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Air Pollution and Lung Cancer: Diesel Exhaust, Coal Combustion[1,2]

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In 1946, when the causes of lung cancer were much less well understood than they are now, a meeting was held by the British Medical Research Council to review hypotheses to explain the remarkable increase in the death rates from lung cancer and to determine strategy. Stocks came away from the meeting to study the community aspects of air pollution, which he did by extending his series of correlation studies, Kennaway to conduct studies of carcinogens in the air, and Hill to carry out a study of smoking in relation to lung cancer. It is now known, of course, that cigarette smoking is by far the most important cause of lung cancer and that about a dozen occupational exposures are also established as causes of this disease. There has been continuing uncertainty about the role of general air pollution. During the past few years, this uncertainty has been compounded with anxiety that the increasing use of diesel-powered vehicles might lead to a deterioration in air quality and, with it, an increase in the incidence of lung cancer. The purpose of this paper is to assess the current role of air pollution as a factor in lung cancer and specifically the contribution of diesel exhaust emissions to the incidence of that disease.

INTRODUCTION

Reasons for believing that air pollution might be an important factor in the development of lung cancer were first, the presence in polluted air of known human carcinogens, such as benzpyrene (35) and, second, pronounced urban/rural gradients of mortality. In England and Wales, death rates for both men and women were highest in the most densely populated conurbations, progressively lower in cities of smaller size, and lowest in the rural areas. One reason for questioning the simple view that these differences could be due to pollution was that the gradient was often greatest where urban pollution was lowest (Copenhagen, Denmark, for example).

An early study by Stocks (32) showed that lung cancer mortality was inversely correlated with the number of hours of sunshine per year. Subsequently, some correlation was seen between lung cancer mortality and measurements of smoke in a number of localities in England and Wales (33). International comparisons (34) suggested that pollution might play a considerable part in lung cancer mortality differences between countries after confounding factors such as smoking, social class, and population density had been taken into account. How successfully these confounding factors were in fact allowed for is debatable. It has, for

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example, become clear that per capita consumption of cigarettes, the index of smoking which is usually available in ecological studies, is too crude to provide an adequate allowance for this most important confounder. Similar doubts also apply to indices of socioeconomic circumstances.

Stocks (31, 33) suggested that benzpyrene in the air was an important factor in the development of lung cancer. Carnow and Meier (2) followed up on this suggestion. They analyzed lung cancer rates in relation to smoking (based on cigarette sales) and benzpyrene (based on a weighting of urban and rural values) in the 48 contiguous states of the United States. They drew the seemingly rather bold conclusion that an increase of 1 ng/1,000 m^3 of benzpyrene would increase the lung cancer death rate by 5%. One reason for skepticism about the validity of this conclusion is that in standardized metropolitan statistical areas, where estimates of benzpyrene concentrations might be expected to have more meaning than over a whole state, there is no substantial correlation between levels of benzpyrene and mortality rates for lung cancer (20).

A fillip to the hypothesis that air pollution was an important factor in the genesis of lung cancer was provided by international immigrant studies. The observations that immigrants from Britain to New Zealand (11), South Africa (5), Australia (6) and Canada (4) had higher death rates from lung cancer than the indigenous populations of those countries, that the rates were higher in those who immigrated at age 30 and over than in those who immigrated at younger ages, and that differences in smoking habits could not explain these differences suggested the importance of environmental exposure before migration. It was fashionable to speak of a "British urban factor" to which coal smoke pollution was an important contributor. However, it is fairly well established that immigrants are not a representative sample of their native countrymen. Moreover, allowance for differences in smoking habits was not as adequate as was claimed, and little attention was given to the jobs which migrants took up after migration.

Perhaps the best studies on urban/rural differences in lung cancer mortality in the United States which provide good evidence on smoking are those carried out by Haenszel and his colleagues (14, 15). These workers studied a 10% sample of white lung cancer deaths during 1958. They compared the smoking habits of these cases with those observed by the National Center for Health Statistics using data from the National Health Interview survey. After allowing for cigarette smoking, a consistent effect of urban residence was still apparent in men, but this was small and inconsistent in women (Fig. 1). The larger differences between urban and rural standardized mortality ratios (SMRs) observed among men compared with women suggest either an inadequate allowance for smoking or the importance of occupational exposures, which might be expected to be greater in men than in women. The smoking data relied heavily on current smoking habits. This would overlook the importance of cigarette smoking in early adult life, which recently has been stressed (7). Haenszel's studies also drew attention to the higher risks of lung cancer in U.S. farm-born and foreign residents of large metropolitan areas and also in more mobile persons in the population.

Hitosugi (22) conducted a study of air pollution, smoking, and lung cancer in Japan, which can be compared with that carried out by Haenszel and his col-

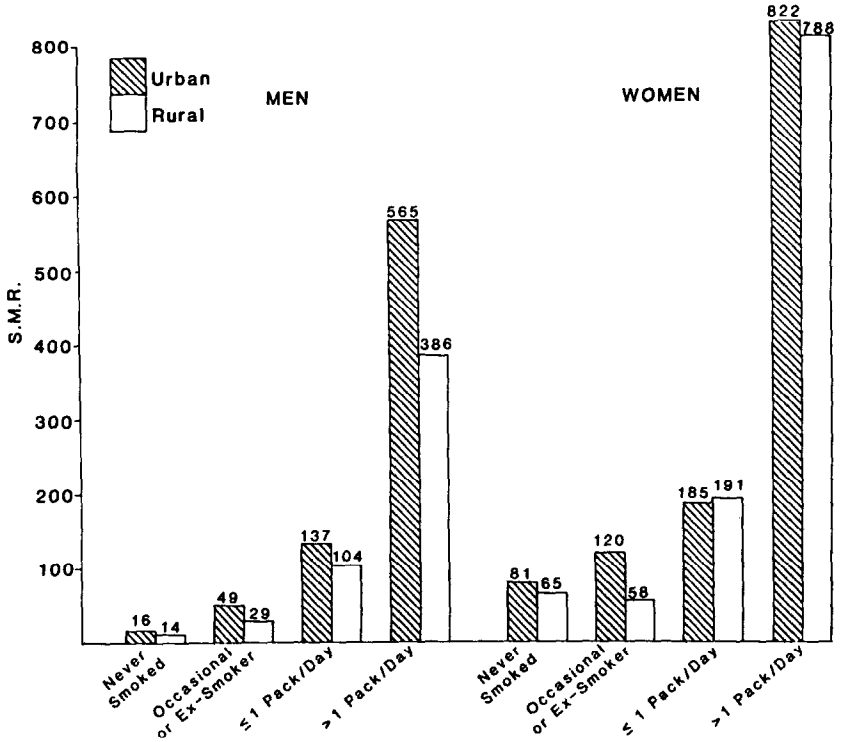

FIG. 1. Standardized mortality ratios in white men and women in the United States, 1958. [Haenszel *et al.* (14,15).] Reprinted with permission.

leagues in the United States. Smoking habits and exposure to air pollution of lung cancer cases in two cities in Japan were compared with the experience of a random sample of adults ages 35–74 years from the same cities. Lung cancer death rates were significantly higher in smokers in each area of pollution. The rates also increased slightly with the extent of pollution among smokers, but not among nonsmokers.

Smoking and place of residence were reported in the 10-year follow-up of a stratified probability sample of the Swedish population covering about 55,000 persons by Cederlof and his colleagues (3). Persons who lived in three cities (Gothenburg, Malmö, and Stockholm), in smaller towns, and in rural areas at the beginning of the study in 1963 were compared. Among nonsmokers, no tendency toward an urbanization effect was seen in either men or women. Among smokers, an association between lung cancer and urbanization, however, was observed. Lung cancer was more common in men residing in the three cities than in those living in the other towns, and these rates, in turn, were higher than the rural rates. The contribution of occupational exposures to the urban excess in smokers was not addressed in this study.

A Karolinska Institute Symposium on air pollution and cancer was held in Stockholm, March 8–11, 1977. The symposium provided a valuable summary of

TABLE 1
LUNG CANCER[a] BY AREA OF RESIDENCE OF BRITISH PHYSICIANS 1951 TO 1976

Area	Observed	Expected	Ratio
Conurbations	152	153.65	0.99
50–100,000	94	88.04	1.07
<50,000	108	109.46	0.99
Rural areas	76	78.85	0.96

Doll and Peto, 1981

[a] Standardized for age and smoking.

the views of many of the leading experts. The general conclusions (1) were that cigarette smoking was the predominant cause of lung cancer and an important component of the urban/rural differences. Combustion products of fossil fuels in the ambient air, probably acting together with cigarette smoke, had been responsible for cases of lung cancer in large urban areas; 5 to 10 cases per 100,000 males per year was suggested as a likely estimate. There is, however, considerable uncertainty about the magnitude of the urban excess that can be attributed to air pollution. Doll, in a review (9) of the epidemiology presented at the Symposium, concluded that in the absence of cigarette smoking, the combined effects of all atmospheric agents could not be responsible for more than 5 cases of lung cancer per 100,000 persons per year in European populations. And how many of these 5 cases are really due to nutritional or occupational differences, passive smoking in the home, or imperfect allowance for smoking still is debatable.

Two more recent pieces of evidence seem particularly important to this question. The first is the 20-year follow-up (7) of the mortality of British physicians (8); the second is the American Cancer Society analysis of half a million men. Table 1 shows the comparison of observed and expected deaths from lung cancer standardized for age and smoking among British physicians according to the area where they lived. There is no evidence that area of residence influenced mortality. The standardized mortality ratios are almost identical and near unity in all areas. Dealing with a homogeneous occupational group eliminates any problem of occupational exposure. However, it might be argued that the results cannot be extrapolated from this single social class, to which the study was limited, to all social classes.

The second study addresses these problems. Hammond and Garfinkel (16) classified their sample of men according to occupational exposure to dust, fumes, vapors, gases, or X rays. Table 2 shows that after allowing for such occupational exposures, the effect of place of residence was small. Table 3 gives estimates of the type and amount of pollution in those cities for which data were available. Again, there appears to be a clear effect of occupation but only a trivial effect of place of residence. This probably explains the earlier observation of Hammond and Horn (17) that even after standardizing for cigarette smoking, lung cancer rates were higher in urban than rural areas (39). These studies suggest that after smoking and occupational exposures are allowed for, general air pollution, if it contributes at all to lung cancer mortality, exerts only a small effect.

TABLE 2
OBSERVED AND EXPECTED LUNG CANCER DEATHS BY PLACE OF RESIDENCE AND BY OCCUPATIONAL EXPOSURE TO DUSTS, FUMES, GASES, OR X RAYS

Area	Occupationally exposed			Not occupationally exposed		
	Observed	Expected	Ratio	Observed	Expected	Ratio
Metropolitan						
1,000,000 and over	165	134.1	1.23	281	285.7	0.98
<1,000,000	166	145.4	1.14	271	280.5	0.97
Nonmetropolitan	245	251.0	0.98	382	413.5	0.92
Rural	143	146.1	0.98	182	214.4	0.85

Hammond and Garfinkel, 1980

The effect of reduction in smoke pollution on mortality from lung cancer was explored by Higgins (21). A greater reduction in lung cancer was observed from 1960 to 1970 among men ages 25 to 54 years in London than in other parts of England and Wales. Little difference was observed in women over a similar period. It seemed possible that this trend might reflect an earlier and somewhat greater decline in smoke pollution in London than in other parts of the country. Subsequent analysis of trends in lung cancer mortality in relation to changes in pollution in the different regions of England and Wales, however, have failed to provide support for this hypothesis. They have also indicated that it is almost impossible to allow adequately for changes in cigarette consumption and, in particular, such imponderables as the reduction of tar and nicotine in cigarettes.

TABLE 3
OBSERVED AND EXPECTED LUNG CANCER DEATHS BY PLACE OF RESIDENCE AND BY OCCUPATIONAL EXPOSURE TO DUSTS, FUMES, GASES, OR X RAYS

Pollution	No. of Cities	Occupationally exposed			Not occupationally exposed		
		Observed	Expected	Ratio	Observed	Expected	Ratio
Particulates							
High							
(130–180 $\mu g/m^3$)	8	45	32.9	1.37	66	73.9	0.89
Moderate							
(100–129 $\mu g/m^3$)	11	21	18.8	1.12	39	49.5	0.79
Low							
(33–99 $\mu g/m^3$)	14	48	37.4	1.28	110	100.1	1.10
Benzene sol.							
High							
(8.5–15.0 $\mu g/m^3$)	9	28	21.0	1.33	52	51.5	1.01
Moderate							
(6.5–7.9 $\mu g/m^3$)	10	44	32.7	1.35	65	75.1	0.87
Low							
(3.4–6.9 $\mu g/m^3$)	12	33	29.2	1.13	76	81.8	0.93

Hammond and Garfinkel, 1980

DIESEL EMISSIONS

The review conducted by the Health Effects Panel of the NRC–NAS Diesel Impact Study Committee (27) pointed out that there have been few studies that permit one to draw any definite conclusions on the potential hazards of exposure to diesel emissions. The review of the Royal College of Physicians (30) noted that the increase in the use of diesel fuel in Britain followed, rather than preceded, the striking rise in lung cancer mortality and therefore could not have been a major factor in the etiology of that disease. Possibly the earliest observation on this topic was made by Doll in his review of occupational lung cancer. He observed (10) that police traffic controllers, who might be expected to be more highly exposed to traffic pollution (including diesel emissions) than other people, had no excess risk of dying from lung cancer.

In the United States, Heuper (19) noted high lung cancer rates among transportation workers exposed to (a) the exhaust from gasoline and diesel engines, (b) petroleum lubricants, and (c) dust from asphalt roads. He noted that 75% of the deaths among transport workers occurred among the 25% of the group who were employed on the railroads. Among Finnish railroad workers, higher malignant disease rates occurred in engineers as compared with trainmen or clerks (18). In the Los Angeles County Cancer Surveillance Program (26), increased SMRs were noted in truck drivers (165), auto repair workers (146), and transportation workers (127), although in the absence of smoking information, too much should not be made of these relatively small excess risks. No doubt, too, exposures other than diesel emissions may have been involved. In a comprehensive study of the mortality of U.S. coal miners carried out by Rockette (29), 22,998 miners covered by the UMWA health and retirement fund were followed from January 1959 to December 1971. Overall, there was a modest elevation of the SMR for lung cancer (112.5) which Rockette commented was "well within the range of what might result from differences in residence or smoking habits of the control study groups." Exposure to diesels in the coal mines was not considered by Rockette. But subsequently, it was pointed out (12) that 95% of all dieselized coal mines in the United States are in the states of Washington, Montana, Wyoming, Utah, Colorado, New Mexico, and Alaska. This latter district had the lowest death rates from all causes, from all cancers, and from cardiovascular disease, and the second lowest death rate from respiratory cancer of all 10 coal mining districts. Thus, the evidence from diesel exposure in the mines does not suggest that these emissions pose a hazard to the employees.

Leupker and Smith (25) studied mortality of members of the International Brotherhood of Teamsters whose employers contributed to the Central States, Southeast, and Southwest Areas Health and Welfare fund. The population during May 1976 comprised 184,435 members from 33 contiguous states. Mortality for 3 months (May to July 1976) was compared with that for the U.S. population in 1974. Mortality for all causes other than respiratory tract cancer and motor vehicle accidents was lower among Teamsters than among the U.S. population. Higher mortality was reported for respiratory tract cancer and the difference reached statistical significance in the 50- to 59-year-old age group. The authors

speculated that this excess might be caused by "environmental exposure (e.g., to diesel exhaust fumes)," but no evidence was presented to support this suggestion. They recognized, however, that without knowledge of smoking habits, no firm inference could be made about such environmental exposures.

On the assumption that oat- or small-cell lung cancer is more likely to result from occupational exposures than from smoking, Wegman and Peters (38) used the Massachusetts Tumor Registry to compare the frequency of various occupations in 100 cases of oat-cell lung cancer with that found in an equal number of cases of cancer of the central nervous system. Smoking and lifetime occupational histories were taken from cases and controls and "usual" occupation was extracted from the death certificate of any man who had died. The tumor registry abstract revealed that eight (10%) cases but only one (1%) control had worked in transportation. But, when more detailed information on occupation from all sources of information was compared, this difference disappeared almost entirely. Furthermore, a higher proportion of the oat-cell cancer cases were smokers. All in all, this study provides little support for an association of oat-cell lung cancer with transportation work, let alone with diesel emission exposures.

A number of studies have been carried out to investigate the specific relationship between exposure to diesel emissions and lung cancer. In 1959, Kaplan (24) reviewed the Baltimore and Ohio Railroad Relief Department's medical records. Deaths that had occurred between January 1953 and December 1958 were tabulated. Comparison was made with expectations derived from the American Cancer Society. Workers were categorized into three groups on the basis of their liability to exposure to diesel exhaust: (a) operating (engineers, firemen, motormen, road brakemen, etc.); (b) non-operating (mechanical service workers in shops or roundhouses who might be exposed to diesel smoke to a lesser degree, but who were exposed to coal smoke, soot, dust, lubricating oil, and welding fumes); and (c) non-operating (rarely exposed to noxious fumes at work: clerks, janitors, agents, bridge inspectors, and port captains). The death rates for lung cancer were lower in all three groups than in the United States as a whole and there was no suggestion that the rates were higher in those most exposed to diesel exhaust. Again, the lapse time may have been too short to show any effect.

A study of mortality among potash miners and millers from eight companies in New Mexico was described by Waxweiller and his colleagues (37). The cohort comprised 2,743 underground and 1,143 surface potash workers between 1940 and 1967. Follow-up of mortality to July 1, 1967, was carried out. There was no evidence of any increased risk of respiratory cancer in these workers among either the surface or underground groups. There was, however, a modest excess of other respiratory diseases (ICD codes 470–479 and 500–527) among the underground workers; whereas 4.57 deaths were expected, 11 were found. The excess was significant at the 5% level. The authors noted that 5 of the 11 deaths occurred in men with pneumoconiosis, which was thought to have been due to employment in silicious rock before potash mining.

The authors noted that in two of the mines, diesel engines had been a major source for underground transportation, in one mine for 10 and in the other for 18 years. They stated that no causes of death differed significantly between miners

TABLE 4
DEATHS, ILL-HEALTH RETIREMENTS, AND TRANSFERS TO ALTERNATIVE WORK DUE TO LUNG CANCER IN LONDON TRANSPORT AUTHORITY MALE STAFF AGES 45–64

Group or staff	Man-years at risk	Deaths, ill-health retirements, and transfers to alternative work: 1950–1954	
		Number[a]	Annual rate per 1,000
Motormen and guards (London Transport Railways)	8,253	11 (10)	1.3
Engineering staff (trolley bus depots)	5,529	10 (9)	1.8
Engineering staff in bus garages (Chiswick Works)	9,979	12 (12)	1.2
Central bus drivers	33,466	23 (17)	0.7
Central bus conductors	16,978	18 (15)	1.1
Engineering staff	18,140	22 (21)	1.2
		Deaths 1950–53	
Population	Male population[b] ages 45–64	Number	Annual rate per 1,000
England and Wales	19,947,000	26,689	1.3
Greater London	3,834,000	6,292	1.6

Source. Raffle (1957).
[a] Deaths are in parentheses.
[b] 1950–1953.

who had worked in dieselized as compared with non-dieselized mines. They commented, however, that there might have been insufficient lapse time since the start of diesel usage for excess deaths to have occurred from diseases characterized by long latency, such as lung cancer.

A study of lung cancer in relation to diesel exposure among London Transport workers was originally reported by Raffle (28) as an illustration of the use to which good industrial records can be put to answer questions and further research. The main assumption was that mechanics (or engineering staff) working in bus garages were exposed to an excess of diesel exhaust. If such exposure increased the risk of lung cancer, then these workers would be expected to have a higher incidence than the general population or than other men working for London Transport. Table 4 shows the evidence presented by Raffle. Jobs are listed in rank order from least to most diesel emission exposures. The overall annual rates compare favorably with those for all England and Wales or Greater London, which is possibly more appropriate. When Raffle presented this table,

TABLE 5
LUNG CANCER CASES AMONG LONDON TRANSPORT STAFF IN RELATION TO THE NUMBER EXPECTED ON THE BASIS OF GREATER LONDON DEATH RATES (1950–1974, MALES AGES 45–64 ONLY)

Job category	Man years at risk	Expected deaths	Observed cases	Mortality ratio (%)
Motormen and guards	35,610	67.7	59	87
Engineers, central works	30,031	63.2	42	66
Bus drivers	175,909	346.8	259	75
Bus conductors	93,095	174.5	130	75
Engineers, garages	86,054	197.1	177	90
Total	420,699	849.2	667	79

Source. Waller, 1981.

the period of follow-up was short, possibly too short compared with the latency of lung cancer for many cases to have developed. The study has therefore been extended, and the results covering a 25-year period have been reported by Waller (36) (Table 5).

Waller used Greater London to derive his expected number of deaths. Note that the SMRs for each job category were less than 100. Furthermore, the SMR of those most exposed to diesel emissions was almost identical to the SMR for those with the least exposure. The two main weaknesses in this study are first, that there has never been adequate allowance for smoking and, second, that those who left the industry voluntarily or on normal retirement were not followed. To the extent that London Transport has a good pension system, this weakness may have been exaggerated. Even allowing for the deficiencies in the data, it is hard to believe that diesel emissions pose much of a risk for lung cancer.

Howe and his colleagues (23) recently have reported a study of 43,826 male pensioners of the Canadian National Railroad Company. Cancer mortality from 1965 to 1977 was related to diesel and other occupational exposures. The likelihood of exposure to diesel emissions was categorized on a qualitative basis for each job in which the man was employed immediately before retirement. Three categories were used: not exposed, possibly exposed, and probably exposed. No quantitative measurements were made, and no attempt was made to validate the classification, nor was duration of employment considered. An elevated risk of lung cancer was observed in men thought to be exposed to diesel exhaust emissions and a significant dose–response relationship was noted, the relative risks being 1.00, 1.20, and 1.35 for the three categories of exposure, respectively. Similar relative risks were observed for exposure to coal dust which the authors point out might have been expected, since during the transition from steam to diesel power, most members of the cohort who were exposed to diesel emissions also would have been exposed to coal dust. A problem arose because of confounding with asbestos. Exclusion of occupational groups that involved maintenance, how-

ever, made little difference to the relative risks. Individuals who worked in maintenance could not be specifically identified, and it is uncertain how adequately exclusion of the group would eliminate asbestos confounding. Smoking histories were not available, but since the relative risks of smoking-related cancers other than lung were 1.00, 1.08, and 1.16 for the diesel-exposed groups, the authors concluded that smoking could not completely explain the apparent association with diesel emissions. It is debatable how well this comparison eliminates confounding by smoking, especially since the relative risks of emphysema were suggestive of a smoking effect: 1.00, 1.35, and 1.44 across the categories. Further reasons for caution in drawing conclusions about the effect of diesel emissions on lung cancer from this study are that only last job was used, and no consideration was given to duration of exposure. It is to be hoped that the authors will continue to explore the possible confounding of smoking and asbestos exposure in this interesting study.

Schenker and his colleagues (31) are conducting a retrospective cohort mortality study of 60,000 railroad workers ages 45 to 64 through the Railroad Retirement Board. Men with at least 10 years of exposure are being followed from January 1, 1967, to December 31, 1979. Comparison by diesel exposure with that expected for the U.S. population is being made. A pilot study of 2,662 subjects, 2,519 of them white males, has been presented. Broken down into high and low diesel exposure groups and using a Cox "proportional hazards model," the relative risk of lung cancer was 1.50 for low and 2.77 for the high diesel exposure groups. There is probable confounding of the higher risk estimate with asbestos, which is being investigated, but the finding in the less exposed group is believed to indicate a true risk. However, it is not clear that smoking has been adequately allowed for to date.

Finally, a historical cohort mortality study of 34,156 male members of the heavy construction equipment operators union with potential exposure to diesel exhaust emissions was made by Milby (13). The cohort comprised all members of the International Union of Operating Engineers, locals 3 and 3A, who were members for 1 year or more between January 1, 1964, and December 31, 1978. Vital status has been established for 95% of the cohort, 10% of whom were found to have died. A survey of a small sample of the cohort suggested that smoking habits were similar to those reported by the National Center for Health Statistics for the male population. Consequently, it is thought that smoking will not confound any occupational relationship. Comparison of mortality of the cohort with that expected for all U.S. white males indicated that the overall mortality was significantly below expectation. On the other hand, mortality from certain specific causes of death, including lung cancer, was raised among retired workers and certain other subgroups. Whether the raised SMRs are due to exposure to diesel emissions is not yet clear, and a more detailed presentation of the findings is awaited with interest.

In summary, the evidence suggests that general air pollution is a rather unimportant factor in the etiology of lung cancer. The possibility, particularly of its interaction with cigarette smoking, cannot, however, be excluded. The effect of exposure to diesel exhaust emissions is uncertain. Earlier investigations were

essentially negative, but some recent studies have suggested that such exposures may cause a small increase in lung cancer risk. However, exposure to diesel exhaust is often confounded with exposure to asbestos, and to date, the elimination of such confounding has not been completely achieved. In addition, adequate allowance for cigarette smoking in assessments of diesel emissions needs to be made. Further studies differentiating diesel emission and asbestos exposures more completely and, if possible, with better allowance for cigarette smoking are needed.

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