Lung Cancer and Air Pollution

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Epidemiologic studies over the last 40 years suggest rather consistently that general ambient air pollution, chiefly due to the incomplete combustion of fossil fuels, may be responsible for increased rates of lung cancer. This evidence derives from studies of lung cancer trends, studies of occupational groups, comparisons of urban and rural populations, and case–control and cohort studies using diverse exposure metrics. Recent prospective cohort studies observed 30 to 50% increases in lung cancer rates associated with exposure to respirable particles. While these data reflect the effects of exposures in past decades, and despite some progress in reducing air pollution, large numbers of people in the United States continue to be exposed to pollutant mixtures containing known or suspected carcinogens. It is not known how many people in the United States are exposed to levels of fine respirable particles that have been associated with lung cancer in recent epidemiologic studies. These observations suggest that the most widely cited estimates of the proportional contribution of air pollution to lung cancer occurrence in the United States based largely on the results of animal studies, may be too low. It is important that better epidemiologic research be conducted to allow improved estimates of lung cancer risk from air pollution among the general population. The development and application of new epidemiologic methods, particularly the improved characterization of population-wide exposure to mixtures of air pollutants and the improved design of ecologic studies, could improve our ability to measure accurately the magnitude of excess cancer associated with air pollution. — Environ Health Perspect 103(Suppl 8):219–224 (1995)

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Introduction

In 1975 in a collection of essays on cancer etiology and control, Pike et al. (*1*) devoted seventeen pages to air pollution. Ten years later a similar collection, *Cancer: Risks and Prevention* (*2*), devoted no pages to air pollution. An optimist might conclude that this is evidence of progress; i.e., that in the intervening decade we had been able to conclude to the satisfaction of most observers that air pollution does not cause cancer and that further research would not be informative. A more likely explanation, however, is that early evidence for an effect of air pollution on lung cancer was overwhelmed by an increasing appreciation of the role played by cigarette smoking in the vast majority of lung cancers.

Many scientists took the view that epidemiology could not reliably detect what must certainly be small relative increases against the high background rate attributable to etiologies involving cigarette smoking. In addition, over that same period some progress had been made in improving air quality in some areas of the United States. This led some health professionals to conclude that the problem, even if real, need not concern us in the future. This view is too sanguine because epidemiologic studies over the last 40 years suggest rather consistently that general ambient air pollution may be responsible for increased rates of lung cancer. The results of more recent studies, which use improved research methods, corroborate earlier findings. Moreover, despite some progress in reducing air pollution, large numbers of people in the United States continue to be exposed to pollutant mixtures containing known or suspected carcinogens.

Ambient air, particularly in densely populated urban environments, contains a variety of known human carcinogens, including organic compounds such as benzo[a]pyrene (B[a]P) and benzene, inorganic compounds such as arsenic and chromium, and radionuclides (*3*). These substances are present as components of complex mixtures, which may include carbon-based particles that absorb organic compounds; oxidants such as ozone; and sulfuric acid in aerosol form. The combustion of fossil fuels for power generation or transportation is the primary source of many organic and inorganic compounds, oxidants, and acids, and contributes heavily to particulate air pollution in most urban settings. The radionuclides result from fuel combustion, as well as from mining operations. Over the past 20 to 30 years there have been changes in some indices of air quality. According to a 1980 report from the Council for Environmental Quality, levels of B[a]P in urban air decreased 70% between 1970 and 1980 (*4*), and levels of total suspended particles decreased approximately 7% between 1973 and 1978. Data from the Six Cities Study suggest that this trend continued through the 1980s (*5*). However, the relative contribution to urban air pollution from mobile sources has increased. While there have been declines in levels of total suspended particles across the United States, Six Cities Study data suggest that the fine particle fraction might not have changed markedly in some urban areas during the past decade (*5*). Data collected by Cass et al. (*6*) in Los Angeles indicate that elemental carbon levels, two-thirds of which are derived from diesel exhaust, declined in most areas between 1958 and 1981 but increased in areas undergoing rapid growth.

Since the mid-1980s there has been a large increase in the number of epidemiologic studies that report health effects associated with particulate air pollution. Health effects have been observed at levels common in many U.S. cities. Although the

biological mechanisms involved are poorly understood, recent epidemiologic evidence suggests that respirable particulate air pollution is a risk factor for respiratory morbidity and cardiopulmonary mortality. Acute and long-term exposure to elevated levels of particulate air pollution has been associated with a wide range of acute and chronic non-malignant respiratory health end points, including *a*) declines in lung function, *b*) increased incidence and duration of respiratory symptoms, *c*) exacerbation of asthma, *d*) restricted activity, *e*) increased hospitalization for respiratory disease, and *f*) increased cardiopulmonary mortality (*7,8*).

The consistency of the findings of epidemiologic studies regarding the effects of combustion-source particulate air pollution across a wide range of nonmalignant health end points suggests the toxic properties of this pollution. In addition, experimental toxicology studies have documented the mutagenic and carcinogenic properties of combustion-source air pollution such as diesel exhaust, which is ubiquitous in urban and highway environments (*9*). Several lines of epidemiologic evidence also suggest that exposure to outdoor air pollution increases the rate of lung cancer. We will review this evidence, focusing on studies of occupational groups, comparisons of urban and rural populations, between-community studies, and case–control and cohort studies, including three recent prospective cohort studies. In addition, we will discuss the plausibility of ambient air pollution–lung cancer effect by summarizing epidemiologic evidence of relative risks of various types of exposure to combustion-source pollutants. Finally, we will identify additional epidemiology research needs.

Occupational Exposure

Lung cancer rates are increased among occupational groups exposed to combustion products of fossil fuels over a wide range of exposure. Coal combustion-related air pollution exposures can be very high for top-side coke oven steel mill workers. A series of studies of coke-oven workers by Lloyd and colleagues (*10*) and Redmond (*11*) observed that increasingly greater excesses of lung cancer were associated with workers exposed to low, medium, and high exposures. There was also an increase in lung cancer associated with increased length of exposure (Table 1). Relative risks of respiratory cancer ranged from approximately 2 to 16, depending on the level and length of exposure.

Various groups of workers occupationally exposed to diesel exhaust, such as

Table 1. Relative risks of death from respiratory cancer (1953–1970) for coke-oven workers in Allegheny County, Pennsylvania.

Level of exposure, work area	Length of employment		
	5+ years	10+ years	15+ years
Low (side only)	1.79	1.99	2.00
Medium (part-time topside)	2.29	3.07	4.72
High (full-time topside)	9.19	11.79	15.72

Adapted from Redmond (*11*).

railroad, bus garage, and dock workers, as well as truckers, have been observed to be at increased risk of lung cancer in multiple studies (*12*). For example, a recent case–control study of Teamster Union members in the midwestern United States found excess lung cancer among mechanics, and short- and long-haul truckers (*13*). The lung cancer rate ratios for employment in various jobs were associated with present-day levels of elemental carbon, which is a relatively specific marker for diesel exhaust and was measured in the repair shops, truck cabs, and in highway and residential environments (Table 2) (*14*). The present-day levels of elemental carbon in truck cabs were slightly higher on average than highway background but on average 4 times greater than those in residential neighborhoods in the Midwest. However, recently assembled data suggest that between 1958 and 1981, Los Angeles residents may have been exposed to ambient air pollution levels as high or higher than those currently associated with truck driving (*6*).

Environmental Exposure

Various studies that contrasted lung cancer rates between urban and rural environments generally have found evidence of

Table 2. Lung cancer relative risk estimates for Teamster job categories.

Job category	Elemental carbon, $\mu g/m^3$	Adjusted RR estimate
Unexposed	1.1–2.5	1.0
Truckers, long haul	3.8	1.3
Truckers, city	4.0	1.3
Mechanics	12.0	1.7

RR, risk ratio. Data from Steenland (*14*).

increased lung cancer among urban dwellers (Table 3). Although many of these studies attempted to control for cigarette smoking, Doll and Peto (*19*) have suggested that the remaining urban–rural difference may be due to urban dwellers starting to smoke at younger ages, as cigarette smoking became increasingly prevalent in the early 20th century. Dean (*24*) controlled for age at initiation of smoking and found that the urban–rural difference persisted. Moreover, cancer incidence data collected by the International Agency for Research in Cancer (IARC) over the past decade continue to show evidence of urban-rural differences in lung cancer rates with urban to rural rate ratios between 1.0 and 1.9 (*28*). Furthermore, studies of population migration from high-exposure countries to lower exposure countries suggest that migrants have lasting risks related to their country of origin and previous exposures(*29–31*). However, urban–rural differences may partially reflect unaccounted for differences in smoking habits, occupational exposures, migration patterns, or other factors related to population density.

Several studies have compared lung cancer rates between areas with differing levels of air pollution (Table 4). These studies found evidence of similar to slightly higher relative excesses of lung cancer in more

Table 3. Urban–rural differences in lung cancer.

Studies	Population	Cases	Rate ratio
Cohort studies			
Hammond and Horn (*15*)	U.S. veterans (1952–1955)	448	1.3
Buell et al. (*16*)	California residents (1957–1962)	304	1.3
Hammond (*17*)	U.S. residents (1959–1965), unexposed/exposed to dust and fumes	1510	1.1/1.3
Cederlof et al. (*18*)	Swedish men (1963–1972)	116	1.4
Doll and Peto (*19*)	British physicians (1951–1971)	401	1.0
Tenkanen and Teppo (*20*)	Finnish men (1964–1979), smokers/nonsmokers	233	1.1/1.9
Case–control studies			
Stocks and Campbell (*21*)	British men (1952–1954)	725	1.7
Haenszel (*22*)	U.S. white men (1958)	2381	1.4
Haenszel and Taeuber (*23*)	U.S. white women (1958–1959)	749	1.3
Dean (*24*); Dean et al. (*25*)	Irish men/women nonsmokers (1960–1962)	3040	2.1/1.3
Hitosugi (*26*)	Japanese men/women (1960–1966)	259	1.8/1.2
Samet et al. (*27*)	New Mexico residents >25 years in urban counties (1980–1982)	422	1.2–1.4

Table 4. Ecologic studies of air pollution and lung cancer.

Study	Locale	Average PM_{10} in high exposure region, $\mu g/m^3$	Rate ratio
Henderson et al. (32)	Los Angeles	53–64	1.3
Buffler et al. (33)	Houston	41–46	1.9
Archer (34)	Utah	47–53	1.6

polluted areas than the urban–rural studies revealed. However, incidence, exposure, and covariate data were all on the aggregate, or ecologic, level, so interpretation of these results is complicated by several factors, including the inability to account adequately for individual- and between-area differences in other risk factors (35).

Two studies took advantage of "natural experiments." Stevens and Moolgavkar (36) deduced that there had been declines in lung cancer incidence among nonsmoking males in England and Wales coincident with substantial declines in levels of particulate and sulfur dioxide pollution that resulted from the implementation of nationwide air pollution control measures.* In another study, Archer (34) analyzed respiratory cancer mortality in two Utah counties with very low smoking rates. These two counties were similar in many respects, with low and nearly equal respiratory cancer mortality rates until a steel mill constructed during World War II caused substantial increases in air pollution in one of them. The subsequent differences in lung cancer incidences were substantial within about 15 years after the increase in air pollution and have persisted. A third neighboring county, unaffected by the steel mill's pollution but with higher smoking rates, had higher lung cancer rates than either of the other two counties—underscoring the profound effect cigarette smoking has on lung cancer risks.

Case–Control and Recent Prospective Cohort Studies

Several case–control and cohort studies used air pollution monitoring data to estimate the exposures of study subjects (Table 5). The case–control studies found relative increases in lung cancer risks after

*Although the incidence rates of cancer began their decline within a few years of the reduction in air pollution, critics have challenged the interpretation of this temporal association. The authors countered that reductions in lung cancer risk in ex-smokers have been observed within 2 years of quitting smoking (36).

adjustment for age [Wallace et al., unpublished data;(1–8,10–27,29–49)], smoking (38–40), and occupational exposure (37–39) similar to those observed in the urban–rural and ecologic studies.

Three recent prospective cohort studies have estimated the effect of air pollution exposure on lung cancer mortality. The study by Abbey et al. (41) followed a cohort of Seventh Day Adventists, whose extremely low prevalence of smoking and uniform, relatively healthy dietary patterns reduce the potential for confounding by these factors. Excess lung cancer was observed in relation to both particle and ozone exposure.

Dockery et al. (5) recently reported the results of a 14- to 16-year prospective follow-up of 8111 adults living in six U.S. cities, that evaluated associations between air pollution and mortality (Table 5). Mortality was ascertained through 1989 and TSP, PM_{10}, $PM_{2.5}$, SO_4, H^+, SO_2, NO_2, and O_3 levels were monitored. Mortality risk was estimated via Cox proportional hazards regression modeling while directly controlling for individual differences in age, sex, cigarette smoking, Body Mass Index (BMI), education, and occupational exposure.

The largest estimated mortality risk was associated with cigarette smoking. However, after controlling for individual differences in age, sex, cigarette smoking, BMI, education, and occupational exposure, differences in relative mortality risks across the six cities were strongly associated with differences in pollution levels experienced in those cities. Associations between mortality risk and air pollution were strongest for respirable particles and sulfates. Air pollution was positively associated with lung cancer mortality and cardiopulmonary disease mortality but not with mortality associated with the combined effects of

other causes. After adjustment for the other risk factors, a 37% excess lung cancer risk was observed for a difference in fine particle pollution equal to that of the most polluted versus the least polluted city.

Another recent study evaluated particulate air pollution as a predictor of mortality in a prospective study of U.S. adults (43). This study linked ambient air pollution data from 151 U.S. metropolitan areas with risk factor data for 552,138 adults enrolled in the American Cancer Society/Cancer Prevention Study II. It assessed the vital status of participants from 1982 to 1989 through the use of personal inquiries and automated linkage supplied by the National Death Index. Data were analyzed using multivariate Cox proportional hazards regression modeling. The study controlled for individual differences in age, sex, race, cigarette smoking, pipe and cigar smoking, exposure to passive cigarette smoke, occupational exposure, education, BMI, and alcohol use.

All-cause and cardiopulmonary mortality were consistently associated with sulfate and fine particulate pollution levels. Lung cancer mortality was associated with combustion-source air pollution when sulfates was used as the index but not when fine particles was used as the index. To evaluate whether this inconsistency was a result of the use of different study areas or different pollution measures, sulfate pollution measures were included in models, which only used data from the 47 metropolitan areas that had both sulfate and fine particulate measures. The results were similar to those from the initial analysis, which suggests that the inconsistency was not attributable to differences among study areas, but that lung cancer seems to be more strongly associated with sulfate particles than with the more general index of fine particulate mass.

Table 5. Case–control and cohort studies of air pollution and lung cancer.

Study	Locale	Exposure classification	Rate ratio, 95% CI
Case–control			
Pike et al. (37)	Los Angeles	Residence in high pollution benzo[a]pyrene area	1.3, NA
Vena (38)	Buffalo, NY	>50 years residence in elevated TSP areas	1.7, 1.0–2.9
Jedrychowski et al. (39)	Cracow	Residence in elevated TSP and SO_2 areas	1.5, 1.1–2.0
Katsouyanni et al. (40)	Athens	Lifelong residence in high pollution areas	1.1, NA
Cohort			
Abbey et al. (41)	California	PM_{10} 42 day/year > 100 $\mu g/m^3$	1.5, 0.9–2.4
		O_3 500 hr/year > 100 ppb	2.3, 0.9–5.3
Dockery and Spengler (42)	6 U.S. cities	Residence in high sulfate or fine particulate pollution areas	1.4, 0.8–2.3
Pope et al. (9)	151 U.S. cities	Residence in high sulfate particulate pollution areas	1.4, 1.1–1.7
		Residence in high fine particulate pollution areas	1.0, 0.8–1.3

When sulfate particulate pollution was used as the index of exposure, estimated pollution-related mortality risk was as high for those who had never smoked as it was for smokers, and as high for women as it was for men. Therefore, although the increased risk associated with air pollution was small compared with that of cigarette smoking, the results of this study suggest that the association between pollution and mortality was not likely to be a result of inadequate control of smoking. Study conclusions supporting this assertion include: *a*) that the associations with air pollution persisted after controlling for cigarette smoking status, pipe and/or cigar smoking, years smoked, cigarettes smoked per day for both current and former smokers, and hours per day exposed to passive cigarette smoke; and *b*) that associations were as large among those who had never smoked as they were for smokers. However, the small number of lung cancer deaths among nonsmokers, even in this large cohort, resulted in limited statistical power to observe lung cancer–air pollution effects only among those who had never smoked.

Potential interactions between ambient air pollution and other risk factors such as cigarette smoking remains largely unknown. Earlier reviewers have noted a greater than additive relation between air pollution and cigarette smoking, which suggests both independent and joint effects [Wallace et al., unpublished data; (*31,35,46*)]. The combined effect of the small number of nonsmoking-related lung cancer cases and the relatively small effect of air pollution compared with those for cigarette smoking results in imprecise estimates of joint effects.

Plausibility of Ambient Air Pollution–Lung Cancer Effect

Combustion-source ambient air pollution has been associated with a wide range of nonmalignant health end points, suggesting toxic properties of this pollution, although the biological mechanisms involved are not well understood. Also, animal studies have documented mutagenic or carcinogenic activity for a wide range of combustion-source particles, including those from the burning of tobacco, coal, diesel fuel, wood, and complex urban or industrial mixtures. Differences in the apparent magnitudes of cancer risks associated with exposure to different combustion-source air pollutants are partially attributable to differences in relative toxic and carcinogenic activity of the pollutant. The level of lung exposure,

or dose, also plays an important role in determining the relative risk of various types of exposure.

Table 6 summarizes epidemiologic evidence of relative risks of various types of exposure to combustion-source pollutants. It suggests that breathing fine or respirable particles from a wide variety of combustion sources increases the risk of lung cancer. The range of relative risk summarized in Table 6 may be interpreted as reflecting not only the differences in relative carcinogenic activity of the pollutant but also the level of exposure, or dose. Cigarette smoking represents the high end of combustion-source particle exposure, and involuntary exposures to ambient air pollution and environmental tobacco smoke represent the low end of exposure.

The similar lung cancer risk estimates for environmental tobacco smoke and ambient air pollution may reflect similar differences in exposure. Spengler et al. (*44*) estimated that, on the average, a home with one smoker has concentrations of respirable particulate matter about 20 μg/m³ higher than homes without smokers. Similar or somewhat larger differentials in exposure exist between U.S. cities rated as high and low polluted . For comparison, in the Harvard Six Cities Study, the range of PM_{10} was 18 to 47 μg/m³ and the range of $PM_{2.5}$ was 11 to 30 μg/m³. In the CPS-II

study the range of $PM_{2.5}$ was 9 to 34 μg/m³. Cigarette smoke has little impact on outdoor pollution concentrations. Outdoor combustion-source particulate air pollution penetrates the walls of residences and among individuals from nonsmoking homes, the indoor, outdoor, and personal exposures are similar and highly correlated [Wallace et al., unpublished data; (*42,45*)]. Estimated effects of ambient air pollution and environmental tobacco smoke can be viewed as at least partially complementary, which suggests that there are small lung cancer effects at relatively low levels of exposure to combustion-source air pollution.

Estimates of the population-attributable risk of lung cancer associated with air pollution made during the past decade were calculated using markedly different methods, and their results span an order of magnitude (Table 7). For example, Doll and Peto (*19*) used estimates of benzo[*a*]-pyrene in urban air and extrapolated from occupational studies of polycyclic aromatic hydrocarbon (PAH)-exposed workers. They estimated that less than 1% of future lung cancer would be attributable to air pollution from the burning of fossil fuels, although they noted that perhaps 10% of current lung cancer in large cities may be due to air pollution. In 1990, the U.S. Environmental Protection Agency (U.S. EPA) estimated that 0.2% of all cancer, and

Table 6. Summary of epidemiologic evidence of relative risks of various types of exposure to combustion-source pollutants.

Exposed groups	Primary combustion sources	Exposure indicators or indexes	Rate ratio
Cigarette smokers	Tobacco	Smoking status and history, cigarettes/day	7.0–22.0
Coke-oven workers	Coal	Job classifications	2.5–10.0
Railroad workers, truck drivers, diesel mechanics	Diesel	Job classifications	1.2–2.6
Residents of areas with high ambient air pollution	Complex mix from coal, wood, diesel, gasoline	Respirable, fine, or sulfate particle concentrations	1.0–1.6
Nonsmokers exposed to environmental tobacco smoke	Tobacco	Smoking status of family members and co-workers	1.0–1.5

Table 7. Estimates of the population attributable risk (PAR%) of lung cancer due to air pollution in the U.S. population.

Source	Method	Estimated attributable risk
Doll and Peto (*19*)	Extrapolation from occupationally exposed groups using past and then-current levels of B[*a*]P	0 to 2% overall (10% of current lung cancer in urban areas) 1% of future lung cancer
Karch and Schneiderman (*47*)	Relative risk of urban vs rural residents from Hammond and Garfinkel (1978) and proportion of 1980 population residing in urban areas	12% of 1980 lung cancer 10 to 19% of future lung cancer based on 1980 levels of TSP
U.S. EPA (*46*)	Summation of numbers of cancers attributable to > 20 individual pollutants from toxicologic and other data	<1% given current levels of pollution

probably less then 1% of lung cancer, could be attributed to the effects of air pollution (46). This estimate was obtained by applying the unit risks for over 20 known or suspected human carcinogens found in outdoor air to estimates of the ambient concentrations and numbers of individuals potentially exposed. The unit risks were derived either from animal experiments or extrapolation from studies of workers exposed to higher concentrations. Karch and Schneiderman (47) using data from the ACS volunteers study and the U.S. Census Bureau, estimated that the urban factor accounted for 12% of lung cancer in 1980. They predicted that 1980 levels of suspended particulates would be associated with a lung cancer rate ratio of 1.3, slightly less than the 47% increase observed for total suspended particles in the recent Six Cities Study, which reported mortality through 1989. Each of these estimates of attributable risk is subject to considerable error in terms of both the relative magnitude of effect and the proportion of the population assumed to be exposed. However, there is no reason to prefer estimates based on extrapolation from animal or occupational studies rather than those resulting from direct observation of the populations at risk.

Epidemiology Research Needs

Direct epidemiologic observation of exposed populations can provide the best information for evaluating the magnitude of air pollution-related excess lung cancer if we can make more valid and precise estimates of the increases in lung cancer associated with air pollution and of the numbers of people exposed. Clearly, better data to support these estimates are needed.

While most studies have made some attempt to address confounding from cigarette smoking and occupation, almost none have addressed possible bias associated with the measurement errors in exposure and covariates. Such bias, even if it is of equal magnitude among those with and without disease, can produce either spuriously high or low estimates of the lung cancer rate ratio in multivariable data. The problem is that few if any studies have collected the data necessary to quantify this bias and to determine its direction. Future studies should develop methods and collect data that can be used to quantify exposure measurement error and compute adjusted effect estimates.

Better estimates of the magnitude of effect will require large-scale epidemiologic studies. Large numbers of cases will be necessary to measure the effects of air pollution and to measure joint effects of air pollution and factors such as occupation and smoking. These studies will probably require pooling data from multiple locales. For these studies to be maximally informative we must develop and apply improved epidemiologic methods. The development and application of new designs and statistical methods for air pollution studies, therefore, should be supported. Navidi and Thomas (48) and Prentice and Sheppard (49) have described hybrid studies that combine ecologic-level contrasts of air pollution effects between cities with individual-level data on covariates. These studies combine the strengths of both ecologic and individual-level studies. Studies employing these designs could contrast the effect of exposure to pollutant mixtures in terms of lung cancer incidence among different cities while effectively controlling confounding by cigarette smoking, diet, and other factors and adjusting for exposure measurement error.

Methods for the retrospective estimation of lifetime exposure to air pollutants should be developed and tested so that large case–control and retrospective cohort studies can be conducted. These methods could be based on combinations of time-activity data and data from national aerometric databases such as those maintained by U.S. EPA. This effort should include development of methods to characterize, quantify, and adjust for exposure measurement error. Current work on biologic markers of exposure to and molecular effects of PAHs (50) represents one approach to improving epidemiologic methods in this area.

The air pollution mixtures in various U.S. population centers should be characterized both in terms of physical and chemical constituents and in terms of sources of major constituents. If possible, retrospective characterization of levels of certain constituents should be accomplished. This information would aid greatly in the interpretation of between-city epidemiologic contrasts. For lung cancer epidemiology, both urban and relatively clean areas with established population-based tumor registries should be targeted for study.

Conclusions

The epidemiologic evidence suggests that combustion-source air pollution contributes to the occurrence of lung cancer among the general population. These results are consistent with studies of other types of exposure to combustion-source pollution such as occupational exposures and exposures to environmental tobacco smoke. The excess lung cancer risk associated with ambient air pollution is small compared with that from cigarette smoking. However, given the ubiquity of combustion-source ambient air pollution exposure, the contribution of this exposure across a population may be of public health importance. Errors in the measurement of air pollution exposure and in the measurement of other risk factors including cigarette smoking continue to limit our ability to quantify the magnitude of the excess lung cancer risks associated with air pollution. It is important to conduct additional research that addresses these concerns.

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