

## Lung Cancer and Diesel Exhaust: A Review

Kyle Steenland, PhD

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The evidence from animal studies indicates that organic extracts of diesel particulate are mutagenic and carcinogenic. Of four animal inhalation studies, two have been positive and two have been largely negative. The most recent data indicate that inhalation studies may be positive only with high doses of exhaust. Human studies of diesel-exposed occupations have been inconclusive. These studies have focused on truck drivers, bus drivers and garage workers, railroad workers, and heavy equipment operators. Most human studies have not been able to estimate exposure to diesel exhaust. Negative studies have frequently suffered from insufficient potential latency. Positive studies have often failed to control for smoking, and have sometimes involved confounding occupational exposures. In general, the occupational epidemiology of diesel-exposed workers is made difficult by the fact that many of the suspected toxic components of diesel-exhaust are also present in cigarette smoke and in ambient air. There are two ongoing epidemiologic studies in the United States, focusing on railway workers and truck drivers, which attempt to overcome prior difficulties. Preliminary data from the study of truck drivers indicates an excess of lung cancer among workers in the trucking industry compared to the U.S. population, but these data need to be controlled for smoking and analyzed according to diesel exposure

**Key words:** lung cancer, diesel exhaust, epidemiology, occupation

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### INTRODUCTION

The possible association of lung cancer and diesel exhaust has become an important public health question in recent years for three reasons. First, extracts of diesel exhaust are both mutagenic and carcinogenic. Although far from conclusive, recent evidence also suggests that inhaled diesel exhaust may be a lung carcinogen in rodents exposed over a long period to high doses. Second, although the current lull in the oil crisis has slowed the trend of increasing diesel use in automobiles, it remains a possibility that future oil shortages will dictate more use of fuel-efficient diesel engines, substantially increasing the levels of diesel exhaust in urban air. Third, substantial numbers of workers are occupationally exposed. The largest groups include truck drivers, truck and bus mechanics, railroad workers, miners, and heavy equipment operators.

The purpose of this paper is to review the animal evidence of carcinogenicity, to discuss problems in assessing exposure to diesel exhaust in human epidemiologic

National Institute for Occupational Safety and Health, Cincinnati, Ohio.

Address reprint requests to Dr. Steenland at Mailstop R-15, National Institute for Occupational Safety and Health, 4676 Columbia Parkway, Cincinnati, Ohio 45226.

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studies, to review the human epidemiology on lung cancer, and to discuss ongoing epidemiologic research.

## ANIMAL TESTS

Diesel exhaust is characterized by levels of particulate which are 20–100 times higher than those found in gasoline exhaust [Springer and Stahman, 1977]. This particulate is a product of the high temperatures of diesel engines and is primarily made up of elemental carbon. Heavier organic molecules, such as polycyclic aromatic hydrocarbons, do not volatilize but instead are adsorbed to the elemental carbon. This particulate is often of respirable size and can accumulate in the lung, where the aromatic hydrocarbons can slowly leach into lung tissue. Among the polycyclic and nitro-polycyclic hydrocarbons are a number of known mutagens and carcinogens. The organic extracts from the particulate were shown to be carcinogenic in rodents in skin test conducted in the 1950's [Kotin et al, 1955]. In recent years, organic extracts of diesel exhaust have been shown to be mutagenic in a wide variety of systems [Lewtas, 1983; Wei and Shu, 1983]. Most recently, attention has focused on the nitroarenes, thought to account for much of the mutagenic potential of diesel exhaust; some of the individual nitroarenes are known to be carcinogenic in rodents [Pederson and Siak, 1981; Rosenkranz 1984].

Four rodent inhalation studies have tested the carcinogenic effects of diesel exhaust itself. In considering these studies and the doses used, it is worth noting that workplace concentrations of particulate from diesel exhaust may range from 0.1–2.0 mg/cubic meter air. While there is no general occupational standard for respirable particulate, the American Conference of Government Industrial Hygienists (ACGIH) recommends 5 mg/cubic meter as the maximum level for nuisance dust [ACGIH, 1983].

A preliminary report from a study sponsored by General Motors indicated that 90 Fischer 344 rats were exposed to diesel exhaust at 250, 750, and 1500  $\mu\text{g}$  of respirable particulate per cubic meter, for 20 h per day for 15 months, followed by 8 months of nonexposure [White et al, 1983]. Among these 90 rats, five lung tumors occurred. No tumors occurred in a 30-animal control group. The tumors in the exposed group did not show a dose-response; however, the lack of a dose-response is not that unusual given the small number of total tumors and the relatively low levels of exhaust.

An Environmental Protection Agency (EPA) animal study also investigated carcinogenicity [Pepelko and Peirano, 1983]. Strain A mice were exposed via inhalation to 6 mg/cubic meter of diesel exhaust for 8 h per day, 7 days a week, for various intervals of time which averaged about 9 months. No increase in tumors over controls was observed. In the same study by EPA, lung tumor incidence was also evaluated in both male and female Sencar mice exposed via inhalation, from conception until 15 months. Concentrations were 6 mg/cubic meter for the first 12 weeks and 12 mg/cubic meter thereafter. The female mice, but not the males, showed a significant increase in lung tumors.

Another recently completed animal inhalation study by NIOSH was negative for carcinogenesis (results presented at a public meeting in Cincinnati on March 29, 1984). This study involved 288 Fischer 344 rats of both sexes who were exposed to room air, to diesel exhaust, to coal dust, and to a combination of coal dust and diesel

exhaust. The rats were exposed to diesel exhaust at a level of 2 mg of respirable particulate per cubic meter of air for 7 h per day, 5 days per week for 2 years. There were no excess neoplasms in the exposed group.

Ideally a bioassay to test carcinogenic potential should involve a life-long treatment at a range of doses, including high doses approximating the maximum tolerated dose (perhaps 10–15 mg/cubic meter in rodents). The animal inhalation studies conducted to date have not always fulfilled those requirements. There are four new rodent inhalation studies of diesel exhaust, which are either recently completed or nearing completion, but which have not yet been published. Lovelace Inhalation Toxicology Research Labs (Albuquerque, NM), in research supported by the Department of Energy, exposed male and female rats to 350, 3,500, and 7,000  $\mu\text{g}/\text{cubic meter}$  of exhaust for 7 h a day, 5 days a week for 30 months. There were approximately 130–140 animals in each treatment group (presentation by Dr. Jo Mauderly, Nov. 11–15, 1985, meeting of the American Association of Aerosol Research, Albuquerque, NM). These data indicated a highly significant increase in lung tumor incidence in the group of rats exposed at the highest dose. Malignant tumors occurred in 1.4% of the controls, 0.7% of the lowest exposure group, 0.7% of the middle level exposure group, and 9.8% of the highest exposure group. For total tumors, the numbers were respectively 1.4%, 0.7%, 4.6%, and 16.1%, reflecting a number of benign tumors in the two highest exposure groups.

Two European studies (Battelle in Geneva, Fraunhofer Institute in Hanover) and one U.S. study (General Motors) are also concluding. Preliminary data from the Battelle study were presented at the Albuquerque meeting (cited above) and indicated an increase of tumors in the most highly exposed animals.

In summary, the data from animals exposed to diesel exhaust via inhalation yield differing results. Although the picture is not yet clear, and results of more studies will be known shortly, it appears that positive results will be obtained if rodents are exposed to high enough levels for a long enough period.

## PROBLEMS IN ASSESSING HUMAN EXPOSURE TO DIESEL EXHAUST

A major difficulty in human occupational studies has been the measurement of actual diesel exposure. Many studies have simply compared men in certain job categories, presumably exposed to diesel fumes, to some standard (presumably nonexposed) population. Even when diesel fumes have been measured, there is no standard method for such measurements. From an industrial hygiene standpoint, it is not clear exactly which substance to measure when assessing occupational exposure to diesel exhaust. Diesel exhaust is composed of gases (nitrogen oxides, carbon monoxide), and various hydrocarbons bound to a carbon core. Most studies have reported levels of particulate, but such particulate is generated by many sources other than diesel engines. Attention has been focused on the PAHs (polycyclic aromatic hydrocarbons) and nitro-PAHs in the exhaust, because many of these compounds are known carcinogens. However, there are no standard methods of measuring PAHs, and PAHs are also emitted by many sources other than diesel (eg, cigarette smoke).

One alternative method of estimating diesel exposure is the measurement of elemental carbon, which is the core of the diesel exhaust particle. After hydrocarbons are burned off, the amount of elemental carbon can then be measured [Cadle et al,

1980]. Elemental carbon may be a better measure than other alternatives because, unlike other compounds, it is particularly characteristic of diesel exhaust.

Finally, of course, even if valid current measurements can be obtained, retrospective studies of diesel-exposed cohorts face the usual problem of trying to estimate via current measurements the exposures of interest which occurred 20 years ago.

## **HUMAN EPIDEMIOLOGY**

### **Miners**

Some underground miners are exposed to diesel exhaust, but for a variety of reasons this occupational group has not been studied to assess whether diesel exhaust is associated with lung cancer.

The introduction of diesel engines into metal mines dates from the early or mid-1960's. Currently in the US there are approximately 250 permanently operating underground metal mines, and 135 intermittently operating metal mines (personal communication, Peter Turcic, MSHA). There are approximately 20,000 miners in these mines, but not all are currently working. Although virtually all of these mines use diesel equipment, it is difficult to estimate how many of these miners are actually exposed to diesel fumes. Furthermore, the still relatively short potential latency period limits the usefulness of a cohort of metal miners.

The introduction of diesel engines into coal mines is an even more recent phenomenon, and still is quite limited. As of 1983, approximately 1,000 diesel units were in place in underground coal mines in the U.S., up from about 200 units in 1977 [Daniel, 1984]. The number of units per mine varies greatly; one mine may account for over 100 units.

### **Truck Drivers**

Table I lists 14 studies assessing lung cancer among truck drivers. Nine of these studies were positive and statistically significant, while five were not. Of those which were not significant, several did show an excess. Often those studies which were not significant lacked power to detect a relative risk on the order of 1.5.

None of these studies measured the exposure of truck drivers to diesel exhaust. In the only published exposure data for the trucking industry, Ziskind et al [1978] found that 40% of 88 trucks in their survey had a significant entry of diesel fumes into the cab of the truck due to leaks in the cab. They also found that over 50% of the trucks tested while idling had about 0.5 ppm of nitrogen oxide in the cab, and 30% had levels over 1 ppm. No quantifiable relationship between nitrogen oxide and particulate was presented by Ziskind et al.

Besides leaky cabs, truck drivers are presumably more highly exposed than the general population due to the higher background levels of diesel exhaust on highways. Older trucks also had their exhaust directly under the cab.

Overall, the studies in Table I appear to indicate an excess of risk of lung cancer among truck drivers, on the order of 30–50%. Despite the large numbers of studies of truck drivers, it is not clear whether this excess is due to smoking or some other factor. Most of these studies did not control for smoking, while those that did often lacked statistical power. Data on diesel exposure was not available in any of these studies.

The following data may help illustrate the possible confounding effect of smoking. A national survey of a random sample of 32,000 U.S. white males, done in 1970, indicated that 31% were current smokers, while among 920 white male truck drivers the percentage of current smokers was 39%. Such a difference in smoking habits might account for a 10–20% excess in lung cancer rates among truck drivers vs the national population [Axelson, 1978]. It should be noted that truck drivers may often smoke in confined spaces (truck cabs), which might make actual smoking differences more extreme than they would otherwise appear.

It should be noted that of the larger studies in Table I which did adjust for smoking, most showed that the lung cancer excess persisted after smoking adjustment [Walrath et al, 1985; Dubrow and Wegman, 1984; Williams et al, 1977]. One study [Damber and Larsson, 1985] was negative after smoking adjustment for men who died under age 70 but was positive for men who died after age 70.

### **Bus Company Employees in London**

There are only three studies of workers for bus companies which have information on lung cancer, all done in London. Table II describes these studies. The two principal studies are by Waller et al [1980] and Rushton et al [1983]. While both studies are generally negative, no firm conclusions can be drawn.

The large study by Waller et al [1980] is unusual in that it did include some exposure data. General area samples for benzo(a)pyrene indicated that exposure was only slightly higher for garage workers than for the general urban public. This study is limited because deaths were ascertained largely among the currently employed. Furthermore, due to limitations of the data, no analyses by duration of exposure or latency were possible. The authors defined five job categories ranked by presumed diesel exposure. No clear trends of lung cancer with job category were apparent. Harris [1983] has analyzed this study in detail, from the point of view of risk assessment. More data on diesel exposure in bus garages were recently published by Waller et al [1985].

The study by Rushton et al [1983] covered only transport workers in the garages (bus drivers excluded), but did include terminated and retired employees. The average length of follow-up was only 6 years. There was no excess of lung cancer in the cohort as a whole. A significant excess lung cancer mortality was observed in this study for those members of the cohort in the “general hand” category (42% of the cohort), but no details are given on what type of exposures were involved in this presumably heterogenous category. Furthermore, this positive finding was not controlled for smoking. On the other hand, the study was negative for bus mechanics (27% of the cohort), who presumably were among the more highly exposed. This study is weakened in that there is no analysis by duration of employment or time since first employment.

### **Railroad Workers**

There have been three studies of railroad workers (Table III). Two of these studies [Kaplan, 1959; Hueper, 1955] probably lacked sufficient latency to observe an effect, given that diesel engines replaced steam engines gradually from the 1930's to the 1950's on the railroad. The study by Howe et al [1983] was positive but has some limitations, as discussed below.

TABLE I. Studies of Lung Cancer Among Truck Drivers

First author (yr)	Population	Study Design	Results*	Comment
Dubrow [1976]	Massachusetts male deaths 1971-73	SMOR	173*	Crude adj for smok using survey, crude exp defn using death certs
Milham [1983]	Washington male deaths 1950-79	PMR	118*	No smok adj, crude exp defn with death certs, short latency, other smok related diseases elevated
Reg General [1978]	English and Welsh male deaths 1970-72	PMR	123*	No smok adj, crude exp defn from death certs (last occ), other smoking related dis elevated, adj made for class
Peterson [1980]	California male deaths 1959-61	PMR	130*	Crude exp defn from death certs, no smok adj, little latency
Menck [1976]	Los Angeles male deaths 1971-72	SMR	165*	No smok adj, crude exp defn from death certs, pop at risk estimated
Williams [1977]	432 Lung ca cases from 3rd Natl Cancer Survey	Case-contr	1.52	Good data on smok and occup, moderate power
DeCoufle [1977]	Male lung ca cases at Roswell Park 1956-65	Case-contr	0.92	Smok adj, exp defn as ever employed, little latency, low power
Leupker [1978]	Teamster deaths 1976	SMR	1.21	No retirees, no smok adj, no analysis by job
Morton [1982]	1,600 Male lung ca cases in Portland 1968-72	SMR	1.82*	Pop at risk estimated, no smok adj, exp defn from death certs

(continued)

TABLE I. Studies of Lung Cancer Among Truck Drivers (continued)

First author (yr)	Population	Study Design	Results*	Comment
Milne [1983]	Lung cancer cases in 1 California county 1958-62	Case-Contr	1.6*	No smok adj, exp defn from death certs, little latency
Walrath [1985]	1,454 Truck drivers in 1950s followed to 1970	SMR	1.49*	Good data on smok and occup in '50s, short latency
Hall [1984]	502 Male lung ca cases diagnosed in 1970 in NY	Case-contr	1.4	Good data on occup, no adj for smok due to small no., moderate power
Damber [1985]	467 Male lung ca cases in Sweden 1971-77	Case-contr	0.9 (smok) 5.4 (nonsmok)	Good data on smok and occup, moderate power, sig excess among men who died after age 70, no info when diesel use began
Ahlberg [1981]	161 Lung ca cases in Sweden 1961-73	Case-contr	1.33*	No smok adj, exp defn from death certs

\*Statistical significance,  $p < 0.05$ . Results indicate odds ratios in case-control studies, odds ratios in SMOR (standardized mortality ratio) studies, proportionate mortality ratio in PMR (proportionate mortality ratio) studies, and rate ratios in SMR (standardized mortality ratio) studies.

TABLE II. Studies of Lung Cancer Among London Transport Workers

First author (yr)	Population	Study design	Results	Comment
Raffle [1957]	Males aged 45-65 who worked during 1950-55, 84 lung ca deaths	SMR	No excess	Diesel buses introduced from 1935-52, potential latency short, six job categories, none with excess, few dths in each category, no retirees, no exp or smok data
Waller [1980]	Males aged 45-64 during 1950-74, 667 lung ca deaths	SMR	No excess	Five job categories, none with excess, no smok data, no retirees, no exp data
Rushton [1983]	8,590 Garage workers who were employed for at least 1 year from 1967-75, followed over same yrs, 102 lung ca deaths	SMR	No overall excess	Several job categories, only "gen hand" (46% of cohort) had a sig excess, (SMR = 133), no details on "gen hand" category, no data on exp, smok, or avg length of employment

TABLE III. Studies of Lung Cancer Among Railroad Workers

First author (yr)	Population	Study design	Results	Comment
Kaplan [1957]	Railroad workers active and retired followed 1953-58, 154 lung ca deaths	SMR	No excess	Workers divided into three job categories, ranked by presumed exposure, no excess in any group compared to, U.S., possibly inadequate latency or duration of exp (diesels introduced 1935-58), no exp or smok data
Hueper [1955]	Railroad workers followed from 1939-50	MR?	Apparent excess	Methods unclear, 75% of all lung ca deaths found in operating personnel who formed only 25% of total personnel, possibly inadequate latency or duration of exposure
Howe [1983]	Railroad pensioners retired prior to 1965 and alive, plus retirees from 1965-77, followed 1965-77, 933 lung ca deaths	SMR	Apparent excess, dose-response	Three exp groups (exp, non-exp, possibly exp) based on job title at retirement, lack of detailed history made control of asbestos exp limited, no analysis by duration of emp or latency, other smoking-related causes of death also elevated in exp group

Howe et al. [1983] studied retired pensioners who died between 1965–77. These workers were classified, based on their last reported jobs, as nonexposed, possibly exposed, and probably exposed to diesel exhaust. No details were given about which jobs were classified into which exposure groups. Standardized mortality ratios (SMRs) for lung cancer for these groups were calculated by comparison with the Canadian population and these SMRs were compared, using the nonexposed group as a baseline. In this latter comparison a significant dose–response (1.00, 1.20, and 1.35) was observed. Steam engines using asbestos were gradually replaced by diesel engines, a process culminating in the 1950's. The authors attempted to separate asbestos-exposed workers from others, but this attempt is not convincing, due to the lack of detailed work histories for this cohort. Furthermore, no data were presented on the smoking habits of the cohort. SMRs for smoking-related diseases (emphysema, laryngeal cancer, esophageal cancer, buccal cancer) showed positive trends when the three diesel exposure categories were examined, raising the possibility that the “dose–response” evident for diesel exposure may in fact have been due to smoking.

### **Heavy Equipment Operators**

Wong et al [1985] conducted a mortality study of 32,000 heavy equipment operators exposed to diesel exhaust and employed for at least 1 year between 1964–78. These investigators did not find an excess of lung cancer, but they did find an increasing trend in lung cancer with increasing latency and increasing duration of employment. Furthermore, they did find a significant excess of lung cancer among 1,345 dead retirees (SMR = 164,  $p < 0.01$ ). When these retirees were restricted to those who lived to age 65, to correct for earlier deaths which presumably were a selected population of sick people, an excess was still observed (SMR = 130,  $p < 0.05$ ).

Although total length of union membership was known, approximately 30% of the cohort had no detailed work histories, and no job category could be determined for them. The remainder had partial work histories, and their jobs were classified and ranked according to presumed diesel exposure. While analysis by job category showed no increased lung cancer among those who presumably had higher levels of diesel exposure, those with no work history showed a significant increase in lung cancer compared to the U.S. population.

Given that most of these men worked outdoors, this cohort may have had relatively low levels of diesel exposure. No environmental data were presented in this report. The authors had no historical smoking data, but they did conduct a survey of 107 currently employed members, and this data indicated that these men had smoking habits which did not differ significantly from the smoking habits of the 1970 U.S. population as determined by the National Center for Health Statistics survey of that year.

### **Case–Control Study of Lung Cancer and Diesel Exposure**

Hall and Wynder [1984] published a report of a case–control study of lung cancer and diesel-exposed occupations. Approximately 500 cases and 500 hospital controls, who were diagnosed in or about 1980, reported their major lifetime occupation. These were then grouped by the investigators according to presumed diesel exposure. After correction for smoking, no significant excess risk was observed for diesel-exposed occupations. The major limitation in this study is that the exposure

classification was necessarily quite crude. Other limitations were that only 64% of the eligible subjects were interviewed and that the power of the study was relatively low to detect excess risks, especially for specific occupations.

## ONGOING STUDIES OF LUNG CANCER AND DIESEL EXPOSURE

There are two ongoing studies in the U.S. which should help clarify the issue of whether exposure to diesel exhaust is associated with lung cancer.

Schenker et al [1984] are completing a large cohort mortality study of 60,000 railroad workers, who are known to have exposure to diesel exhaust while working in the railroad industry [Schenker and Speizer, 1980]. Relative exposure rankings among various jobs were determined using sampling results from current workers and historical reconstruction.

Using the above cohort, Schenker et al [1984] have reported findings from a pilot study of 2,519 white male railroad workers (exposed and nonexposed). These men were aged 45–64, and all had at least 10 years' service by 1967. The exposed group had an SMR for respiratory cancer of 0.94 when compared to the national population. However, when the exposed were compared directly to the nonexposed, standardizing for age and calendar time, the exposed showed a rate ratio for lung cancer of 1.42 (0.45–2.39).

While no smoking data were available for workers who were not current, the investigators plan to estimate the effect of smoking by considering the smoking habits of the currently employed, and by conducting case-control studies. Asbestos is another potential confounder in this cohort.

In a collaborative project being conducted by researchers from NIOSH and NCI, this author and others are studying the mortality of approximately 10,000 long-term Teamsters who died in 1982–83 in the Central States region. These men have held the following jobs: truck drivers (69%), dockworkers (5%), mechanics (6%), and jobs outside the trucking industry (20%). All these men have worked minimally for 10 years in a Teamster job, and most have been lifetime Teamsters. Teamster records include a detailed work history.

Exposure to diesel exhaust will be determined by sampling current workers as well as historical reconstruction. Workers in the Teamsters outside the trucking industry will be considered nonexposed, with the exception of workers in presumably diesel-exposed jobs such as heavy equipment operators. A relative ranking of exposure among long-haul truck drivers, city truck drivers (often using gasoline-powered vehicles), dockworkers, and mechanics will be developed.

Our study includes two components. First, a random sample of 2,000 deaths will be chosen, and smoking histories of these individuals will be obtained from next-to-kin. This series of deaths will be used to conduct a proportionate mortality analysis for a variety of individual causes of death, while controlling for smoking.

In a preliminary proportionate mortality analysis, approximately 10,000 death certificates of Teamsters were reviewed. These deaths represent all the 1982–83 deaths among long-term Teamsters in the Central States region. Using the occupational data on the death certificates, these deaths were classified into truck drivers, mechanics in trucking firms, dockworkers in trucking firms, and other Teamsters who were not in the trucking industry and whose lifetime occupation did not involve

exposure to diesel fumes. Any death certificate with data insufficient to classify the individual (eg, a mechanic for whom it was not clear whether he worked in the trucking industry) or whose job was likely to involve exposure to diesels outside the trucking industry (eg a crane operator in construction) was excluded. A proportionate mortality analysis for lung cancer was conducted for these deaths, using the NIOSH life-table program [Waxweiler et al, 1983], in which the proportion of death due to lung cancer in the study population was compared to the proportion expected based on the U.S. population.

The results in Table IV indicate that among our study population truck drivers have an approximately 50% excess risk of lung cancer, confirming the results of numerous other studies. Mechanics (who may have the highest exposures to diesel exhaust) show a more than two-fold risk, compared to the U.S. population. Dockworkers show an approximately 30% excess risk, while Teamsters outside the trucking industry show an approximately 15% excess risk. It must be stressed that these results are preliminary data based on the relatively crude occupational classifications available on death certificates. Furthermore, they are not controlled for smoking. Most of these excesses are in the range which could be explained by increased smoking by Teamsters.

The second and more important component of the NIOSH-NCI study is a case-control study of lung cancer, controlling for smoking. Approximately 1,500 lung cancer deaths in 1982-83 will be compared to 1,500 other deaths. Analysis will estimate the lung cancer risk of various job categories in the trucking industry compared to nontrucking jobs. In addition, analyses based on a relative ranking of jobs by diesel exposure will also be conducted. It is this component of the NIOSH study which will hopefully provide the best data to test the hypothesis of an association between lung cancer and diesel exhaust.

## CONCLUSION

Diesel exhaust is known to contain carcinogenic compounds which are attached to respirable particulate, and there are animal data to indicate that diesel exhaust can act as a lung carcinogen. Hence there is legitimate concern which has prompted numerous epidemiologic studies regarding the possible association of lung cancer and diesel exhaust. These studies to date have been inconclusive. Many of these studies have suffered from problems common in retrospective occupational studies. It is usually quite difficult to accurately assess historical exposures. Given that the introduction of diesel engines into a number of occupations is relatively recent, insufficient potential latency is also a factor.

The epidemiology of lung cancer and diesel exhaust is particularly difficult because gasoline exhaust, cigarette smoke, and general urban air pollution contain many of the same suspect compounds which are contained in diesel exhaust. Further-

**TABLE IV. Proportionate Mortality Ratios for Teamsters 1982-83 Deaths, by Job Category**

Job category (n)	PMR <sup>a</sup>	95% CI <sup>b</sup>
Mechanics (255)	2.26	1.62-3.09
Truck drivers (5,834)	1.54	1.44-1.66
Dockworkers (490)	1.32	0.99-1.75
Others (nontrucking) (1,064)	1.16	0.95-1.42

<sup>a</sup>PMR, proportionate mortality ratio.

<sup>b</sup>CI, confidence interval.

more, occupational exposures to diesel exhaust are usually relatively low, and measurement techniques are not standardized. Therefore, beyond the usual problems in assessing exposure in retrospective studies, the measurement of current exposure to diesel exhaust poses particular problems.

Judging from the literature to date, if diesel exhaust is a human lung carcinogen, it appears to be a relatively weak one (at the levels of occupational exposure which have been studied). Hence, control over smoking, a major confounder, is especially important. It is difficult to control for smoking in retrospective occupational cohort studies [Steenland et al, 1984], although such a study design has important advantages over population-based case-control studies (higher exposures, better power). Case-control studies nested within cohorts exposed in the past may provide a better approach, especially if there is a group of nonexposed or minimally exposed within the cohort.

## REFERENCES

- American Conference of Government Industrial Hygienists (1983): Threshold limit values for chemical substances and physical agents in the work environment with intended changes for 1983-1984. ACGIH.
- Ahlberg J, Ahlbom A, Lipping H, Ahlbom A, Norell S, Osterblom L, (1981): Cancer among professional drivers. *Laekartidningern* 78: 1545-1546.
- Axelsson O (1978): Aspects on confounding in occupational health epidemiology. *Scan J Wk Env Hlth* 4:85-89.
- Cadle S, Groblicki P, Stroup D, (1980): Automated carbon analyzer for particulate samples. *Analy Chem* 52: 2201-2206.
- Damber L, Larsson L (1985): Professional driving, smoking, and lung cancer: a case-referent study, *Brit J Ind Med* 42: 246-252.
- Daniel JH (1984): The use of diesel-powered equipment in US underground coal operations. Presented at the American Mining Congress International Coal Show, Chicago, Illinois: Bureau of Mines, Dept. of Interior, May 3, 1984.
- DeCoufflé P, Stanislawczyk K, Houten L (1977): A retrospective survey of cancer in relation to occupation, NIOSH report No 77-178.
- Dubrow R, Wegman D (1984): Occupational characteristics of cancer victims in Massachusetts, 1971-1973. NIOSH Publication No. 84-109.
- Hall N, Wynder E (1984): Diesel exhaust exposure and lung cancer: a case-control study, *Env Res* 34: 77-86.
- Harris J (1983): Diesel emissions and lung cancer. *Risk Analysis* 3,2: 83-100.
- Hueper W (1955): A quest into environmental causes of carcinoma of the lung. Public Health Monograph No. 36, U.S. Dep HEW, PHS.
- Howe G, Fraser D, Lindsay J, Presnal B, Zhang Yu S (1983): Cancer mortality in relation to diesel fume and coal exposure in a cohort of retired railway workers, *JNCI* 6: 1015-1019.
- Kaplan I (1959): Relationship of noxious gases to carcinoma of the lung in railroad workers. *JAMA* 171: 2039-1019.
- Kotin P, Falk H, Thomas M (1955): Aromatic hydrocarbons. *Arch Ind Hlth* 11: 113-120.
- Leupker R, Smith M (1978): Mortality in Unionized Truck Drivers. *JOM* 10: 677-682.
- Lewtas J (1983): Evaluation of the mutagenicity and carcinogenicity of motor vehicle emissions in short-term bioassays. *Env Hlth Per* 47: 141-152.
- Menck H, Henderson B (1976): Occupational Differences in Rates of Lung Cancer. *JOM* 12: 797-799.
- Milham S (1983): Occupational Mortality in the State of Washington, 1950-1979. NIOSH publication. No. 83-116.
- Milne K, Sandler D, Everson R, Brown S. (1983): Lung Cancer and Occupations in Alameda County: A Death Certificate Case-Control Study. *Am J Ind Med* 4: 566-575.
- Morton W, Treyve E (1982): Histologic differences in occupational risks of lung cancer incidence. *Am J Ind Med* 3: 441-457.

- Pederson T and Siak J (1981): The role of nitroaromatic compounds in the direct-acting mutagenicity of diesel particle extracts. *J. Applied Tox* 1,2: 54-60.
- Pepelko W, Peirano W (1983): Health Effects of Exposure to Diesel Engine Emissions. *J Am College Tox* 2,4: 253-306.
- Peterson G (1980): Occupational mortality in the state of California 1959-61. NIOSH publication No. 80-104.
- Raffle, P (1957) The health of the workers. *Br J Ind Med* 14: 73-80.
- Rosenkranz H (1984) Mutagenic and carcinogenic nitroarenes in diesel emissions: risk identification. *Mut Res* 140: 1-6.
- Rushton L, Alderson M, Nagarajah C (1983): Epidemiological survey of maintenance workers in London Transport Executive bus garages and Chiswick Works. *Brit J Ind Med* 40: 340-345.
- Schenker M, Speizer R (1980): A Retrospective Cohort Study of Diesel Exhaust Exposure. In: Pepelko W, Danner R, Clarke N (eds): *Railroad Workers: Study Design and Methodologic Issues*. "Health Effects of Diesel Engine Emissions: Proceedings of an International Symposium, Vol. II," EPA-600/9-80-057B, Nov 1980, pp. 114-126.
- Schenker M, Smith T, Munoz A, Woskie S, Speizer F (1984): Diesel exposure and mortality among railway workers: results of a pilot study. *Brit J Ind Med* 41: 320-327.
- Springer V, Stahman R (1977): Diesel car emissions—emphasis on particulate and sulfate, Society of Automotive Engineers Report No. 770818: pp 1-32.
- Sterling T, Weinkham J (1976): Smoking characteristics by type of employment. *JOM* 11: 743-754.
- Steenland K, Beaumont J, Halperin H (1984): Methods of control for smoking in occupational cohort mortality studies. *Scand J Work Environ Health* 10: 143-149.
- Waller RE (1985): Trends in lung cancer in London in relation to exposure to diesel fumes. In: Pepelko W, Danner R, Clarke N (eds): "Health Effects of Diesel Engine Emissions: Proceedings of an International Symposium, Vol. II," EPA-600/9-80-057B, Nov. 1980, pp 1085-1099.
- Waller R, Hampton L, Lawther P (1985): A further study of air pollution in diesel bus garages. *Brit J Ind Med* 42: 824-830.
- Walrath J, Rogot E, Murray J, Blair A (1985): Mortality patterns among US veterans by occupation and smoking status. NIH Publication No. 85-2756. Wash, DC: U.S. Govt Printing Office.
- Waxweiler R, Beaumont J, Henry J, Brown D, Robinson C, Ness G, Wagoner J, Hyg S, and Lemen R (1983): A modified life-table analysis system for cohort studies. *JOM* 2: 115-124.
- Wei E, Shu H (1983): Nitroaromatic carcinogens in diesel soot: a review of laboratory findings. *AJIH* 9: 1085-1088.
- White H, Vostal J, Kaplan H, Mackenzie W (1983): A long-term inhalation study evaluates the pulmonary effects of diesel emissions. *J Appl Tox* 6: 332.
- Williams R, Stegens N, Goldsmith J (1977): Associations of cancer site with occupation and industry from the 3rd National Cancer Survey interview. *JNCI* 4: 1147-1185.
- Wong O, Morgan R, Kheifets L, Larson S, Whorton D (1985): Mortality among members of a heavy construction equipment operators union with potential exposure to diesel exhaust emissions. *Brit J Ind Med* 42: 435-448.
- Ziskind R, Carlin T, Ballas J (1978): "Evaluating toxic gas hazards in heavy duty diesel truck cabs." Proceedings; 4th Joint Conference on Sensing Environmental Pollutants. American Chemical Society, pp 377-383.