

Economic Effects of Environmental Tobacco Smoke  
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*Abstract: Environmental tobacco smoke (ETS) has been shown to be associated with increases in rates of cancer, morbid conditions of the respiratory and cardiovascular system and increases in the rates of spontaneous abortion and perinatal mortality. The authors combine exposure data, data on increased morbidity and medical and indirect cost data, all derived from published reports, to estimate the total economic cost of ETS exposure in the United States. Total annual costs for conditions with well-documented increases in morbidity, excluding economic losses related to pregnancy and the newborn, are estimated at over \$5 billion in direct medical costs and over \$5 billion in indirect costs.*

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### **Executive Summary**

Cigarette smoking has long been identified as a major cause of preventable death and has been factored into underwriting decisions and individual risk ratings. The 2004 Surgeon General's Report (U.S. Dept. of Health and Human Services, 2004) reiterates that over

400,000 Americans die each year as a result of cigarette smoking and that, on average, a smoker loses about 12-13 years of life expectancy.

In this paper the authors combine published data on mortality and morbidity associated with exposure to secondhand smoke with published estimates of medical costs for the related conditions and standard estimates of economic value to derive estimates of the medical and other costs associated with exposure to secondhand smoke. We performed a literature review of the effects of ETS on mortality and morbidity, and on the basis of the available data, we calculated quantitative estimates of total ETS-related excess morbidity and mortality in the U. S. population. As documented in Appendix I, chronic exposure to secondhand smoke has been established as a cause for many of the same diseases caused by active smoking. While the number of deaths caused by chronic exposure to secondhand smoke is substantially less than the number caused by active smoking, the public health concern is elevated because secondhand--smoke deaths are occurring among individuals who have decided not to smoke, and thus their increased risk for disease and death is involuntary. We have also identified areas for consideration by insurance companies that might wish to evaluate the feasibility of using exposure to ETS as an underwriting criterion.

In terms of relative harm caused by active smoking versus chronic exposure to secondhand smoke, there are not any clearly agreed-to metrics, however most scientists would agree that the risk of death from chronic exposure to secondhand smoke is likely an order of magnitude lower than that of active smoking. The Centers for Disease Control and Prevention (CDC) estimates 440,000 active smokers (out of 50 million) die per year compared to around 50,000 passive smokers (out of 150 million). Thus, while deaths from passive smoking are tragic, real and preventable, their actuarial impact is less (possibly by an order of magnitude) compared to deaths of active smokers.

## **Introduction**

Exposure of nonsmokers to ETS is a source of widespread excess morbidity and mortality, imposing significant costs on nonsmokers and society as a whole. Exposure to ETS is defined as the exposure of a nonsmoker to the combustion products of cigarettes and other tobacco products. Typically, former smokers are excluded from the group of nonsmokers for which the effects of exposure or absence of exposure to ETS are compared. Definitions in the literature have slight variations, as presented in detail in Appendix 4, but the different definitions are consistent enough to permit aggregation of the results of various studies of mortality and morbidity. A special situation is the case of a fetus of a smoking mother. The literature typically classifies the effects of smoking on the fetus of a smoking mother as an effect of smoking, rather than as exposure to ETS. While the effects of ETS are subtle in comparison to active smoking, the number of people exposed is so large that the costs are substantial. A major conclusion of this paper is that in the United States, the annual costs of excess medical care, mortality and morbidity caused by ETS exceed \$10 billion.

Deleterious effects of smoking have been recognized and well-documented for over forty years (U.S. Dept. of Health, Education and Welfare, 1964). It is estimated that over 400,000 Americans die each year from the effects of smoking (Centers for Disease Control and Prevention, 1993). Exposure to ETS could be anticipated to have similar effects at a lower level of frequency and severity, and published literature now gives overwhelming evidence for these effects. In the many thousands of published articles on the effects of tobacco smoke relatively little attention has been devoted to quantification of the economic consequences of the increases in morbidity and mortality related to ETS. This paper is an analysis of these economic effects.

Extensive research has been carried out, and hundreds of papers have been published on the hazardous chemical components of ETS, their absorption by the human body and the hazardous nature of these chemicals. These studies have included controlled laboratory experiments as well as analyses of exposure to smoke in the environment. While these studies demonstrate the basis for a causal link between exposure to ETS and its harmful effects, they do not form the direct basis for our conclusions. Our analysis is based on the hundreds of studies that have documented a quantitative relationship between health problems and exposure to ETS by relating the degree or type of exposure to a measure of excess morbidity. This latter category of research is the basis of our estimates of the economic costs of exposure to ETS.

Determination of causation is a complex process that goes far beyond analysis of statistical data. (See, for example, Hitchcock, 2002.) Occurrence of a condition such as lung cancer may arise from a concurrence of several conditions, such as age, genetic predisposition and exposure to toxins such as ETS. For the purpose of measuring economic effects we sought to evaluate the difference in cost that would occur if exposure to ETS were eliminated. We believe that the types of controlled studies included in our analysis permit such an evaluation. It is not necessary, for this purpose, to identify an inventory of conditions bearing on the probability of occurrence of morbid conditions. The fact that the level of exposure of the population to ETS is controllable, for example through legal restriction on the locations where smoking is permitted, and that reduction in exposure, other things being equal, would lead to a decrease in morbidity and mortality, justify the isolation of ETS as an element of the causes of these morbid conditions. The publications that were the basis for our estimates of excess morbidity and mortality were published in peer-reviewed journals that insist on adequate control of confounding variables in the research that they publish. Consequently, the strong statistical association and the elimination of other potential causes results in the conclusion of a causal relationship between ETS exposure and certain diseases. For some diseases, there continues to be controversy about whether ETS actually causes the disease, or aggravates existing disease. Whether ETS is the primary cause or aggravates disease, we believe that the cost increases and economic effects estimated in this paper would not be incurred, but for exposure to ETS.

The source of environmental tobacco smoke can be separated into two components, mainstream smoke and side-stream smoke. Mainstream smoke is generated by the smoker drawing air through the cigarette. Side-stream smoke goes directly into the

surrounding air from the combustion of the cigarette. Mainstream and side-stream smoke have essentially the same chemical components, but in different relative concentrations (National Research Council, 1986). The main source of smoke exposure for smokers comes from mainstream smoke. In addition, most of the particulate components of mainstream smoke stay in the lungs of the smoker, so that the mainstream smoke component of environmental tobacco smoke is quite different from the smoke originally inhaled by the smoker. For these reasons the smoke to which nonsmokers are exposed through ETS is different from the smoke to which smokers are exposed. This difference would have to be considered in any application of dose-response theory to extrapolate from the effects of smoke on smokers to the effects of ETS on nonsmokers.

Our analyses are based on studies of the effects of actual ETS exposure, rather than on the basis of extrapolations of effects of smoking on active smokers. We have not attempted to quantify or apply a dose-response relationship to smoke exposure, nor have we considered issues related to differences between mainstream and side-stream smoke. The data that we have relied on are based on research on the actual effects of exposure to ETS, rather than extrapolations of results for active smokers. The technology to obtain quantitative measurement of exposure to tobacco smoke at low doses, using serum cotinine levels, has been developed fairly recently. Most of the available data on morbidity caused by ETS relate the effects to qualitative definitions of ETS exposure. Our analyses are based on qualitatively defined exposure of the population to ETS, rather than on cotinine levels. We have reviewed the literature on dose-response relationships, as discussed further in the body of this paper. The current state of knowledge does not permit dose-response relationships to be used to draw a quantitative relationship between the effects of active smoking and the effects of exposure to ETS.

According to the most authoritative estimate (Centers for Disease Control and Prevention, 2002) the annual medical and economic costs directly attributable to active smoking in the United States are \$150 billion. While the costs related to ETS, as determined in this paper, are a fraction of this, they still represent an enormous toll on society. In addition these costs are important because exposure to ETS has been shown to be more amenable to reduction than active smoking has been. Further, exposure to ETS is involuntary, so it may be appropriate to have a lower tolerance for these externally imposed costs than about the costs of direct smoking. Differences in ETS exposure have not, to our knowledge, been directly incorporated into insurance company underwriting procedures, which could be refined on the basis of ETS cost estimates.

### **Published Research on Environmental Tobacco Smoke**

Hundreds of original research articles have been published on the effects of ETS. These articles vary in their approach to the subject in several important ways. For example, most involve evaluation of health effects in humans on the basis of surveys, but some involve controlled experiments on animals. As discussed below, there are several different approaches to defining and measuring exposure to ETS. The differences in the definition of exposure to ETS are not significant. There can be significant differences in how exposure is quantified, for example by the number of smokers in a nonsmoker's

household, or the number of cigarettes smoked by members of a nonsmoker's household, by the number of hours per day during which a nonsmoker can smell tobacco smoke in his or her environment, and more recently by the concentration of cotinine in the blood serum.

There is evidence of publication bias (Misakian & Bero, 1998), exhibited in the fact that there is a strong relationship between the type of research sponsor and the conclusions reported, as well as in the fact that stronger conclusions appear to be more likely to be published, and to be published sooner, than weak conclusions. Publication bias is not unique to studies of ETS, but has been found to exist in many fields, and is an issue in any review of published research. Numerous articles have been sponsored by organizations opposed to smoking as well as by tobacco interests. There is no lack of published articles exhibiting observations contrary to the consensus that ETS exposure is harmful, and we included the findings of these articles in our determinations of morbidity rates on the same basis as articles with positive findings. Given the fact that the health effects of exposure to ETS have been studied for over 40 years, the lag in publication of studies with less statistically significant results is a minor issue. If articles showing negative results were systematically excluded from publication, we would expect to see that the results of published articles would show a pattern resulting from such a cutoff, but this was not the case. In fact, there is a relatively symmetrical distribution of published results around the mean. The validity of conclusions on the effects of ETS exposure in relation to publication bias was studied by Bero, et al. (1994), and publication bias was found not to invalidate the conclusions regarding the health effects of ETS. This article examines the tobacco industry's claim that publication bias against negative studies invalidates the risk assessment of ETS exposure conducted by the U.S. Environmental Protection Agency and other reviews of the health effects of ETS. The article discusses the determination of the number of published original research articles that tested the hypothesis that ETS exposure is associated with adverse health effects and that reported statistically significant "positive" or non-significant "negative" results; the number of articles that concluded that ETS is a health risk; and unpublished studies on the effects of ETS on health. Articles were identified by a computerized and non-computerized search of the medical literature supplemented with material obtained from the tobacco industry. Articles were classified as peer-reviewed journal articles or articles from sponsored symposia. The study considered the statistical significance of results reported in the article and whether or not the article concluded that ETS exposure is a health risk. The paper concludes that there is no publication bias against statistically non-significant results on ETS in the peer-reviewed literature. The high proportion of articles in symposia that reach the conclusion that ETS is not harmful primarily results from the inclusion of review articles, rather than from original research. We have concluded that, even with some evidence of publication bias, valid conclusions can be drawn from the published, peer-reviewed literature.

An emotional public debate has surrounded research into the health effects of exposure to tobacco products. People have become polarized on both sides of a debate about whether and how to reduce the use of tobacco, and the resulting exposure of both users and non-users. At times the controversy has degenerated into *ad hominem* attacks from both

sides. Our response to this environment has been to include in our analyses the combined results of studies that were published in peer-reviewed journals, whether such results found a positive, negative or insignificant relationship between ETS exposure and the particular morbidity under study. We have not attempted to determine the motivations of researchers, but have depended on the peer-review process to select scientifically grounded articles.

Most published articles focus on one or a few specific health effects in relation to a specific definition of exposure. In many cases it is not possible to determine all of the health effects that were monitored, but only those that are reported, which could be a proper subset of the health conditions that were considered. Given the variety of issues and the technical challenges of evaluating these issues in hundreds of articles, it is fortunate that several well-funded, independent studies of the literature on the health effects of smoking have been conducted over the past forty years, most recently with the publication of the IARC report *Tobacco Smoke and Involuntary Smoking* (International Agency for Research on Cancer, 2004), which covers research articles published through 2002. All of the major independent studies of the literature have come to consistent conclusions. As a result, we are confident that these studies provide a solid foundation for a quantitative analysis of the economic effects of exposure to ETS, and have used them as the basis of our analysis. Appendix 1 presents brief summaries of the findings of well over 100 articles and reviews on the principal morbidities associated with exposure to ETS. In view of the quality and extent of the work that has already been done, we have not attempted to independently assess the scientific merit of the articles reviewed in the IARC, California EPA, and surgeon general studies.

### **Population Exposure to Environmental Tobacco Smoke**

In the U. S. population exposure to ETS can be determined from the National Health and Nutrition Examination Surveys (NHANES), periodic surveys of a statistical sample of the population conducted since 1956 by the CDC. These surveys cover approximately 10,000 people and have included questions related to exposure to tobacco smoke for many years and serum cotinine laboratory analyses since 1988.

Exposure to ETS has been measured in several different ways. The principal measures of exposure are either self-reported descriptions of exposure in the home, at work or in social situations or laboratory measurement of metabolites of nicotine, principally cotinine. In this paper we consider tobacco smoke to be synonymous with cigarette smoke. Other sources of tobacco smoke are a relatively minor component of the total, so this simplification has no practical effect on our findings. The most common exposure situations that have been studied are having a smoking spouse or, in the case of children, a smoking parent, possibly with a measure of the number of cigarettes smoked by the smoker or the number of smokers in the household, exposure in the workplace, or overall descriptions of the degree of exposure to smoke, such as being able to smell tobacco smoke at certain times. The chemical measurement of exposure is currently based on cotinine, a metabolite of nicotine associated quantitatively with the amount of tobacco smoke absorbed by the body, and amenable to measurement at extremely low

concentrations. An analysis of NHANES data (Pirkle, et al., 1996) yields quantitative relative exposure levels for the principle qualitatively characterized ETS exposure groups, home exposure only, work exposure only, and both home and work exposure, as well as the proportion of the U.S. population in each group. The geometric mean serum cotinine concentrations for nonsmokers, age 17 and higher, are shown in Table 1. Levels for individuals age 4 through 16 are shown in Table 5.

Table 1. Geometric Mean Serum Cotinine Concentration ( $\mu\text{g}/\text{mL}$ ) and Sample Size for Individuals Age 17 and Higher, NHANES III, 1988-1991.

<b>Exposure Group</b>	<b>Serum Cotinine Concentration</b>	<b>Sample Size</b>	<b>Group Percentage</b>
No known exposure	0.000124	3,154	40.7
Work exposure only	0.000318	779	10.1
Home exposure only	0.000700	315	4.1
Home and work exposure	0.000926	246	3.2
Active smokers	0.30000*	3,246	41.9
Total		7,740	100.0

\* Estimate. Smoker cotinine levels vary with the number of cigarettes smoked per day.

The percentages in Table 1 are computed in relation to the total population. Many of the discussions of ETS focus on exposure among nonsmokers, and use percentages of the nonsmoking population for purposes of discussion. These rates may be obtained from Table 1 by dividing by the percentage of nonsmokers, 58.1 percent, in that table. The resulting values are shown in Table 2.

Table 2. Geometric Mean Serum Cotinine Concentration ( $\mu\text{g}/\text{mL}$ ) in Relation to Non-smoking Population for Individuals Age 17 and Higher, NHANES III, 1988-1991.

<b>Exposure Group</b>	<b>Serum Cotinine Concentration</b>	<b>Group Percentage</b>
No known exposure	0.000124	70.0
Work exposure only	0.000318	17.4
Home exposure only	0.000700	7.1
Home and work exposure	0.000926	5.5

*The Second National Report on Human Exposure to Environmental Chemicals* (Centers for Disease Control and Prevention, 2003) presents data on serum cotinine levels among non-smokers in terms of the concentrations at certain percentiles. If we assume that the geometric mean concentrations for the groups in Table 2 approximate the concentration for the midpoint of the group, and work from highest to lowest concentrations, we obtain the percentiles shown in Table 3. For example, the highest concentration would correspond to the 97.1 percentile, computed as  $100 - (5.5/2)$ , and the next highest concentration would correspond to the 90.9 percentile, computed as  $100 - 5.5 - (7.1/2)$ .

Table 3. Geometric Mean Serum Cotinine Concentration ( $\mu\text{g}/\text{mL}$ ) for Estimated Percentiles of the Nonsmoking Population for Individuals Age 17 and Higher, NHANES III, 1988-1991.

<b>Exposure Group</b>	<b>Serum Cotinine Concentration</b>	<b>Estimated Percentile</b>
Home and work exposure	0.000926	97.1
Home exposure only	0.000700	90.9
Work exposure only	0.000318	78.7
No known exposure	0.000124	35.0

*The Second National Report on Human Exposure to Environmental Chemicals* presents (in its Table 60) the data shown in Table 4 below.

Table 4. Geometric Mean Serum Cotinine Concentration ( $\mu\text{g}/\text{mL}$ ) by Percentiles of the Nonsmoking Population for Individuals Age 20 and Higher, NHANES III, 1999-2000

<b>Percentile</b>	<b>Serum Cotinine Concentration</b>	<b>Percentile</b>
95	0.001480	95
90	0.000630	90
75	0.000167	75
50	< 0.000050	50

Perhaps by coincidence the percentiles in Table 4 are rather close to those in Table 3, making possible a comparison of exposure changes over the ten-year period between the midpoints of the two surveys. We have assumed that the percentiles continue to approximate the levels of exposure from home and work, home, and work exposure respectively. We observe that the exposure for the majority of the population who are not aware of ETS exposure has declined significantly. The CDC found in this report that the median serum cotinine concentration declined 67 percent. The median individuals are in the group that is not knowingly subject to ETS exposure. A decline of about half (recognizing slightly different percentiles) is observed at the 78.7<sup>th</sup> (respectively 75<sup>th</sup>) percentile, which we assume represents work exposure. Exposure at home, and of those exposed at home and at work, has not significantly changed, given the statistical uncertainties of measurement.

For purposes of our later calculations, we have assumed that the relative rates of exposure to ETS among nonsmokers follow the rates in Table 1, while recognizing that the percentage of active smokers has dropped to 23.35 percent. On this basis we calculate that the nonsmokers who are exposed to ETS comprise 22.96 percent of the U.S. population.

Table 5. Geometric Mean Serum Cotinine Concentration ( $\mu\text{g}/\text{mL}$ ) and Sample Size for Individuals Age 4 through 16, NHANES III, 1988-1991.

Exposure Group	Serum Cotinine Concentration	Sample Size	Group Percentage
No known exposure	0.000117	1,450	57.1
Home exposure only	0.001040	981	38.6
Active smokers	0.30000*	108	4.3
Total		2,639	100.0

\* Estimate. Smoker cotinine levels vary with the number of cigarettes smoked per day.

Of the several chemical biomarkers for smoking exposure serum cotinine is the best currently available. Cotinine is a metabolite of nicotine. It is uniquely associated with exposure to nicotine, which, for practical purposes, is associated uniquely with tobacco. We have assumed that the level of exposure to nicotine in tobacco smoke can be used to represent the level of exposure to other components of smoke, as we would expect nicotine to be represented as a relatively constant component of smoke, and there is no evidence to the contrary. We have not, however, based our overall estimates on cotinine levels, as most of the research on ETS was conducted before the development of the laboratory methods currently used to measure cotinine. We have used serum cotinine measurements only to allocate total excess morbidity among individuals exposed at home only, at work only, and both at work and at home. The sensitivity of tests for cotinine has progressed to the point that serum cotinine concentrations as low as 0.00005 micrograms per milliliter can be reliably measured. At this level of sensitivity half of the U. S. population shows measurable serum cotinine. This is a significant decrease from the results of studies conducted 15 years ago, which had detected serum cotinine in 90 percent of the U.S. population. The biological half-life of serum cotinine in the human body is typically 15 to 19 hours. Thus, the level of serum cotinine measures the integrated average exposure of the individual over a period of two or three days. This is a very short period in relation to the long-term exposure necessary to cause many of the smoking-related diseases, but it allows the status reported in response to surveys to be tested objectively. In addition, there is substantial evidence that individual exposure to ETS remains relatively stable over time.

For analysis of the effects of ETS to be meaningful it is necessary to eliminate active smokers from the observed population. Some observers have expressed concern that the self-reported data on the individual's own nonsmoking status may be inaccurate. The serum cotinine data show a very clear separation in measurements between smokers and nonsmokers, so this allows an evaluation of the self-reported survey data. As noted below, an error rate of about 1 percent in self-reported smoking status is supported by studies of blood chemistry. If individuals who are misclassified as nonsmokers are included in studies of ETS, the effect would depend on the number included in the group believed to be exposed to ETS versus the number included in the group believed not to be exposed to ETS. The effect on the measured relative risk would be the relative risk for smokers, times the excess of the proportion of smokers included in the ETS-exposed group versus the unexposed group. We have no basis to estimate this difference. The total effect, split between the two groups, is the proportion of erroneously classified

smokers (i.e. 1 percent) times the relative risk for smokers. Since this is well below the additional risk from ETS exposure for any of the conditions evaluated in this paper, we have accepted the additional risk levels indicated by the published research.

A recent report (Pirkle, et al., 1996) supports several conclusions about exposure to ETS.

- Nicotine in foods (such as tomatoes, potatoes, eggplant and green peppers) is a negligible contributor to serum cotinine levels (less than 0.00002  $\mu\text{g/ml}$ ).
- The geometric mean concentration of serum cotinine for nonsmokers was 0.00010 to 0.00030  $\mu\text{g/ml}$ , compared with approximately 0.3  $\mu\text{g/ml}$  for smokers. There is a gap in serum cotinine concentration between the maximum concentration for nonsmokers and the minimum concentration for smokers. Virtually all nonsmokers have less than 0.010  $\mu\text{g/ml}$  of serum cotinine, and virtually all smokers have more than 0.015  $\mu\text{g/ml}$ .
- Mis-reporting of smoking status (smokers denying that they smoke) in the National Health and Nutrition Examination Survey is less than 3 percent, and is 1.3 percent for adults. This is generally consistent with, while somewhat lower than, the results of a number of other studies.
- Quantitative exposure to ETS among nonsmokers, as measured by serum cotinine concentration, is strongly correlated with self-described home and work exposure. While half of the U.S. population is exposed to tobacco smoke, those who report exposure at work, at home or both have higher levels of serum cotinine than those who report no such exposure.
- The lowest level of exposure to ETS among individuals reporting some exposure is among those reporting exposure at work but not at home. Those reporting exposure at home, but not at work have about twice this level of exposure, and those reporting exposure at both work and home have three times the average level of exposure as among those exposed only at work.

On the basis of the above we have concluded that self-reported levels of exposure are a reasonable basis for estimating exposure to and health effects of ETS, and could be a basis for commensuration of the results of surveys based on different definitions of exposure. Almost all of the published research on the health effects of ETS is based on qualitative descriptions of exposure. While it is now possible to quantify exposure using tests for cotinine, this is an area for future research. In the absence of quantitative exposure data we have combined various groups of individuals into one category of “exposed” to project total health effects. In other words, we did not distinguish among the categories of “exposed at work only,” “exposed at home only,” and “exposed both at work and at home.” No doubt there are differences in the levels of health effects for these groups, but distinguishing among them would require additional data to be gathered.

We have not attempted to isolate the effects of exposure to ETS from the effects of other air pollutants and environmental exposures to which people are exposed. In typical indoor environments where smoking is permitted, several studies have found that the level of pollutants associated with tobacco smoke far exceeds the level of all other air

pollutants. While there may be an association between exposure to ETS and exposure to other air pollutants or environmental chemicals, in view of the studies that compared the levels of other indoor pollutants in comparison to ETS, we felt that the level of other exposures was generally much lower. In addition, the research studies on which our estimates are based generally considered the question of exposure to other pollutants, and controlled for this potentially confounding variable. The issue of whether and how confounding variables were considered was part of the review process by the independent agencies such as the CDC and the World Health Organization for evaluating the original research papers. We relied on these reviews in our selection of papers used for mortality and morbidity estimates.

### **Changes in ETS Exposure in the United States**

During the approximately 40-year period over which the principal studies of ETS have been conducted, there have been major changes in the smoking habits of the U. S. population. Based on data presented by the CDC, the percentage of smokers in the population has declined from 42.4 percent in 1965 to 23.35 percent in 2000. This has necessarily resulted in a reduction in exposure to ETS, since there are fewer sources of smoke to cause exposure. At the same time the exposure of the nonsmoking population has changed even more dramatically as a result of restrictions on the locations where people are permitted to smoke, as well as an increased awareness that ETS can be harmful to health, causing both smokers and nonsmokers to reduce the exposure of nonsmokers to ETS. Key events, such as the first U.S. surgeon general's report, the ban on tobacco broadcast advertising, and increases in the federal cigarette tax were each followed by a decrease in tobacco consumption. *The Second National Report on Human Exposure to Environmental Chemicals* has new, expanded data that will facilitate determination of trends in serum cotinine levels by many cohorts based on characteristics such as age and sex. The report shows that since the early 1990s, median serum cotinine levels in nonsmokers in the United States have decreased by 58 percent in children, 55 percent in adolescents, and 67 percent in adults.

In view of the substantial reductions in ETS exposure over the past 40 years, most notably in the last decade, and the fact that many ETS-related morbidities are related to long-term exposure, any estimate of ETS-related morbidity, mortality and cost depends on the time period over which exposure is evaluated. We have chosen to base our estimates on the currently prevailing levels of exposure to ETS.

### **Health Effects of ETS Exposure**

As documented in Appendix I, exposure to ETS is associated with increased morbidity and mortality from cancer of various organ systems as well as from pulmonary and cardiovascular conditions. Prenatal and perinatal health effects have also been documented in the references in Appendix I. Many other adverse health effects of exposure to ETS have been studied and documented in the literature, but the quantitative results from research to date remain imprecise. Our analysis of economic effects is limited to effects for which a positive association has been quantitatively documented.

Some of the effects of exposure to ETS are subtle, and would require extremely large sample sizes to produce quantitative conclusions about excess mortality or morbidity. Our methodology does not assign a cost to conditions for which the excess mortality or morbidity is not statistically significant, even though we are sure that some excess costs exist for conditions that do not have a statistically demonstrated rate of excess occurrence. For example, studies of rates of breast cancer, brain tumors, leukemia, lymphomas and certain childhood cancers are suggestive of increased risk associated with ETS, but the results are not consistent enough to provide quantitative estimates of relative risk. As a result, our estimates of the excess mortality and morbidity caused by exposure to ETS are a lower bound for the total effects of exposure. We do not have a basis to quantify this understatement, but believe that the principal adverse health effects of ETS have been identified and quantified in this paper.

Appendix 1 shows the range of morbidities that have been found to have increased incidence in relation to ETS exposure, and briefly summarizes the findings of principal studies for each morbidity. Most of the research studies define an occurrence of disease as a diagnosis coded within certain categories by reference to an international disease classification system, most commonly the ICD-9 system. For example, a case of lung cancer would be defined as a diagnosis within a set of ICD-9 codes consistent with that disease, represented by codes that begin with 162. Table 6 summarizes the relative risk for the morbidities with greatest economic impact for individuals exposed to ETS. Relative risk levels in Table 6 have been calculated by combining the reviewed studies, weighted by the expected number of base-rate cases. Appendix 3 illustrates this calculation for lung cancer. For each study we calculated the expected base-rate cases as the actual number of cases divided by the relative risk found by the study. The total actual cases divided by the total base-rate cases yields the overall relative risk. This calculation method was necessary because some of the studies did not publish the size of the population subject to study. Some conditions that are strongly related to ETS exposure have been omitted from the analysis because their total economic impact is much less than that of the included conditions. For example, cancer of the nasal sinus is almost three times as likely to occur among individuals exposed to ETS as it is among the unexposed population, but this relatively uncommon cancer has a much smaller impact than lung cancer, for which the risk ratio is 120 percent, but for which the base rate is larger.

The medical costs of an individual with a given morbidity are strongly related to existing comorbidities. Generally the studies on effects of exposure to ETS have not isolated the effects of comorbidities, and the cost data that we used were population averages, which do not provide a basis for estimating the effects of comorbidities. Comorbidities would be considered and included in the analysis of the well-designed case control studies, so their effect would be taken into account on an overall basis. Our results are based on an implicit assumption that the level of comorbidities in the exposed and unexposed population is not substantially different.

Further research is needed on the effects of ETS on respiratory infections, including the common cold, bacterial pneumonia and tuberculosis. Researchers have noted some

increases in rates for these conditions in relation to ETS exposure, but we have been unable to identify adequate evidence to quantify the economic effects related to respiratory infections.

The degree to which ETS increases the incidence of a particular morbidity is measured by the relative risk. Relative risk is the ratio of the rate of morbidity among an exposed group to the rate in a corresponding but unexposed group. For example, we expect 1.22 times as many cases of lung cancer among a population exposed to ETS as would be observed if the population were not exposed. Table 6 presents the results of our analysis and summary of published research on the relative risk for the principle morbidities that have been identified as being exacerbated by ETS.

Table 6. Estimated Relative Risk by Morbidity for Individuals Exposed to ETS

Category	Morbidity	Relative Risk
Cancer	Lung cancer	1.22
	Cervical cancer	1.41
Respiratory system	Asthma	1.44
	Otitis media	1.52
	Chronic pulmonary disease	1.83
Cardiovascular system	Coronary heart disease	1.10
Perinatal manifestations	Low birth weight	1.22
	Spontaneous abortion/perinatal mortality	1.54
Postnatal manifestations	Sudden Infant Death Syndrome	1.80

### **Dose-Response Relationships for Tobacco Smoke Exposure**

The existence of a dose-response relationship is considered to be a necessary condition for a causal relationship to exist between the dose and the response. In this context a dose-response relationship means that there is a statistically significant increase in the response related to an increase in the dose. The existence of a dose-response relationship in this sense does not mean that a mathematical function can be determined to link dose and response. Most, but not all, of the studies of effects of ETS have evaluated the dose-response relationship, and have obtained positive results. For example, for individuals exposed at home, the number of cigarettes smoked by other household members has been used to measure dose. The workplace environment does not lend itself to such a clear-cut measure of exposure, and dose-response in the workplace has not been as extensively studied.

There is a wide gap between the level of exposure of active smokers to smoke components and the exposure of individuals exposed to ETS. Within the narrow range of ETS exposures, the dose response relationship in most studies appears to be a relatively simple, nearly linear relationship, but there is no established dose-response relationship that would include both active smokers and nonsmokers exposed to ETS. As noted in the report on ETS by the National Research Council (1986), if individuals with a high sensitivity to the irritating effects of ETS are more likely to be nonsmokers, then the

population to which a dose-response curve would be applied would be different for active smokers and for nonsmokers. Consequently, there would be some question as to whether a single dose-response relationship could be applied to both groups.

For purposes of estimating the economic effects of the significant reduction in exposure among non-smoking individuals who are not aware of their exposure, in the absence of any other model we have assumed a linear dose-response relationship at the levels of exposure prevalent among nonsmokers.

### **Sensitivity of Estimates to Changes in Assumptions**

The estimates derived in this paper are based on a number of assumptions. These assumptions affect the estimated number of cases for each morbidity, the age at onset, the medical costs, the number of years of life lost, the number of years of disability, the number of working years lost, the economic productivity of employment and of services, the projection of economic productivity into the future and the rate used for discounting future economic losses to present value. Even where there is only one reasonable assumption, given the data considered, there may be uncertainty in the values used because of the limitations of sample sizes in the various studies, for example where the relative risk of a given condition is based on a limited number of research studies.

None of the assumptions stands out as leading to particular sensitivity in the results. For example, the uncertainty in relative risk for a particular morbidity, arising from the limitations of sample size, translates directly into corresponding uncertainty in the estimated economic costs.

A further source of uncertainty is the computation of rates for the unexposed population from current population rates that include smokers, ETS exposed nonsmokers, and non-exposed nonsmokers. For example, most cases of lung cancer in the United States are attributable to smoking. In order to determine the base rate of lung cancer among the unexposed population, one has to eliminate the smoking-related cases. We have used a relative risk of 8.0 (i.e., smokers get lung cancer at eight times the rate of unexposed individuals) to carry out this calculation for lung cancer. The relative risk for lung cancer for active smokers compared to nonsmokers has been estimated in various studies from 3.5 to 10 or more. For example, see the 1990 Surgeon General's Report (Office on Smoking and Health, 1990). If we change this assumption to 7.0, fewer smoking-related cases would be eliminated and the base rate would accordingly be increased, leading to an increase in the computed number of ETS cases, which are based on the population rate times the corresponding relative risk factor. In the case of lung cancer a change in the relative risk for smokers from 8.0 to 7.0 would ultimately lead to a 10-percent increase in our estimate of ETS-related lung cancer cases.

In the case of each assumption we have tried to make choices that neither overstated nor understated the economic costs.

## **Quantitative Analysis of Excess Morbidity and Mortality**

We have based our estimates of excess cases of morbid conditions that are expected to arise annually from ETS exposure of the U. S. population and the exposure indicated by NHANES III adjusted for subsequent reductions in ETS exposure. Our estimates of the relative risks of morbid conditions among people exposed to ETS are based on our compilation of published reports.

Typical studies of the effects of ETS on the occurrence of medical conditions compare the rate among a group of people exposed to ETS to the rate among a group matched for other risk factors, but not exposed to ETS. The resulting data provide the best available evidence of the relative risk related to ETS, but do not provide an unexposed population base rate, as the unexposed sample may not be representative of the population as a whole. Population rates include the rates among active smokers, which, for the conditions of interest, are generally much higher than the rate among people who are not exposed at all to tobacco smoke. Therefore, the conversion of ETS relative risk to excess cases involves adjusting the population rate to remove the effects of smoking, and then applying the relative risk taking into account the rate of exposure to obtain ETS-related cases.

The total number of cases among exposed individuals is determined by multiplying the relative risk for exposed individuals times the base rate of occurrence for unexposed individuals, and multiplying by the exposed population. The number of ETS-related cases is determined by subtracting the number of cases that would be obtained from the base rate from the total number of cases. This approach implicitly assumes that the age distribution of exposed individuals is similar to the age distribution of the population as a whole, since the base rate depends on the age distribution of the population, especially for conditions, such as cancer, that have highly age-dependent rates. We believe that the assumption that the age distributions of exposed and unexposed individuals are similar is reasonable for adults, but we are not aware of any existing research on the question of the relationship among adults between smoking exposure and age. Data on exposure of children show that there is an inverse relationship between age and exposure. Younger children exhibit higher concentrations of serum cotinine than exhibited by older children. Exposure of children is essentially limited to children in households with smokers, but adults from nonsmoking households are potentially subject to exposure at work. The principal morbid conditions to which children are subjected through exposure to ETS are not as age-related as cancer, so we did not feel that it was necessary to attempt to model the age distribution of children's exposure. The strength of the evidence of the relationships between ETS exposure and morbid conditions varies by the condition considered. The strongest relationship is for lung cancer, for which the number of studies is large, and for which clear evidence of a causal relationship is corroborated by laboratory studies. In comparison, the evidence for other conditions is less strong, but still convincing. For example, our evaluation of excess asthma incidents is based on a compilation of four studies covering approximately 6,000 cases, while our lung cancer

estimate is based on over 40 independent original studies covering 13,000 cases. The evidence for all of the conditions included in our cost estimates is based on a number of statistically significant findings with a clear and consistent indication of increased risk. We omitted conditions for which the consensus of published reports was not clear.

We have used two different approaches to the measurement of the cases of morbidity produced by exposure to ETS. Some conditions have an occurrence measured by a specific diagnosis at a point in time. These conditions include cancer and conditions related to the newborn. We have measured these conditions in terms of the number of new diagnoses per year. The medical costs are measured in terms of the present value of the total lifetime cost of treatment. Other conditions, typically of a chronic nature, are of more gradual onset, such as chronic pulmonary disease and asthma. The medical costs of these conditions are more readily quantified in terms of the total caseload in the population. The medical costs are measured in terms of the annual cost of treatment of the chronic condition among the population.

Exposure to ETS is believed to increase the number of cases of asthma and to exacerbate existing cases. Most of the research on the relationship between ETS and asthma does not distinguish between initiation of a new case and exacerbation of an existing case, so we were unable to determine separate estimates for these two effects. The total estimated economic impact of ETS through asthma can be determined without concern about this distinction, because research on the subject generally measures the number of medical incidents, such as hospital admissions, among exposed and unexposed populations. Therefore, by estimating the effect as the cost of excess incidents of medical care, we are able to determine the overall increase in cost of asthma associated with ETS, combining the costs of the two categories of cases.

Spontaneous abortion is not subject to mandatory reporting in the United States. Measurement of the rate of spontaneous abortion is made difficult by the fact that many women are unaware of pregnancies terminated at a very early stage. According to the National Institutes of Health Medline Web site (National Institutes of Health, 2004) the rate of spontaneous abortion of known pregnancies is approximately 10 percent, occurring during weeks seven to 12 of pregnancy. The same reference cites a rate of up to 50 percent of fertilized eggs that die and are lost before the woman knows that she is pregnant. We have used an estimate of 15 percent of pregnancies as the base rate of spontaneous abortion and perinatal mortality. Perinatal mortality is generally less than five per 1,000 births, and is less than the uncertainty in the rate of spontaneous abortion. Research on the effects of ETS has generally dealt with the combined rate, so we have considered them together in a single combined rate.

Table 7. Estimated Number of Additional New Cases of Selected Acute Conditions in the United States Associated with Exposure to ETS

Category	Morbidity	Excess Cases (per 100,000 exposed)
Cancer	Lung cancer	3.71
	Cervical cancer	1.26
Respiratory system	Otitis media	2.67
Perinatal manifestations	Low birth weight	2.90
	Spontaneous abortion/perinatal mortality	104
Postnatal manifestations	Sudden Infant Death Syndrome	391 <sup>†</sup>

<sup>†</sup> total U.S. cases per year

Table 8. Estimated Number of Additional Population Cases of Selected Chronic Conditions in the United States Associated with Exposure to ETS

Category	Morbidity	Excess Cases (per 100,000 exposed)
Respiratory system	Asthma	1,879*
	Chronic obstructive pulmonary disease	1,423
Cardiovascular system	Coronary heart disease	728

\* equivalent of excess medical incidents

The number of cases in Tables 7 and 8 are calculated by applying the respective relative risk values to the population base rates. Population base rates are actual population rates reduced by the estimated cases related to active smoking and ETS. For example, according to the American Lung Association (2004) there are 16.4 million cases of chronic obstructive pulmonary disease (COPD) in the United States. This is equivalent to a population rate of 56.2 per thousand. The relative risk of COPD for smokers is estimated at 10 times the base rate, and for nonsmokers exposed to ETS it is 1.83, as shown in Table 7. If the portions of the population that are smokers, non-exposed nonsmokers, and nonsmokers exposed to ETS are denoted by  $p_s$ ,  $p_{ns}$ , and  $p_{ETS}$ , respectively, the base rate is  $r_{COPD}$ , and relative risks are denoted by  $rr_s$  and  $rr_{ETS}$ , and recognizing that the relative risk for unexposed nonsmokers is, in effect, one, then the population rate for COPD can be computed as follows:

$$\text{Population Rate} = r_{COPD} \times (p_s rr_s + p_{ns} + p_{ETS} rr_{ETS})$$

Solving for  $r_{COPD}$  using the above values for the population rate and for  $rr_s$  and  $rr_{ETS}$ , and with  $p_s = 0.2335$ ,  $p_{ns} = 0.5369$  and  $p_{ETS} = 0.2296$  as stated above, we have

$$r_{COPD} = (\text{Population Rate}) / (p_s rr_s + p_{ns} + p_{ETS} rr_{ETS})$$

Subject to a slight difference due to rounding, the above calculation yields a base population rate for COPD in the absence of tobacco smoke equal to 0.01715.

Given the population base rate, the number of additional cases per 100,000 exposed as shown in Table 8 is computed as follows:

$$100,000 \times I_{\text{COPD}} \times (rr_{\text{ETS}} - 1) = 1,423$$

### **Economic Costs of Excess Morbidity**

The cost of excess morbidity consists of direct medical costs, costs associated with disability, and the opportunity costs of unpaid caregivers. We have made estimates of the first two categories of costs, but we have not attempted to estimate the indirect costs associated with unpaid caregivers.

Two measures of tobacco-related excess medical costs are in current use in the literature. One is the fraction of observed costs that is attributable to tobacco smoke. The second is the difference between actual medical costs and the costs that would exist if the population were not exposed to smoke. The first measure incorporates, implicitly, the so-called “death benefit,” which is the reduction of medical costs produced by the early death of those affected by smoking-related disease (Zeger, et al., 2000). The second measure compares actual expenses with an unobservable quantity, medical costs in the absence of tobacco smoke. Our approach in this paper is to measure the first quantity, referred to as the net attributable cost, for medical expenses. In addition, however, we estimate the economic loss caused by early death.

The medical costs of ETS-related morbidity arise from a combination of the excess occurrence of morbidity associated with exposure to ETS and the medical costs associated with these conditions as they arise from exposure to ETS. Johnson, et al. (2003) conducted a case-matched analysis of medical costs of two major categories of morbidity attributable to active smoking, viz., respiratory conditions and cardiovascular conditions. Their analysis was based on the National Medical Expenditure Survey (NMES) of the Agency for Health Care Research and Quality. This survey includes a series of questions on active smoking, which they could use as a basis for their analysis. The major findings of the analysis of Johnson, et al. for all ages combined are presented in Table 9 below.

Table 9. Percent of Cases and Expenses Attributable to Smoking in the 1987 NMES

Disease	Cases	Expenses
Respiratory	70.2	53.4
Cardiovascular	19.6	13.4

In Table 9 the amounts are expressed as percentages. We have used the term “respiratory” to represent the total number of cases and expenses from lung and laryngeal cancer and chronic obstructive pulmonary disease. We have used the term “cardiovascular” to represent the total number of cases and expenses from cardiovascular disease, stroke and certain other cancers. As the table shows, the fractions of costs

attributable to smoking are similar to, but approximately 25 percent to 30 percent less than the fractions of attributable morbidity. The differences are two to four times the standard error of measurement. The reasons for these differences are not known, but may arise from the different underlying health status of those exposed to smoke, and differences in the severity of disease, including the possibility of higher rates of mortality causing earlier death among cases attributable to smoking.

The NMES does not include questions about exposure to ETS, and no other resource exists that would enable a case-matched analysis of ETS-related costs at this time. Therefore we have estimated the medical costs related to ETS on the basis of the attributable fraction of morbidity. Many of the conditions studied are strongly related to smoking, so the population average cost is relatively close to the cost for smoking-related cases. For example, in the case of lung cancer, the great majority of cases is smoking-related, so the costs for the population are essentially smoking-related costs. The results of Johnson, et al. would indicate that for conditions with a small smoking-related component the use of population average costs could result in an overstatement in our cost estimates, but we believe the use of the attributable fraction of cases as a basis for determining the attributable fraction of expenses is the best estimate of expenses for ETS exposure available at this time. This is an area for future research when resources enabling such an analysis become available.

The U.S. Environmental Protection Administration (2002) has produced estimates of direct medical costs for several diseases related to various pollutants. The estimates of interest in relation to ETS are shown in Table 10. These estimates are adjusted from the date of the EPA analysis (i.e., 1996) to 2004 costs by using the medical cost component of the Consumer Price Index.

We have not attempted to quantify the direct medical costs associated with spontaneous abortion, perinatal mortality and Sudden Infant Death Syndrome (SIDS), but the related economic loss from SIDS is included in the estimates of economic losses caused by ETS.

Table 10. Estimated Lifetime Direct Medical Costs of Selected Diseases

<b>Morbidity</b>	<b>Average Total Cost</b>	<b>Discounted Cost (at 5%)</b>
Lung cancer	80,980	76,921
Cervical cancer	16,518	16,518

Table 11 indicates the direct per case or per incident medical costs associated with selected diseases. The cost data are assembled from various sources. For example, the cost per year for chronic obstructive pulmonary disease is based on the total direct U.S. medical cost of \$20.9 billion divided by the number of cases (16.4 million) (American Lung Association, 2004). Coronary heart disease average total cost is also determined on the basis of the total annual direct U.S. medical cost of \$66.3 billion divided by the number of cases, 13.2 million (American Heart Association, 2004). The otitis media case count is based on a case frequency estimate by Biles, et al. (1980) applied to current population values, and the total cost estimate of Gates (1996) adjusted for inflation.

Costs for low birthweight are based on the estimate of Lewit, et al. (1995), adjusted for inflation.

Table 11. Estimated Annual or per-Incident Direct Medical Costs of Selected Diseases

<b>Morbidity</b>	<b>Average Total Cost</b>
Otitis media	297*
Chronic obstructive pulmonary disease	1,274
Coronary heart disease	5,023
Low birth weight	33,550*

\* per incident

Taking into account the number of excess cases per 100,000 exposed individuals, the population exposure to ETS, and the medical costs associated with the various conditions, we have estimated the total direct medical costs of the conditions considered as presented in Table 12. The direct medical costs for asthma are estimated on the basis of population total annual asthma cost data from the National Institutes of Health (National Heart, Lung, and Blood Institute, 1999), multiplied by the proportion of asthma cases attributable to exposure to ETS.

As an example of the calculations performed to obtain the values in Table 12, we present the calculation for lung cancer. From Table 7 the additional annual cases of lung cancer per 100,000 exposed individuals are 3.71. The exposed group, as discussed following Table 1, is 22.96% of the U.S. population. Using 292 million as the U.S. population, this produces an estimate of 67 million people. Multiplying the cases per 100,000 exposed times the number of people exposed we estimate that there are 2,488 cases per year on the basis of current exposure. As stated in Table 10, the present value of direct medical costs per case of lung cancer is \$76,921. Multiplying the number of cases by the cost per case we estimate that the annual present value of lung cancer costs resulting from exposure to ETS is currently \$191 million.

Table 12. Estimated Direct Medical Cost of Exposure to ETS per Year for the U.S. Population, Based on Present Values

<b>Category</b>	<b>Morbidity</b>	<b>Cost (\$1,000,000)</b>
Cancer	Lung cancer	191
	Cervical cancer	14
Respiratory system	Asthma	773
	Otitis media	53
	Chronic pulmonary disease	1,215
Cardiovascular system	Coronary heart disease	2,452
Perinatal manifestations	Low birth weight	284
<b>Total</b>		<b>4,982</b>

The costs estimated in Table 12 are concentrated in the segment of the U.S. population with exposure to environmental tobacco smoke, which, on the basis of Table 1 and the current level of active smoking, currently comprises 22.96 percent of the population, as

noted following Table 1. The excess direct medical cost per person exposed is approximately \$80 per year. Assuming a linear dose-response relationship at the dose levels for ETS exposure, the cost per year for nonsmokers exposed at home and at work is about \$150 per person. In comparison to total health care expenses reported by the Medical Expenditure Panel Survey (Olin and Machlin, 2003), this represents an increase of 6 percent in the cost of medical care caused by exposure to ETS at home and at work.

### **Economic Costs of Excess Mortality and Disability**

Our estimate of the economic value of each life-year lost is comprised of three components, lost wages, lost fringe benefits and lost services. The results are presented in Table 13. Each component of economic productivity is based on population averages. No attempt has been made to take into account the relationship between exposure to ETS and economic status. No reduction is taken for personal consumption, because it is assumed that personal consumption would have had corresponding value to the deceased individual. Services are valued at the currently prevailing federal minimum wage times the typical number of hours devoted to family services, and are valued for the remaining population life expectancy at the age at death. Lost wages are valued on the basis of the worklife expectancy at the age at death. Projected economic output is increased by projected inflation of 2 percent and discounted to present value at 5 percent. For childhood deaths the economic loss is projected over a full normal lifespan.

The average annual wage for all occupations in the United States was \$34,020 in 2001 (U.S. Dept. of Labor, 2001). We have adjusted this to a value of \$38,134 for 2004 by applying the June-to-June changes of the Employment Cost Index of the Bureau of Labor Statistics.

The average number of future years in the workforce is referred to as the worklife expectancy of the individual. This is affected by a combination of the survival probability of the individual and the rate of participation in the labor force by age. According to data provided by the U.S. Bureau of Labor Statistics (Smith, 1986), labor force participation by women has been increasing rapidly. Participation of men declined, but not as much as the increase for women. For purposes of the current estimates we have used the average of the worklife expectancy values presented by Smith for men and women. The use of worklife expectancy is consistent with the use of annual wages, since both are measured in terms of full-time, year-round employment.

Lost fringe benefits are measured as the employer cost of benefits, excluding the value of time not worked and miscellaneous benefits such as severance and discounts on products. We assume that the employer cost of benefits can be used as a proxy for part of the economic productivity of the individual. Based on data published by the U.S. Chamber of Commerce (U.S. Chamber of Commerce, 2001) the average cost of such benefits for all companies in the United States is 26.8 percent of payroll, or \$9,833 per full year worked at 2003 average wage rates.

Bryant, Zick and Kim (1992) presented an analysis of the number of hours spent on household work in relation to a number of demographic factors. Among the factors identified as significant, the number of hours is strongly related to the age and sex of the individual, the number and ages of children in the household. Data are presented only for individuals up to age 65. Since the incidence of ETS related mortality tends to be concentrated at older ages, we used data for individuals with no children under 18 years of age. We used a rough average based on this analysis of 1,700 hours per year for women, and 700 hours per year for men, resulting in an overall average based on equal numbers of men and women of 1,200 hours per year. At the current federal minimum wage rate, this has a value of \$6,180 per year of life.

Our estimate of the economic losses associated with low birth weight and SIDS are based on future earnings, fringe benefits and services assuming a normal lifetime, and assuming that both work and services start at age 20, net of the present value of costs, estimated on the basis of information from the U.S. Department of Agriculture at \$200,000, of raising a child. For low birth weight the economic costs associated with excess mortality are considered, but not costs associated with impaired function. In simple terms, the estimated economic losses connected with low birth weight and sudden infant death syndrome measure the potential economic contributions of the individuals who could have survived, but for the adverse effects of exposure to ETS. We have not included an estimate of the economic loss associated with spontaneous abortion, as we do not intend to speculate about what effects might ensue from a lost pregnancy.

Economic losses for respiratory system conditions are based on lost work time as a result of illness. Cancers of the respiratory system are treated under the heading of cancer. No increase in mortality is included in our estimates for respiratory system conditions, excluding cancer.

Because of decreased exposure of nonsmokers to ETS both direct medical costs and indirect costs associated with ETS are less than they would have been at the higher exposure levels prevalent 10 years ago. Reductions in exposure have produced current total savings of direct medical costs and indirect costs amounting to approximately \$5 billion annually compared to expected costs at the former levels of exposure. This estimate is based on the reduction in exposure to ETS as shown in Tables 3 and 4, for work exposure and for those nonsmoking individuals who were not aware of ETS exposure, but assumes no change in exposure at home, and negligible change for those exposed at home and at work. It is further based on an assumption of a linear dose-response relationship at the levels of exposure of nonsmokers to ETS, evidenced by serum cotinine concentrations less than 0.0015 micrograms per milliliter.

To obtain the above estimated savings we used serum cotinine as a proxy for ETS exposure, and estimated the average serum cotinine of nonsmokers. Serum cotinine of individuals exposed at home and at work, or at home only, was estimated at 0.00832 micrograms per milliliter for 12.6 percent of the nonsmoking population. This exposure was assumed unchanged. Exposure of individuals exposed at work only was assumed to have resulted in the decrease of serum cotinine levels from 0.000318 micrograms per

milliliter to 0.000167 micrograms per milliliter as shown in Tables 3 and 4. Exposure of individuals who were not aware of their exposure was assumed to have been reduced by 67 percent from the earlier level of 0.000124 micrograms per milliliter, as determined in *The Second National Report on Human Exposure to Environmental Chemicals*. The combined result is an estimated reduction of average serum cotinine among nonsmokers from 0.000247 to 0.000162 micrograms per milliliter, a 34 percent reduction. On the basis of a linear dose-response relationship this implies that current costs are 34 percent less than they would have been if ETS exposure levels had not been reduced.

Table 13. Estimated Economic Value of Lost Wages, Fringe Benefits, and Services per Year for the U.S. Population Excluding Infants, Based on Present Values

Category	Morbidity	Cost (\$million)
Cancer	Lung cancer	469
	Cervical cancer	110
Respiratory system	Asthma (disability only)	161
	Chronic pulmonary disease	886
Cardiovascular system	Coronary heart disease	2752
Perinatal manifestations	Low birth weight	174
Postnatal manifestations	Sudden infant death syndrome	131
Total		4,683

As an example of the calculations embodied in Table 13, consider the component arising from lung cancer cases in the 50- to 55- age group. We have assumed that 6.56 percent of lung cancer deaths occur in this age group, and that the individuals in this group would have had a work-life expectancy of 7.2 years and a life expectancy of 29.7 years if they had not had lung cancer. We used a simplified approach to valuing the economic loss by valuing an annuity for the work-life expectancy on the basis of current average wages and fringe benefits, and annuity for the life expectancy based on expected services at the current minimum wage, in both cases using inflation and discount factors cited above.

### **Use of ETS Exposure in Insurance Underwriting**

Smoking has been used as an underwriting criterion for insurance for at least 30 years. The mortality of insured smokers has been observed to be approximately twice that of nonsmokers at a wide range of ages. These considerations naturally lead to the question of whether exposure to ETS might also be used as an underwriting criterion. The lower magnitude of ETS effects, combined with the fact that exposure to ETS is correlated with criteria already used lead to the conclusion that the value of ETS exposure as an underwriting criterion is substantially less than the value of active smoking.

The design of an underwriting process involves a balance of costs and benefits. The determination of the number of rating classes and the choice of criteria for assignment to various classes are related design decisions. The desirability of including an

underwriting criterion related to exposure to ETS depends on the integrated design of a class structure and rating criteria.

### Life Insurance Underwriting

In the case of life insurance, a key element of the design of the underwriting system is the decision as to whether or not most applicants will qualify for the lowest price class. For the great majority of companies, even with a preferred risk underwriting category, a relatively large number of applicants, such as 50 percent or more, will qualify for the lowest price class. In this case, if the cost of including an underwriting criterion is less than the related reduction in claim costs, the criterion can be considered for inclusion in the underwriting process. Typically, underwriters prefer that the reduction in claims exceed the cost by a wide margin, as the complexity of the underwriting process can have a negative effect on sales.

An exception to the underwriting philosophy described above could occur in the case of companies for which only a small number of applicants qualify for preferred-risk life insurance pricing. The cost-benefit relationship for these companies may not follow the pattern described in the preceding paragraph, as the price is more sensitive to the underwriting classification. We do not consider this category of companies further, as its underwriting structure is more involved with their target markets and need for competitive premium rates than with selection of risks.

Assuming that the underwriting criteria are intended to limit claim costs in relation to a predetermined price for a standard risk, we can evaluate the cost-benefit relationship for using ETS exposure as an underwriting criterion. As explained below, because of the low concentration of cotinine associated with exposure to ETS it is much more expensive to use cotinine to evaluate exposure of nonsmokers to ETS than to use a cotinine test as a screen for smokers. This implies that the criteria used for exposure to ETS may need to be based on a questionnaire, rather than a laboratory test. In addition, it would seem, as a practical matter, that the questionnaire would have to focus on current, rather than historical, exposure. The definitions of ETS exposure, other than a definition based on cotinine, are generally qualitative, and somewhat subjective. It may be difficult to apply such a criterion without a high risk of evasion on the part of applicants. In insurance underwriting the test for cotinine is based on a urine specimen, rather than on the use of a blood sample.

The preferred basis for the use of cotinine levels in scientific studies is serum cotinine. This is because that value is more consistently related to nicotine exposure than is urine cotinine, because the relative urine volume affects the concentration of cotinine in the urine. On the other hand, for underwriting purposes urine cotinine has the advantage of being concentrated through preferential excretion to a level approximately ten times the level of serum cotinine for the individual. When urine cotinine is used as a screening test for smokers, the variability of concentration in relation to nicotine exposure is not an issue, because the difference in levels between smokers and nonsmokers is so great that the risk of error is very low. If cotinine were to be used for underwriting nonsmokers the

variability of urine cotinine concentration in relation to exposure might make it necessary to rely on a test of serum cotinine, since the degree of exposure would be of specific interest.

We did not allocate excess mortality related to ETS exposure to age groups, so we do not, at this time, have a computation of the relative increase in mortality caused by exposure to ETS. In the course of our analyses we computed ETS-related excess mortality from cancer, heart disease and chronic obstructive pulmonary disease at a level of approximately 40,000 additional deaths per year. In comparison to about two million annual deaths in the United States, this indicates that, on a very crude, overall basis the excess mortality is in the range of values that would seem reasonable. This increase in mortality may currently be included with the rates assigned to existing underwriting criteria that are related to socioeconomic level or occupation, or to existing morbidity already induced by exposure to ETS. At this level of excess mortality it would not seem practical to use ETS exposure as an underwriting criterion. While the excess mortality estimated in this paragraph would hardly be considered a precise estimate, it is enough to provide a general indication of the potential usefulness of ETS exposure as an underwriting criterion.

#### Medical Expense Insurance Underwriting

If exposure to ETS were to be included in an underwriting system for individual medical expense insurance, we would assume that the system already includes a classification for smokers vs. nonsmokers. Many family policies that would cover individuals exposed to ETS would include at least one active smoker, so would be underwritten on the basis of the costs for smokers. Therefore, somewhat less than the 23 percent of the population exposed to ETS would be in the category of policies including ETS exposure but not active smokers.

If, within the narrow range of ETS exposures, a linear dose-response is assumed, and on the basis of exposure amounts in Table 1, nonsmokers exposed to ETS at work only would account for 36.2 percent of the excess medical cost due to ETS for individuals 17 years of age and older. This would be approximately \$1.8 billion, incurred by the 10.1 percent of the population exposed to ETS at work only. This represents about 2.8 percent excess expenses for this group. This represents about one-fifth of the excess cost for individuals who are overweight, or 8 percent of the excess cost for individuals who are obese. This level of excess cost would place a tight constraint on the level of administrative effort that could be justified to include it in the underwriting system. This is, of course, a decision for each company. The potential excess costs associated with chronic exposure to secondhand smoke need to be weighed against the systems costs necessary to assess and administer a differential rating system.

On the basis of a hypothetical linear dose-response relationship excess costs incurred by individuals exposed to ETS at home but not at work would be 5.6 percent of the cost for unexposed individuals, and the excess cost for those exposed both at home and at work would be 8.4 percent of the cost for unexposed individuals.

## Use of Cotinine for Underwriting Exposure to ETS

Serum cotinine levels in nonsmokers are typically below 0.001 microgram per milliliter, about one three-hundredth of the level in smokers. Testing for concentrations this low currently requires sophisticated methods that involve liquid chromatography, or the use of radioactive materials and mass spectrometry, a different and much more sophisticated test than is needed to screen for smokers. No commercial provider of such tests exists. If a commercial market for such tests were to develop, the cost would be relatively high compared to other tests used in underwriting. The cost of such a test, if available, would currently be much higher than the typical laboratory tests used for underwriting. As noted above, current tests for cotinine in underwriting are based on a urine specimen. In situations in which no blood specimen is currently taken, there would be an issue of additional cost as well as the possibility that the customer might view the test as more invasive.

To the extent that there is latency in the health effects of ETS exposure the underwriting criterion necessary to screen out individuals subject to latent conditions would have to be an approach based on exposure over time, and not just current levels of exposure. This limits the effectiveness of a test for serum cotinine.

### **Summary and Conclusion**

Exposure to Environmental Tobacco Smoke is a public health problem with an economic impact in the United States of many billions of dollars per year. Exposure has been greatly reduced over the last 15 years, resulting in a savings of \$5 billion annually, as explained above. This reduction in morbidity and the resulting reduction in economic losses is a major public health victory. It results from a combination of a reduction in the percentage of smokers in the population with a greater reduction in the rate at which nonsmokers are exposed to smoke. The victory may be temporary, however, if current levels of smoking among adolescents lead to future increases in the smoking percentage for the population as a whole, or if rules and attitudes that limit ETS exposure are relaxed.

The benefits of recent reductions in exposure to ETS are not felt equally throughout the population. Young children of smoking mothers continue to be exposed at a higher level than any other group of nonsmokers, and the reductions in exposure for this segment of the population are small. The reductions have not occurred equally for all ethnic and socioeconomic groups (Wortley, et al., 2002). There is work yet to be done to extend the benefits of reductions in exposure to all groups.

The authors hope that the data presented in this paper can be used by insurance companies and employers in evaluating the effects of exposure to ETS on medical costs

and in public-policy discussions to help to quantify the smoking-related costs borne by the nonsmoking population.

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Appendix 1

**Summary of Research by Morbidity**

Morbidity	Impact of ETS Exposure	Papers	Findings
<p><b>Perinatal manifestations</b></p>	<p>(1) Fetal Growth                      - birthweight                      - low birthweight                      - growth retardation                      - prematurity</p>	<p>MacMahon <i>et al.</i> (1966)</p>	<p>The study found an 86.8-gram decrement associated with any paternal smoking for female infants, with a slightly lower decrement for male infants (-78 grams).</p>
		<p>Comstock and Lundin (1967)</p>	<p>The mean birth weight of infants with smoking fathers and nonsmoking mothers was 42 grams less than that of infants whose parents both did not smoke.</p>
		<p>Underwood <i>et al.</i> (1967)</p>	<p>The authors found that mean birth weight was decreased only 3-7 grams depending on the amount smoked by the father.</p>
		<p>Borlee <i>et al.</i> (1978)</p>	<p>There was an association between paternal smoking and birth weight.</p>
		<p>Magnus <i>et al.</i> (1984)</p>	<p>Maternal smoking is significantly associated with birth-weight decrements.</p>
		<p>Karakostov (1985)</p>	<p>The author reported an 84-gram weight decrement in infants of women exposed to ETS during pregnancy compared to infants whose parents were both nonsmokers.</p>
		<p>Rubin <i>et al.</i> (1986)</p>	<p>The independent decrement in birth weight per cigarette (or cigar or pipe bowl) smoked daily by the father was 6.1 grams (<math>p &lt; 0.03</math>).                      The decrement seen with maternal smoking was 9.2 grams per cigarette per day (adjusted for paternal smoking and other variables).</p>

		MacArthur and Knox (1987)	The authors found 14-gram decrement in mean birth weight if the father smoked.
		Schwartz-Bickenbach et al. (1987)	Those infants with smoking fathers and nonsmoking mothers weighed on average 205 grams less than infants whose parents did not smoke. The decrement associated with maternal smoking was on the order of 400 grams.
		Campbell <i>et al.</i> (1988)	Current paternal smoking status was associated with a 113-gram decrement in birth weight, about one-half the effect of maternal smoking (-253 grams) in all births.
		Brooke <i>et al.</i> (1989)	The authors found a difference in mean birth weight (adjusted to 40 weeks) associated with ETS exposure of 18 grams in nonsmokers and 39 grams in smokers.
		Chen <i>et al.</i> (1989)	Mean birth weight was decreased only 9-11 grams, depending on the amount smoked by the spouse and was decreased 4-15 grams, depending on the amount smoked by all family members. The authors found no evidence of a dose-response relationship of amount smoked by the father or by all household members to rates of low birth weight (LBW).

		Saito (1991)	Among infants whose father smoked, but whose mother did not smoke during pregnancy, there was a decrement in mean birth weight of 33.4 grams ( $p < 0.05$ ) compared to infants of nonsmoking parents. Among infants whose parents both smoked, the mean birthweight was further decreased 66 grams. The rate of prematurity did not vary by paternal smoking status.
		Mathai <i>et al.</i> (1990 and 1992)	Adjusting for multiple confounders, the mean decrement was 63 grams because of ETS exposure.
		Zhang and Ratcliffe (1993)	There was a crude weight decrement of 26 grams associated with paternal smoking. The rates of LBW at term and intrauterine growth retardation (IUGR), were similar whether the father was a smoker or a nonsmoker.
		Martinez <i>et al.</i> (1994)	The rates of LBW at term and Infant birth weight significantly decreased with increasing paternal smoking;
		Yerulshalmy (1971)	The proportion of LBW infants from pregnancies in which the husband smoked was increased significantly compared to those in which the husband did not smoke.
		Mau and Netter (1974)	The investigators found slight increases in each outcome IUGR, prematurity and LBW among infants of fathers who smoked more than 10 cigarettes per day.

		Nakamura <i>et al.</i> (1988)	Focusing on nonsmoking mothers only, the crude rates for positive paternal smoking status were increased for LBW, and slightly for pre-term and SGA births.
		Mathai <i>et al.</i> (1992)	The rate of prematurity was increased somewhat with ETS exposure.
		Martin and Bracken (1986)	There was no association of prematurity with ETS exposure.
		Ogawa <i>et al.</i> (1991)	The crude and adjusted odds ratio for LBW at term did not indicate any increased risk with ETS exposure.
		Lazzaroni <i>et al.</i> (1990)	Mean birth weight of infants of women exposed to ETS was reduced 51 grams, which was not statistically significant.
		Ahlborg and Bodin (1991)	Among these working women, home exposure was associated with a 34-gram decrement in mean birth weight, but workplace exposure was not associated with a birth weight reduction.
		Fortier <i>et al.</i> (1994)	ETS exposure at home only was not associated with IUGR. The risk of IUGR associated with workplace-only exposure was slightly greater, and showed evidence of a slight dose-response trend with heavier exposure. Women exposed both at home and at work had IUGR rates more similar to the at-home-only exposed women. ETS exposure at any location was not associated with pre-term birth.

		Mainous and Hueston (1994)	Comparing mean birth-weight, women in the highest exposure category had infants that weighed on average 84 grams less than infants in the very low exposure category.
		Chen and Pettiti (1995)	There was no indication of an increased risk of term IUGR with greater exposure.
		Roquer <i>et al.</i> (1995)	The rate of IUGR was about doubled with ETS exposure.
		Hauth <i>et al.</i> (1984)	The authors reported that infants of passive smokers had similar birth weights to those of nonsmokers.
		Ueda <i>et al.</i> (1989)	The LBW of infants of women with higher cotinine levels was lower than that of infants of women with lower cotinine levels.
		Eskenazi <i>et al.</i> (1995)	Examining cotinine as a continuous variable, there was a 1-gram weight decrement for each nanogram per milliliter increase in cotinine. The authors also found a slight increase in LBW associated with ETS exposure, but found no effect on gestational age or prematurity.
	(2) Spontaneous abortion & perinatal mortality (stillbirths & neonatal deaths)	Comstock and Lundin (1967)	The authors reported “no significant differences” in the rates of stillbirth by paternal smoking status. Neonatal death rates were elevated in infants with nonsmoking mothers and smoking fathers compared to infants with no parental smokers. Neonatal death rates were highest when both parents smoked.
		Tokuhata (1968)	The results showed that husbands’ smoking status was unrelated to fetal loss.

		Yerushalmy (1971)	He found higher mortality rates among LBW births to couples in which the father was a smoker, particularly among blacks.
		Mau and Netter (1974)	The authors found an increased rate of perinatal mortality among pregnancies where the father smoked 10 or more cigarettes per day, both for all women and for nonsmoking women.
		Koo <i>et al.</i> (1988)	Women whose husbands had ever smoked were 40 percent more likely to have had a miscarriage or abortion and twice as likely to have had a dilation and curettage (D & C) than wives of nonsmokers.
		Ahlborg and Bodin (1991)	ETS exposure (any versus none) was not found to be associated with excess risk for hospital-ascertained intrauterine deaths (spontaneous abortions plus stillbirths) among nonsmoking mothers.
		Windham <i>et al.</i> (1992)	This study lends some support to the findings of Ahlborg and Bodin (1991) of an increased risk of fetal death associated with ETS exposure.
		Windham <i>et al.</i> (1995)	The finding of an association between ETS exposure and spontaneous abortion was not confirmed.
	(3) Congenital malformations - neural tube defects (e.g., anencephaly, spina bifida) - cleft palate - defects of the genitourinary and	Mau and Netter (1974)	The rates of severe malformations among all newborns increased with amount smoked by the father. No association was found with maternal smoking.
		Holmberg and Nurminen (1980)	Maternal smoking showed a greater association with central nervous system defects.

	the cardiovascular systems	Hearey <i>et al.</i> (1984)	Paternal smoking was the only variable found significantly associated with neural tube defects.
		Seidman <i>et al.</i> (1990)	The authors noted non-significant increases in rates of minor and major malformations associated with heavy paternal smoking.
		Savitz <i>et al.</i> (1991)	A dose-response relationship for smoking one pack or more per day was suggested only for the clefts and urethral stenosis.
		Zhang <i>et al.</i> (1992)	The overall odds ratio of birth defects and paternal smoking was slightly elevated with little evidence of a dose-response effect.
		Shaw and Wasserman (1993) Wasserman <i>et al.</i> (1994)	Exposure to others' smoke at work, or at places other than home, led to slightly increased risks among infants of maternal nonsmokers, as well as among smokers.
<b>Postnatal Manifestations</b>	(1) Sudden Infant Death Syndrome (SIDS)	Bergman and Wiesner (1976)	They found an overall crude odds ratio (OR) for any maternal smoking during pregnancy of 2.2, and for any maternal smoking after pregnancy of 2.4.
		McGlashan (1989)	There was virtually complete overlap between the women who smoked during pregnancy and who smoked during the infants' first year of life, with ORs for both forms of maternal smoking of 1.9.
		Mitchell <i>et al.</i> (1991)	Any maternal smoking in the 2 weeks prior to interview had an OR of 1.8.

		Nicholl and O’Cathain (1992)	Smoking by the partner was associated with an OR of 1.6 whereas smoking (during pregnancy) by the mother was 2.1. Prenatal exposure to maternal active smoking components may be more important for younger infants, and postnatal ETS exposure more important for older infants.
		Schoendorf and Kiely (1992)	The risk of SIDS among infants of women who smoked both during and after pregnancy was 3.1 and 3.1 for whites and blacks respectively.
		Mitchell <i>et al.</i> (1993)	There was a strong dose-response relationship observed between amount smoked by the mother and risk of SIDS. The researchers also found a significant relationship between recent paternal smoking and SIDS, with a crude OR of 2.4 and an adjusted OR of 1.4. When parents were included, there was a strong dose-response relationship between the number of household smokers and the risk of SIDS.
		Mitchell <i>et al.</i> (1995)	There was no discernible pattern in risk between locations or between smoking frequency, with ORs associated with maternal smoking ranging from 1.7 to 3.0. Mothers who smoked but claimed they never did so in the house had a higher risk of SIDS than did mothers who smoked in the house.

		Haglund <i>et al.</i> (1995)	The authors found that the winter season and maternal smoking were both independent risk factors for SIDS, but that the excess risk due to smoking did not vary by season. The excess relative risk of smoking was approximately 3.5 for early SIDS deaths (7-90 days) and 2.5 for late SIDS deaths (91-364 days).
		Klonoff-Cohen <i>et al.</i> (1995)	Measures of ETS exposure were associated with increased SIDS risk. Increased risk was also seen with increasing number of household smokers and with total cigarette exposure per day.
		Blair <i>et al.</i> (1996)	The study documented significant, dose-related increases in SIDS risk for the three main measures of household smoke that were examined: the number of smokers in the household, the hours of smoke exposure to the infant daily, and the number of cigarettes smoked daily in the household. They also demonstrated elevated risk in families where the mother was a nonsmoker and either the father or another family member smoked.
	(2) Neuropsychological development	Makin <i>et al.</i> (1991)	Scores of an extensive neuropsychological test of children of ETS-exposed mothers tended to fall between those of children of non-exposed and active-smoking mothers.

		<p>Eskenazi and Trupin (1995)</p>	<p>Children whose mothers were exposed to ETS during pregnancy did not have Raven or PPVT scores that differed significantly from the scores of children with no smoking exposure.</p> <p>The OR for “active” behavior among children whose mothers were exposed to ETS during pregnancy was somewhat elevated.</p> <p>Raven and PPVT scores for children with prenatal or postnatal exposure only were not significantly lower than scores for children with no smoking exposure. In contrast, Raven and PPVT scores for children with both pre- and postnatal exposure were lower than those for children with no exposure.</p>
		<p>Rantakallio (1983)</p>	<p>Both maternal and paternal smoking were significant predictors of the school performance score.</p>
		<p>Bauman <i>et al.</i> (1989)</p>	<p>The authors found an inverse relationship between family smoking and their test scores of the California Achievement Test (CAT).</p>

		Bauman <i>et al.</i> (1991)	For all Raven and PPVT exams, mean scores were 3-10 percent lower in children of current smokers, independent of maternal smoking status during pregnancy. There was little difference in mean scores by prenatal smoking status after stratification by current parental smoking. Current parental smoking had a negative effect on PPVT and Raven scores, whereas the mother's smoking during pregnancy had a negligible effect.
		Baghurst <i>et al.</i> (1992)	The authors found postnatal smoking had a negative effect on the Bayley and the McCarthy scores.
		Weitzman <i>et al.</i> (1992)	Smoking was associated with BPI score in a dose-related manner in two groups of children with mothers who smoked: children whose mothers smoked after pregnancy only, and children whose mothers smoked both during and after pregnancy.
	(3) Postnatal physical development	Eskenazi and Bergmann (1995)	Children of ETS-exposed pregnancies were on average 0.4 cm higher than non-smoke-exposed children.
		Rona <i>et al.</i> (1981 and 1985)	Rona <i>et al.</i> (1981) found an inverse association between the number of people smoking more than five cigarettes per day at home and the standardized height of the child. Children of smokers were slightly but significantly shorter than non-exposed children.

		Chinn and Rona (1991)	Postnatal ETS exposure has no effect on children's height.
		Rantakallio (1983)	The adjusted height decrement associated with maternal smoking during pregnancy was approximately -0.9 cm.
		Berkey <i>et al.</i> (1984)	The authors found a very significant dose-related decrease in height with increasing current maternal cigarette consumption.
<b>Reproductive effects</b>	(1) Female fertility and fecundability	Tokuhata (1968)	The crude odds ratio for fertility among couples in which the wife did not smoke and the husband did smoke was calculated as 0.67.
		Olsen (1991)	Current smoking by the woman's partner was associated with a delay to conception in the pregnancies of both smoking and nonsmoking women.
		Wilcox <i>et al.</i> (1989)	The authors found that women exposed to ETS as children became pregnant faster than unexposed women. In utero exposure (exposure due to the woman's mother smoking during pregnancy) to maternal smoking showed a weak association with reduced fecundability.
		Weinberg <i>et al.</i> (1989)	The authors reported that in utero exposure reduced fecundability.
		Schwingl (1992)	Childhood ETS exposure (one or two parents smoking) was associated with fecundability ratios (FRs) of 1.1-1.2. Current smoking by the daughters was not associated with fecundability.

	(2) Other Female Reproductive Effects - menopause - rates of menstrual disorders	Everson <i>et al.</i> (1986)	The authors reported an association of ETS exposure and lower age at menopause. The authors found that childhood exposure to paternal smoking was not associated with early menopause.
<b>Respiratory health effects</b>	(1) Acute Health Effects - asthma (exacerbation) - respiratory infections (children) - otitis media (OM) (children) - sensory irritation and annoyance (eye irritation, nasal irritation, alteration of sensory thresholds, odor annoyance)	Evans <i>et al.</i> (1987b)	ER (emergency room) visits were positively associated with reported ETS exposure
		Chilmonczyk <i>et al.</i> (1993)	ETS exposure was found to be associated with increased frequency of asthma exacerbations in a dose-dependent manner.
		Murray and Morrison (1989)	Children of smoking mothers had more severe asthma than children of nonsmoking mothers.
		O'Connor <i>et al.</i> (1987)	Maternal smoking emerged as a significant ( $p = 0.02$ ) predictor of forced expiratory volume (FEV), after adjusting for predicted FEV.
		Strachan and Carey (1995)	While paternal smoking was unrelated to the outcomes examined, maternal smoking of >10 cigarettes/day was significantly related to the combined category of frequent wheezing plus speech-limiting attacks. After adjusting for numerous other household factors, the odds ratio for maternal current smoking was still elevated, but no longer significant.

		F.D. Gilliland et al. (2003)	Children's asthma status affected their response to ETS. Children without asthma also had an increased risk if exposed to two or more smokers (RR=1.44, 95 percent CI: 1.04, 2.00). Therefore, ETS exposure is associated with increased respiratory-related school absenteeism among children, especially those with asthma.
		Jindal <i>et al.</i> (1994)	In comparison with a non-exposed group of 100 patients, the ETS- exposed group showed significantly lower forced expiratory lung function indices.
		Bailey <i>et al.</i> (1990)	The investigators found no relationship between asthma severity and passive smoking,
		Ehrlich <i>et al.</i> (1992)	There was no significant difference between acute and non-acute asthmatics in relation to maternal smoking.
		NRC (1986) U.S. DHHS (1986) U.S. EPA (1992)	ETS exposure increases the risk of acute lower respiratory disease in young children by 1.5 to twofold.
		Douglas <i>et al.</i> (1994)	Maternal smoking was associated with a significantly increased frequency of respiratory illness in the second, but not the first year of life.

		Nicholson et al. (2003)	The study shows a small but sustained increase in prevalence of wheeze in Irish schoolchildren when compared to previous Irish studies carried out by Loftus in 1993 and Taylor in 1996. In this study, male sex is a significant risk factor for the development of both wheezing and rhinitis but not eczema. 782 (41 percent) of children were exposed to passive smoking at home and they found that passive smoking at home is a risk factor for the development of wheeze, but not eczema or rhinitis. Maternal smoking during pregnancy was not found to be significantly associated with wheeze, contrary to a large U.K study.
		U.S. EPA (1992)	...there was “good evidence demonstrating a significant increase in the prevalence of middle ear effusion in children exposed to ETS,” but only “some evidence [for] acute middle-ear infections” (acute otitis media).
		Fleming <i>et al.</i> (1987)	For children under 5 years old, maternal smoking was significant risk factor for upper respiratory tract infection, but not otitis media.
		Teele <i>et al.</i> (1989)	Parental smoking was significant risk factor for acute otitis media (OM) in children under one year only.
		Takasaka (1990)	No association was reported for ETS exposure and OM.

		Kallail <i>et al.</i> (1987)	There was a non-significant excess of ETS exposure among the children with hearing problems who were later confirmed by physicians to have "middle-ear problems."
		Ra (1992)	ETS exposure at home was associated with a 4.9-fold increase in hearing loss.
		Collet <i>et al.</i> (1995)	Trend of increasing risk of recurrent OM with increasing number of cigarettes smoked. No effect of paternal smoking.
		Ey <i>et al.</i> (1995)	Maternal smoking of 20 cigarettes/day associated with increased risk of recurrent OM. No effect of paternal smoking.
		Weber <i>et al.</i> (1976, 1984, 1987), Muramatsu <i>et al.</i> (1983)	The authors exposed volunteers to progressively increasing concentrations of ETS; as exposure duration and intensity increased, subjects began to report subjective eye irritation and blink rate also increased.
		Basu <i>et al.</i> (1978)	The tear film was less stable after ETS exposure.
		Bascom <i>et al.</i> (1991), Willes <i>et al.</i> (1992)	As a group, historically ETS sensitive, but not ETS non-sensitive, subjects showed significant increases in nasal airway resistance (NAR) by rhinomanometry after 15-minute exposures to STS at levels chosen to simulate a smoking lounge.
		Ahlstrom <i>et al.</i> (1987)	Both active and passive smokers reported lower perceived odor intensities (i.e., were less sensitive) than nonsmokers.
	(2) Chronic health effects	U.S. EPA (1992)	ETS is a risk factor for inducing new cases of asthma.

<ul style="list-style-type: none"> <li>- asthma (induction)</li> <li>- chronic respiratory symptoms (children)</li> <li>- decreased lung development (children)</li> <li>- chronic pulmonary disease and respiratory symptoms (adults)</li> </ul>	Bråbäck <i>et al.</i> (1995)	There were findings of other significant associations between maternal smoking and respiratory symptom indices in Poland and Estonia, but not in Sweden.
	Cuijpers <i>et al.</i> (1995)	This study reported significant associations between ETS exposure and cough for 11-20 cigarettes per day in boys, but not for <11 or >20 cigarettes per day, and it found no significant associations for girls.
	Moyes <i>et al.</i> (1995)	They reported that parental ETS exposure was related to nocturnal cough, nasal symptoms and wheeze in the older (ages 13-14) but not the younger (ages 6-7) children.
	Forastiere <i>et al.</i> (1992)	The authors found significantly elevated odds ratios of respiratory illness in relation to the children's exposure to passive smoking.
	Mannino <i>et al.</i> (1996)	They found that ETS-exposed children had 21 percent more restricted activity days, 31 percent more days of bed confinement, and 39 percent more days of school absence than those not exposed.
	Sherrill <i>et al.</i> (1992)	There were no statistically significant ETS-related effects on FEV or forced vital capacity (FVC) in males, though females whose parents both smoked tended to have a slower rate of growth in FEV, and those exposed to maternal smoking tended to have a lower FVC than the non-exposed.

		Wang <i>et al.</i> (1994)	Both current maternal smoking and pre-school exposure to maternal smoking were significant predictors of the children's pulmonary function. Early maternal smoking was also associated with a small increase in FVC, which was statistically significant only in children aged 11 to 18. In children aged 6 to 10, current maternal smoking was related to slower growth rates of both FVC and FEV, and in older children, with a reduction in the growth rate of FEF <sub>25-75</sub> (expiratory flow during the middle half of a forced vital capacity (FVC) maneuver -- an indicator of the caliber of the more peripheral, mid-sized to smaller airways).
		Enstrom and Kabat (2003)	For participants followed from 1960 until 1998 the age adjusted relative risk (95-percent confidence interval) for never smokers married to ever smokers compared with never smokers married to never smokers was 1.27 (0.78 to 2.08) for chronic obstructive pulmonary disease among 9619 men, and 1.01 (0.94 to 1.08), 0.99 (0.72 to 1.37), and 1.13 (0.80 to 1.58), respectively, among 25,942 women. The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect.

		Rona and Chinn (1993)	The investigators found significant associations of maternal smoking with reduced FEF <sub>25-75</sub> and FEF <sub>75-85</sub> in boys, but not girls. The authors also found an association between reduced childhood lung function and maternal but not paternal smoking.
		Cunningham <i>et al.</i> (1994 and 1995)	The investigators found decrements in FEV <sub>0.75</sub> , FEV <sub>1</sub> , FEV <sub>1</sub> /FVC, PEFR, FEF <sub>25-75</sub> , and FEF <sub>65-75</sub> that were highly significantly related to both maternal smoking during pregnancy and to maternal ETS exposure in the year preceding the examination.
		White and Froeb (1980)	For both sexes, nonsmokers who reported chronic workplace exposure to ETS had, as a group, significantly lower FEF <sub>25-75</sub> (forced expiratory flow during the middle half of the forced vital capacity maneuver) and FEF <sub>75-85</sub> values than did non-ETS exposed nonsmokers.
		Comstock (1981)	Most respiratory tract symptoms were equally prevalent among those exposed and not exposed.
		Kauffman <i>et al.</i> (1983 and 1989)	For most lower respiratory symptoms, odds ratios comparing passive smokers with nonsmokers were elevated, but not to a statistically significant degree.

		Kentner <i>et al.</i> (1984)	Whereas active and prior smokers had significantly lower age- and sex-standardized ventilatory parameters, no such changes were evident among persons whose sole lifetime exposure was passive smoking.
		Brunekreef <i>et al.</i> (1985)	Analyzed cross-sectionally, those women 40 to 60 years of age at the time of the latest measurement who were passively exposed to cigarette smoke in the home had significantly lower peak expiratory flow (PEF) than did the unexposed group.
		Kalandidi <i>et al.</i> (1987 and 1990)	A significant trend was observed in the odds ratio for chronic obstructive pulmonary disease (COPD) and spousal smoking as the husband's estimated (during-marriage) total cigarette consumption increased.
		Euler <i>et al.</i> (1988)	Self-reported ETS exposure, both in the workplace and in the home, was significantly related to self-reported symptoms of COPD (breathlessness, sputum production and wheezing).
		Masi <i>et al.</i> (1988)	There was a significant decrement in FEF <sub>25-75</sub> , FEF <sub>50</sub> , and residual volume as a function of cumulative lifetime exposure to ETS at home (but not at work) among males only. Females showed a significant trend toward lower carbon monoxide diffusion capacity with increasing cumulative ETS exposure at work (but not at home).

		Hole <i>et al.</i> (1989)	Age-, height-, and sex-adjusted FEV <sub>1</sub> values were significantly lower among those exposed to ETS compared to those not exposed.
		Masjedi <i>et al.</i> (1990)	Significantly lower spirometric values (FEV <sub>1</sub> , FVC, and FEF <sub>25-75</sub> ) were observed for those who reported ETS exposure at work (not home) versus those who did not. Among females, no systematic differences were found with ETS exposure at either work or home.
		Jaakkola <i>et al.</i> (1995)	Jaakkola and colleagues found that work-related ETS exposure during the study period was associated with a slight, but significant increase in the rate of decline of FEV <sub>1</sub> .
		Dayal <i>et al.</i> (1994)	Dayal et al. found a significant relationship between reported obstructive lung disease and household ETS exposure involving one or more packs a day.
		Xu and Li (1995)	Xu and Li reported reduced levels of FEV <sub>1</sub> and FVC associated with ETS exposure, which were statistically significant in never-smoking men, but not women.
		Robbins <i>et al.</i> (1993)	Robbins et al. reported that several, but not all, qualitative measures of ETS exposure were associated with an increased relative risk of developing AOD, including adult plus childhood exposure, but not childhood or adult exposure alone.

		Greer <i>et al.</i> (1993)	The incidence of “definite asthma” by reported symptoms or reported physician diagnosis during the 10-year interval was significantly related to occupational ETS exposure.
		Ng <i>et al.</i> (1993)	The authors reported increased risks of chronic respiratory symptoms and reduced FEV <sub>1</sub> associated with household ETS exposure.
<b>Carcinogenic effects</b>	(1) All cancers (combined)	Sandler <i>et al.</i> (1989)	Exposure to ETS did not increase the risk for all cancers combined in nonsmoking men and nonsmoking women after adjusting for age, marital status, education and housing quality. In men and women, there was no association between ETS exposure and risk of nonsmoking-related tumors.
		Reynolds <i>et al.</i> (1987)	Nonsmoking women whose husbands smoked showed a OR of 1.68 for all cancers combined compared to women whose husbands did not smoke.
		Sandler <i>et al.</i> (1985a and b)	Among lifetime nonsmokers, there was a significant twofold increased risk associated with spouses' smoking after adjustment for gender, race and age.
		Neutel and Buck (1971)	There was a small increased risk for all cancers combined among children whose mothers smoked compared to children whose mothers did not smoke.
		Pershagen <i>et al.</i> (1992)	There was no association between maternal smoking and risk of all cancers combined.

		Golding <i>et al.</i> (1990)	Maternal smoking remained statistically significant in logistic regression analysis when other risk factors were controlled for.
		Stjernfeldt <i>et al.</i> (1986a; 1986b; 1992)	There was some suggestion of an increased risk for all cancers combined in relation to mother's smoking during pregnancy.
		McKinney and Stiller (1986)	Maternal smoking habits during pregnancy were not associated with risk for all cancers combined.
		Buckley <i>et al.</i> (1986)	There was no association between maternal smoking during pregnancy and risk of all cancers combined. Paternal smoking during the index pregnancy was also not associated with all childhood cancers combined.
		John <i>et al.</i> (1991)	The data suggest an increasing trend in risk with increasing amounts smoked by mothers, but not by fathers.
	(2) Lung cancer	Stockwell <i>et al.</i> (1992)	Compared to unexposed individuals who had no household ETS exposure, women who were exposed to husbands' smoking had ORs of 1.6 for those who had ever been exposed and 2.2 for those with 40 or more smoke-years of exposure after adjustment for age, race, and education.
		Brownson <i>et al.</i> (1992)	There was no association between risk of lung cancer and ETS exposure from parents or other household members during childhood.

		<p>Fontham <i>et al.</i> (1991 and 1994)</p>	<p>Spousal smoking was associated with a statistically significant increased risk of lung cancer.</p> <p>Exposure to other sources of ETS during adult life were also associated with an increased risk of lung cancer.</p> <p>In this study, ETS exposure during childhood/adolescence from father, mother or other household members was not associated with risk of lung cancer.</p>
		<p>Kabat <i>et al.</i> (1995)</p>	<p>There were no significant associations between spouses' smoking and risk of lung cancer in male or female subjects.</p> <p>Household exposure was not significantly associated with risk of lung cancer.</p> <p>Workplace ETS exposure was not associated with increased risk of lung cancer in males or females in this study. There were small increased risks for lung cancer associated with ETS exposures in social situations and inside cars.</p> <p>Exposure to ETS during childhood was not associated with any increased risk in males, but it was associated with an increased risk in females of borderline statistical significance.</p>

		Liu <i>et al.</i> (1993)	Compared to nonsmoking women who were not exposed to husband's smoking, women exposed to one to 19, and 20+ cigarettes per day of husband's smoking showed ORs of 0.7 and 2.9, respectively after adjusting for education, occupation and living area. Risk of lung cancer was increased in association with living in a house with poor air circulation.
		Schwartz <i>et al.</i> (1996)	After adjustment for age, race and sex, exposure to ETS at home was not a significant risk factor for lung cancer, while exposure to ETS at work was of borderline statistical significance.
		Ko <i>et al.</i> (1997)	Risk of lung cancer in nonsmoking women was not associated with ETS exposure from parents, cohabitants or coworkers, but there was a small non-significant increased risk associated with ETS exposure from spouses.
		Cardenas <i>et al.</i> (1997)	In the analyses based on spousal smoking habits, never smoking women married to smokers showed a small increased risk of lung cancer. There was an increasing trend of risk associated with number of cigarettes smoked by spouses.

		Enstrom and Kabat (2003)	For participants followed from 1960 until 1998 the age adjusted relative risk (95-percent confidence interval) for never smokers married to ever smokers compared with never smokers married to never smokers was 0.75 (0.42 to 1.35) for lung cancer. The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect.
	(3) Nasal sinus, cervical and bladder	Hirayama (1983 and 1984)	The author reported an increased risk of para-nasal sinus cancer among nonsmoking women exposed to husbands' smoking.
		Fukuda and Shibata (1988 and 1990)	Exposure to ETS was associated with a small, non-significant increased risk of nasal cancer. Active smoking was associated with a non-significant increased risk of nasal cancer in women.
		Sandler (1985a)	Spouses' smoking habits were associated with an increased risk of cervical cancer in nonsmokers after adjustment for age, race, education and smoking habits of parents. Husbands' smoking also increased risk of cervical cancer in women who were smokers. Maternal smoking was not associated with risk of cervical cancer whereas paternal smoking was associated with a statistically non-significant increased risk.

		Slattery <i>et al.</i> (1989)	Among nonsmokers, ETS exposure inside and outside of the home was associated with a significantly increased risk with adjustment for potential confounders which included age, education, church attendance and number of sexual partners of the woman.
		Coker <i>et al.</i> (1992)	There was no significant or consistent association between ETS exposure at work or at home and risk of cervical cancer/intraepithelial Neoplasia (CIN).
		Kabat <i>et al.</i> (1986)	For nonsmoking males, there was a non-significant increased risk of bladder cancer associated with ETS exposure at home but not at work, whereas among nonsmoking females, a non-significant increased risk was observed for ETS exposure at work but not at home.
		Burch <i>et al.</i> (1989)	There was no association between risk of bladder cancer and ETS exposure at home or at work.
	(4) Breast cancer	Hirayama (1984)	Nonsmoking women whose husbands smoked showed a small, non-significant increased risk of breast cancer.
		Wells (1992)	Compared to nonsmoking women married to never smokers, the age-adjusted ORs were 1.62 among nonsmoking women married to smokers, 0.64 among smoking women married to nonsmokers and 1.51 among smoking women married to smokers.

		Smith <i>et al.</i> (1994)	Although there was an increased risk of breast cancer associated with childhood ETS exposure, adult exposure to ETS from partners, from other smokers at home and at work, and total lifetime exposure, there was no consistent dose trend of increasing risks with increasing levels of any of these sources of ETS exposure.
		Morabia <i>et al.</i> (1996)	Compared to nonsmoking women who were not exposed to any ETS, the OR was 2.6 for women who were exposed to passive smoking from spouses and 2.3 for women who were ever exposed to passive smoking from all sources combined.
	(5) Stomach cancers	Hirayama (1984)	The author didn't find an association between ETS exposure and risk of stomach cancer in nonsmokers.
	(6) Brain tumors	Howe <i>et al.</i> (1989)	Maternal and paternal smoking during index pregnancy was associated with a small, non-significant increased risk of brain tumor.
		Gold <i>et al.</i> (1993)	There was no association between risk of childhood brain tumor and maternal or paternal smoking at any time, specifically during the year the index child was born, or in the two years before the index child was born.
		McCredie <i>et al.</i> (1994)	Increased risks were found in relation to smoking by either parent and to mothers' smoking.
		Kuijten <i>et al.</i> (1990)	Mothers' smoking and mothers' exposure to side-stream smoke were not associated with risk of astrocytoma.

(7) Leukemia, lymphomas and non-Hodgkin's lymphomas	Neutel & Buck (1971)	The rate of leukemia in children was higher among mothers who smoked compared to mothers who did not smoke.
	Pershagen <i>et al.</i> (1992)	There was no increased risk associated with mothers' smoking during pregnancy for lymphatic leukemia when year and county of birth, birth order of index subject and maternal age were adjusted for in the analysis.
	Magnani <i>et al.</i> (1990)	No association between parental smoking and risk of leukemia in children was found in a hospital-based case-control study conducted in the main pediatric hospital in Turin, Italy between 1981 and 1984.
	McKinney and Stiller, (1986)	The authors found a 90-percent increase in risk of lymphomas in subjects whose mothers' smoked one to 10 cigarettes/day during pregnancy, but there was no increased risk for subjects whose mothers who smoked more.
(8) Other rare childhood cancers - neuroblastoma - Wilms' tumor of the kidney - Germ cell tumors - Bone and soft-tissue sarcomas	Kramer <i>et al.</i> (1987)	A small increased risk was observed for mothers' smoking during pregnancy and at any time prior to conception of the index child. Fathers' smoking during the two years prior to birth of the index child conferred a similar increase in risk.
	Bunin <i>et al.</i> (1987)	There is no association between maternal smoking during pregnancy and risk of Wilms' tumor.

		Grufferman <i>et al.</i> (1982)	Risk of rhabdomyosarcoma (RMS) was not related to mothers' smoking at any time, or mothers' smoking during the pregnancy of the index subject. On the other hand, fathers' smoking was a statistically significant risk factor.
		Magnani <i>et al.</i> (1989)	No association between paternal and maternal smoking habits and risk of RMS and non-RMS-soft tissue sarcomas (STS) was reported by Magnani <i>et al.</i> (1989).
<b>Cardiovascular Health Effects</b>	Coronary heart disease (CHD) - myocardial infarction (MI) - angina pectoris (AP) - sudden unexpected death (SUD)	Hirayama (1981, 1984, 1990)	Compared to nonsmoking women married to nonsmokers, women married to exsmokers or smokers of one to 19 cigarettes/day, and smokers of 20+ cigarettes/day, showed relative risks of 1.10, and 1.31, respectively, for coronary heart disease (CHD).
		Garland <i>et al.</i> (1985)	The age-adjusted mortality rates were 1.2, 3.6 and 2.7, respectively, for women married to nonsmokers, ex-smokers, and current smokers.
		Svensden <i>et al.</i> (1987)	Compared to men married to nonsmokers, men married to smokers showed a higher risk of death from CHD and for fatal and nonfatal CHD combined after adjusting for other risk factors for heart disease.
		Helsing <i>et al.</i> (1988)	The adjusted relative risk for any ETS exposure was 1.31 for men and 1.24 for women.

		<p>Hole <i>et al.</i> (1989)</p>	<p>Passive smokers compared to controls did not differ in self-reported prevalence of angina and the relative risk estimate was not significantly changed after adjusting for potential confounders including age, sex, social class, diastolic blood pressure, serum cholesterol and body mass index.</p> <p>The risk for angina was elevated for those more highly exposed to ETS smoking. Compared to women with no exposure, the relative risk was 2.09 for women with low ETS exposure and 4.12 for women with high ETS exposure. Risk for angina (adjusted for age and sex) increased 2 percent for passive smokers, 67 percent for active smokers living with nonsmokers, and 98 percent for active smokers living with smokers compared to controls.</p>
		<p>Humble <i>et al.</i> (1990)</p>	<p>Nonsmoking women married to current smokers showed higher risks for all CHD mortality than women married to never smokers.</p>

		Butler (1988)	<p>Based on 87 CHD deaths in nonsmoking women, those married to ex-smokers did not show any elevation in risk compared to nonsmoking women married to nonsmokers.</p> <p>Years of working with a smoker was also not associated with risk of CHD in men, but was associated with an increased risk of CHD in women, after adjusting for age. Exposure to ETS at home and at work increased the risk of heart disease mortality in nonsmoking women but not in nonsmoking men.</p>
		LeVois and Layard (1995)	<p>Nonsmoking men and women whose spouses were former smokers showed risks for CHD that were close to 1.0. After adjusting for age and race, there was no association between any ETS exposure from spouses and risk of CHD mortality in men or in women. However, in both men and women, there was some increase in risk when amount smoked by spouses was considered.</p>

		Steenland <i>et al.</i> (1996)	<p>Small increased risks for CHD mortality in men and women in association with current exposure to spouses' smoking were found.</p> <p>There was, however, no association between risk of CHD mortality in nonsmoking men and women and being married to spouses who were former smokers.</p> <p>There was some suggestion of a trend of increasing risk of CHD mortality in nonsmoking men and women with increasing number of years exposed to spouses' smoking. There is also some suggestion that the risk of CHD mortality associated with exposure to a smoking spouse was more apparent in individuals who had heart disease at baseline.</p>
		Kawachi <i>et al.</i> (1997)	<p>Exposure to ETS was associated with increased risks of both nonfatal MI and fatal CHD events.</p> <p>Self-reported duration of years lived with a smoker was also associated with an increased risk of incident CHD events although there was not a smooth trend of increasing risk with increasing years of exposure.</p>
		Lee <i>et al.</i> (1986)	<p>There was no association between this combined index of ETS exposure and risk for CHD in men and women.</p>
		He <i>et al.</i> (1989)	<p>A significant, threefold increase in relative risk for CHD was observed for nonsmoking women whose husbands were smokers.</p>

		Dobson <i>et al.</i> (1991a)	ETS exposure at work was not associated with risk of heart attack in men or in women.
		Jackson <i>et al.</i> (1991), Jackson (1989)	The age- and social-class-adjusted OR for myocardial infarction (MI) in relation to ETS exposure at home (and/or work) was 2.7 in females and 1.03 in males.
		La Vecchia <i>et al.</i> (1993)	Compared to subjects married to never-smokers, the adjusted OR for acute MI associated with being married to ex-smokers was 0.91 and the OR for those married to current smokers was 1.21.
		He <i>et al.</i> (1994)	Risk of CHD was significantly increased in relation to ETS exposure from husbands (defined as living with a smoking husband for over five years) and at work (defined as working with smoking coworkers for over five years). The risks increased approximately twofold for ETS exposure from husbands only and at work only, and by fourfold for exposures both at work and from husbands. There were also significant trends of increasing risks with increasing intensity (amount smoked daily, number of smokers) and duration (in years) of ETS exposure at work.
		Layard (1995)	There was no association between exposure to spouse's smoking and risk of CHD death in men or in women. Analysis by amount smoked by spouses also did not reveal any association between amount smoked by the spouses and risk of CHD mortality.

		<p>Muscat and Wynder (1995)</p>	<p>Adult ETS exposure was associated with an elevated risk of MI in men and in women.  Exposure to ETS at work was associated with a small increased risk in men but not in women, whereas exposure to ETS in transportation was associated with an increased risk in women but not in men.</p>
		<p>Enstrom and Kabat (2003)</p>	<p>For participants followed from 1960 until 1998 the age adjusted relative risk (95 percent confidence interval) for never smokers married to ever smokers compared with never smokers married to never smokers was 0.94 (0.85 to 1.05) for coronary heart disease. The results do not support a causal relation between environmental tobacco smoke and tobacco related mortality, although they do not rule out a small effect.</p>

## Appendix 2

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## Appendix 3

### Example of Relative Risk Calculation—Lung Cancer

The studies of relative risk of lung cancer among nonsmoking adults currently exposed to ETS can be grouped into two major categories, nonsmoking adults exposed to ETS from a smoking spouse, and nonsmoking adults exposed to ETS at work. We computed average relative risk on a combined basis. The number of expected cases was similar for the two groups, and there was not a clear pattern of difference between the results by gender or type of exposure, so we considered it appropriate to weight the results on the basis of the total number of expected base-case lung cancer cases, which is a measure of the total exposure to risk in the absence of ETS.

There were 13,164 cases of lung cancer covered by the studies included. The principal results of the various individual studies are shown in Tables 1 through 5 of this appendix, and summarized in Table 6. The average relative risk from exposure to ETS was 1.22.

The relative risk compares the rate of lung cancer for nonsmokers exposed to ETS with the rate found in a matched population of nonsmokers not exposed to ETS. Once the relative risk is determined, the number of expected cases among nonsmokers exposed to ETS is the product of the number of expected cases on the basis of the base rate times the relative risk. The excess of this amount over the expected base cases is the expected excess cases arising from ETS exposure.

Appendix 3 Table 1: Relative Risk from Smoking Spouse – Female Cohort Studies

First Author	Year	Lung Cancer Cases	Computed Relative Risk	Expected Base
Garfinkel	1981	153	1.18	129.66
Hirayama	1984	200	1.45	137.93
Butler	1988	8	2.02	3.96
Cardenas	1997	150	1.20	125.00
Nishino	2001	24	1.90	12.63
<b>Total</b>		535		409.18

Appendix 3 Table 2: Relative Risk from Smoking Spouse – Female Case-Control Studies

First Author	Year	Lung Cancer Cases	Computed Relative Risk	Expected Base
Chan	1982	84	0.75	112.00
Correa	1983	22	2.07	10.63
Trichopoulos	1983	62	2.13	29.11
Buffler	1984	41	0.80	51.25
Kabat	1984	24	0.79	30.38
Lam	1985	60	2.01	29.85
Garfinkel	1985	134	1.23	108.94
Wu	1985	29	1.20	24.17
Akiba	1986	94	1.52	61.84
Lee	1986	32	1.03	31.07
Koo	1987	86	1.55	55.48
Pershagen	1987	70	1.03	67.96
Humble	1987	20	2.34	8.55
Lam	1987	199	1.65	120.61
Gao	1987	246	1.19	206.72
Brownson	1987	19	1.52	12.50
Geng	1988	54	2.16	25.00
Shimizu	1988	90	1.08	83.33
Inoue	1988	22	2.55	8.63
Kalandidi	1990	90	1.62	55.56
Sobue	1990	144	1.06	135.85
Wu-Williams	1990	417	0.79	527.85
Liu	1991	54	0.74	72.97
Brownson	1992	431	0.97	444.33
Stockwell	1992	210	1.60	131.25
Du	1993	75	1.19	63.03
Liu	1993	38	1.66	22.89
Wang	1996	135	1.11	121.62
Fontham	1994	651	1.26	516.67
Kabat	1995	67	1.10	60.91
Sun	1996	230	1.16	198.28
Ko	1997	105	1.24	84.68
Boffetta	1998	509	1.20	424.17
Zaridze	1998	189	1.63	115.95
Rapiti	1999	41	1.02	40.20
Zhong	1999	407	1.15	353.91
Wang	2000	233	0.90	258.89
Lee	2000	268	2.20	121.82
Johnson	2001	71	1.20	59.17
<b>Total</b>		5,753		4,887.99

Appendix 3 Table 3: Relative Risk from Smoking Spouse – Male Cohort Studies

<b>First Author</b>	<b>Year</b>	<b>Lung Cancer Cases</b>	<b>Computed Relative Risk</b>	<b>Expected Base</b>
Hirayama	1984	64	2.25	28.44
Cardenas	1997	97	1	97.00
<b>Total</b>		161		125.44

Appendix 3 Table 4: Relative Risk from Smoking Spouse – Male Case-Control Studies

<b>First Author</b>	<b>Year</b>	<b>Lung Cancer Cases</b>	<b>Computed Relative Risk</b>	<b>Expected Base</b>
Correa	1983	8	1.97	4.06
Buffler	1984	11	0.51	21.57
Kabat	1984	12	1	12.00
Akiba	1986	19	2.1	9.05
Lee	1986	15	1.31	11.45
Kabat	1995	39	1.63	23.93
Boffetta	1998	141	1.65	85.45
<b>Total</b>		245		167.51

Appendix 3 Table 5: Relative Risk from Work Exposure

First Author	Year	Gender	Lung Cancer Cases	Computed Relative Risk	Expected Base
Kabat	1984	Men	25	3.27	7.65
		Women	53	0.68	77.94
Koo	1984	Women	88	1.19	73.95
Garfinkel	1985	Women	134	0.93	144.09
Wu	1985	Women	29	1.30	22.31
Lee	1986	Men	10	1.61	6.21
		Women	15	0.63	23.81
Butler	1988	Men	7	1.72	4.07
		Women	8	1.47	5.44
Shimizu	1988	Women	90	1.20	75.00
Kalandidi	1990	Women	90	1.70	52.94
Wu-Williams	1990	Women	417	1.20	347.50
Wang	1996	Women	135	0.89	151.69
Fontham	1994	Women	651	1.39	468.35
Kabat	1995	Men	41	1.02	40.20
		Women	58	1.15	50.43
Reynolds	1996	Women	528	1.56	338.46
Zaridze	1998	Women	189	0.88	214.77
Janerich	1990	Both	191	0.91	209.89
Schwartz	1996	Both	257	1.50	171.33
Ko	1997	Women	105	1.10	95.45
Boffetta	1998	Women	509	1.19	427.73
		Both	650	1.17	555.56
Rapiti	1999	Both	58	1.10	52.73
Zhong	1999	Women	504	1.70	296.47
Wells	1998	Both	835	1.39	600.72
Kreuzer	2000	Both	292	1.03	283.50
Lee	2000	Women	268	1.20	223.33
Wang	2000	Both	233	1.56	149.36
<b>Total</b>			6,470		5,170.87

Appendix 3 Table 6: Summary of Results for Lung Cancer

Type of Study	Lung Cancer Cases	Expected Base	Relative Risk
Smoking Spouse			
Female Cohort	535	409.18	1.31
Female Case-Control	5,753	4,887.99	1.18
Male Cohort	161	125.44	1.28
Male Case-Control	245	167.51	1.46
Exposed At Work	6,470	5,170.87	1.25
<b>Total</b>	13,164	10,761	1.22

## Appendix 4

### **Definitions of Exposure to Environmental Tobacco Smoke**

Definitions of exposure to environmental tobacco smoke have varied slightly in different studies. In the interest of completeness we present the definitions used in three major analyses of ETS effects.

#### **California Environmental Protection Administration Definition of ETS Exposure**

“ETS is also called ‘second-hand smoke’, and ETS exposure is frequently used interchangeably with ‘involuntary smoking’ and ‘passive smoking.’ ETS is formed from the smoldering of a cigarette or other tobacco product, and from smoke exhaled by the smoker (NRC, 1986). There are other minor contributors such as the smoke that escapes while the smoker inhales, and some vapor-phase components that diffuse into the environment. Once released into the environment of the smoker, components are diluted by the ambient air, diffusing in and being transported through it. These smoke constituents may also aggregate with other components in the air, and further age and change in character. This complex mixture is defined as ETS, and inhalation of it, as ETS exposure. In some ways this may be an overly restrictive definition when it comes to assessing effects from prenatal smoke exposures. Because the fetus cannot actively smoke, all of its exposure to tobacco smoke constituents is ‘passive’ or ‘involuntary.’ Nonetheless, exposure of the fetus due to maternal smoking during pregnancy is not considered to be ETS exposure in this report.”

#### **Centers for Disease Control and Prevention Definition of ETS Exposure**

“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). Secondhand smoke contains a complex mixture of more than 4,000 chemicals, more than 50 of which are cancer-causing agents (carcinogens). People are exposed to secondhand smoke in the home, workplace, and in public venues such as bars, bowling alleys and restaurants.”

#### **U.S. Environmental Protection Administration Definition of ETS Exposure**

“Secondhand smoke is a mixture of the smoke given off by the burning end of a cigarette, pipe, or cigar, and the smoke exhaled from the lungs

of smokers. This mixture contains more than 4,000 substances, more than 40 of which are known to cause cancer in humans or animals and many of which are strong irritants. Secondhand smoke is also called environmental tobacco smoke (ETS); exposure to secondhand smoke is called involuntary smoking or passive smoking.”