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Lung cancer and exposure to diesel exhaust among bus garage workers

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GUSTAVSSON P, PLATO N, LIDSTRÖM E-B, HOGSTEDT C. Lung cancer and exposure to diesel exhaust among bus garage workers. *Scand J Work Environ Health* 1990;16:348—54. Mortality and cancer incidence was investigated among the 695 bus garage workers employed as mechanics, servicemen, or hostlers for at least six months in five bus garages in Stockholm between 1945 and 1970. The exposure to diesel exhaust and asbestos was estimated by industrial hygienists. A small excess of lung cancer mortality was found in the cohort when occupationally active men in Stockholm were used as the reference group. A case-referent study was performed within the cohort, six referents being selected for each of the 20 lung cancer cases. The lung cancer risk increased with increasing cumulative exposure to diesel exhaust, but not with cumulative asbestos exposure. The relative risk for lung cancer among the highly exposed men was 2.4 (95 % CI 1.3—4.5) as compared with those with low exposure. The study indicates that exposure to diesel exhaust increases the risk for lung cancer.

Key terms: asbestos, epidemiology, humans, polycyclic aromatic hydrocarbons, PAH.

Exhausts from diesel-fueled vehicles have long been considered a potential cancer hazard due to their chemical composition and mutagenic activity in bacterial test systems (1).

An increased risk for lung cancer was found among railroad workers exposed to diesel exhaust in the United States (2, 3) and Canada (4). A small excess of lung cancer was also found among persons with a history of exposure to diesel exhaust in a case-referent study from the United States (5), while another American case-referent study was negative in this respect (6). No increase in the occurrence of lung cancer was found for London transport workers (7) nor for employees at a Swedish bus company (8).

The carcinogenic activity of diesel exhaust has been supported by several recently completed animal inhalation studies (9—13). The mutagenic and carcinogenic properties of diesel exhaust are probably mainly caused by polycyclic aromatic hydrocarbons (PAH), and especially the nitroarenes seem to have a high mutagenic potency (14).

The present study was initiated to investigate the risk of lung cancer in relation to diesel exhaust and asbestos in a cohort of bus garage workers with varying degrees of exhaust exposure.

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Subjects and methods

Population

All the men who had worked as a mechanic, serviceman, or hostler for at least six months in any of five bus garages in Stockholm from 1945 to 1970 were included in the study. Altogether 708 individuals were identified from company records.

Information on name, personal identification number, workplace, job type, and beginning and end of work periods were collected from the records. Life outcome was traced via a computerized register of the living population, via death and burial books of the clerical parishes, and via Stockholm City archives. Underlying causes of death were obtained from Statistics Sweden, and cancer incidence data were obtained from the Swedish Cancer Registry. Mortality was investigated from 1952 to 1986, and cancer incidence from 1958 to 1984.

Seven cohort members were deceased before 1952, and six had emigrated. These 13 individuals were excluded from the cohort. Life outcome could be ascertained at the end of follow-up as of 31 December 1986 for all the remaining 695 individuals.

Exposure assessments

Diesel-powered buses were first introduced in Stockholm in the beginning of the 1930s. During World War II all buses were fueled with generator gas. Since 1945 all buses equipped with combustion engines have been diesel fueled. The intensity of the exposure to diesel exhaust and asbestos, specific for workplace, work task, and calendar-time period, was assessed by industrial hygienists and structured in a job-exposure matrix.

Diesel exhaust

The diesel-exhaust exposure assessments were based on (i) the emission of diesel exhaust, depending on the number of buses, engine sizes, engine running time, and fuel types, (ii) ventilatory equipment and air volume of the garages, and (iii) job types and work practices. Detailed historical data for these parameters were available, but only few exposure measurements had been performed. No single substance is accepted as a marker of diesel-exhaust exposure, although several such substances have been proposed (eg, respirable dust, elemental carbon, nitrous oxides, and PAH (15, 16)). Thus the available data permitted relative exposures to be estimated, but not absolute exposure levels.

The diesel-exhaust exposure level (exposure intensity) for every work period in the work history was classified on a ratio scale of six degrees. The exposure intensity was set at zero for the unexposed work operations and at 1.0 for the lowest exposures greater than zero. Each successive degree of the scale corresponded to a 50 % increase in intensity: 1.5, 2.25, 3.38, and 5.06.

Asbestos

Personal sampling of asbestos exposure during brake repair in bus garages in Stockholm was performed in 1987, including reconstruction of earlier exposure conditions (Plato N, unpublished data). These data, and the results of exposure investigations from other bus garages (17—20), were used to estimate the exposure to asbestos for the cohort members, and the time-weighted annual mean exposures were classified on a scale of three degrees: 0, 1 and 2. Level 1 corresponded approximately to a level of 0.08 fibers · ml^{-1} of air and level 2 to 0.16 fibers · ml^{-1}.

Cumulative exposures

We calculated indices of cumulative exposures to diesel exhaust and asbestos by multiplying the exposure level by the duration in years for every work period in the work history. The contributions from all work periods were then added for the cumulative exposures to diesel exhaust and asbestos for every individual in the cohort. The diesel-exhaust exposure index ranged from 0 to 112 (median 13), and the asbestos exposure index from 0 to 86 (median 19).

For the case-referent analysis, the diesel-exhaust exposure index was subdivided into four classes (0—10, 10—20, 20—30, and >30) representing each class by a score value ("diesel-exhaust score") of 0, 1, 2, or 3, respectively. The asbestos exposure index was subdivided into the following four classes: 0—20, 20—40, 40—60, and >60.

Analysis

Cohort and case-referent techniques were used for the analysis. In the cohort analysis the observed mortality was compared with that of a local reference population (the population of greater Stockholm). A second set of local reference rates was also used, with adjustment for occupational activity. This standardization for occupational activity was performed in order to reduce the bias from the "healthy worker effect" (21). Data on occupational activity were recorded in the population and housing census of 1960, and the mortality from 1961 to 1965 among employed and unemployed persons, specific for age group, sex, and cause of death, has been published by Statistics Sweden (22). From these data, the ratio of the mortality among the employed to that in the general Swedish population was calculated, specific for age group, sex, and cause of death. The ratios were subsequently applied to (multiplied by) the local death rates. The obtained rates reflect the local mortality with adjustment for occupational activity. The adjustment was specific for cause of death and age, but approximate with regard to geographic region and trends in the ratios with calendar time. The procedure for the calculation of the adjusted rates has been described elsewhere (23). The reference rates for cancer incidence were based on national statistics (24).

Expected numbers of deaths and cancers were computed according to the person-year method (25), counting years at risk for each individual, specific for sex, five-year age classes, and five-year calendar-time classes. Both the diesel-exhaust and asbestos exposure indices were time-dependent and were recalculated for every year during the allocation of person-years, and the contributions were referred to the appropriate stratum of the respective exposure index (25).

The "occupational mortality analysis program" (OCMAP-PC) (26) was used for the mortality analyses, and the "epidemiology in Linköping" (EPILIN) program (27) was used for the analysis of cancer incidence.

Dose-response relationships were investigated in a case-referent analysis. A full cohort analysis of internal dose-response relationships using the Cox proportional hazards model (25) was considered. However, the involvement of two time-dependent measures of dose (diesel-exhaust and asbestos exposure indices) led to complicated calculations that were not feasible with the available software. A case-referent analysis, matched with regard to age, permitted calculation and analysis of cumulative exposures without need for dynamic (iterative) recalculation of the exposure indices, as would have been required in a full cohort design. Selection of an appropriate number of controls maintains validity and causes only a small loss of precision (25). This design still permitted the use of both dead and incident lung-cancer cases as end points in the study, which was not the case in the analyses for

standardized mortality ratio (SMR) using external reference rates.

The cases and referents were selected from the cohort. All individuals with a primary form of lung cancer (eighth revision of the International Classification of Diseases, ICD 8 = 162.1, mesotheliomas not included) identified in the register of causes of deaths or in the cancer register were selected as cases. Altogether 20 cases were identified, 12 were registered in both registers, five were registered only in the register of causes of deaths, and three only in the cancer register.

Six referents were selected for every case. The referents were drawn at random and without replacement from those being noncases at the time of diagnosis of the case and born within ±2 years of the case, according to the method described by Breslow & Day (25). The cumulative exposures of the referents were truncated at the time of the diagnosis of the case.

The relative risk for lung cancer was approximated by the odds ratio, calculated by conditional logistic regression (28), with the matched sets kept together. The EGRET (epidemiologic graphics, estimation, and testing) program (29) was used, and the main results were confirmed with the epilog program (30).

Results

The overall mortality in the cohort equaled the expected when the local rates, adjusted for occupational activity, were used as the reference (table 1). There were 17 lung-cancer deaths, whereas 13.9 would be expected. Altogether, 73 cases of ischemic heart disease were found, whereas 64 were expected. Four cases of esophageal cancer were found, and 2.1 were expected. The observed numbers for other causes of death were close to those anticipated.

Analysis of the lung cancer mortality by increasing cumulative diesel-exhaust exposure indicated no definite evidence for a dose-response relationship, and neither was a dose-response trend found for ischemic heart disease (table 2). The SMR values for both diseases were lowest in the lowest dose category, however. The analysis by cumulative asbestos dose gave no indication of a dose-response for lung cancer (table 3).

The analysis of cancer incidence confirmed the mortality findings, and no additional excesses were found (table 4). Several histological types of lung cancer were represented, and also two mesotheliomas, but the numbers were too few to permit conclusions regarding excesses of individual types.

Table 1. Mortality among bus garage workers, 1952—1986. The reference rates were based on local mortality and on local mortality adjusted for occupational activity. There were 21 317.5 person-years of observation (O = observed number of deaths, E = expected number of deaths, SMR = standardized mortality ratio, 95 % CI = 95 % confidence interval).

Cause of death*	O	Stockholm, occupationally active			Stockholm, general population		
		E	SMR	95 % CI	E	SMR	95 % CI
All malignant tumors (140—209)	62	56.8	109	84—140	60.0	103	79—132
Esophageal cancer (150)	4	.	..	.	2.1	193	53—494
Stomach cancer (151)	5	..	..	..	5.1	97	32—227
Bowel cancer (152—153)	1	..	..	..	3.8	26	7—147
Rectal cancer (154)	4	..	.	..	2.3	177	48—453
Liver cancer (155)	3	.	.	..	2.0	150	31—493
Pancreatic cancer (157)	2	..		.	4.4	46	5—165
Lung cancer (162)	17	13.9	122	71—196	14.8	115	67—184
Prostatic cancer (185)	6	..	..	.	5.0	119	44—259
Bladder cancer (188)	1	..	.	..	1.9	52	1—288
Kidney cancer (189)	2		..	..	2.8	72	9—259
Brain tumors (191)	4	.	..	..	1.8	220	60—563
Hematopoietic cancer (200—209)	6	4.9	123	45—268	5.1	117	43—255
Nervous diseases (320—389)	5	..		..	2.6	190	62—442
Circulatory diseases (390—458)	96	90.4	106	86—130	104.7	92	74—112
Ischemic heart disease (410—414)	73	64.1	114	89—143	72.8	100	78—126
Cerebrovascular diseases (430—438)	10	11.7	85	41—157	14.0	72	34—132
Respiratory diseases (460—519)	10	7.0	143	68—262	11.5	87	42—160
Asthma, bronchitis, emphysema (490—493)	4	..	..	..	5.3	75	20—192
Digestive diseases (520—577)	8	13.2	61	26—120	15.6	51	22—101
Liver cirrhosis (571)	5	8.0	63	20—146	9.1	55	18—128
Genitourinary diseases (580—629)	2	2.6	75	9—274	3.2	63	8—230
Violent death and intoxication (E800—E999)	21	23.4	90	56—137	25.1	84	52—128
Suicide (E950—E959)	11	9.8	113	56—201	10.5	105	52—187
All causes (0—999)	208	204.3	102	88—117	235.9	88	76—101

* Code of the eighth revision of the International Classification of Diseases in parentheses.

Table 2. Mortality by cumulative exposure to diesel exhaust. The reference rates were based on data from occupationally active men in Stockholm. (O = observed number of deaths, E = expected number of deaths, SMR = standardized mortality ratio)

Cause of death[a]	Diesel-exhaust exposure index[b]								
	0—10			10—30			>30		
	O	E	SMR	O	E	SMR	O	E	SMR
All causes (0—999)	68	80.9	84	61	49.4	123	79	74.1	107
All malignant tumors (140—209)	19	21.4	89	16	13.5	119	27	21.9	123
Lung cancer (162)	5	5.15	97	5	3.3	152	7	5.5	127
Ischemic heart disease (410—414)	20	23.7	84	21	14.8	142	32	25.6	125

[a] Code of the eighth revision of the International Classification of Diseases in parentheses.
[b] Index of the cumulative exposure to diesel exhaust; see the Subjects and Methods section.

Table 3. Mortality by cumulative exposure to asbestos. The reference rates were based on data from occupationally active men in Stockholm. (O = observed number of deaths, E = expected number of deaths, SMR = standardized mortality ratio)

Cause of death[a]	Asbestos exposure index[b]								
	0—20			20—40			>40		
	O	E	SMR	O	E	SMR	O	E	SMR
All causes (0—999)	99	92.3	107	41	44.9	91	68	67.3	101
All malignant tumors (140—209)	25	24.3	103	12	12.4	97	25	20.1	124
Lung cancer (162)	5	5.8	86	6	3.0	197	6	5.1	118
Ischemic heart disease (410—414)	34	26.6	128	15	13.9	108	24	23.6	102

[a] Code of the eighth revision of the International Classification of Diseases in parentheses.
[b] Index of the cumulative exposure to asbestos; see the Subjects and Methods section.

Table 4. Incidence of cancer among the bus garage workers, 1958—1984. The reference rates were based on national statistics. There were 16 695 person-years of observation. (O = observed number of cases, E = expected number of cases, SMR = standardized mortality ratio, 95 % CI = 95 % confidence interval)

Cancer site[a]	O	E	SMR	95 % CI
All malignant tumors (140—209)	91	88.6	103	83—126
Esophagus (150)	4	1.2	327	89—837
Stomach (151)	6	6.7	90	33—196
Colon (153)	4	6.3	64	17—163
Rectum (154)	4	4.5	88	24—226
Liver (155)	2	2.3	88	10—318
Pancreas (157)	2	3.5	57	6—207
Nose and nasal sinuses (160)	1	0.3	376	9—2096
Larynx (161)	1	1.2	83	2—460
Lung (162)[b]	17	10.6	161	94—257
Squamous-cell carcinoma	5	4.2	118	38—275
Undifferentiated oat-cell carcinoma	7	4.4	160	64—329
Adenocarcinoma	2	1.3	152	17—547
Prostate (177)	15	15.7	96	54—158
Kidney (180)	3	4.3	70	14—204
Bladder (181)	4	6.1	66	18—168
Malignant melanoma (190)	5	2.1	237	76—554
Skin tumors, melanoma excluded (191)	2	2.6	78	9—282
Nervous system (193)	6	3.2	190	70—415
Hematopoietic organs (200—209)	6	7.5	80	29—175

[a] Code of seventh revision of the International Classification of Diseases in parentheses.
[b] Including two cases of mesothelioma and one case of alveolar-cell cancer.

Table 5. Number of living and deceased persons with lung cancer (cases) and referents from the cohort according to the cumulative diesel-exhaust exposure. (RR = relative risk, 95 % CI = 95 % confidence interval)

	Diesel-exhaust exposure index[a]				Total
	0—10	10—20	20—30	>30	
Cases	5	2	3	10	20
Referents	45	15	18	42	120
Total	50	17	21	52	140
Crude RR	1[b]	1.20	1.50	2.14	
RR from logistic regression	1[b]	1.27	1.56	2.63	-
Fitted RR from weighted regression	1[b]	1.34	1.81	2.43	-
95 % CI	—	1.09—1.64	1.20—2.71	1.32—4.47	-

[a] See the Subjects and Methods section for a definition of the index.
[b] Reference category.

The numbers of cases and referents are presented n table 5, and the details of the conditional logistic egression are presented in table 6. In model 1 the diesel-exhaust index score was introduced as factors (indicator variables 0 and 1). The relative risk for lung cancer increased with increasing exposure to diesel exhaust, but the confidence intervals were wide (figure 1).

The diesel score was alternatively introduced as an unfactored variable (regression model 2). The obtained regression coefficient of 0.32 corresponded to a relative risk of 1.37, interpreted as a 37 % increase in risk for an increase in the dose index score by one unit. The 95 % confidence interval (95 % CI) for this estimate was narrower than that of model 1, but included a relative risk of unity.

The asbestos exposure did not seem to influence the lung cancer risk (models 3 and 4).

There were indications of a dose-response relationship regarding diesel exhaust and lung cancer. (Compare models 1 and 2 in table 6.) The confidence intervals were wide, though, since the number of cases in each exposure group was small. Rothman has described a method for exploring the dose-response trend in a situation of this type (31). The regression coefficients (ie, the logarithms of the relative risks) of model 1 seemed to be nearly linearly correlated with the diesel-exhaust index score, and a weighted regression was performed with the use of the inverse of the variance of the estimates as weights. A regression coefficient of 0.2971 with a standard error of 0.0306 was obtained. This figure can be interpreted as an increase in lung cancer risk of 35 % for an increase of one level in the diesel-exhaust index score, and it corresponds closely to the relative risk of 1.37 obtained in model 2 (table 6). The fitted relative risks from the weighted regression and 95 % confidence intervals (31) are shown in table 5.

Table 6. Results of the logistic regression. (RR = relative risk, 95 % CI = 95 % confidence interval)

Parameter	β	se(β)	RR	95 % CI
Model 1				
Diesel-exhaust index[a]				
0—10	0[b]	—	1.0[b]	—
10—20	0.236	0.922	1.27	0.21—7.72
20—30	0.447	0.776	1.56	0.34—7.16
>30	0.969	0.649	2.63	0.74—9.42
Model 2				
Diesel-exhaust score[a]	0.318	0.211	1.37	0.91—2.07
Model 3				
Asbestos index[a]				
0—20	0[b]	—	1.0[b]	—
20—40	0.511	0.618	1.67	0.50—5.60
40—60	0.230	0.699	1.26	0.32—5.00
>60	0.184	0.789	1.20	0.26—5.64
Model 4				
Asbestos score[a]	0.054	0.232	1.06	0.67—1.66

Explanation of models.

Model 1:

Logit $P(x) = \alpha + \beta_1 x_1 + \beta_2 x_2 + \beta_3 x_3$, where

$x_1 = 1$ when diesel exposure = 10—20, else $x_1 = 0$
$x_2 = 1$ when diesel exposure = 20—30, else $x_2 = 0$
$x_3 = 1$ when diesel exposure >30, else $x_3 = 0$

Model 2:

Logit $P(x) = \alpha + \beta_1 x_1$, where

x_1 takes the value of the diesel score: 0, 1, 2 or 3.

Models 3 and 4 are analogous, replacing the diesel-exhaust index with the asbestos index.

[a] See the Subjects and Methods section for a definition of the index or score.
[b] Reference category

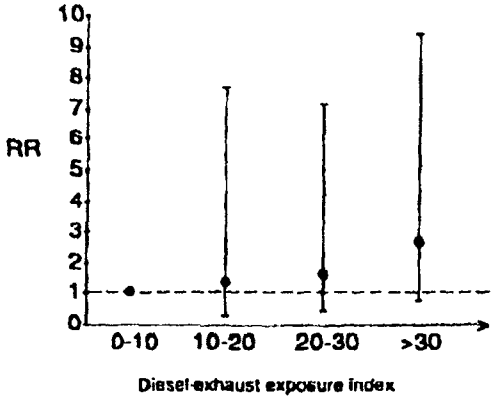


Figure 1. Estimates of the relative risk (RR) of lung cancer with logistic regression subdivided by cumulative exposure to diesel exhaust. The 95 % confidence intervals are shown.

Discussion

The overall lung cancer mortality in the cohort was slightly increased (SMR 122). In other words, an excess of the same magnitude as in other studies of workers exposed to diesel exhaust was found. The SMR for lung cancer incidence was higher (table 2). The SMR obtained in the mortality analysis was probably the more valid one since it was standardized for geographic variations in the background rate. The lung cancer rate in Stockholm is higher than the national average.

The case-referent analysis indicated a risk excess among the highly exposed, as compared with the lowly exposed, workers, and a dose-response relationship was present. The logistic regression indicated a lung cancer risk that was 2.6 times higher for highly exposed workers than for the lowly exposed workers, although with a wide confidence interval. When data from all the dose groups were combined in the weighted linear regression, a risk excess of 2.4 was found for the highly exposed workers (95 % CI 1.3—4.5). This regression model was thus more effective but also more restrictive since a linear relationship of the logarithms of the relative risks was assumed. This condition is met in the material however; the observed relative risks corresponded closely to those predicted by the regression (table 5). The crude relative risks (table 5) were somewhat lower, probably because precision was lost when the matching was dissolved.

The dose-response trend in the SMR analysis was weak. The case-referent analysis utilized both deceased and living lung cancer cases, while the cohort analysis was limited to the deceased cases. The additional incident cases were all highly exposed to diesel exhaust.

No effect from exposure to asbestos was found. The mean asbestos exposure index was 27, and the maximum was 86. These index levels correspond to a cumulative asbestos exposure of approximately 2.2 and 6.0 fibers · ml^{-1}-years, respectively. Such an exposure

would be anticipated to increase the lung cancer rate by only a few percent (32) and would not be detected in a study of this size. There were two persons with pleural mesothelioma, with cumulative asbestos exposures of 2.2 and 3.9 fibers · ml^{-1}-years, respectively. They were employed by the bus company at 27 and 30 years of age, and the tumors occurred at the ages of 63 and 64, respectively. One of them was an electrician, having worked in the electrical workshop in one of the garages. Both may have been exposed to asbestos during previous employments.

Misclassification

The exposure estimations were based on job tasks, workplace, and time periods, and the actual exposures to both diesel exhaust and asbestos have probably varied within these subgroups of the cohort. However, the exposures were classified without knowledge of the life outcome of the individuals, and thus the misclassification is independent of the outcome and may only force the relative risk towards unity (31).

The classification of the outcome (ie, the information in the registers of deaths and cancer incidence) is generally considered to be accurate (33), and since any misclassification is likely to be independent of the exposure of the individuals, it would also tend to underestimate an excess risk.

Confounding

Data on smoking habits were not possible to obtain in this retrospective study, and uncontrolled effects from tobacco smoking cannot be ruled out so far as the cohort study is concerned. In the case-referent study, however, it was not probable that the smoking habits differed substantially between the workers with high and low exposure, since both groups belonged to the same occupational category. A risk excess of 2.4 is unlikely to be explained even by extreme smoking habits (34) if it is assumed that the group with low exposure had normal smoking habits.

Risk estimation

The results of the study support the assumption that high cumulative exposure to diesel exhaust increases the risk for lung cancer. The risk excess among the highly exposed workers was greater than in other studies, possibly because of the detailed exposure assessments, which gave a good contrast between the workers with high and those with low exposure.

Total dust levels were measured during bus service and repair in one of the garages in 1981, 1982, and 1989. In 1981, personal, time-weighted 8-h samples showed a level of 1.2—1.4 mg/m^3 (Jacobsson, unpublished data). In 1982, during 4—8 h of sampling, the levels were 0.5—1.0 mg/m^3 (Bergström, unpublished data), and in 1989 they were 0.3—0.9 mg/m^3 (Axelsson, unpublished data). Total dust is also generated from sources other than the diesel engines, however, and the proportion of inorganic material in the dust was determined by etching of the samples in 1981 and 1989. The proportion varied from 50 to 62 % in 1981 and from 60 to 70 % in 1989, findings indicating that most of the total dust was not generated by the diesel engines.

Substitution of these approximate data into the job-exposure matrix and into the regression model indicates a doubled lung cancer risk after 20 years of work in bus garages with a total dust level of 0.9 mg/m^3.

The present study supports the view that diesel exhaust constitutes a cancer hazard for exposed workers. The magnitude of the risk excess can only be estimated approximately, however, due to the limited size of the study group.

Acknowledgments

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