobacco smoke, serum cotinine levels track exposure to tobacco smoke and its toxic constituents.

- In 1991, data showed that nearly 90 percent of the U.S. population had measurable levels of serum cotinine in their blood. The Centers for Disease Control and Prevention's National Report on Human Exposure to Environmental Chemicals found more than a 75 percent decrease in median cotinine (metabolized nicotine) levels for nonsmokers in the United States since 1991.

- Children and teenagers, 3-19 years old, had higher levels of cotinine than did adults, 20 years old and above.

- Involuntary exposure to ETS remains a common, serious public health hazard that is entirely preventable by adopting and enforcing appropriate regulatory policies. Smokefree environments are the most effective method for reducing ETS exposure. Healthy People 2010 objectives address this issue and seek optimal protection of nonsmokers through policies, regulations, and laws requiring smoke-free environments in all schools, work sites, and public places.

- California, Connecticut, Delaware, Maine, Massachusetts, New York, and Rhode Island meet the nation's Healthy People 2010 objective to establish

smoke-free indoor air laws covering public places and worksites. Because of comprehensive state laws, virtually all indoor workplaces in these states are now smoke free, including restaurants and bars.

- The dramatic declines in serum cotinine levels among nonsmokers are a good indication that efforts to ensure clean indoor air through smoking restrictions in workplaces, restaurants and other public places are working. However, there are still too many people, especially young people, who continue to be exposed to environmental tobacco smoke (ETS).

Title: Tobacco Smoke - Cotinine

Publication Date: January 2003

Agency or Author: National Center for Environmental Health, Centers for Disease Control and Prevention

Purpose: This document provides the serum cotinine levels for the U.S population. Included in this information are age-grouped mean cotinine levels based upon the National Health and Nutrition Examination Survey in 1999-2000. Major conclusions are that cotinine levels are found across all age groups and are in fact higher among adolescents and children.

Tobacco Smoke

Cotinine

CAS No. 486-56-6

General Information

Tobacco use is the most important, preventable cause of premature morbidity and mortality in the United States. The consequences of smoking and the use of smokeless tobacco products are well known and include an increased risk for cancer, emphysema, and cardiovascular disease. For example, lung cancer is the leading cancer-related killer of both men and women in the United States, and smoking is by far the leading cause of lung cancer.

Environmental tobacco smoke (ETS) is a known human carcinogen, and persistent exposure to ETS is associated with an increased risk for lung cancer and other diseases. Children are at particular risk from ETS, which may exacerbate asthma among susceptible children and greatly increase the risk for lower respiratory-tract illness, such as bronchitis and pneumonia, among young children.

Cotinine is a major metabolite of nicotine and is currently regarded as the best biomarker in active smokers and in nonsmokers exposed to ETS. Measuring cotinine is preferred over measuring nicotine because cotinine persists longer in the body. Cotinine can be measured in serum, urine, saliva, and hair. Nonsmokers exposed to typical levels of ETS have cotinine levels of less than 1

Table 60. Cotinine

Geometric mean and selected percentiles of serum concentrations (in ng/mL) for the non-smoking U.S. population aged 3 years and older, National Health and Nutrition Examination Survey, 1999-2000.

	Geometric mean (95% conf. interval)	Selected percentiles (95% confidence interval)						Sample size
		10th	25th	50th	75th	90th	95th	
Total, age 3 and older	*	< LOD	< LOD	.059 (<LOD-.070)	.236 (.180-.310)	1.02 (.740-1.27)	1.96 (1.64-2.56)	5999
Age group								
3-11 years	*	< LOD	< LOD	.109 (.064-.180)	.500 (.290-1.02)	1.88 (1.19-3.09)	3.37 (1.79-4.23)	1174
12-19 years	*	< LOD	< LOD	.107 (.080-.163)	.540 (.371-.762)	1.65 (1.25-2.11)	2.56 (2.35-3.23)	1773
20 years and older	*	< LOD	< LOD	< LOD	.167 (.137-.200)	.630 (.520-.863)	1.48 (1.23-1.77)	3052
Gender								
Males	*	< LOD	< LOD	.080 (.060-.100)	.302 (.220-.390)	1.20 (.890-1.56)	2.39 (1.78-3.06)	2789
Females	*	< LOD	< LOD	< LOD	.179 (.135-.250)	.850 (.590-1.14)	1.85 (1.41-2.37)	3210
Race/ethnicity								
Mexican Americans	*	< LOD	< LOD	< LOD	.139 (.107-.182)	.506 (.340-.813)	1.21 (.813-1.84)	2242
Non-Hispanic blacks	*	< LOD	< LOD	.131 (.110-.150)	.505 (.400-.625)	1.43 (1.22-1.66)	2.34 (1.89-2.97)	1333
Non-Hispanic whites	*	< LOD	< LOD	.050 (<LOD-.070)	.210 (.150-.313)	.950 (.621-1.40)	1.92 (1.54-2.74)	1949

< LOD means less than the limit of detection, which is 0.05 ng/mL.

* Not calculated. Proportion of results below limit of detection was too high to provide a valid result.

Z-130

ng/mL, with heavy exposure to ETS producing levels in the 1-10 ng/mL range. Active smokers almost always have levels higher than 10 ng/mL and sometimes higher than 500 ng/mL.

Interpreting Serum Cotinine Levels Reported in the Table

Table 60 presents data for the U.S. nonsmoking population aged 3 years and older. For these results, nonsmoking is defined as a serum cotinine level less than or equal to 10 ng/mL. Choosing a cutoff of 15 ng/mL makes little difference in the results. The LOD for these measurements was 0.050 ng/mL.

From 1988 through 1991, as part of NHANES III, CDC determined that the median level (50^{th} percentile) of cotinine among nonsmokers in the United States was 0.20 ng/mL (Pirkle et al., 1996). Table 60 shows that the median cotinine level in 1999-2000 has decreased to 0.059 ng/mL—more than a 70% decrease. This reduction in cotinine levels suggests a dramatic reduction in exposure of the general U.S. population to ETS since the period 1988-1991. Compared with results for the period 1988-1991 for population groups defined by age, gender, and race/ethnicity (Pirkle et al., 1996), cotinine levels declined in all categories.

Covariate-adjusted geometric means were not calculated because more than 40% of the population had cotinine levels less than the LOD. At comparable percentiles, men have higher cotinine levels than women, and non-Hispanic blacks have higher levels than non-Hispanic whites or Mexican Americans. Higher levels of cotinine have been reported for non-Hispanic blacks (Caraballo et al., 1998). As seen previously (Pirkle et al., 1996), males continue to have higher levels than females, and people aged 20 years and older have lower levels than those younger than 20 years of age.

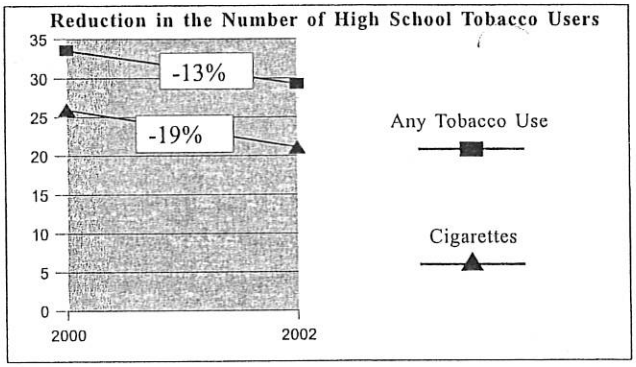
Kansas Department of Health & Environment

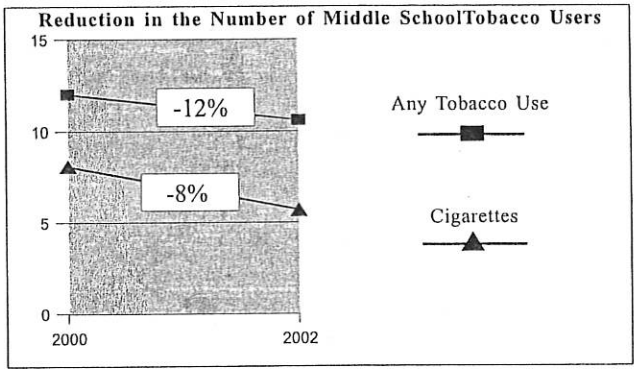
September 2003

Fewer Teens Using Tobacco in 2002

Teen tobacco use is declining

Between 2000 and 2002, the number of kids who use tobacco in Kansas declined by 12% among 6th -8th graders and 13% among 9th -12th graders. **This represents 4,400 fewer kids using tobacco.** This decline includes a 19% decrease in the number of 9th - 12th graders who smoke cigarettes and an 8% decrease in the number of 6th - 8th graders who smoke cigarettes, indicating early success of the Kansas Tobacco Use Prevention Program and its partners. TASK, a statewide youth movement, was created to promote tobacco free teens by uniting communities to create one strong voice standing against the promotion and use of tobacco products. Initially formed in 1998 as a part of the Tobacco Free Kansas Coalition, TASK has evolved into an active entity heavily involved with the prevention of Kansas teen tobacco use. Also contributing to the decline in tobacco use was the $.55 cigarette excise tax increase seen in Kansas in 2002/2003.





Measuring success

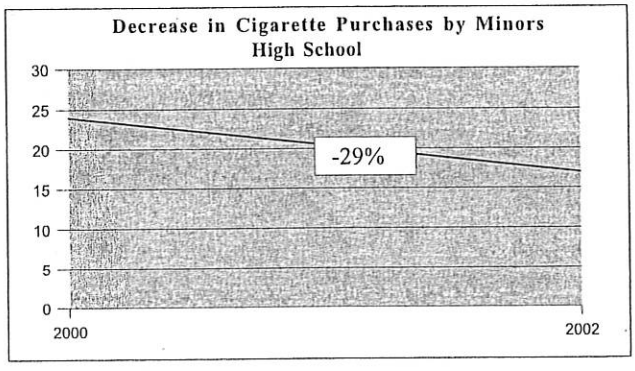
The Kansas Youth Tobacco Survey (KYTS) was conducted in 2000 to provide baseline data and again in 2002 to track changes in youth tobacco use attitudes and behaviors. The survey was administered in randomly sampled middle schools and high schools across Kansas. The KYTS is part of a national effort to obtain data on youth knowledge, behaviors and attitudes toward tobacco. The Kansas results have been reviewed and validated by the Centers for Disease Control and Prevention.

Key results

- 29% reduction in the number of high school seniors who smoke cigarettes
- 12% decline in the number of 6th - 8th graders who have ever tried smoking cigarettes
- 21% reduction in the number of 9th - 12th graders who smoke 5 or more cigarettes per day

Fewer minors are buying cigarettes.

There was a 29% decrease in the number of minors who reported purchasing cigarettes in a store between 2000 and 2002.



KansasTobaccoUse PreventionProgram
Kansas Department of Health and Environment

1000 SW Jackson, Suite 230
Topeka, KS 66612-1274
(877) 602-0368
www.kdhe.state.ks.us/tobacco

2432

Title: Secondhand Smoke and Children's Health

Publication Date: 1999

Agency or Author: World Health Organization

Purpose: This document summarizes the findings of various studies and their conclusions concerning Environmental Tobacco Smoke (ETS) and children's health. This document proves that multiple studies across multiple types of agencies have come to the same conclusions: exposure to ETS has negative health impacts on children

2-134

Second Hand Smoke and Children's Health

A Summary of Research Findings

From: Consultation Report. International Consultation on Environmental Tobacco Smoke (ETS) and Child Health. Tobacco Free Initiative, World Health Organization, June, 1999, p. 17

Report [1]	Lower respiratory tract infections [2]	Middle ear disease	Chronic respiratory symptoms	Asthma	Lung function	Sudden infant death syndrome (SIDS)
United States Surgeon General (1986)	More frequent in children whose parents smoke	Suggestive evidence that middle ear effusion [3] is more common in children whose parents smoke	More frequent in children whose parents smoke	Not reviewed	Small decrements in children whose parents smoke	Not reviewed
United States Environmental Protection Agency (1992)	ETS is causally associated with increased risk	ETS is causally associated with increased prevalence of middle ear effusion	ETS is causally associated with increased prevalence	ETS is causally associated with additional episodes and increased severity of symptoms in asthmatic children; suggestive evidence that ETS causes new cases of asthma	ETS is causally associated with small reductions	Strong evidence that maternal smoking increases the risk of SIDS. Data inadequate to assess specific role of ETS
California Environmental Protection Agency (1997) [4]	ETS is causally associated	ETS is causally associated	ETS is causally associated	ETS is causally associated with asthma exacerbation and induction	Suggestive evidence of causal association with ETS	ETS is causally associated
Australian National Health and Medical Research Council (1997) [5]	ETS has cause-and-effect relationship	Causal link between ETS and middle ear effusion	Not reviewed	Causal relationship between ETS and asthma	Association with ETS exposure	Causal association with ETS
United Kingdom Scientific Committee on Tobacco and Health (1998)	ETS is a cause	Parental smoking causes acute and chronic middle ear disease	Convincing evidence that parental smoking increases risk	ETS is a cause of asthma attacks	Not reviewed	ETS has cause-and-effect association

[1] See bibliography in report for details of source.
[2] In infants and very young children.
[3] i.e., fluid in the middle ear, or "glue ear".
[4] The report also concluded that exposure of pregnant nonsmokers to ETS is causally associated with reduced foetal growth and that there is suggestive evidence that ETS is causally associated with adverse impacts on cognition and behaviour.
[5] The report also concluded that there is suggestive evidence that exposure of pregnant nonsmokers to ETS causes reduced foetal growth.

Title: Cigarette Use Among High School Students – United States, 1991-2003

Publication Date: June 2004

Agency or Author: Morbidity and Mortality Weekly Report

Purpose: This document summarizes the national trend information for youth cigarette use for the past 12 years. This document breaks down use by gender, race/ethnicity, and grade. Overall use of cigarettes peaked during the mid to last 90's and has been declining ever since. However in spite of the decline experienced in youth rates, 1 in 5 high school students still report that they smoke.

MMWR™
Morbidity and Mortality Weekly Report
Weekly June 18, 2004 / Vol. 53 / No. 23

Cigarette Use Among High School Students — United States, 1991–2003

Cigarette use is the leading preventable cause of death in the United States (*1*). One of the national health objectives for 2010 is to reduce the prevalence of current cigarette use among high school students to ≤16% (objective no. 27-2b) (*1*). To examine changes in cigarette use among high school students in the United States during 1991–2003, CDC analyzed data from the national Youth Risk Behavior Survey (YRBS). This report summarizes the results of that analysis, which indicated that although 1) the prevalence of lifetime cigarette use was stable among high school students during the 1990s and 2) the prevalence of both current and current frequent cigarette use increased into the late 1990s, all three behaviors had declined significantly by 2003. Prevention efforts must be sustained to ensure this pattern continues and the 2010 objective is achieved.

The national YRBS, a component of CDC's Youth Risk Behavior Surveillance System, used independent three-stage cluster samples for the 1991–2003 surveys to obtain cross-sectional data representative of public and private school students in grades 9–12 in all 50 states and the District of Columbia. During 1991–2003, sample sizes ranged from 10,904 to 16,296, school response rates ranged from 70% to 81%, student response rates ranged from 83% to 90%, and overall response rates ranged from 60% to 70%. For each cross-sectional national survey, students completed an anonymous, self-administered questionnaire that included identically worded questions about cigarette use.

For this analysis, temporal changes for three behaviors were assessed: 1) lifetime cigarette use (i.e., ever tried cigarette smoking, even one or two puffs), 2) current cigarette use (i.e., smoked cigarettes on ≥1 of the 30 days preceding the survey), and 3) current frequent cigarette use (i.e., smoked cigarettes on ≥20 of the 30 days preceding the survey). For current cigarette use, temporal changes and subgroup differences in 2003 were analyzed by sex, race/ethnicity, and grade. Data are presented only for non-Hispanic black, non-Hispanic white, and Hispanic students because the numbers of students from other racial/ethnic groups were too small for meaningful analysis.

Data were weighted to provide national estimates, and SUDAAN was used for all data analyses. Temporal changes were analyzed by using logistic regression analyses that assessed linear and quadratic time effects simultaneously and controlled for sex, race/ethnicity, and grade. Quadratic trends indicated significant but nonlinear trends in the data over time. When a significant quadratic trend accompanied a significant linear trend, the data demonstrated a nonlinear variation (e.g., leveling off or change in direction) in addition to an overall increase or decrease over time. T-tests were used to examine differences in current cigarette use in 2003 by sex, race/ethnicity, and grade. All results are statistically significant ($p<0.05$) unless otherwise noted.

Significant linear and quadratic trends were detected for lifetime and current cigarette use. The prevalence of lifetime cigarette use, although stable during the 1990s, declined significantly, from 70.4% in 1999 to 58.4% in 2003 (Table 1). The prevalence of current cigarette use increased from 27.5% in 1991 to 36.4% in 1997 and then declined significantly to 21.9% in 2003. A significant quadratic trend was detected for current frequent cigarette use; the prevalence increased from 12.7% in 1991 to 16.7% in 1997 and 16.8% in 1999, then declined significantly to 9.7% in 2003.

INSIDE

DEPARTMENT OF HEALTH AND HUMAN SERVICES
CENTERS FOR DISEASE CONTROL AND PREVENTION

2-136

The *MMWR* series of publications is published by the Epidemiology Program Office, Centers for Disease Control and Prevention (CDC), U.S. Department of Health and Human Services, Atlanta, GA 30333.

SUGGESTED CITATION
Centers for Disease Control and Prevention. [Article Title]. MMWR 2004;53:[inclusive page numbers].

Significant linear and quadratic trends were detected in current cigarette use among both sexes (Table 2). Among female students, the prevalence of current cigarette use peaked during 1997–1999 and then declined significantly to 21.9% in 2003. Among male students, the prevalence of current cigarette use peaked in 1997 and then declined significantly to 21.8% in 2003. Similarly, among white, white female, Hispanic, Hispanic female, Hispanic male, and 9th- and 11th-grade students, current cigarette use prevalence peaked by 1997 and then declined significantly in 2003. Significant quadratic trends were detected among white male, black, black female, black male, and 10th- and 12th-grade students, indicating that the prevalence of current cigarette use peaked by 1999 and then declined significantly.

During 2003, white students were significantly more likely than black and Hispanic students to report current cigarette use. More white female students than black and Hispanic female students and more Hispanic female than black female students reported current cigarette use. The prevalence of current cigarette use was not significantly different among white, black, and Hispanic male students. By grade level, significantly more 10th-, 11th-, and 12th-grade students than 9th-grade students and more 12th-grade than 10th-grade students reported current cigarette use.

Reported by: *Office on Smoking and Health; Div of Adolescent and School Health, National Center for Chronic Disease Prevention and Health Promotion, CDC.*

Editorial Note: The findings in this report indicate that the prevalence of current cigarette use has declined substantially since the late 1990s and is at the lowest level since YRBS was initiated in 1991. These findings are consistent with trends observed in other national surveys, although the other surveys suggest the rate of decline might be slowing (*2–4*). Factors that might have contributed to the decline in cigarette use include 1) a 90% increase in the retail price of cigarettes during December 1997–May 2003 (*5*), 2) increases in school-based efforts to prevent tobacco use, and 3) increases in the proportion of young persons who have been exposed through the mass media to smoking-prevention campaigns funded by states or the American Legacy Foundation (*6*). Factors that might have slowed the rate of decline in cigarette use among young persons include 1) tobacco industry expenditures on tobacco advertising and promotion, which increased from $5.7 billion in 1997 to $11.2 billion in 2001 (*7*); 2) reductions in Master Settlement Agreement funds used for tobacco-use prevention; and 3) the frequency with which smoking was depicted in films (*8*).

2-137

TABLE 1. Percentage of high school students who reported lifetime cigarette use*, current cigarette use†, and current frequent cigarette use§, by category — Youth Risk Behavior Survey, United States, 1991–2003¶

Category	1991		1993		1995		1997		1999		2001		2003	
	%	(95% CI**)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)
Lifetime	70.1	(±2.2)	69.5	(±1.4)	71.3	(±1.7)	70.2	(±1.9)	70.4	(±3.0)	63.9	(±2.1)	58.4	(±3.1)†† §§
Current	27.5	(±2.7)	30.5	(±1.9)	34.8	(±2.2)	36.4	(±2.3)	34.8	(±2.5)	28.5	(±2.0)	21.9	(±2.1)†† §§
Current frequent	12.7	(±2.2)	13.8	(±1.7)	16.1	(±2.6)	16.7	(±1.9)	16.8	(±2.5)	13.8	(±1.6)	9.7	(±1.4)§§

* Ever tried cigarette smoking, even one or two puffs.
† Smoked cigarettes on ≥1 of the 30 days preceding the survey.
§ Smoked cigarettes on ≥20 of the 30 days preceding the survey.
¶ Linear and quadratic trend analyses were conducted by using a logistic regression model controlling for sex, race/ethnicity, and grade. Prevalence estimates shown here were not standardized by demographic variables.
** Confidence interval.
†† Significant ($p<0.05$) linear effect.
§§ Significant quadratic effect.

TABLE 2. Percentage of high school students who reported current cigarette use*, by sex, race/ethnicity†, and grade — Youth Risk Behavior Survey, United States, 1991–2003§

Characteristic	1991		1993		1995		1997		1999		2001		2003	
	%	(95% CI¶)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)	%	(95% CI)
Sex														
Female	27.3	(±3.4)	31.2	(±2.1)	34.3	(±3.2)	34.7	(±2.8)	34.9	(±2.6)	27.7	(±2.1)	21.9	(±2.8)** ††
Male	27.6	(±3.1)	29.8	(±2.3)	35.4	(±2.4)	37.7	(±2.7)	34.7	(±3.0)	29.2	(±2.6)	21.8	(±2.1)** ††
Race/Ethnicity														
White, non-Hispanic	30.9	(±3.3)	33.7	(±2.2)	38.3	(±2.7)	39.7	(±2.4)	38.6	(±3.2)	31.9	(±2.3)	24.9	(±2.4)** ††
Female	31.7	(±4.6)	35.3	(±2.6)	39.8	(±3.5)	39.9	(±3.2)	39.1	(±3.5)	31.2	(±2.5)	26.6	(±3.7)** ††
Male	30.2	(±3.8)	32.2	(±2.7)	37.0	(±3.3)	39.6	(±3.8)	38.2	(±3.7)	32.7	(±3.0)	23.3	(±2.5)††
Black, non-Hispanic	12.6	(±2.5)	15.4	(±2.5)	19.2	(±3.2)	22.7	(±3.8)	19.7	(±4.1)	14.7	(±2.8)	15.1	(±2.8)††
Female	11.3	(±2.3)	14.4	(±2.7)	12.2	(±3.1)	17.4	(±3.9)	17.7	(±3.5)	13.3	(±3.4)	10.8	(±2.9)††
Male	14.1	(±4.5)	16.3	(±4.2)	27.8	(±5.5)	28.2	(±5.5)	21.8	(±7.1)	16.3	(±3.2)	19.3	(±3.7)††
Hispanic	25.3	(±2.8)	28.7	(±2.9)	34.0	(±5.3)	34.0	(±2.7)	32.7	(±3.8)	26.6	(±4.3)	18.4	(±2.3)** ††
Female	22.9	(±3.8)	27.3	(±3.9)	32.9	(±5.6)	32.2	(±3.7)	31.5	(±4.6)	26.0	(±3.7)	17.7	(±2.1)** ††
Male	27.9	(±3.6)	30.2	(±3.4)	34.9	(±8.7)	35.5	(±3.6)	34.0	(±4.5)	27.2	(±7.0)	19.1	(±3.5)** ††
Grade														
9th	23.2	(±3.8)	27.8	(±2.4)	31.2	(±1.6)	33.4	(±5.1)	27.6	(±4.0)	23.9	(±2.9)	17.4	(±2.4)** ††
10th	25.2	(±2.7)	28.0	(±3.3)	33.1	(±3.8)	35.3	(±4.1)	34.7	(±2.5)	26.9	(±3.2)	21.8	(±2.9)††
11th	31.6	(±3.8)	31.1	(±3.2)	35.9	(±3.8)	36.6	(±3.6)	36.0	(±3.0)	29.8	(±3.7)	23.6	(±3.2)** ††
12th	30.1	(±4.4)	34.5	(±3.8)	38.2	(±3.6)	39.6	(±4.9)	42.8	(±5.5)	35.2	(±4.1)	26.2	(±2.8)††

* Smoked cigarettes on ≥1 of the 30 days preceding the survey.
† Numbers for other racial/ethnic groups were too small for meaningful analysis.
§ Linear and quadratic trend analyses were conducted by using a logistic regression model controlling for sex, race/ethnicity, and grade. Prevalence estimates shown here were not standardized by demographic variables.
¶ Confidence interval.
** Significant ($p<0.05$) linear effect.
†† Significant quadratic effect.

The findings in this report are subject to at least two limitations. First, these data apply only to youths who attend high school. Nationwide, among persons aged 16–17 years, approximately 6% were not enrolled in a high school program and had not completed high school (*9*). Second, the extent of underreporting or overreporting in YRBS cannot be determined, although the survey questions demonstrate test/retest reliability (*10*).

Although the declines in cigarette use are encouraging, prevention efforts must be sustained if the nation is to reach its 2010 national health objective. In 2003, approximately one in five high school students were current smokers, and one in 10 were current frequent smokers. Reducing the prevalence of cigarette use further among young persons will require continued efforts in 1) devising targeted and effective media campaigns, 2) reducing depictions of tobacco use in entertainment media, 3) instituting campaigns to discourage family and friends from providing cigarettes to young persons, 4) promoting smoke-free homes, 5) instituting comprehensive school-based programs and policies in conjunction with supportive community activities to prevent smoking initiation and encourage smoking cessation, and 6) decreasing the number of adult smokers (e.g., parents) to present more nonsmoking role models.

References

1. U.S. Department of Health and Human Services. Healthy People 2010 (conference ed., 2 vols.). Washington, DC: U.S. Department of Health and Human Services, 2000.
2. Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. Monitoring the future: national results on adolescent drug use—overview of key findings. Bethesda, Maryland: National Institute on Drug Abuse, 2004; NIH publication no. 04-5506.

Z-138

3. Substance Abuse and Mental Health Services Administration. Overview of findings from the 2002 National Survey on Drug Use and Health. Rockville, Maryland: U.S. Department of Health and Human Services, 2003; DHHS publication no. (SMA) 03-3774.
4. CDC. Tobacco use among middle and high school students—United States, 2002. MMWR 2003;52:1096–8.
5. U.S. Department of Labor. Consumer price index—all urban consumers. U.S. city average, cigarettes. Washington, DC: U.S. Department of Labor, Bureau of Labor Statistics, 2004. Available at http://data.bls.gov/labjava/outside.jsp?survey=cu.
6. Farrelly MC, Healton C, Davis KC, Messeri P, Hersey JC, Haviland ML. Getting to the truth: evaluating national tobacco countermarketing campaigns. Am J Public Health 2002;92:901–7.
7. Federal Trade Commission. Cigarette report for 2001. Washington, DC: Federal Trade Commission, 2003. Available at http://www.ftc.gov/os/2003/06/2001cigreport.pdf.
8. Dalton MA, Sargent JD, Beach ML, et al. Effect of viewing smoking in movies on adolescent smoking initiation: a cohort study. Lancet 2003;362:281–5.
9. Kaufman P, Alt MN, Chapman CD. Dropout rates in the United States: 2000. Washington, DC: U.S. Department of Education, National Center for Education Statistics, 2001; report no. NCES 2002-114.
10. Brener ND, Kann L, McManus T, Kinchen SA, Sundberg EC, Ross JG. Reliability of the 1999 Youth Risk Behavior Survey questionnaire. J Adolesc Health 2002;31:336–42.

Diminishing Racial Disparities in Early-Onset Neonatal Group B Streptococcal Disease — United States, 2000–2003

Increased use of intrapartum antibiotics to prevent perinatal group B streptococcal (GBS) disease during the 1990s led to substantial declines in the incidence of GBS disease in newborns (*1*). Despite this success, at the end of the 1990s, early-onset GBS disease (in infants aged <7 days) continued to be a leading infectious cause of neonatal mortality in the United States, and black infants remained at higher risk than white infants (*1*). In 2002, CDC and the American College of Obstetricians and Gynecologists (ACOG) revised guidelines for prevention of early-onset GBS disease to recommend late prenatal screening of all pregnant women and intrapartum antibiotic prophylaxis (IAP) for GBS carriers (*2,3*). These guidelines were expected to result in further declines in early-onset disease (*4*). This report updates early-onset incidence trends since 1999 analyzed by using population-based, multistate data from the Active Bacterial Core surveillance (ABCs)/Emerging Infections Program Network. The results of the analysis indicated that 1) after a plateau in early-onset disease incidence during 1999–2002, rates declined 34% in 2003 and 2) although racial disparities in incidence persist, rates for blacks now approach the 2010 national health objective of 0.5 cases per 1,000 live births (*5*). Continued implementation of screening and prophylaxis guidelines by clinicians and public health practitioners should lead to further declines in racial disparities.

ABCs conducts active, laboratory-based surveillance for all cases of invasive GBS, including periodic audits to ensure completeness of case finding. A case of early-onset GBS disease was defined as isolation of GBS from a normally sterile site (e.g., blood or cerebrospinal fluid) in a neonate aged 0–6 days residing in an ABCs area. Participating areas during 2000–2003 were Connecticut, Maryland, Minnesota, and selected counties in California, Colorado (beginning in 2001), Georgia, New York, Oregon, and Tennessee, representing a population that produced 419,062 live births in 2001. Of the 2001 live-birth cohort, 73% were white, 20% were black, and 7% were of other races; 15% were of Hispanic origin. The incidence of early-onset disease was calculated by using live-birth data for 2000 and 2001 from ABCs states' vital statistics or the National Vital Statistics Report (available at http://www.cdc.gov/nchs/data/nvsr/nvsr51/nvsr51_02.pdf). Incidence for 2002 and 2003 were calculated by using 2001 live-birth data. Incidence of GBS disease from earlier surveillance years was derived from data published previously (*1*) using comparable methods. A total of 184 (13.2%) of 1,397 cases with missing or unspecified race data during 1996–2002 were matched with birth records to improve the completeness of race reporting. Remaining cases of unknown race (during 1996–2002, a total of 77 [5.5.%] of 1,397; in 2003, a total of 21 [15.7%] of 134) were distributed on the basis of the known race distribution within each county and included in all reported rates. To assess the impact of the August 2002 guidelines, incidence in 2003 was compared with the average incidence for 2000 and 2001; 2002 was considered a transition year.

During 2000–2003, a total of 701 cases of early-onset GBS disease were reported in the surveillance areas (Table). Outcome was known for 676 (96.4%) cases; the case-fatality ratio was 6.5%. A total of 150 (21.4%) infants were born before 37 weeks' gestation; among these preterm infants, the case-fatality ratio was 22.7%.

During 1999–2001, early-onset disease incidence remained nearly constant, with an average of 0.47 cases per 1,000 live births. In 2003, the overall disease incidence was 0.32 (Figure 1), representing a 34% (95% confidence interval [CI] = 20%–46%) decline in incidence since 2000–2001. The incidence in 2003 varied geographically, from 0.53 in Tennessee to 0.14 in Oregon (Table). Rates in Georgia decreased significantly compared with the 2000–2001 baseline ($p<0.01$), and rates in Tennessee decreased marginally ($p = 0.06$).

During 1999–2001, disease incidence remained stable for both black and white populations, and rates among black

Title: Various

Publication Date: Various

Agency or Author: Various

Purpose: Included are the findings of two primary research articles concerning the impact of clean indoor air laws on the restaurant and hospitality industry: one from El Paso, Texas and the other from Lawrence, Kansas. Also included are two summary articles that give the results of comprehensive literature reviews concerning clean indoor air laws and economic impacts. Finally, included are the abstracts from four primary research articles concerning the economic impact of clean indoor air laws.

The conclusion of the scientific body of evidence is that no negative economic impacts have been found concerning the implementation of clean indoor air laws. Studies to date have found that there is either no impact on revenue or there is an increase in revenue when adjusted for possible outside factors following the implementation of clean indoor air laws.

Additional information can be found on the included data CD.

Impact of a Smoking Ban on Restaurant and Bar Revenues — El Paso, Texas, 2002

Smoke-free indoor air ordinances protect employees and customers from secondhand smoke exposure, which is associated with increased risks for heart disease and lung cancer in adults and respiratory disease in children (*1,2*). As of January 2004, five states (California, Connecticut, Delaware, Maine, and New York) and 72 municipalities in the United States had passed laws that prohibit smoking in almost all workplaces, restaurants, and bars (*3*). On January 2, 2002, El Paso, Texas (2000 population: 563,662), implemented an ordinance banning smoking in all public places and workplaces, including restaurants and bars. The El Paso smoking ban is the strongest smoke-free indoor air ordinance in Texas and includes stipulations for enforcement of the ban by firefighting and law enforcement agencies, with fines of up to $500 for ordinance violations (*4*). To assess whether the El Paso smoking ban affected restaurant and bar revenues, the Texas Department of Health (TDH) and CDC analyzed sales tax and mixed-beverage tax data during the 12 years preceding and 1 year after the smoking ban was implemented. This report summarizes the results of that analysis, which determined that no statistically significant changes in restaurant and bar revenues occurred after the smoking ban took effect. These findings are consistent with those from studies of smoking bans in other U.S. cities (*5–8*). Local public health officials can use these data to support implementation of smokefree environments as recommended by the Task Force on Community Preventive Services (*9*).

To study the impact of the El Paso smoking ban on all sectors of the local restaurant and bar industry, TDH and CDC obtained quarterly sales tax reports and monthly mixed-beverage tax receipts from the Texas Comptroller of Public Accounts. The sales tax reports provided revenue data for restaurants, bars, and retail businesses, grouped by Standardized Industrial Classification (SIC) codes. Categories were created for restaurants (SIC codes 5812, 5816, and 5817) and bars (SIC codes 5813 and 5814) (*10*). The sales tax reports included revenue generated by sales of meals and sales of beer and wine for establishments with beer and wine retailer permits; sales tax revenue data were used for 1990–2002. Other restaurant and bar revenue data came from reports filed by holders of mixed-beverage permits. The state's mixed-beverage gross receipts tax, enacted in 1994, is levied on revenue generated by sales of alcoholic beverages (e.g., liquor, beer, and wine) and nonalcoholic beverages and ice used in mixed drinks. Mixed-beverage revenue data were used for 1995–2002.

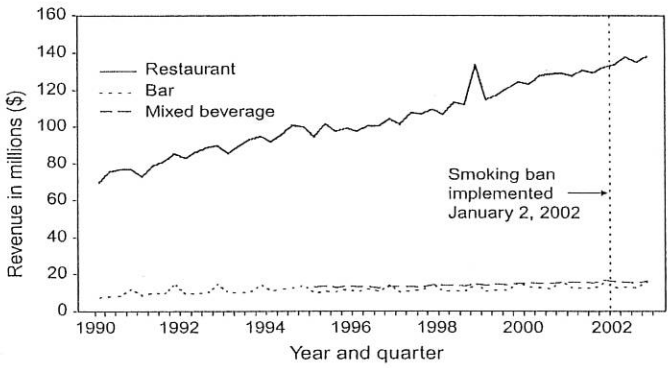
Multiple linear regression analysis was used to examine the effect of the El Paso smoking ban on changes in revenue over time. The following independent variables were considered: a variable indicating whether the smoking ban was in force, an ordinal variable to represent secular time, and three variables to indicate during which one of four calendar quarters the revenue data were collected. Two regression models were created for each of the following primary dependent variables: 1) revenue subject to sales tax from all restaurants and bars, restaurants only, and bars only; and 2) revenue subject to the mixed-beverage tax. For each category, the first model examined the association between the smoking ban and revenue, and the second examined the association between the smoking ban and the fraction of revenue as a percentage of El Paso's total retail revenues (SIC codes 5211–5999). This fraction accounts for economic variation that might impact revenue in all sectors of the retail economy (*6*).

Two sets of statistics were used to evaluate the quality of the models. The Durbin-Watson statistic was calculated for each model to determine if first-order autocorrelation was present. Variance inflation factors were examined to determine if multicollinearity was present in any of the models.

Restaurant, bar, and mixed-beverage revenues varied by quarter; in all categories, revenues usually were higher during the fourth quarter (October–December) of each year (Figure 1). During all four quarters, bar and mixed-beverage revenues accounted for approximately 1% of total retail revenues (Figure 2).

None of the regression models for restaurant, bar, or mixed-beverage revenues or for such revenues as percentages of total retail revenue over time showed any statistically significant changes after the smoking ban was implemented (Table). In

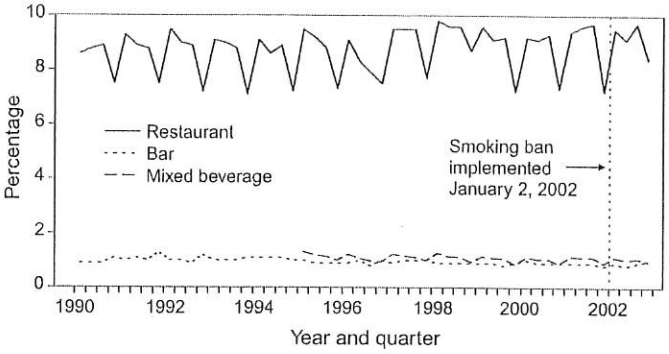
FIGURE 1. Restaurant, bar, and mixed-beverage* revenues, before and after implementation of smoking ban, by quarter — El Paso, Texas, 1990–2002



* Mixed-beverage revenue data were available only for 1995–2002.

2-141

FIGURE 2. Restaurant, bar, and mixed-beverage* revenues as percentage of total retail revenues, before and after implementation of smoking ban, by quarter — El Paso, Texas, 1990–2002



* Mixed-beverage revenue data were available only for 1995–2002.

addition, the results did not change when revenues were adjusted for inflation, and adjusting for changes in price did not change the results (*8*). In all models, the variance inflation factors had values of <2 for each of the independent variables, indicating that multicollinearity was not present, and the Durbin-Watson statistics indicated that none of the autocorrelations was statistically significant (Table).

Reported by: *P Huang, MD, Texas Dept of Health. AK De, PhD, Div of Applied Public Health Training, Epidemiology Program Office; ME McCusker, MD, EIS Officer, CDC.*

Editorial Note: No decline in total restaurant or bar revenues occurred in El Paso, Texas, after the city's smoking ban was implemented on January 2, 2002. These findings are consistent with the results of studies in other municipalities that determined smoke-free indoor air ordinances had no effect on restaurant revenues (*2,5–8*). Despite claims that these laws especially might reduce alcoholic beverage revenues (*2*), the mixed-beverage revenue analyses indicate that sales of alcoholic beverages were not affected by the El Paso smoking ban.

The findings in this report are subject to at least three limitations. First, because sales tax reports lag revenue collection by 6 months, sales tax data were available for only 1 year after the El Paso smoking ban was implemented. However, analyses from other cities that included data for several years after a smoking ban was enacted indicated no declines in restaurant or bar revenues (*6–8*). Revenue data from El Paso will be monitored for any changes in restaurant and bar revenues. Second, because limited revenue data for El Paso were available, methods that might provide better estimates of the impact of the ban could not be used. Regression models measuring changes in slope for revenues before and after implementation of smoke-free indoor air ordinances might provide better estimates of how these ordinances affect revenues (*8*); time-series models also might produce better estimates. When more information becomes available, these models should be applied to the El Paso data. Finally, because the SIC code–based restaurant and bar categories are not mutually exclusive, certain bars were included in the restaurant category created for this analysis. However, mixed-beverage tax data, which provide a more precise measure of alcohol-related revenue, support the finding that bar revenues were not affected by the smoking ban.

Opponents of smoke-free indoor air ordinances have claimed that enacting smoke-free indoor air ordinances will harm restaurant and bar revenues (*2*). However, the findings in this report indicate that, in El Paso, Texas, restaurant and bar revenues were not affected by the smoking ban. Such analyses of

TABLE. Impact of a smoking ban on restaurant, bar, and mixed-beverage revenues* — El Paso, Texas, 2002

Revenue type	Mean revenue per quarter ($)	Effect of ban		Model fit†	
		Change in revenue§ ($)	(95% CI¶)	R^2	Durbin-Watson**
Restaurant	104,749,601	1,336,331	(-3,189,740–5,862,402)	0.96	1.76
% of total retail	*8.8*	*0.2*	*(-0.7–1.1)*	*0.21*	*2.05*
Bar	11,454,957	9,211	(-1,959,153–1,977,576)	0.43	2.03
% of total retail	*1.0*	*0.03*	*(-0.1–0.1)*	*0.29*	*1.70*
Total	**116,204,559**	**1,269,532**	**(-4,632,656–7,171,720)**	**0.95**	**2.08**
% of total retail	***9.7***	***0.3***	***(-0.6–1.2)***	***0.15***	***2.02***
Mixed beverage	14,187,573	-276,505	(-909,710–356,700)	0.83	1.89
% of total retail	*1.1*	*0.03*	*(-0.1–0.2)*	*0.46*	*1.70*

* Restaurant and bar revenues are from sales tax data for 1990–2002; mixed-beverage revenues are from mixed-beverage gross receipts tax data for 1995–2002.
† P values were all nonsignificant (p<0.01).
§ Change in revenue indicates the value of the coefficient for the indicator variable representing the El Paso smoking ban in each model. All p values for this coefficient were nonsignificant (p>0.1).
¶ Confidence interval.
** None of the Durbin-Watson results indicates a significant autocorrelation. In a model with three independent variables and 52 observations (i.e., restaurant and bar models), <1.67 indicates significant positive autocorrelation and >2.58 indicates significant negative autocorrelation. In a model with three independent variables and 32 observations (i.e., mixed-beverage models), the critical values are <1.65 and >2.76, respectively.

2-142

economic data can provide local policymakers with statistical evidence to evaluate the merit of implementing smoke-free indoor air ordinances in their communities.

Acknowledgments

This report is based on contributions by M Boerm, P Gingiss, Univ of Houston; Research Div and Open Government Section, Office Texas Comptroller of Public Accounts.

References

1. California Environmental Protection Agency. Health effects of exposure to environmental tobacco smoke. Sacramento, California: California Environmental Protection Agency, Office of Environmental Health Hazard Assessment, 1997. Available at http://www.oehha.org/air/environmental_tobacco/finalets.html.
2. Glantz SA. Smoke-free restaurant ordinances do not affect restaurant business. Period [Editorial]. J Public Health Manag Pract 1999;5:vi–ix.
3. American Nonsmokers' Rights Foundation. Clean indoor air ordinance counts summary. Berkeley, California: American Nonsmokers' Rights Foundation, 2004. Available at http://www.no-smoke.org/mediaordlist.pdf.
4. Gingiss PM, Roberts-Gray C, Boerm MC, et al. Texas smoke-free municipal ordinance database. Houston, Texas: University of Houston, 2002.
5. CDC. Assessment of the impact of a 100% smoke-free ordinance on restaurant sales—West Lake Hills, Texas, 1992–1994. MMWR 1995; 44:370–2.
6. Glantz SA, Smith LR. The effect of ordinances requiring smoke-free restaurants and bars on revenues: a follow-up. Am J Public Health 1997;87:1687–93.
7. Glantz SA. Effect of smokefree bar law on bar revenues in California. Tob Control 2000;9:111–2.
8. Glantz SA, Charlesworth MA. Tourism and hotel revenues before and after passage of smoke-free restaurant ordinances. JAMA 1999; 281:1911–8.
9. Task Force on Community Preventive Services. Recommendations regarding interventions to reduce tobacco use and exposure to environmental tobacco smoke. Am J Prev Med 2001;20:10–5.
10. Occupational Safety and Health Administration. Standard Industrial Classification (SIC) System Search. Washington, DC: U.S. Department of Labor, Occupational Safety and Health Administration, 2003. Available at http://www.osha.gov/oshstats/sicser.html.

Effect of New Susceptibility Breakpoints on Reporting of Resistance in *Streptococcus pneumoniae* — United States, 2003

In January 2003, the National Committee for Clinical Laboratory Standards (NCCLS) finalized new breakpoints for defining the susceptibility of *Streptococcus pneumoniae* isolates to cefotaxime and ceftriaxone (*1*). The former breakpoints were based on attainable concentrations of these antibiotics in cerebrospinal fluid (CSF) and the level at which it was thought that meningitis treatment failed because of elevated minimum inhibitory concentrations (MICs). The new breakpoints differ for *S. pneumoniae* isolates causing meningitis and those causing nonmeningeal clinical syndromes. To assess the effect of these new criteria on reporting of nonsusceptible *S. pneumoniae* isolates, CDC analyzed cefotaxime MIC data from the Active Bacterial Core Surveillance (ABCs) of the Emerging Infections Program (EIP) Network during 1998–2001. This report summarizes the results of that analysis, which indicated that after the new criteria were applied, the number of isolates defined as nonsusceptible to cefotaxime decreased 52.1%–61.2% for each year. Laboratory reports for clinicians should include interpretations using the new breakpoints for meningitis and nonmeningeal syndromes for all non-CSF isolates.

During 1998–2001, ABCs/EIP surveillance areas from eight states (California, Connecticut, Georgia, Maryland, Minnesota, New York, Oregon, and Tennessee) conducted surveillance for invasive pneumococcal disease. Surveillance populations ranged from approximately 17.4 million in 1998 to 18.6 million in 2001 (*2*). A case of invasive pneumococcal disease was defined as isolation of *S. pneumoniae* from a normally sterile site in a resident of a surveillance area. Isolates were tested for susceptibility at reference laboratories by using NCCLS methods (*1*). Isolates were considered to be nonsusceptible to an antibiotic if they met intermediate or resistant criteria by MIC testing. Under the former criteria, susceptible, intermediate, and resistant MIC breakpoints for cefotaxime and ceftriaxone were $\leq$0.5, 1, and $\geq$2 μg/mL, respectively, for all pneumococci. Under the new criteria, isolates from CSF or other body sites where meningitis is suspected maintain the old breakpoints, but isolates causing nonmeningeal syndromes have breakpoints of $\leq$1, 2, and $\geq$4 μg/mL, respectively.

During 1998–2001, the number of *S. pneumoniae* isolates collected annually ranged from 3,128 to 3,961 (Table). Approximately 95.6% of isolates collected caused nonmeningeal clinical syndromes such as pneumonia with bacteremia. The percentage of isolates causing meningitis ranged from 4.4% in 1998 to 5.5% in 2000.

The percentage of isolates causing nonmeningeal syndromes that were nonsusceptible to penicillin ranged from 24.3% in 1998 to 26.5% in 2000. Penicillin nonsusceptibility was consistently higher among isolates causing meningitis (Table). The susceptibility breakpoints for penicillin remain unchanged and are the same for isolates causing both meningitis and nonmeningeal syndromes.

Under the former breakpoints, the percentage of isolates causing nonmeningeal syndromes that were nonsusceptible to cefotaxime ranged from 13.8% in 1998 to 16.7% in 2000 (Table). Cefotaxime nonsusceptibility was consistently higher among isolates causing meningitis. When the new breakpoints were applied, the percentage of isolates causing invasive

2-143

The Economic Impact of Indoor Smoking Bans

October 13, 2004

Michael H. Fox, Sc.D.
Associate Professor
Department of Health Policy and Management
University of Kansas Medical Center
913 588-2687
mfox2@kumc.edu

Research Director
Research and Training Center on Independent Living
University of Kansas, Lawrence, Kansas
785 864-0587

2-144

The Economic Impact of Indoor Smoking Bans

I. Background

State or community-wide ordinances banning indoor smoking in public places have gained increased momentum in recent years as clear scientific evidence has established the link between exposure to second-hand smoke (SHS) and increased risk of disease and illness. The CDC (2001) estimates over 3,000 lung cancer deaths and 62,000 deaths from coronary heart disease in adult nonsmokers are attributable to second-hand smoke in the U.S. each year, with estimates of respiratory illnesses affecting hundreds of thousands of persons, and consequent health care costs in the billions. According to recent data cited by the CDC (10/2004), California, Connecticut, Delaware, Maine, Massachusetts, New York, and Rhode Island as well as 336 other municipalities elsewhere in the U.S. have passed laws prohibiting smoking in virtually all public places, including restaurants and bars.

While no recent literature credibly argues against the dangers of second-hand smoke exposure, there is literature that argues against the need for ordinances banning indoor smoking, primarily based on three factors:

- the *freedom* of smokers to smoke in public places and freedom of workers to choose not to work in places where smoking is allowed;
- the *scientific validity* of studies that cite little or no economic hardship in places where ordinances have banned smoking; and
- the need for *private markets* to accommodate the demands of a diverse clientele, including both smokers and non-smokers. This is closely aligned with what many would term *business rights.*

II. Summarizing Economic Arguments Against Indoor Smoke-free Policies

A. The Quality and Objectivity of the Research

A recent exhaustive review of 97 studies evaluating the economic impact of smoke-free policies led by an Austrialian research team (Scollo, et.al. 2003) and published in a sponsored journal of the British Medical Association looked closely at the quality and funding sources of each research study.

"Quality" was determined by having independent reviewers use four criteria:

- use of objective data,
- use of multiple observation points over time,
- use of statistical methods that control for random events, and
- use of statistical methods that control for economic trends.

In addition, funding sources were considered as being either connected to the tobacco industry or not.

They found:

- One of 31 (3%) tobacco industry supported studies has been published in a peer reviewed journal, compared to 23 of 60 (38%) of non-industry funded studies;

- None (0/31) of the tobacco industry funded studies met all four independent criteria of methodological quality and 84% (26/31) met none of the criteria, while 35% (21/60) of studies that were not supported by the tobacco industry met all criteria.

- Of these 21 non-tobacco funded studies meeting all criteria for quality, **none** (0) reported a negative economic impact of smoking bans, while four (4) reported a positive impact, with business growth occurring after a ban.

- 94% (29/31) of tobacco industry supported studies concluded that there would be a negative economic impact after enactment of a smoke free policy, though none of these studies is based upon the use of objective (i.e., non survey) data. One of 35 studies (3%) funded by a source independent of tobacco industry funding reported a negative impact.

IN SUMMARY, TOBACCO FUNDED RESEARCH LOOKING AT ECONOMIC IMPACT IS LESS SCIENTIFICALLY VALID THAN RESEARCH FUNDED BY INDEPENDENT SOURCES

B. Dunham and Marlow Studies:

Two economists have written extensively on the economic impact of smoking Bans (Dunham and Marlow, 2000) disputing research that suggests no negative effects to smoke-free policies and supporting arguments for free markets to determine appropriate controls. Most recently, results of a seven year old survey of restaurant owners in Wisconsin were published in an economic journal as an "industry overview" (Dunham and Marlow, 2003). The authors highlighted the fear and uncertainty among bar and restaurant owners about the impacts of a possible ban and argued for the freedom of the market to determine what was best to minimize potential economic hardship. An earlier published article contends that sales and tax revenues are inappropriate measures of economic impact because of their inability to distinguish what the authors feel are "differential effects" that might influence some establishments more than others.

The primary difficulties with these studies and with other work undertaken by these two economists are:

1. Their reliance upon survey data that are highly subjective and play to the fear of business owners speculating on unknown consequences or disruptions to their past practice;
2. Their dismissal of economic outcomes such as sales and tax revenues as appropriate measures to determine success or failure of smoking bans;
3. Their inability to control for time using multiple observation points in any of their own economic forecasting;

4. Their use of citations that themselves have no published or in some cases even unpublished (Evans, www.speakup.org/pdf/misc003.pdf, March 1997) sources;
5. The self-stated objectivity of the economists with respect to undertaking scientific research. In the case of Mr. Dunham, he holds a masters degree in business and economics and is president of a self proclaimed "guerrilla economics" consulting company whose marketing logo is "Economics of Crisis and Controversy" (http://www.guerrillaeconomics.com/). In the case of Dr. Marlow, a more centrist economist (Brad DeLong, of the Univ. of California, http://econ161.berkeley.edu/movable_type/2003_archives/000811.html) has characterized him as being among the "Gamma Gradient" of far right supply side economists in our country who represent a fringe element within the Republican party who interpose political philosophy freely with economic theory in expressing their ideas. In a recent letter to President Bush, Dr. Marlow and this group of economists enthusiastically endorsed what they termed the "fiscally responsible" economic policies of this Administration. Yet the letter was signed by only four of seventeen past members and chairs of Republican Councils of Economic Advisors.

IN ADDITION TO LACKING OBJECTIVITY, DUNHAM AND MARLOW HAVE NOT MADE A SCIENTIFICALLY VALID CASE FOR ECONOMIC HARDSHIP AMONG BUSINESSES AFFECTED BY INDOOR SMOKING ORDINANCES, RELYING AS THEY DO UPON SURVEY DATA THAT REQUIRE RESPONDENTS TO PREDICT THEIR OWN ECONOMIC CONSEQUENCES TO CHANGES IN THEIR BUSINESS CLIMATE.

III. Recent Economic Arguments that Support Indoor Smoke-Free Policies

A. A recent issue of Morbidity and Mortality Weekly Reports, an official publication of the Centers for Disease Control and Prevention (CDC), reports on the impact of a smoking ban on restaurant and bar revenues in El Paso, Texas, tracking revenues from 1990 and through the end of 2002, the first year in which the ban occurred. Data were controlled for inflation and seasonal factors. No significant changes in revenues occurred among restaurants, bars or mixed beverage establishments affected by the ban.

Other economic studies, such as that undertaken by the Worker's Compensation Board of British Columbia (2001), identify lower operating costs for businesses operating in a smoke-free environment, including a reduced need for improved ventilation systems, reduced health claims and absenteeism among employees, reduced building insurance and cleaning costs, and other related savings.

Similar results have been found in studies of the influence of a ban in California, communities in Texas, North Carolina, Arizona and Massachusetts and in New York City, also cited in the CDC article or the Canadian Worker's Compensation study. In fact, there appears to be consistent evidence that supports either no effect of a smoking ban on a community's economic health

2-147

or an improvement, based on scientific studies that use objective, non-opinion data such as tax or business revenues to evaluate outcomes. In the first year after the smoke-free ordinance took effect in NYC, for example, tax revenues from all bars and restaurants rose 12%. A study published in 2001 by Cremieux and Ouellette found that in Quebec restaurants, anticipated costs of regulating smoking in restaurants were not realized – there was essentially no impact due to infrastructure costs, decreased productivity, or decreased patronage. They concluded that smoking regulations do not impose undue economic hardship on the industry. Another study published in 2002 by Bartosch and Pope came to the same conclusion – no measurable difference in meals tax revenue was found in Massachusetts following implementation of a highly restrictive restaurant smoking policy.

THERE IS NO CREDIBLE EVIDENCE TO SUPPORT THE SOMETIMES PASSIONATELY EXPRESSED VIEW THAT SMOKE-FREE ORDINANCES WILL HARM BUSINESS. INDEED, IT APPEARS THAT MANY STUDIES SUPPORT EVIDENCE OF BUSINESS GROWTH.

IV. Learning from the Lawrence, Kansas Experience: The Mayor's Task Force, Economic Impact Statement, March 31, 2004

The author of the Economic Impact Statement, a restaurant owner who was a member of the Mayor's Task Force, raised numerous points leading to the conclusion that a full impact, exemption free smoking ban would lead to a 10% reduction in alcohol beverage sales and $2.5 million loss in revenues to businesses in Lawrence.

Point: While community-wide studies show little or no overall effect of smoke-free ordinances, the influence on sales and profits for individual restaurants can be substantial. Because of this "macro" approach to researching the economic impact, these studies are "not particularly useful."
Response: Community-wide actions require community-wide ways of measuring these actions. While the author is correct in assuming that there will be losers, there will also be winners, and on balance the evidence overwhelmingly points to no long term community-wide business hardships resulting from smoke-free policies enacted elsewhere.

Point: 54% of restaurant owners and 81% of bar owners "indicated profit reductions" based on smoking bans.
Response: This is incorrect on two levels: research on which this is based is seriously flawed, and the data used from this bad research are incorrect. First, the study (Dunham and Marlow, 2003, discussed above) is a survey of perceptions of respondents in which results are pooled between those in which some unspecified "ban" occurred – even though none appear to be documented in Wisconsin according to the American Nonsmoker's Rights Foundation's listing of smoke-free ordinances – and those in which persons

2-148

were asked to predict what would happen if government imposed a "ban." The author of the Economic Impact Statement should have used the figure in this survey referring to restaurants affected by the "ban": 38% of restaurants – not 54% - in which they claimed a ban would lead to loss in revenues. 55% claimed no loss or increase in revenues, using the original data cited. ***In other words, bad research was misquoted to suggest that more than half of restaurants experiencing "government bans" would lose money.***

Point: Smokers consume more food and drink than non-smokers.
Response: While evidence suggests that this is true, there are also over twice as many adults who are non-smokers as there are smokers and they represent a potential market share who can make up for any drop in consumption by smokers. Also, this is assuming that smokers will simply drop their patronage or spending on food or drink based on the requirement to smoke outdoors. There is no evidence that this will occur.

Point: Draft beer sales dropped by 13% in British Columbia during a four month experiment with a smoke free policy.
Response: The independent evaluation of this four month trial, however, came to the conclusion that there would be no long-term economic impacts of continuing a smoke free policy, and recent regulations in BC now call for either smoke-free accommodations or construction of well ventilated, separately walled designated smoking rooms in restaurants and bars in BC in which employees who elect to work cannot spend more than 20% of their work time.

Point: Restaurant sales increases in California lag behind those of Lawrence because of the state-wide smoking ban.
Response: There are many factors that could explain this, but no existing research which attributes this difference to smoking policy.

Point: Examples of economic growth in the hospitality industry are slanted towards new, largely national chains which can afford to be more flexible to changing business conditions, including smoke-free policies.
Response: There will be individual businesses adversely affected, but those who can adapt to changing business conditions should remain competitive. Many other factors go into competitiveness with national chains, including product quality, location, pricing, marketing strategy, customer loyalty, etc.

Point: Lawrence will lose $2.5 million in sales if a smoking ban is enacted.
Response: There remains absolutely no evidence to justify this prediction. At the time of this writing, efforts to place the smoking ordinance on a ballot to be placed to a vote have not received the required 3,750 signatures from registered voters in the city of Lawrence. Newspaper articles that suggest an economic effect of the ordinance are completely equivocal.

V. Summary

Evaluating the existing literature on economic impact of indoor smoking bans leads to the following:

1. Though no studies are without limitations, the overwhelming majority of studies that maintain a rigorous scientific element suggest that the economic impact of a smoking ban is minimal if it exists at all;

2. The leading researchers who appear to argue consistently against smoking bans give little evidence of objectivity in their work in this or other areas they are involved in;

3. Arguments that mask the economics of the issue, such as smoker/non-smoker or business "rights," are issues entirely separate from those having to do with economic impact, and should be separated out from any discussion of them.

References

U.S. Environmental Protection Agency (2004). Health Effects/Adult Nonsmokers.
http://www.epa.gov/smokefree/healthrisks.html#Other%20Studies

U.S. Centers for Disease Control and Prevention (CDC). Morbidity and Mortality Weekly Report. Impact of a Smoking Ban on Restaurant and Bar Revenues – El Paso, Texas, 2002. February 27, 2004; 53(7):150-152.

U.S. Centers for Disease Control and Prevention (CDC). Morbidity and Mortality Weekly Report. State-Specific Prevalence of Current Cigarette Smoking Among Adults, and Policies and Attitudes About Secondhand Smoke – United States, 2000. December 14, 2001; 50(49): 101-105.

U.S. Centers for Disease Control and Prevention (CDC). Morbidity and Mortality Weekly Report, Recommendations and Reports. Strategies for REducign Exposure to Environmental Tobacco Smoke, Increasing Tobacco-use Cessation, and Reducing Initiation in Community Health Care Systems. November 10, 2000; 49(RR-12): 1-11.

Scollo M., Lal A., Glantz S. Review of the Quality of Studies on the Economic Effects of Smoke-free Policies on the Hospitality Industry. *Tobacco Control.* March 2003 v12 i1 p13(8). COPYRIGHT 2003 British Medical Association.

Dunham J., Marlow M. Smoking Laws and their Differential Effects on Restaurants, Bars, and Taverns. *Contemporary Economic Policy* 2000;18:326-333.

2-150

Dunham J., Marlow M. The Effects of Smoking Laws on Seating Allocations of Restaurants, Bars, and Taverns. *Economic Inquiry* 2000;38:151-157.

Dunham J., Marlow M. The Economic Incidence of Smoking Laws. (Industry Overview). *Applied Economics,* Dec. 15, 2003;35(18):1935-1943.

Workers Compensation Board of British Columbia . The Economic Impacts of the Proposed Amendment to the ETS Regulation. February, 2001.

Cremieux P., Ouellette P. Actual and Perceived Impacts of Tobacco Regulation on Restaurants and Firms. *Tobacco Control.* March 2001; v10i2 p33-37.

Bartosch W., Pope G. Economic Effect of Restaurant Smoking Restrictions on Restaurant Business in Massachusetts, 1992 to 1998. *Tobacco Control.* June 2002; v11i3 p38-42.

ANR AMERICANS FOR NONSMOKERS' RIGHTS
Defending your right to breathe smokefree air since 1976

ECONOMIC IMPACT OF SMOKEFREE LAWS: CASE STUDIES

December 2004

All reliable economic impact studies on business show either no economic effect or a positive one after a smokefree law goes into effect. When the issue of smokefree air arises, the tobacco industry will work hard to create dissent and fear. Their goal is to convince business owners and residents that the sky will fall if a smokefree law passes. Since 1987, the tobacco industry and smokefree opponents have consistently claimed that smokefree laws lead to a decrease in business in restaurants, bars, bingo halls, and billiard halls, usually by 20-50%, with an accompanying decrease in employment. These claims are totally unfounded. On the contrary, the number of peer-reviewed economic studies showing that smokefree laws have either no economic effect, or a positive one, continues to mount as more communities pass and implement strong smokefree laws. Going smokefree is good for health and good for business. Period.

State

- **New York:** Contrary to arguments of smokefree opponents that smokefree air puts bars and pubs out of business, there was no reported sharp decline in the number of bars following the law's implementation. In fact, the number of bars in the state has increased by 3.5%, from April 2002 to May 2004. New York's comprehensive law took effect on June 23, 2003.[1]

- **California:** According to the California Board of Equalization, the Golden State's hospitality sector continues to grow since the California Clean Indoor Air Act was enacted in 1994. Sales tax data show an increase in annual sales from $7.16 billion in 1997 for establishments selling beer and wine to $9.6 billion in 2002. For establishments selling all kinds of alcohol, sales increased from $8.64 billion in 1997 to $11.3 billion in 2002. In 2003, the Board's Employment Development Department reported that the number of individuals employed in California's bars and restaurants had about 200,500 more employees than they did in 1995, before the smokefree policy took effect.[2]

- **Delaware:** Despite predictions that the smokefree law would have dire economic effects on the hospitality sector, comparative data compiled by the Delaware Division of Public Health and Division of Revenue shows that business remains steady. In fact, data shows the number of restaurant, tavern, and taproom licenses in Delaware has increased since the smokefree law took effect. The number of issued restaurant licenses increased from 3,291 in November 2002 to 3,323 in October of 2003. Employment within the hospitality industry increased, as well, from 27,900 individuals employed in food service and drinking establishments in September 2002 to 28,100 in September 2003.[3]

- **Massachusetts:** A systematic statewide comparison of 239 communities in Massachusetts revealed that local smokefree laws do not harm businesses. Taxable meals receipts data was collected for over 1,000 restaurants between 1992 and 1999. Contrary to restaurateur predictions, researchers found that restaurant sales in towns with strong smoking restrictions experienced a slightly faster rate of growth than restaurant sales in towns without such

2530 San Pablo Avenue, Suite J • Berkeley, California 94702 • (510) 841-3032 / FAX (510) 841-3071
www.no-smoke.org • anr@no-smoke.org

restrictions. Included in the study was an analysis of the effect of comprehensive ordinances on communities bordering towns without similar smoking restrictions. The data revealed that this factor "failed to have a statistically significant effect on meals receipts."[4]

- **Texas:** Clean indoor air ordinances were passed in Arlington, Austin, Plano, and Wichita Falls between July 1994 and March 1996. Researchers evaluated the effect of these ordinances on restaurant sales using restaurant and retail tax data. Information was collected from the first quarter of 1987 through the last quarter of 1999. Despite variations in the municipalities' geographic, demographic, and economic composition, no detrimental effect on restaurant sales was found to have resulted from the ordinances in any of the four cities studied.[5]

Local

- **New York City:** Business is booming in New York City's bars and restaurants with tax receipts up 12% since the introduction and enactment of the city's Smoke-Free Indoor Air law in March 2003. Figures from the city's Department of Finance show $12 million paid in taxes from bars and restaurants from April through September of 2003, compared to $10.8 million in 2002. Department of Finance Commissioner, Martha E. Stark said one early economic trend was encouraging since the policy was introduced last March: "New York's bars and restaurants paid the city 12% more in business taxes in the months since the ban began than they did in the corresponding six-month period in 2002." In addition, a 2003 New York City Department of Health and Mental Hygiene study designed to measure the ordinance's effect on employment rates in smokefree establishments, found a gain of 10,000 jobs since the implementation of the smokefree air act.[6]

- **Minot, North Dakota:** After analyzing six years of data collected by the Office of the North Dakota Tax Commission, a study conducted by the Minot State University College of Business and the North Dakota Center for Persons with Disabilities, found "no adverse change in restaurant sales because of [Minot's] restaurant no-smoking ordinance," which went into effect on January 1, 2002. Data was collected from the first quarter of 1997 through the fourth quarter of 2002, and figures were analyzed using linear regression analysis – a statistical technique that adjusts for normal fluctuations in sales due to economic trends and seasonal patterns.[7]

- **Fort Wayne, IN:** Hudson Institute Fellow, William Styring, investigated the impact of a 1998 smoking ban on restaurant revenues in Fort Wayne. Sales tax data was collected between 1987 (twelve years before the ordinance was enacted) and 2000 (two years after the ordinance was enacted). No statistically significant variation in revenues was found.[8]

- **Boulder, Colorado:** According to GASP (Group to Alleviate Smoking Pollution) of Colorado, sales tax revenues continued to grow in Boulder after the passage of the smokefree restaurant ordinance in 1995. Revenues from January through October of 1997 were up 3.14%, 1998 revenues were up 4.83%, and 1999 revenues were up 4.31%. The Boulder city finance department referred to the 1999 restaurant sales as a positive "strength."[9]

- **Dane County, Wisconsin:** In 1992, the city of Madison and several surrounding towns in Dane County passed ordinances restricting smoking in restaurants. A report on the impact of these laws found that between 1992 and 1997, per capita restaurant expenditures rose at a higher rate within the county than in the rest of the state. Meanwhile, employment in restaurants grew faster than in any other Madison industry. Furthermore, the number of voluntary smokefree restaurants in Dane County areas *not* covered by the ban grew from 4 in 1993 to 89 in 1997.[10]

- **Corvallis, Oregon:** A July 1998 smokefree law in Corvallis bars did not harm business, concluded a study conducted by the Pacific Research Institute in Eugene. Sales data was collected from September 1997 through September 1999 and compared to data collected in nearby communities where similar smokefree laws were not in place. Researchers concluded that smokers did not abandon Corvallis bars and restaurants, and that revenues from the nonsmoking majority replaced any loss of business from smokers. Furthermore, Corvallis showed no decline in malt beverage sales relative to surrounding communities.[11]

- **Chapel Hill, NC:** Researchers at UNC-Chapel Hill examined restaurant sales data between 1990 and 1997 in ten counties; five with comprehensive smoking ordinances and five similarly situated counties with weak or no smoking ordinances. No differences were found in restaurant sales between the two groups.[12]

- **Flagstaff, AZ:** A study conducted by researchers at Northern Arizona University found that Flagstaff's smokefree restaurant ordinance had no adverse effect on restaurant sales, as measured by tax data from January 1, 1990 (3.5 years before the enactment of the smokefree ordinance) to December 31, 1994 (1.5 years after enactment). Using four different methods of analysis, the study compared Flagstaff restaurant and retail sales with sales in two similar Arizona cities, three counties, and the entire state of Arizona.[13]

- **West Lake Hills, TX:** Researchers at the Centers for Disease Control and Prevention used sales tax data to analyze the impact of a 100% smokefree ordinance on restaurant sales in West Lake Hills. Data was collected for a 17-month period preceding the enactment of the ordinance and for a 19-month period following the enactment. Multiple linear regression techniques were used to account for seasonal variations and temporal economic trends. The study concluded, "The total sales of the restaurants did not decrease after implementation of the ordinance."[14]

- **Beverly Hills and Bellflower, CA:** The California cities of Beverly Hills and Bellflower repealed their smokefree restaurant ordinances following opposition organized by the tobacco industry. Studies have since shown that, contrary to tobacco industry claims, there was no detectable drop in restaurant sales during the time the ordinances were in effect, nor was there an increase in restaurant sales following reversal of the 100% smokefree ordinances.[15,16,17]

International

- **British Columbia, Canada:** On January 1, 2000, the Workers' Compensation Board (WCB) of British Columbia amended its workplace smoking laws to include the hospitality industry. The following March, the amendment was overturned in court pending further public consultations. A study conducted by Pacific Analytics Inc analyzed both the real (two month) and potential economic impact of the amendment at the request of the WCB. Researchers concluded that the amendment would have had no long-term impact on employment or restaurant sales. A new amendment prohibiting smoking in all hospitality and entertainment facilities went into effect in April 2002.[18]

REFERENCES

[1] RTI International, "First Annual Independent Evaluation of New York's Tobacco Control Program," *New York State Department of Health*, November 2004. Accessed on November 29, 2004. Download at http://www.health.state.ny.us/nysdoh/tobacco/reports/docs/nytcp_eval_report_final_11-19-04.pdf.

[2] California State Board of Equalization: California Department of Health Services, Tobacco Control Section, November 2002; State of California, Employment Development Department, *Labor Force Statistics*, November 2003.

[3] [n.a.], "Delaware's Clean Indoor Air Act: The 1st Anniversary Story," *Delaware Division of Public Health and Delaware Division of Revenue*, 2004.

[4] Bartosch, William, and Pope, Gregory, "The Economic Effect of Restaurant Smoking Restrictions on Restaurant Business in Massachusetts 1992-1998: Final Report", *Center for Health Economics Research*, submitted to Massachusetts Department of Public Health, November 27, 2000.

[5] Hayslett, and Huang, "Impact of Clean Indoor Air Ordinances on Restaurant Revenues in Four Texas Cities," *[n.s.]*, March 21, 2000.

[6] Shanahan, C., "New York bars and restaurants 'not hit by smoking ban'" *Irish Examiner*, January 7, 2004; New York City Department of Health and Mental Hygiene, "Initial effects of New York City smoking ordinance," July 23, 2003.

[7] Moseley, F.; Buettner-Schmidt, K., "The Economic Impact of Minot's Smoke-Free Restaurant Ordinance," Minot, North Dakota: *Minot State University, College of Business & ND Center for Persons with Disability*, June 5, 2003.

[8] Styring, "A Study of the Fort Wayne (IN) Restaurant Smoking Ban: Has it Impacted the Restaurant Business?" May 2001.

[9] [n.a.], "Boulder's Smoke-Free Ordinance Makes Good Cents for Restaurants and Bars," *GASP of Colorado*, 2000.

[10] Dresser, "Clearing the Air: The Effect of Smoke free Ordinances on Restaurant Revenues in Dane County," *Tobacco-Free Wisconsin Coalition,* January 1999.

[11] Dresser, Boles, Lichtenstein and Strycker, "Multiple Impacts of a Bar Smoking Prohibition Ordinance in Corvallis, Oregon," *Pacific Research Institute*, [n.d.].

[12] Goldstein and Sobel, "Environmental Tobacco Smoke Regulations Have Not Hurt Restaurant Sales in North Carolina," *North Carolina Medical Journal, 59(5): 284-288*, September/October 1998.

[13] Sciacca and Ratliff, "Prohibiting Smoking in Restaurants: Effects on Restaurant Sales," *American Journal of Health Promotion, 12(3): 176-184,* January/February 1998.

[14] [n.a.], "Assessment of the Impact of a 100% Smoke-Free Ordinance on Restaurant Sales," *Morbidity and Mortality Weekly Report, 44:370-372*, 1995.

[15] Hinderliter, de Llamas and Associates, Glendora, CA, November 8, 1991.

[16] Glantz and Smith, "The Effect of Ordinances Requiring Smokefree Restaurants on Restaurant Sales," *American Journal of Public Health 84:1081-1085*, 1994.

[17] [n.a.], "The 30 Percent Myth," *Consumer Reports*, May 1994.

[18] Pacific Analytics Inc., "The Economic Impacts of the Proposed Amendment to the ETS Regulation," *The Workers Compensation Board of British Columbia*, February 2001.

2-156

Summary of Studies Assessing the Economic Impact of Smoke-Free Policies in the Hospitality Industry – includes studies produced to February 2004*

Produced by Michelle Scollo and Anita Lal,
VicHealth Centre for Tobacco Control, Melbourne, Australia

April 2004

VicHealth Centre for Tobacco Control
The Cancer Council Victoria
100 Drummond St
Carlton, Vic
Australia
61 3 9635 5123

http://www.vctc.org.au/tc-res/Hospitalitysummary.pdf

Last record added 28 April 2004; Last minor revision to this paper: 28 April 2004

Background

Well-designed studies on the economic impact of policy changes: 1. are based on objective measures; 2. use data several years before and after policy implementation; 3. use appropriate statistical tests which test for significance, controlling for underlying trends and fluctuations in data; and 4. control for changes in economic conditions [1].

A large number of studies have examined the effect of smoke-free policies in the hospitality industry. Studies vary greatly in methodological quality. To facilitate greater analysis of methodological quality and overall trends in findings, we have compiled and summarised the publication details, key features and findings of all available studies.

We attempted to locate all studies in the English language that purported to predict or assess the economic impact of smoke-free policies in the hospitality industry [1]. In late November 2001, we searched Medline, Science Citation Index, Social Sciences Citation Index, Current Contents, PsychInfo, and Healthstar using the terms smok* and restaurants, bars, hospitality, economic, regulation and law. We also included unpublished studies; these studies were predominantly funded by the tobacco industry or organizations linked to the tobacco industry. These were located from a compilation by the Alberta Tobacco Control Centre [2], by a request to members of the International Union Against Cancer's International Tobacco Control Network (GLOBALink), and an examination of hospitality industry websites and the websites of tobacco companies based in major English speaking countries, including the Philip Morris "Options" website, www.pmoptions.com. We also conducted an Internet search with the Google search engine, using the terms "smok* bans" and "restaurants" or "bars", limited by the terms "economic impact" or "study". Since December 2001, we have added further studies as we have become aware of them through monitoring of media reports and alerts on tobacco related publication by the Centers for Disease Control.

Each study was summarised and the following details tabulated: study author and year published; date and location of policy implementation; nature of policy implemented; publisher name and type; funding source indicated; nature of outcome measure used; the type of analysis used; whether economic trends were controlled for; a brief description of the findings; and whether the study was peer-reviewed [2]. Where the source of funding was unclear, we systematically searched previously secret tobacco industry documents made available as part of settlement agreements between tobacco companies the US attorneys general [3, 4] and accessible through www.tobaccoarchives.com .

Both authors examined each of the reports. Each author made an independent assessment of whether or not study authors had concluded that the actual or potential impact of the smoke-free policies on the measures studied was negative.

Findings

Characteristics and results of each of the studies are tabulated in **Attachment Tables 1** and **2**. **Attachment Table 1** includes studies using objective outcome measures such as sales tax receipts, business registrations, or employment levels. **Attachment Table 2** includes studies using subjective outcome measures such as patron or proprietor predictions and estimates. Some studies included both objective measures and subjective measures. In this case, findings about objective measures are tabulated in **Attachment Table 1**, and findings about subjective measures in **Attachment Table 2**.

- No negative economic impact from the introduction of smoke-free policies in restaurant and bars is indicated by the 21 studies where findings are based on an objective measure such as taxable sales receipts, where data points several years before and after the introduction of smoke-free policies were examined, where changes in economic conditions are appropriately controlled for, and where appropriate statistical tests are used to control for underlying trends and fluctuations in data. Just a few studies using objective measures have found negative effects. Each of these is methodologically flawed.

- Studies concluding a negative economic impact have predominantly based findings on outcomes predicted before introduction of policies, or on subjective impressions or estimates of changes rather than actual, objective, verified or audited data. These studies were funded predominantly by the tobacco industry or organisations allied with the tobacco industry. Almost none of the studies finding a negative impact are published in peer-reviewed journals.

The key characteristics and findings of each of the studies are summarised in **Tables 1** and **2** below.

Table 1. Studies using objective measures to assess economic impact of smoke-free policies in the hospitality industry

	Control for economic conditions		*Do not control for economic conditions*	
	No effect, or positive effect	**Negative effect**	**No effect, or positive effect**	**Negative effect**
Studies funded from sources other than the tobacco industry				
Taxable sales receipts	Bartosch & Pope, (1995)[5] **Bartosch & Pope (1999)** [6] **Bartosch & Pope (2002) [7]** Bialous & Glantz (1997) [8] * Dresser (1999) [9] Ferrence et al (2003) [10] **Glantz & Charlesworth (1999) [11]** **Glantz & Smith (1994) [12]** **Glantz & Smith (1997) [13]** Glantz (2000) [14] Glantz & Wilson-Loots (2003) [15] **Goldstein & Sobel (1998)[16]** Hayslett & Huang (2000) [17] Huang (2004) [18] **Huang et al (1995)[19]** **Hyland et al (1999)[20]**[a] Hyland (2002) [21] **Hyland (2003) [22]** Maroney et al (1994)[23] Moseley (2003) [24] Pacific Analytics (2001)[25]		California State Board of Equalization (1998)[31] * City of Boulder (1996) [32] Fletcher (1998) [33] New York City Department of Finance (2004) [34]	

	Pope & Bartosch (1997)[26] **Sciacca & Ratliff (1998)[27]** Styring (2001) [28] Taylor Consulting (1993)[29] **Wakefield et al (2002) [30]**			
	No effect, or positive effect	**Negative effect**	**No effect, or positive effect**	**Negative effect**
Sales data other	Bourns & Malcomson 2002 [35] Lal 2003 [36]		* Dresser et al (1999)[37]	
Employment levels	**Hyland et al (2000) [38]** * Bourns & Malcomson 2001[39] * Hild et al 2001[40] *** Hyland & Cummings (1999)[41][b]** * Hyland & Tuk (2001)[42] Hyland (2003) [22]		New York City Department of Health and Mental Hygiene (2003) [43] New York City Department of Finance (2004) [34]	
Number of establishments	*** (Hyland & Cummings (1999)[41]) [b]**		New York City Department of Finance (2004) [34]	
Number of restaurant/bar permits applications			New York City Department of Finance (2004) [34]	
Bankruptcy data	(Bourns & Malcomson 2001[39]) (Bourns & Malcomson 2002)[35]			
Number of Employment insurance claims	(Bourns & Malcomson 2001[39]) (Bourns & Malcomson 2002)[35]			
Studies for which funding source is unknown				
Sales Data				* Pubco 2002 [44]
Studies conducted by organisations or consultants with links to the tobacco industry around the time of the study				

Taxable sales receipts				* Lilley et al (1996)[b] [45] * Masotti et al (1991)*[46] †
Studies funded by tobacco companies or industry groups supported by the tobacco industry				
Taxable sales receipts				* Laventhol et al (1990) [47]
Sales data other				* Applied Economics (1996)[48] Deloitte & Touche LLP [49]
Employment levels				* Lilley et al (1999)[50] * Lilley et al (1996) [51][a]
Number of establishments				* (Lilley et al 1999) [50])

Bold type = peer reviewed; * Use discrete rather than continuous data prior to and after the introduction of policies; † Only weak evidence of connection with the tobacco industry

Table 2. Studies using subjective measures to assess the economic impact of smoke-free policies in the hospitality industry

	Control for economic conditions		*Do not control for economic conditions*	
	No effect or positive effect	**Negative effect**	**No effect or positive effect**	**Negative effect**
Studies funded from sources other than the tobacco industry				
Public self-reported intentions or actual patronage of restaurants/bars			Allen & Markham (2001) [52] August (2000)[53] **Biener & Fitzgerald (1999)[54]** **Biener & Siegel (1997) [55]** **Corsun et al (1996)[56]** Decima Research (2002) [57] Decima Research (2001)[58] Dresser et al (1999)[37] Field Research (1998)[59] Field Research (1997)[60] **Hyland & Cummings (1999)[d] [61]** Lam (1995)[62] McGhee 2002[63] Miller & Kriven (2002) [64] Miller & Kriven (2002)[65] Shapiro, (2001)[66] Styring (2001)[28] **Wakefield et al 1999 [67]**	
Proprietor predictions/ perceptions of sales changes	**Hyland & Cummings[e] (1999)[68]**		(Allen & Markham (2001)[52]) **Cremieux & Oulette (2001)[69]** (Dresser et al (1999)[37]) Edwards (2000)[70] Huron County Health Unit 1999 [71] **Jones et al (1999) [72]** Markham & Tong (2001)[73] Parry et al (2001) [74] **Sciacca & Eckram (1993)[75]** **Sciacca (1996)[76]** **Stanwick (1998)[77]** The Conference Board of Canada (1996)[78]	

			Yorkshire Ash (2001) [79]	
Proprietor predictions/perceptions of cost			**(Cremieux & Oulette (2001) [69])** (The Conference Board of Canada (1996)[78]) Douglas County CHIP (2001) [80]	
Estimated numbers of overseas visitors			Hodges & Maskill (2001)[81]	

April 2003, Vol 93, No. 4 | American Journal of Public Health 611-617

RESEARCH AND PRACTICE

Changes of Attitudes and Patronage Behaviors in Response to a Smoke-Free Bar Law

Hao Tang, MD, PhD, David W. Cowling, PhD, Jon C. Lloyd, MA, Todd Rogers, PhD, Kristi L. Koumjian, MS, Colleen M. Stevens, MSW and Dileep G. Bal, MD, MPH

Hao Tang, David W. Cowling, Jon C. Lloyd, Todd Rogers, Kristi L. Koumjian, and Colleen M. Stevens are with the Tobacco Control Section, California Department of Health Services, Sacramento. Dileep G. Bal is with the Cancer Control Branch, California Department of Health Services, Sacramento.

Correspondence: All requests for reprints should be sent to Hao Tang, MD, PhD, Tobacco Control Section, California Department of Health Services, PO Box 942732, MS 555, Sacramento, CA 94234-7320 (e-mail: htang@dhs.ca.gov).

Objectives. We examined patron responses to a California smoke-free bar law.

Methods. Three telephone surveys measured attitudes and behavior changes after implementation of the law.

Results. Approval of the law rose from 59.8% to 73.2% (odds ratio [OR] = 1.95; 95% confidence interval [CI] = 1.58, 2.40). Self-reported noncompliance decreased from 24.6% to 14.0% (OR = 0.50; 95% CI = 0.30, 0.85). Likelihood of visiting a bar or of not changing bar patronage after the law was implemented increased from 86% to 91% (OR = 1.76; 95% CI = 1.29, 2.40).

Conclusions. California bar patrons increasingly support and comply with the smoke-free bar law.

This article has been cited by other articles:

CONTEMPORARY ECONOMIC POLICY

Contemporary Economic Policy 2004 22(4):520-525; doi:10.1093/cep/byh038

Smoke-free Ordinances Increase Restaurant Profit and Value

Benjamin C. Alamar and Stanton A. Glantz

Abstract

This study estimates the value added to a restaurant by a smoke-free policy using regression analysis of the purchase price of restaurants as a function of the presence of a smoke-free law and other control variables. There was a median increase of 16% (interquartile range 11% to 25%) in the sale price of a restaurant in a jurisdiction with a smoke-free law compared to a comparable restaurant in a community without such a law. This result indicates that contrary to claims made by opponents of smoke-free laws, these laws are associated with an increase in restaurant profitability. (JEL *I120*, *H000*, *D780*)

Abbreviations: BEA: Bureau of Economic Analysis • BLS: Bureau of Labor Statistics • CI: Confidence Interval • GSP: Gross State Product • SDCF: Sellers Discretionary Cash Flow • SHS: Secondhand Smoke • WLS: Weighted Least Squares

2-164

CONTEMPORARY ECONOMIC POLICY

Contemporary Economic Policy 2004 22(4):520-525; doi:10.1093/cep/byh038

Smoke-free Ordinances Increase Restaurant Profit and Value

Benjamin C. Alamar and Stanton A. Glantz

Abstract

This study estimates the value added to a restaurant by a smoke-free policy using regression analysis of the purchase price of restaurants as a function of the presence of a smoke-free law and other control variables. There was a median increase of 16% (interquartile range 11% to 25%) in the sale price of a restaurant in a jurisdiction with a smoke-free law compared to a comparable restaurant in a community without such a law. This result indicates that contrary to claims made by opponents of smoke-free laws, these laws are associated with an increase in restaurant profitability. (JEL *I120*, *H000*, *D780*)

Abbreviations: BEA: Bureau of Economic Analysis • BLS: Bureau of Labor Statistics • CI: Confidence Interval • GSP: Gross State Product • SDCF: Sellers Discretionary Cash Flow • SHS: Secondhand Smoke • WLS: Weighted Least Squares

2-165

The Center For Hospitality Research Cornell Hotel and Restaurant Administration Quarterly

New York's smoke-free regulations: Effects on employment and sales in the hospitality industry

by Andrew Hyland, Vanaja Puli, Michael Cummings, Russ Sciandra

Vol.44, Iss. 3; pg. 9

Executive Summary: Contrary to claims that smoke-free regulations cause decrease in hospitality-industry sales, this study determined that neither sales nor employment is hurt when smoke-free regulations are put in place. That conclusion is based on an examination of changes in restaurants' and hotels' business levels in five populous New York State jurisdictions that have implemented smoke-free regulations. Using state sales data and employment data for eating and drinking establishments and for hotels, the study compared those statistics for the year before the regulation was implemented with the same statistics for the first year following implementation. Instead of damaging hospitality sales and employment, the onset of smoke-free regulations was associated with increases in per-capita taxable sales for eating and drinking establishments and hotels (controlling for other economic factors). Employment rose in hotels, while no measurable change was observed for employment in restaurants operating under smoke-free regulations.

2-166