



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 States¹

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?

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 Institute⁴, there are 69 known or probable carcinogens in cigarette smoke⁵. The complete list of these carcinogens appears in the table below.

LIST OF KNOWN, PROBABLE, AND POSSIBLE CANCER CAUSING CHEMICALS IN SECONDHAND SMOKE	
<u>Polycyclic Aromatic Hydrocarbons</u>	<u>Miscellaneous Organic Compounds</u>
Benz(a)anthracene Benzo(b)fluoranthene Benzo(j)fluoranthene Benzo(k)fluoranthene Benzo(a)pyrene Dibenz(a,h)anthracene Dibenzo(a,l)pyrene Dibenzo(a,e)pyrene Indeno(1,2,3-cd)pyrene 5-Methylchrysenes	Acetamide Acrylonitrile DDT Catechol 1,1-Dimethylhydrazine 2-Nitropropane Ethyl carbamate Ethylene oxide Propylene oxide Methyleugenol MeAaC (2-amino-3-methyl-9-H-pyrido[2,3-b]indole)
<u>N-Nitrosamines</u>	<u>Inorganic Compounds</u>
N-Nitrosodimethylamine N-Nitrosoethylmethylamine N-Nitrosodiethylamine N-Nitrosodi-n-propylamine N-Nitroso-di-n-butylamine N-Nitrosopyrrolidine N-Nitrosopiperidine N-Nitrosodiethanolamine N-Nitrosomonocotinine 4-(Methylnitrosamino)-1-(3pyridyl)-1-butanone	Hydrazine Arsenic Beryllium Nickel Chromium (only hexavalent) Cadmium Cobalt Lead Polonium-210
<u>N-Heterocyclic Amines</u>	<u>Aldehydes</u>
AaC Trp-P-1 Glu-P-1 PhIP IQ Trp-P-2 Glu-P-2	Formaldehyde Acetaldehyde
<u>Volatile Hydrocarbons</u>	<u>Heterocyclic Compounds</u>
1,3-Butadiene Isoprene Benzene Styrene	Quinoline Dibenz(a,j)acridine Benzo(b)furan Dibenz(a,h)acridine Dibenzo(c,g)carbazole Furan
<u>Aromatic Amines</u>	
2-Toluidine 2-Naphthylamine 2,6-Dimethylaniline 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 *10th 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

Related Fact Sheets

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), *10th Report on Carcinogens: Revised December 2002*, December 2002, <http://ehp.niehs.nih.gov/roc/tenth/profiles/s176toba.pdf>.
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- 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.
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- 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.
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- 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. Ekblom, "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 Spontaneous 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*

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