Environmental Tobacco Smoke:
Health Risk or Health Hype?

A Special Report
Prepared by the American Council on Science and Health

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Active smoking has been recognized as a major cause of disease and death for at least 40 years. But in the past 20 years a growing body of evidence has shown that exposure to environmental tobacco smoke (ETS)—in other words, passive smoking—may also be a threat to health.

Scientists have documented the presence of some of the toxic and carcinogenic components of environmental tobacco smoke in the hair and body fluids of nonsmokers exposed to tobacco smoke, and an extensive database on the health effects of ETS now exists. Numerous epidemiological studies have examined the associations between ETS exposure and acute (short-term) and chronic (long-term) health effects. But debates still continue about the validity of the scientific methods used to establish causality for chronic diseases—lung cancer and cardiovascular disease, for example—reported in nonsmokers living or working with smokers. Study limitations and sometimes-equivocal results have led many observers to question whether the health risks that the studies have shown to be associated with environmental tobacco smoke are, indeed, real.

In this report the American Council on Science and Health (ACSH) evaluates the large body of evidence that exists regarding the health effects of ETS. ACSH’s analysis yields the following conclusions:

- Irritation of the eyes, nose, and respiratory tract is the most common and firmly established adverse health effect associated with exposure to ETS.
- Exposed infants and children, in particular, are at increased risk of respiratory infections, middle-ear effusion (fluid inside the eardrum), and the exacerbation of asthma and other respiratory symptoms.
- Exposed adults are at increased risk for respiratory ailments; ETS may aggravate the symptoms of preexisting asthma and emphysema.
- Extensive epidemiological evidence indicates that ETS exposure is a weak risk factor in the development of lung cancer in nonsmokers regularly exposed to ETS in the workplace and/or at home.
- Epidemiological evidence also suggests that ETS is a weak risk factor for heart disease in nonsmoking spouses of smokers and in nonsmokers regularly exposed to ETS in the workplace and/or at home.
- Other reported links between ETS and chronic disease (breast cancer, cervical cancer, and leukemia, for example) have not been scientifically established and are not addressed in this report.
The scientific evidence that tobacco smoke in indoor environments is associated with acute and chronic respiratory illnesses, particularly in children, supports the adaptation of measures designed to reduce or prevent exposure to ETS. Such strategies may include increasing ventilation, eliminating the source of ETS by reducing active smoking, and limiting levels of exposure through indoor smoking restrictions. Prevention efforts should target highly exposed individuals (such as children and nonsmoking spouses of smokers, and workers in smoke-filled workplaces) and certain populations that are especially vulnerable to the risks of ETS.

**INTRODUCTION**

Medical reports linking active smoking and lung cancer began to appear in the 1930s. It was the landmark 1964 Surgeon General’s report on cigarette smoking and health, however, that officially recognized active smoking as the primary cause of lung cancer and a major cause of disease and death in smokers.¹

In the decades since 1964 additional research has addressed the impact of tobacco smoke on nonsmokers who inhale environmental tobacco smoke (ETS) at home, at work, and in other indoor settings.

In 1986 the National Research Council and the U.S. Surgeon General’s Office issued assessments of the health effects of exposure to ETS.²,³ These two reports concluded independently that ETS increases the risk of lung cancer in adult nonsmokers and that there is an increased frequency of respiratory symptoms and lower respiratory tract illnesses among children whose parents smoke. For other diseases, such as heart disease, there were too few studies available in 1986 for researchers to arrive at a conclusion.

Since 1986 the number of studies examining acute and chronic diseases in humans exposed to ETS has more than doubled, resulting in a larger database from which to assess the potential health effects of environmental tobacco smoke. A prominent and controversial 1992 review by the Environmental Protection Agency (EPA)⁴ combined the earlier conclusions of the Surgeon General and the National Research Council with more recent epidemiologic studies. That 1992 EPA study concluded that “ETS is a human lung carcinogen, responsible for approximately 3000 lung cancer deaths annually in U.S. nonsmokers.” Some scientists have challenged this purported association between ETS exposure and lung cancer (among other health outcomes), however. These scientists have argued that the low relative risks from ETS (i.e., the small apparent increases in risk for
exposed versus nonexposed persons) cannot truly be differentiated from no effect at all.

Another large group of epidemiological studies has examined the association between ETS exposure and heart disease. These findings also continue to be the subject of debate and controversy.

In addition to providing a thorough review of the available information on the potential health risks of ETS exposure,* this ACSH Special Report offers a description of the chemistry of ETS; a discussion of some of the ways ETS exposure is measured; and various estimates of how many people are regularly exposed to ETS in the United States. The report concludes with a discussion of future goals and a description of preventive or protective steps that we can take to deal with ETS. ACSH hopes that the information contained in this report will equip readers to make their own informed decisions about environmental tobacco smoke.

**Assessing Exposure to ETS**

**The Chemical Composition of ETS**

ETS is defined as tobacco smoke in the ambient atmosphere—in either indoor or outdoor air. ETS consists of two types of smoke: “side-stream” smoke (the smoke released from the smoldering end of a cigarette) and “mainstream” smoke (the smoke exhaled by a smoker after inhalation).

ETS is a complex mixture of over 4,000 chemicals, in the form of both gases and particulates, produced by the burning materials (tobacco and additives) of a cigarette. Many of the chemicals in ETS have no known health effects or are present in extremely low (and therefore likely harmless) concentrations.5 There are, however, quite a few known toxicants—chemical agents with the potential to cause harm—in ETS.

Among the toxicants in ETS are nitrogen oxides, ammonia, formaldehyde, carbon monoxide, methyl isocyanate, nicotine, and 43 compounds classified by the EPA as known or suspected human carcinogens—cancer-causing agents. These carcinogens include nitrosamines, benzene, benzo(a)pyrene, vinyl chloride, radionuclides, and arsenic. Furthermore, ETS is the only indoor source of some of these compounds, and particular-

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* Beyond its health effects, ETS may be annoying, irritating, and aesthetically unacceptable to many people. This paper addresses only the health effects of ETS, however.
EnvironmenTal TobacCO SMOke:

ly of the group of chemicals known as “tobacco-specific nitrosamines.”

Sidestream smoke and mainstream smoke contain essentially the same toxic agents and carcinogens. Some of these components are dilute in sidestream smoke relative to their concentrations in the mainstream smoke that smokers inhale; but others—among them nicotine, carbon monoxide, ammonia, benzene, and the tobacco-specific nitrosamines—are actually present in higher concentrations in sidestream smoke than in mainstream smoke. The concentrations of these toxicants are higher because of sidestream smoke’s lower combustion temperature and the resultant incomplete combustion of the cigarette materials, and because of the absence of any filtering of the sidestream smoke by the cigarette’s filter.3,5,6

Studies of indoor air quality in commercial and public buildings show that particulate levels in areas where smoking is permitted are considerably higher than in nonsmoking areas.7 A single smoker in a home can double the amount of particulates inhaled by nonsmoking members of the household.8 Smoke particulates also have been shown to alter the environmental and physiological behavior of radon gas (a lung carcinogen), as radon may be suspended on the fine particulates of smoke.9,10 The interaction between radon and tobacco smoke is complex, however, and the health implications remain unclear.

Cigarette smoke is also a significant source of exposure to volatile compounds such as benzene (a human carcinogen).11 Nonsmokers exposed to ETS at work have been shown to have significantly elevated levels of volatile aromatic compounds in their breath.12 Annual average concentrations of benzene and styrene have been shown to be increased by approximately 50 percent in the homes of smokers, and ETS thus may present an important source of exposure to these compounds among nonsmokers living with smokers.12

Measuring Exposure and Absorption of ETS Chemicals

Absorption of tobacco-specific chemicals (e.g., nicotine) following ETS exposure has been documented in a number of studies done among the general population. Nicotine is converted in the body to cotinine; cotinine therefore can be used as an indirect measure of a person’s recent exposure to tobacco smoke. Levels of nicotine in hair and levels of cotinine in such body fluids as saliva and urine have been shown to increase with increasing environmental nicotine levels and with self-reported ETS exposure.13,14 Trace amounts of nicotine are also found in certain vegetables, among them eggplant and green peppers, but a recent large population survey showed that consumption of these nontobacco sources of nicotine was not associated with measurable cotinine levels.15
Cotinine has been detected in 50 percent to 88 percent of nonsmokers\textsuperscript{16} exposed to ETS. It has also been found in the body fluids of infants of smoking parents.\textsuperscript{14} Nonsmokers living with smokers have a median concentration of about 25 nanograms per milliliter (ng/mL) of urinary cotinine;\textsuperscript{17} this level represents about two to three times the level found in nonsmokers living with other nonsmokers.

As would be expected, levels of nicotine exposure resulting from ETS exposure are much lower than levels of nicotine exposure resulting from active smoking. (Typically, serum or urinary cotinine levels are 100 times lower in ETS–exposed nonsmokers than in active smokers.)\textsuperscript{18} Measured exposures to respirable suspended particulates are also higher for nonsmokers reporting exposure to ETS than they are for nonsmokers without reported ETS exposure.\textsuperscript{19}

Numerous epidemiological studies have been conducted in nonsmokers exposed to ETS at home and/or at work (see “Health Effects of ETS,” page 11). In these studies an assortment of methods have been used to assess the ETS exposure levels of study subjects, many of whom have been the nonsmoking spouses of smokers.

When study subjects have been grouped by exposure level, assessment methods have typically relied on the subjects’ recall of the number of cigarettes per day, the number of packs per year, or the total number of years that their spouses smoked. Such methods have some limitations. Some examples follow.

The recall by people diagnosed with lung cancer of past ETS exposure may be influenced by the presence of a disease thought to be potentially related to that exposure. Investigators have very few options, however, when they are trying to assess, in a large number of study subjects, the possible associations between a long-term, low-level environmental exposure and a disease like cancer. This is because there is usually a long period between exposure (in this case, to ETS) and clinical evidence of disease (in this case, a diagnosis of lung cancer).

Nevertheless, the observation of large groups of people with relatively high levels of ETS exposure (nonsmoking spouses exposed in the home for most of their adult lives) provides useful information, both about potential dose–response relationships and about the effects that occur with varying levels of ETS exposure.

When a dose–response relationship can be clearly characterized, it is sometimes possible to determine the doses below which the disease of interest to the researchers would not be expected to occur—in other words, a threshold. A threshold has not been identified for either active smoking or exposure to ETS, however.

Another approach to evaluating the dose–response nature of the potential association between ETS exposure and a specific disease involves estimating passive smokers’ risk based on the risk of the disease
observed in active smokers. This can be done by using “cigarette equivalents.” Some scientists (and the tobacco industry) have argued that non-smokers heavily exposed to ETS breathe in the equivalent of less than one cigarette per day. Such estimates are typically based on nonsmokers’ nicotine and/or cotinine levels, which are typically 1 percent of those found in active smokers.20

Some of the more toxic and carcinogenic compounds in ETS are present in higher concentrations in sidestream smoke than in mainstream smoke, however. The compound 4-aminobiphenyl, for example, is present in passive smokers at levels that are approximately 14 percent of those found in active smokers.20 Thus, if cigarette-equivalent calculations and threshold estimations are based on these other, more toxic and carcinogenic, compounds, ETS exposure may yield a dose equivalent of several cigarettes per day.21

Who Is at Risk of Significant Exposure to ETS?

About 25 percent of the adult population of the United States smokes cigarettes.22 ETS is widely present in homes, in automobiles, in workplaces, and in enclosed public places and is just as widely absorbed by nonsmokers in the general population. All of this has been well documented by air sampling and by the body measurement of such biological markers as nicotine and cotinine. (A biological marker, or “biomarker,” is a physiological or chemical change in the body that signals exposure to a particular agent—in this case, to ETS.)

Field studies, controlled experiments, and mathematical models have all shown that, under typical conditions of smoking and ventilation, ETS diffuses rapidly throughout buildings and homes; that it persists for long periods after active smoking has ended; and that it represents one of the major sources of indoor air pollution in buildings in which smoking occurs.4

Most people spend approximately 80 percent to 90 percent of their time indoors,23 and exposure to ETS in indoor air is common. Some evidence suggests that self-reports of ETS exposure may actually underestimate true exposure levels, as determined through more objective measurements.24 Some individuals reporting no exposure to ETS show low concentrations of cotinine in their urine, indicating that they have, indeed, been exposed to ETS.17

Several recent large population surveys have illustrated that a sizable proportion of the U.S. population is regularly exposed to ETS. A recent study by the U.S. National Center for Health Statistics (NCHS) found that among adults, 20 percent of never-smokers and 23 percent of former smokers were regularly exposed to ETS at home and/or at work.25 In another very large NCHS survey it was determined that 43 percent of U.S.
children aged 2 months to 11 years lived in homes with at least one smoker, and that 37 percent of adult nonsmokers either lived in homes with at least one smoker or reported ETS exposure at work. An analysis done by the U.S. Centers for Disease Control (CDC) reported that of households that included both smokers and children under 18 years of age, almost 90 percent allowed smoking in all or some places inside the home. Overall, the CDC report revealed, approximately 33 percent of adult smokers had children living in their homes. These prevalence data indicate that at least 15 million children are exposed to ETS in the home on a regular basis.

For adult nonsmokers, the relative contribution of work versus home environments to ETS exposure has not been well quantified, and there is probably extremely high variation among individuals. In addition, social settings outside the workplace (e.g., restaurants, bowling alleys, bars) may contribute significantly to ETS exposure of patrons and, especially, employees in these establishments. As discussed in the next section, findings from epidemiological studies have suggested a direct relationship between the extent of ETS exposure and potential health effects among nonsmokers who live and/or work with smokers.

HEALTH EFFECTS OF ETS

There is an extensive database of published literature on the health effects of ETS. There is also substantial agreement in the scientific and public health community that ETS is related to acute (short-term) irritant effects and respiratory effects in children. The role of ETS in the development of longer term diseases such as cancer and cardiovascular disease is more controversial, however.

Health Effects Reported in Children Regularly Exposed to ETS

The finding that ETS causes or aggravates respiratory illnesses in children is virtually undisputed. There is strong epidemiological evidence that children exposed to ETS are at increased risk for lower respiratory tract infections, middle-ear disease, and worsening of asthma symptoms. In addition, recent data suggest that ETS may be a risk factor for Sudden Infant Death Syndrome, or SIDS (see page 13).
Lower Respiratory Tract Infections (Pneumonia and Bronchitis)

For children exposed to ETS at home, a clearly significant (likely not due to chance) dose-related association has been demonstrated between ETS exposure and the frequency of acute lower respiratory tract infections. Increases in the severity and duration of these infections have also been linked with children’s ETS exposure. ETS–exposed children require hospitalization for respiratory infections four times more often than do children not regularly exposed to ETS.

Researchers Strachan and Cook pooled the results from 38 separate studies in a meta-analysis that examined ETS exposures in the home and the incidence of acute lower respiratory tract illnesses in children. Strachan and Cook found that children exposed to ETS at home were 57 percent more likely than nonexposed children to contract lower respiratory tract illnesses during the first three years of life. Respiratory symptoms that were also found to be significantly increased included wheezing, chronic cough, chronic phlegm, and breathlessness.

Middle-Ear Disease

The exposure of children to ETS from parental smoking has been causally associated with an increased frequency and duration of episodes of middle-ear effusion (fluid inside the eardrum). Various studies have attributed a range of from 8 percent to 33 percent of cases of middle-ear effusion to ETS. These studies have controlled for day-care attendance and for other known risk factors for middle-ear disease.

Middle-ear effusion is the most frequent cause of deafness in children. It is also the most common reason for surgical hospitalization of young children. ETS exposure has been linked specifically to hospital admission for tympanostomy (placement of drainage tube in the ear).

In a meta-analysis for ETS exposure and various middle-ear disease outcomes, researchers Strachan and Cook reported a 38 percent increased risk for recurrent otitis media (recurrent middle-ear infections) and a 48 percent increased risk for middle-ear effusion.

Exacerbation of Asthma

There is evidence that exposure to ETS can exacerbate preexisting asthma in children. Increased severity of asthma symptoms or impaired recovery following acute episodes that required hospitalization have been reported in children of parents who smoked. In their 1992 report the EPA estimated that ETS exposure exacerbated asthmatic symptoms in approximately 20 percent of this country’s 2 to 5 million asthmatic children.

Asthmatic children appear to be more susceptible than other people to the pulmonary effects—effects related to the lungs—of ETS. When ETS exposure and nicotine absorption were compared among asthmatic and nonasthmatic children, it was found that even when ETS exposures
were reduced for the asthmatic children, those children still showed higher systemic exposures—that is, higher systemic nicotine levels. This was probably due to the asthmatic children’s decreased pulmonary clearance capabilities.\textsuperscript{45}

When parents of asthmatic children stop smoking in the presence of their children, the severity of the children’s symptoms and other measures of respiratory function show clear improvements.\textsuperscript{46}

\textit{Other Respiratory Effects Reported in Children Exposed to ETS}

Recent reports have linked children’s ETS exposure to an increased incidence of airway complications during general anesthesia.\textsuperscript{47} Among children less than 18 years old, ETS exposure—specifically, having a mother who smokes—has also been shown to be a strong independent risk factor for meningococcal meningitis, a dangerous spinal infection.\textsuperscript{48}

\textit{SIDS and Other Effects in Newborn Infants}

Sudden Infant Death Syndrome (SIDS) is the most common cause of death in infants aged 1 month to 1 year. The available evidence suggests that infants of smoking mothers are at increased risk of SIDS independent of other known risk factors, including low birthweight and gestational age.\textsuperscript{49–52} The published literature does not permit a definitive conclusion, however, as to whether the increased risk is related to exposure to tobacco smoke during pregnancy (\textit{in utero} exposure), following birth (postnatal exposure), or both. Thus, at present there is not enough direct evidence to support postnatal exposure to ETS alone as a risk factor for SIDS.

\textit{Health Effects Reported in Adults Regularly Exposed to ETS}

Exposure to ETS has been shown to aggravate symptoms of preexisting health conditions such as asthma in nonsmoking adults.\textsuperscript{53,54} ETS has also been shown to have subtle but significant effects on nonsmoking adults’ respiratory health, contributing to such symptoms as increased cough, phlegm production, post-nasal drip, chest discomfort, and reduced lung function.\textsuperscript{3,4,55} As with asthmatic children, when exposure to ETS is reduced or eliminated, adults show an improvement in related symptoms.\textsuperscript{53}

In a large cross-sectional population survey, never-smokers exposed to ETS experienced a 27-percent to 43-percent increase in acute adverse health effects—defined as more than one day of restricted activity, bed confinement, or work absence.\textsuperscript{25} Additionally, ETS exposure has been linked to an acceleration of age-related hearing loss.\textsuperscript{56}
A number of epidemiological studies have been conducted for two chronic health endpoints, lung cancer and cardiovascular disease. The findings of the majority of these studies have indicated a possible weak association with exposure to ETS.

While reports have suggested that several other chronic illnesses—specifically, breast cancer, cervical cancer, and leukemia—may be linked to ETS, these associations have not been established scientifically. Therefore, these suggested associations are not discussed in this report.

Lung Cancer

The risk of lung cancer in nonsmokers exposed to ETS has been investigated extensively in over 30 published epidemiological studies. In so-called “cohort studies” the subjects were primarily nonsmoking women married to smokers. These women were followed over time and their lung cancer rates compared with rates in control groups made up of nonsmoking women married to nonsmokers. In so-called “case-control studies” individuals with existing lung cancer were compared with people without lung cancer with respect to the extent and duration of their exposures to ETS. Other studies examined men and women exposed to ETS at work and/or at home.

Twenty-seven out of 30 studies reported a small but positive association between ETS exposure and lung cancer, showing modestly elevated excess risks—typically, 1.2 to 1.3 times the lung cancer rates in the controls—in the ETS–exposed subjects. These numbers translate into an approximate 20- to 30-percent increase in lung cancer risk for nonsmokers exposed to ETS. Only seven of the studies resulted in a statistically significant elevation in risk, however. (The term “statistically significant” indicates that the association between ETS and lung cancer in these studies was likely not a result of chance.) The relationship between estimated extent and duration of ETS exposure (the dose) and lung cancer (the response) was evaluated in 26 of the studies. Although no consistent definition of dose level was used, there was clear evidence of a dose–response trend in 15 of the 26 studies.

An assessment of whether there is, indeed, a causal association between ETS exposure and lung cancer should be based on the available data and on consideration of the following elements:

THE STRENGTH OF THE ASSOCIATION. The strength of the positive association between ETS exposure and lung cancer in nonsmokers is weak. In other words, when the increased risk of lung cancer among nonsmokers exposed to ETS is compared with the risk among nonsmokers not exposed to ETS, the difference in lung cancer risk between these two groups—the so-called “relative risk”—is consistently small.

A relative risk equal to 1 suggests no association between exposure
and outcome. Relative risks for lung cancer typically fall between 1.2 and 1.3. In only 7 of the 30 reviewed lung cancer/ETS studies was the increased risk for those exposed to ETS statistically significantly different from the apparent risk for the controls. In epidemiological studies, finding statistically significant differences between groups — between those exposed and those not exposed, for example — is critical to establishing an association. But finding statistically significant differences is affected by numerous study factors, among them the size of the group being studied (the “sample size”) and the size of the effect being tested (small effects are more difficult to detect than large effects).

**THE CONSISTENCY OF THE RESPONSE.** Although many of the study findings did not attain statistical significance, the clear majority of the epidemiological studies of ETS and lung cancer—studies conducted in different countries by different researchers using a variety of study designs and sample sizes—have shown a positive association, however slight, between ETS exposure and increased risk of lung cancer.

**DOSE–RESPONSE EFFECTS.** When establishing causation, it is important to demonstrate that changes in the amount of exposure to (or changes in the dose of) a particular agent lead to predictable changes in effect—the effect, in this case, being risk of lung cancer. Most (15 out of 26) of the ETS/lung cancer studies in which study subjects were grouped by exposure level (grouped, that is, by such factors as the number of cigarettes smoked per day by the spouse or the number of years the spouse had smoked in the home) have found that risk increases as exposure level increases. Even the risks associated with the greatest levels of exposure to ETS are very small, however, when compared to the risks associated with active smoking.

**BIOLOGICAL PLAUSIBILITY.** It has been convincingly demonstrated that ETS is absorbed by the lungs of nonsmokers and distributed throughout the body. The presence of the same carcinogens in ETS as are found in mainstream smoke and the established causal relationship between active smoking and lung cancer make it plausible that ETS may also be a lung carcinogen. Evidence from studies of ETS or its components conducted in experimental animals and evidence from genetic tests (tests conducted with cultured cells and bacteria) also support the biological plausibility of such an association.88,89

**EFFECT OF CONFOUNDING FACTORS.** With such a small increase in relative risk being found in each study, there exists the possibility that risk factors other than ETS exposure are more common in the exposed groups than in the controls and that these other factors—known as “confounders”—are therefore contributing to the apparent increase in risk of disease. When
adjusted for critical confounding variables such as lifestyle and dietary factors, however, the results across different study populations in different countries change only slightly. This argues against confounding as the sole explanation for the small observed increase in relative risk among ETS–exposed nonsmokers.

**SELECTION BIAS DUE TO MISCLASSIFICATION OF SMOKING STATUS.** For a variety of reasons, a group of people being studied may contain misclassified individuals—people who actually do not belong in the category to which they have been assigned. If not accounted for, the resulting “selection bias” can invalidate the findings of a study. For example, if individual current and ex-smokers (both groups with a greater risk for developing lung cancer than never-smokers) were to be misclassified into a group of never-smokers in a study, this misclassification would artificially increase the apparent cancer risk due to ETS exposure among the never-smokers.

**ACCURACY AND RELIABILITY OF EXPOSURE ASSESSMENT METHODS.** The most important limitation in all studies reporting lung cancer risks among nonsmokers is the lack of quantitative ETS exposure data. Very little detail is available concerning measurements of the intensity and duration of nonsmokers’exposures at home, at work, and in social settings.

Most of the published studies have defined nonsmokers’exposures to ETS on the basis of their living with or being married to smokers. Typically, this information has been ascertained through interviews or written questionnaires. Because most cancers have a long latency period between exposure and development of disease, the development of a more accurate and precise measure of past exposure events is neither likely nor feasible. The best efforts should be made, however, to identify any outside-the-home ETS exposures among a study’s control subjects and to determine the extent to which ETS–exposed subjects have attempted to minimize exposure to their spouses’cigarette smoke.

*One overall conclusion that can be drawn from consideration of the above elements is that exposure to ETS represents a weak risk factor for lung cancer among nonsmokers.*

**Coronary Heart Disease**

Organizations and government agencies such as the American Heart Association, the American Lung Association, the American College of Cardiology, and the U.S. Occupational Safety and Health Administration (OSHA) have all declared exposure to ETS to be a risk factor for heart disease.

In 1986, when reports on environmental tobacco smoke were issued by the National Research Council and the U.S. Surgeon General’s Office, only a few studies on ETS and cardiovascular disease were available in the
published literature. Today, however, the range of published studies on this issue, while not as extensive as the literature concerning the association of ETS with lung cancer, shows that the body of evidence has grown dramatically in the intervening years. As with the association between active smoking and lung cancer, the causal association between active smoking and heart disease is clearly established and has been generally accepted in the scientific community for many years.\textsuperscript{90}

To date 18 epidemiological studies have been published examining the association of ETS exposure with coronary heart disease (CHD). These include nine case-control studies and nine prospective cohort studies.\textsuperscript{76,91–108}

Several of the cohort studies involved the monitoring of very large study populations over periods of many years, in efforts to examine many possible causative factors for heart disease. The study populations lived in the United States, in Europe, in Japan, and elsewhere in Asia.

These studies had varied designs. Some evaluated fatal and/or nonfatal cardiac events. Others controlled or adjusted for various demographic factors such as age, sex, or socioeconomic status and/or established coronary risk factors such as dietary factors, lifestyle, or a family history of excess cholesterol in the blood. Still others made separate studies of non-smokers currently exposed at home or at work and nonsmokers whose histories of ETS exposure were only in the past (in other words, nonsmokers in whom the potential exposure-related cardiovascular effects could have been reversed or repaired after their ETS exposure was either reduced or stopped).

These studies found estimated relative risks for nonsmokers living with current or former smokers (as compared with nonsmokers living with nonsmokers) that ranged from 0.92 to 2.70. Ten of the 18 studies reported a significantly increased risk of cardiovascular disease for nonsmokers living with current or former smokers.

In contrast with the data from many of the lung cancer studies, the data for cardiovascular disease in nonsmokers exposed to ETS do not show as consistent a pattern of dose–response trends. Only four of the 13 studies that evaluated dose–response relationships reported significant trends for increasing cardiovascular disease with increasing ETS exposure.

A number of scientists have pooled the data from the various available coronary heart disease studies. These meta-analyses have consistently found excess risk in the range of 1.2 to 1.5—a level of risk 20 percent to 50 percent higher than that found in the studies’ control populations.\textsuperscript{109–113}

As with the association between ETS and lung cancer, the association between ETS and cardiovascular disease appears to be positive but weak.

The effects of environmental tobacco smoke on the cardiovascular system could arise from many of the constituent chemicals of ETS—among them carbon monoxide, nicotine, and polycyclic aromatic hydrocarbons. Cardiovascular effects could also occur as a result of interactions
between two or more of these constituent chemicals.

Many factors have been identified as important in the development of cardiac events. Some of these factors are related to short-term effects, such as the increased likelihood of blood clots; others are related to long-term changes, such as damage to blood-vessel walls. A number of epidemiological studies that have evaluated the overall risk of heart disease among ETS–exposed persons have also assessed certain of these intermediary health effects.

Some of the intermediate health effects that have been associated with exposure to ETS are described in the paragraphs that follow.

**PLATELET ACTIVATION AND AGGREGATION.** Platelets are components of blood and are necessary for normal clotting. Exposure to ETS activates the platelets and increases what’s known as “platelet aggregation”—clumping of the platelets. Such clumping increases the likelihood of excessive blood clotting and damage to the lining of the coronary arteries—both factors that contribute to heart attacks. Several epidemiological studies have demonstrated this intermediate effect among nonsmokers exposed to ETS.114–117

**CHANGES IN CHOLESTEROL LEVELS.** Exposure to ETS among adolescents whose parents smoke and among adults working in places where smoking is permitted has been associated with changes in cholesterol levels. In particular, ETS–exposed persons show lower levels of high density lipoproteins (HDL), otherwise known as “good” cholesterol. Lower HDL levels increase the risk of atherosclerosis (clogging of the arteries with fatty deposits), which can be a precursor to heart attacks.21

**THICKENING OF CAROTID ARTERY WALLS.** There exist data showing that people exposed to ETS have a significant thickening of the walls of the carotid artery—the main artery leading to the brain. This thickening results in an increased resistance to blood flow—a condition that can be a precursor to stroke.118

**INCREASED PLASMA FIBRINOGEN LEVELS.** Several epidemiological studies have demonstrated that ETS exposure is associated with increased levels of plasma fibrinogen (a protein in the blood that is essential for clotting). Such increases contribute to blood clotting and are associated with higher rates of cardiovascular disease.92,119

**DECREASED OXYGEN TO THE HEART.** Healthy young adults exposed to ETS while exercising experience higher resting heart rates and higher blood carboxyhemoglobin levels. Both of these work to decrease oxygen availability to the tissues and so result in shorter time to exhaustion.120,121
The principal limitations to the ETS/lung cancer studies (see page 14) are also applicable to the studies of ETS exposure and heart disease. This is especially true of the difficulty (or inability) of directly assessing exposure to ETS, which is typically defined as spousal smoking. Despite these limitations, the majority of studies have found a positive correlation between ETS exposure and heart disease.

Another limitation of the heart disease studies has been the difficulty in controlling for all known cardiovascular disease risk factors, among them high blood pressure, elevated serum cholesterol, obesity, stress, and assorted psychosocial factors. Critics often fault studies that fail to account for these confounding variables. Nevertheless, increased risks of comparable magnitude have been observed in studies from different countries. In addition, these epidemiological data are supported by a wealth of pathological evidence—blood-vessel damage, increased blood clotting, and decreases in oxygen supply to the heart—all of which has been linked to ETS exposure. It should be remembered, too, that the increased risks observed in these studies have been small (see pages 14 and 17).

Because of the wide-ranging public health and social policy implications of finding a causal association between ETS and various diseases, interested parties (i.e., pro- and anti-tobacco organizations and individuals) have engaged in protracted and sometimes bitter struggles to promote their particular viewpoints.

The American Cancer Society, the American Lung Association, the American Heart Association, the American Medical Association, the World Health Organization, an assortment of government agencies (the Environmental Protection Agency, the Surgeon General of the United States, the National Institute of Occupational Safety and Health) and many individual scientists have all concluded that ETS exposure is associated with significant health risks.

But some researchers, public-policy organizations, journalists, and the cigarette industry itself have questioned the links between ETS and disease. Another vocal group of critics has been scientists who disagree with the methodologies—especially the quantitative estimations of ETS-related deaths from lung cancer and cardiovascular disease—used in ETS risk assessments.
The tobacco industry and several researchers have claimed that publication bias exists against negative studies of the health effects of ETS. Were all these negative studies to be published, these critics say, they would change the weight of evidence regarding the ETS/health effects association.

A number of studies do exist that show no association between ETS and health effects. More often than not, however, these are studies published as review articles in non–peer-reviewed publications (meeting symposia, for example), and they frequently lack statistical analyses of results.

A 1994 review of published ETS literature by Bero and colleagues indicated that the majority (80 percent) of original journal articles on the subject of ETS and health effects found a positive association, as compared with 51 percent of symposium articles. This finding led Bero and associates to the conclusion that cigarette industry–sponsored ETS symposia publish more negative findings than do journals. Only reanalysis of the raw data from existing studies and additional objective research will determine which of these sources has published the more reliable findings.

In 1986 the International Agency for Research on Cancer (IARC) designated tobacco products and tobacco smoke (both mainstream smoke and ETS) as human carcinogens. Similarly, in 1992 the Environmental Protection Agency classified ETS as a “Class A” carcinogen—a known human carcinogen.

The EPA’s 1992 report at once provided legislators around the country with a critical tool for establishing smoking restrictions. In July 1998, however, North Carolina–based Federal Judge William L. Osteen overturned the EPA’s classification of ETS as a Group A carcinogen. Judge Osteen ruled, first, that the EPA had failed to follow the proper objective procedure for carcinogen classification; and, second, that the data used by the EPA to link ETS and cancer had failed to achieve sufficient statistical significance.

Despite this setback, the labeling of secondhand smoke as an “official” carcinogen is still being considered. In December 1998 the subcommittee of the National Toxicology Program’s Board of Scientific Counselors recommended that ETS be labeled a carcinogen on the federal government’s official list of carcinogenic agents.
The Tobacco Industry Responds

Cigarette companies have waged an aggressive campaign to refute, or at least to minimize, the evidence showing an association between ETS and adverse health effects. The companies’ strategies regarding ETS have somewhat resembled their strategies regarding active smoking: They have conducted private, internal research (some of which has supported the conclusion that ETS has adverse health effects) while publicly denying that any risks have been supported by research.

In 1995 Dr. Stanton Glantz and colleagues reported that in the mid-1970s the cigarette industry had conducted its own research relating to the effects of ETS. Glantz and his team noted that researchers working for the tobacco industry had determined that sidestream smoke produced respiratory irritation, that it contained toxic substances, and that it was carcinogenic to laboratory animals.

The cigarette industry has relied primarily on two approaches in its attempts to “defend” ETS: It has presented the issue as one of individual rights versus overzealous government intervention, and it has questioned the scientific validity of the evidence against ETS.

The industry’s criticisms of the scientific studies of the health effects of ETS, while directed primarily against the lung cancer studies, may be applied to any of the disease outcomes associated with ETS exposure. To a great extent these criticisms are common to environmental epidemiology analyses in general. They are especially pertinent, however, to studies dealing with exposure scenarios similar to those involving ETS: widespread, low-dose exposures.

There is a distinct advantage to using the findings from such studies to estimate risk in a relatively direct manner rather than to extrapolate, either from high-dose human exposures or from findings in experimental animals. Unfortunately, designing epidemiological studies suitable for agents such as ETS is a demanding task. (Concerns specific to the assessment of risks from exposure to ETS are discussed on pages 8–10, in the section on Measuring Exposure and Absorption of ETS Chemicals.)

The tobacco industry has been successful in its efforts to focus the ETS debate exclusively on lung cancer. To counter the growing trend toward indoor smoking restrictions and to allay the public’s fears, the cigarette companies have argued that ETS does not present a “meaningful” lung cancer risk—and therefore does not present a threat to public health.

While this argument may be justified by the apparently weak association between ETS and lung cancer, the argument ignores other, more firmly established health effects connected with ETS (see “Health Effects of ETS,” page 11). Lung cancer should not be the only ETS–related health outcome taken into account when public health policies are being established. At the same time, however, mere aesthetic concerns (such as the simple annoyance of ETS) should not be allowed to replace real public health concerns.
The impact of ETS might best be judged in terms of the degree to which ETS affects the general population. Large percentages of adults and children are regularly exposed to ETS, at home and elsewhere. Because of this, even small increases in the risks of various acute and chronic diseases, if truly associated with ETS exposure, represent significant public health impacts. The health risks associated with ETS are more certain and more prevalent for acute—that is, for irritant—effects. But the data also show that, based on ETS’s reported relationships with lung cancer and heart disease, ETS exposure represents a potentially significant preventable cause of death and disease.

ACSH’s review and analysis of the relevant data yield the following conclusions with regard to potential health effects in persons exposed to ETS:

- Irritation of the eyes, nose, and respiratory tract is the most common and firmly established adverse health effect associated with exposure to ETS.
- Exposed infants and children, in particular, are at increased risk of respiratory infections, middle-ear effusion, and the exacerbation of asthma and other respiratory symptoms.
- Exposed adults are at increased risk for respiratory ailments; ETS may aggravate the symptoms of preexisting asthma and emphysema.
- Extensive epidemiological evidence indicates that ETS exposure is a weak risk factor in the development of lung cancer in nonsmokers regularly exposed to ETS in the workplace and/or at home.
- Epidemiological evidence also suggests that ETS is a weak risk factor for heart disease in nonsmoking spouses of smokers and in nonsmokers regularly exposed to ETS in the workplace and/or at home.

Preventive Measures

Research has indicated that the total removal of tobacco smoke by ventilation is both technically and economically impractical. Because of their small size, ETS particulates disperse rapidly throughout a room. As a result, the simple separation of smokers and nonsmokers within the same airspace, or even the placement of smokers and nonsmokers in separate rooms with a common ventilation system, may reduce but will not eliminate exposure to ETS. This is especially true for workplace settings in which exposure may be prolonged.
ETS can be completely removed from indoor air only by completely removing the source of the ETS. Concentrations of tobacco smoke need not be reduced to zero, however, to prevent the vast majority of acute effects in children and adults or to reduce risks for lung cancer and heart disease.

More data are needed on both the extent and the determinants of ETS exposure. But many factors influencing exposure to ETS, both at home and at work, are already known to us. These include the size of the space involved; the number of smokers in the space; the type and rate of ventilation; and the amount of time smokers and nonsmokers spend in the space.

Three major control measures exist for limiting exposure to ETS and thereby limiting associated adverse health effects:

- **Increasing Indoor Ventilation Rates to Fairly High Levels.** As explained above, this is not always a practicable option.
- **Eliminating the Source of the ETS.** Reducing smoking rates among both adults and young people remains a significant public health challenge. Such smoking-cessation and prevention efforts as restrictions on advertising, increased taxes on tobacco products, and stricter enforcement of age restrictions in retail establishments can all be brought to bear. But health-education materials designed to bring about a reduction in active smoking should also include information on the potential hazards of ETS and should focus particularly on the risks ETS poses to vulnerable populations such as infants and children.
- **Limiting Levels of ETS Exposure by Placing Regulatory Restrictions on Smoking in Schools, in Workplaces, and in Public Spaces.** The public health impact of such policies will necessarily vary, depending on the type of ETS exposure involved. Efforts to reduce exposures to ETS at home and in the workplace are more effective at reducing risk to the general public than are attempts to eliminate such sporadic or short-term exposures as occur outdoors and in public places. It should be remembered, however, that while ETS exposure in a public place such as a restaurant or bar may be “sporadic” to the restaurant’s patrons, it is ongoing for the employees.

Workplace smoking policies have had a major effect on airborne nicotine concentrations. Such concentrations have fallen from a median of 8.6 micrograms per cubic meter (µg/m³) in open offices at worksites that allow smoking to 1.3 µg/m³ at worksites that restrict smoking. Levels have fallen to 0.3 µg/m³ in worksites that have banned smoking outright.

Nonoffice work spaces have similarly been affected: Today, median nicotine concentrations in nonoffice work spaces are 2.3 µg/m³ at worksites that allow smoking, 0.7 µg/m³ at worksites that restrict smoking, and
0.2 µg/m³ at worksites where smoking is banned.\textsuperscript{127}

With more precise data on the distribution of ETS exposure and on ETS exposure levels in the general population, more accurate estimations of the magnitude of the risks of ETS exposure may be possible. In the absence of further data, the current body of scientific evidence supports measures to reduce or prevent exposure to ETS among people who are highly exposed and among those who are particularly vulnerable to the adverse health effects associated with such exposure.

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