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Exposure of Motor Vehicle Examiners to Carbon Monoxide: A Historical Prospective Mortality Study

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Exposure of Motor Vehicle Examiners to Carbon Monoxide: A Historical Prospective Mortality Study

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ABSTRACT. The National Institute for Occupational Safety and Health (NIOSH) investigated the health effects of chronic exposure to low concentrations of carbon monoxide by conducting a historical prospective cohort study of mortality patterns among 1,558 white male motor vehicle examiners who were employed in New Jersey for a minimum of 6 months between 1944 and 1973. Industrial hygiene surveys indicated that the examiners were exposed to carbon monoxide at a time-weighted average (TWA) of 10-24 ppm; the exposure level recommended by NIOSH is 35 ppm TWA. A modified life table technique was used to calculate cause-specific expected deaths for the cohort adjusted for age and calendar time periods. The expected deaths were compared to the number of observed deaths through August 1973. The cohort demonstrated a slight overall increase in cardiovascular disease deaths (124 observed vs. 118.4 expected), but a more pronounced excess was observed within the first 10 yr following employment (28 observed vs. 20.9 expected). A statistically significant excess of cancer mortality was found for motor vehicle examiners after 30 yr latency (13 observed vs. 6.9 expected, $P < .05$); this excess, however, was not confined to any particular organ site. The number of deaths due to accidents was significantly lower than expected (8 observed vs. 19.6 expected, $P < .01$).

THE NUMBER OF PERSONS potentially exposed to carbon monoxide (CO) in the work environment is greater than for any other physical or chemical agent.[1] The National Occupational Hazard Survey (NOHS) estimates the number to be as high as 975,000.[2] Although the adverse health effects of acute exposure to CO have been well documented,[3,4] a controversy still exists whether low-level chronic exposures to CO may present a potential health risk. Various studies have suggested that concentrations of CO as low as 35 ppm may present a possible health risk for individuals with pre-existing cardiovascular disease (CVD).[5-8] This degree of exposure will produce a carboxy-hemoglobin (COHb) level of approximately 5%.

Occupational sources of CO are of major concern when studying the health effects of CO exposure. In the working environment CO levels will exceed those levels normally found in the ambient air to which the general public is exposed. Since automotive exhaust has been identified as a major source of occupational CO exposure, the authors chose to study motor vehicle inspectors employed in safety lane stations in the state of New Jersey. These persons are regularly exposed to automotive exhaust containing CO, as well as to other potentially toxic components of automotive exhaust (Table 1).[9]

Previous studies of workers exposed to automotive exhaust have primarily been cross-sectional morbidity studies of currently employed workers. These studies often omitted former employees, many of whom may

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have left their jobs due to adverse health conditions acquired while employed, thus leading to an underestimation of risk. To avoid such a deficiency, a historical prospective cohort mortality study of both previously and currently employed motor vehicles examiners was undertaken to determine whether there were any significant excesses in cause-specific mortality among workers exposed to automotive exhaust.

METHODS AND MATERIALS

The New Jersey Division of Motor Vehicles was created in 1928, but formal motor vehicle inspections did not occur until December 1937. At that time, 28 inspection stations had been built and strategically placed around the state so that each of the 21 counties had at least one inspection station. Currently, 38 motor vehicle inspection stations are in operation. Most stations are enclosed, except for an opening at each end to allow vehicles to enter and exit; some stations, however, are semi-enclosed, having only a roof, and occasionally, face walls; and six stations are non-enclosed (open).

Subjects. One thousand five hundred fifty-eight white male workers who had been employed for at least 6 months as motor vehicle examiners for the state of New Jersey prior to August 31, 1973, were chosen for study. The study was limited to white males since numbers were too small in the other sex or race groups for meaningful statistical analyses.

Vital status was ascertained for more than 99% of the 1,558 workers who met the study criteria [employed at least 6 months between 1944 and August 31, 1973 (Table 3)]. The 8 individuals for whom vital status could not be determined were assumed to be alive at the end of the study, thereby contributing a maximum number of person-years of observation to the study total. [This assumption tended to slightly increase the overall expected number of deaths, thus making the findings more conservative (see RESULTS)]. Of the workers who met the study criteria, 60% (913) were employed for more than 5 yr and 21% (324) were employed for at least 20 yr. Of the remaining 321 workers, only 14% (45) were employed for less than 1 yr (but longer than 6 months). Approximately 63%

Table 1.—Composition of Automobile Exhaust[9]

Constituent	Concentration (ppm) Minimum	Maximum
Aldehydes	0	300
Carbon dioxide	50,000	150,000
Carbon monoxide	2,000	120,000
Hydrocarbons	100	20,000
Lead compounds	*	*
Nitrogen	780,000	850,000
Oxides of nitrogen	0	4,000
Oxygen	0	40,000
Sulfur dioxide	†	†
Water vapor	50,000	150,000

*Depends on lead additives.
†Depends on sulfur content of fuel.

of the cohort members began employment prior to 1957, thus being able to accumulate a substantial number of person-years at risk.

All vital status information was obtained from the Social Security Administration, Internal Revenue Service, State Bureaus of Motor Vehicles, New Jersey Division of Pensions, U. S. Postal Service, and other various follow-up sources.

Death certificates were obtained for 233 of the 237 individuals known to be deceased (98.3%), and the underlying and contributory causes of death were coded by a qualified nosologist according to the revision of the *Manual of the International Classification of Diseases, Injuries, and Causes of Death* in effect at time of death.[13] These codes were subsequently converted in accordance with the 7th revision. The 4 cases reported as deceased, but for whom no death certificates could be obtained, were coded as "deaths due to unknown causes."

A modified life table analysis based on the Cutler-Ederer Technique[14] was used to obtain person-years of observations. Person-years began for each worker after completing 6

Table 2.—Summary of Carbon Monoxide Air Concentrations* for New Jersey Motor Vehicle Inspection Stations, August 29, 1973–May 10, 1974[10]

		Cars/Lane/hr				Carbon Monoxide TWA (ppm)			
		Mid-90 Percent Range				Mid-90 Percent Range			
	No. of Days Sampled	Low	High	Average	*SD*	Low	High	Average	*SD*
Outdoor stations	16	17	50	35.0	10.4	4	21	10.0	6.4
All other stations	97	15	45	31.1	9.0	11	40	24.4	13.3

*8-hr, time-weighted average (TWA)

months of employment or after January 1944 (whichever was later in time), and ended at the date of death or at the at the study end date of August 31, 1973 (whichever came first). Cause-specific expected deaths for the study population were then obtained by applying death rates, calculated from yearly tallies of United States deaths and decennial census data, to the person-years of observation of the cohort members. The yearly deaths for the U. S. population from 1940 to 1975 were obtained from the annual *Vital Statistics of the United States*, published by the National Center for Health Statistics.[4] The two-tailed Poisson[15] distribution was used to determine significant cause-specific differences between the observed and expected number of deaths.

Measurements. Carbon monoxide measurements were made at 31 of the 38 motor vehicle inspection stations. (Since motor vehicle inspectors frequently moved from station to station and changed specific jobs, analysis was not undertaken according to station type [i.e., open, semi-open, enclosed], nor specific job category.) Area samples were taken by utilizing direct reading CO meters (Ecolyzers) adapted with hoses and placed within the inspector's breathing zone. Data were stored using continuous strip-chart recorders connected to each meter. The average CO concentration for each station was then determined by computing the arithmetic mean of the hourly averages of the strip charts. During August 29, 1973, to May 10, 1974,[10] CO was monitored for 113 days.

In addition to environmental sampling, pre-shift and post-shift carboxyhemoglobin (COHb) tests were administered to 27 volunteer motor vehicle examiners by breath

Table 3.—Vital Status of a Cohort of White Male New Jersey Motor Vehicle Examiners*

Status as of August 31, 1973	Study Cohort Members No.		Study Cohort Members Percent	
Known to be alive	1313		84.3	
Known to be deceased	237		15.2	
Death certificates obtained		233		98.3
Death certificates not obtained		4		1.7
Not known whether alive or dead	8		0.5	
Total	1558		100.0	

*All examiners were employed for at least 6 months.

Table 4.—Mortality Experience of a Cohort of White Male New Jersey Motor Vehicle Exambers*

Cause of Death	7th Revision ICDA Number†	Observed Deaths	Expected Deaths	SMR‡
Tuberculosis	(001-019)	1	3.1	32
All malignant neoplasms	(140-205)	52	47.8	109
Buccal cavity and pharynx	(140-148)	4	1.6	254
Digestive organs and peritoneum	(150-159)	9	14.4	62
Respiratory system	(160-164)	16	15.7	102
Other and unspecified sites	(190-199)	12	5.9	205§
Neoplasms of lymphatic and hematopoietic system	(200-205)	6	4.8	125
Residual	(residual)	5	5.4	93
Diseases of the nervous system	(330-334, 345)	13	16.9	94
Diseases of the circulatory system	(400-468)	124	118.4	105
Arteriosclerotic heart disease	(420)	89	96.4	92
Disease of the arteries and veins	(450-468)	13	5.9	218§
Residual	(residual)	22	16.1	136
Non-malignant diseases of the respiratory system	(470-527)	10	13.1	76
Diseases of the digestive system	(540-581)	7	10.8	65
Accidental deaths	(800-962)	8	19.6	41#
Violent deaths	(963, 970-985)	5	8.4	59
All other causes		11	19.4	57
Unknown causes	(780-793, 795, Blank)	6	2.9	207
All causes		237	260.4	91

*All examiners had been employed at least 6 months.
†International Classification of Diseases adapted for use in the United States.
‡Standard Mortality Ratio = $\frac{\text{Observed Deaths}}{\text{Expected Deaths}}$ X 100)
§$P < .05$
#$P < .01$

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Table 5.—Observed and Expected Mortality by Latency and Cumulative Exposure of a Cohort of White Male New Jersey Motor Vehicle Examiners*

Latency (yr)	Exposure (yr) 0.5 – 9 Obs/Exp	(SMR†)	10 – 19 Obs/Exp	(SMR)	20 – 29 Obs/Exp	(SMR)	⩾ 30 Obs/Exp	(SMR)	Total Obs/Exp	(SMR)
0 – 9	45/55.8	(80)							45/55.8	(80)
10 – 19	29/29.7	(98)	37/59.8	(62)					66/89.5	(73)
20 – 29	24/22.5	(106)	19/14.6	(130)	38/43.0	(88)			81/80.1	(101)
⩾ 30	9/ 7.4	(121)	6/ 4.8	(125)	26/18.6	(139)	4/ 4.1	(98)	45/34.9	(128)
TOTAL	107/115.4	(93)	62/79.2	(78)	64/61.6	(104)	4/ 4.1	(98)	237/260.4	(91)

*All examiners employed at least 6 months.

†Standardized Mortality Ratio $= (\frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100)$.

analysis to measure the rate of CO absorption. Nineteen of the examiners tested were nonsmokers and eight were smokers.

RESULTS

Carbon monoxide exposure. The six outdoor motor vehicle inspection stations had CO exposures in the mid 90% range of 4 to 21 ppm TWA, whereas the semi-open and enclosed stations had levels of 11 to 40 ppm TWA (Table 2). The mean CO exposure in the outdoor stations was 10 ppm TWA, compared to 24.4 ppm TWA at other stations. Although CO levels were generally below the National Institute for Occupational Safety and Health's (NIOSH) recommended standard of 35 ppm TWA, the levels exceeded the recommended standard of 10% of the days sampled. In addition, all stations occasionally experienced peak CO exposures above 200 ppm, the ceiling level recommended by NIOSH.[11]

Carboxyhemoglobin shifts. The average pre-shift COHb level for all 27 volunteers was 3.3% vs. an average post-shift COHb level of 4.7%. This increase in COHb levels was found to be statistically significant at the $P < .01$ alpha level using the paired t distribution. When the same analysis was used for nonsmoking individuals only, the pre-shift COHb levels averaged 2.1%, and the post-shift COHb levels averaged 3.7%. This difference was also noted to be statistically significant at the $P < .01$ alpha level. Smokers did not, however, have significant differences between pre- and post-shift work COHb levels. The average pre- and post-shift COHb levels were 6.1 and 7.0% respectively.

Mortality Data

The total expected number of deaths for the cohort under study, based upon the United States white male death rates, was 260.4 (Table 4).

A detailed analysis of cause-specific mortality showed a slight excess of deaths in the "Diseases of the Cardiovascular System" category [124 observed (obs.) vs. 118.4 expected (exp.)]. Within this category, there was a deficit of deaths due to arteriosclerotic heart disease (89 obs. vs. 96.4 exp.), but a statistically significant excess of deaths due to diseases of the arteries and veins (13 obs. vs. 5.9 exp.; $P < .05$). More specifically, when aneurysms of the heart and symphilitic aneurysms of the aorta were excluded, a statistically significant excess was observed in mortality due to aneurysms of all other sites (7 obs. vs. 2.1 exp.; $P < .05$).

A small excess in the observed number of deaths due to malignant neoplasms (52 obs. vs. 47.8 exp.) appeared to be accounted for by cancers of the buccal cavity and pharynx (4 obs. vs. 1.6 exp.), and to cancers of other and unspecified sites (12 obs. vs. 5.9 exp.; $P < .05$). Included in this latter category were four cancers of the brain, whereas only 1.7 would have been expected. All brain tumors were classified as malignant gliomas. No striking departures from expected mortality were found for cancers of the digestive, respiratory, or hematopoietic systems.

Deficits in deaths were observed for diseases of the nervous system; non-malignant respiratory and digestive system diseases; and for violent and accidental deaths, which was statistically significant (8 obs. vs. 19.6 exp.; $P < .01$), and included motor vehicle, as well as other types of accidental deaths.

A latency (time period since initial employment as a motor vehicle examiner) and cumulative exposure table was generated for the all-cause death category (Table 5). Each cell shows the observed and expected number of deaths, and the standardized mortality ratio (SMR). The interior of the Table allows an examination of the effect of one variable controlling for the other—a useful analysis since the variables are not independent. Within each exposure category, the SMR generally increases with increasing latency. The same is true for each latency category; i.e., the SMR increases with increasing length of exposure. The diagonal of the matrix, however, remains low relative to

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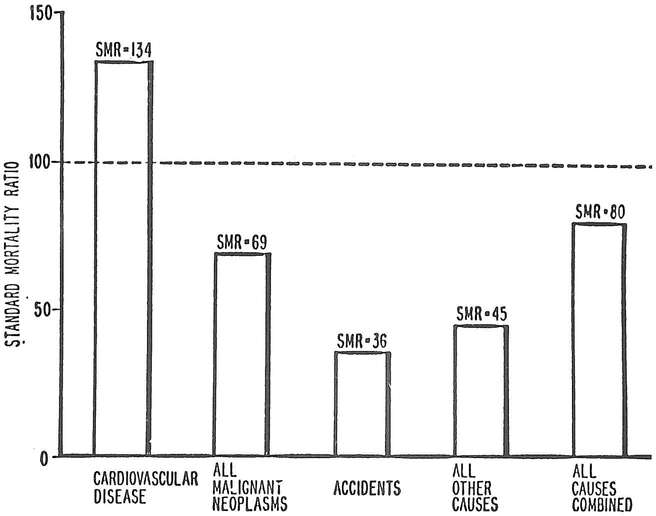

Fig. 1.—Standardized mortality ratio (SMR) for various causes of death within the first 10 yr following employment.

adjacent values, because the diagonal mainly contains person-years of workers still employed, whereas the off diagonal mainly represents person-years of terminated employees.

Within the first 10 yr following initial employment as an examiner, the low SMR (80) for all mortality hides an unusual distribution of SMRs by cause of death (Fig. 1). Cardiovascular disease deaths during this time period had an SMR of 134 (28 obs. vs. 20.9 exp.), whereas all malignant neoplasms had an SMR of 69 (6 obs. vs. 8.7 exp.), accidental deaths had an SMR of 36 (3 obs. vs. 8.4 exp.), and all other deaths combined had an SMR of 45 (8 obs. vs. 17.8 exp.). A further analysis of cardiovascular disease mortality beyond the 10-yr latency period revealed no association with duration of employment or latency (Table 6).

Although only a small increase in the number of cancer deaths was found for the entire cohort (52 obs. vs. 47.8 exp.), the SMR increased with increasing latency. Specifically, a statistically significant excess of cancer was observed for those examiners having accumulated a minimum of 30 yr latency: i.e., 13 obs. vs. 6.89 exp.; $P < .05$ (Table 7). Furthermore, all 13 of these workers had been employed for more than 10 yr. These deaths did not appear to cluster at any organ site (Table 8).

DISCUSSION

The overall deficit of mortality observed in an occupational cohort is not unanticipated. In studies of occupational disease, a deficit of mortality is often seen within the first few years following employment (latency), when compared to the mortality experience of the U.S. population. This phenomenon is referred to as the "healthy worker effect"[16] and reflects that healthy persons self-select themselves into employment. This "effect" is further enhanced by employers' use of pre-employment physical examinations or other health screening techniques. Therefore, for all combined causes of death, the SMR of 80 seen during the first 10 yr following employment was not unexpected. However, the component SMR for cardiovascular disease deaths (134) was unexpected, since the "healthy worker effect" has been most significantly associated with decreased cardiovascular disease mortality.[16-18] It would seem that if the pre-employment medical examinations given to prospective employees were effective in detecting existing cardiovascular disease, then a mortality deficit attributable to this cause would be apparent within the first 5-10 yr following such an examination.

Except for a few intermittent years, pre-employment medical examinations were conducted by the prospective employee's own physician (personal communication, Mr. J. Grandjean, Chief of Bureau of Vehicle Inspection, and Mr. Thomas Barber, Chief Personnel Officer, State of New Jersey). This may have resulted in the hiring of some

Table 6.—Observed and Expected Mortality Due to Cardiovascular Disease by Latency and Cumulative Exposure of a Cohort of New Jersey White Male Motor Vehicle Examiners*

	Exposure (yr)									
Latency (yr)	0.5 – 9 Obs/Exp	(SMR†)	10 – 19 Obs/Exp	(SMR)	20 – 29 Obs/Exp	(SMR)	⩾ 30 Obs/Exp	(SMR)	Total Obs/Exp	(SMR)
0 – 9	28/20.9	(134)							28/20.9	(134)
10 – 19	12/13.0	(92)	21/27.7	(76)					33/40.7	(81)
20 – 29	9/10.8	(83)	11/7.3	(150)	24/21.2	(113)			44/39.3	(112)
⩾ 30	4/3.6	(111)	2/2.4	(83)	11/9.3	(118)	2/2.0	(100)	19/17.3	(110)
TOTAL	53/48.3	(110)	34/37.4	(91)	35/30.5	(115)	2/2.0	(100)	124/118.2	(105)

*All examiners employed at least 6 months.

†Standardized Mortality Rate = $\left(\frac{\text{Observed Deaths}}{\text{Expected Deaths}} \times 100\right)$.

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Table 7.—Observed and Expected Mortality Due to Cancer by Latency of a Cohort of New Jersey White Male Motor Vehicle Examiners*

Latency (yr)	Observed	Expected	SMR†
0–9	6	8.66	69
10 – 19	16	16.32	98
20 – 29	17	15.89	107
⩾ 30	13	6.89	189‡
TOTAL	52	47.76	109

*All examiners were employed at least 6 months.
†Standardized Mortality Ratio = [(Observed Deaths/Expected Deaths) X 100].
‡$P < .05$.

less-than-healthy employees, some of whom may have had pre-existing cardiovascular disease. The possible combination of pre-existing cardiovascular disease with occupational CO exposure may have precipitated deaths due to cardiovascular disease in such workers soon after initial employment. This hypothesis would be consistent with findings by other researchers who have found immediate health effects from CO exposure in persons with pre-existing coronary heart disease.[5-8] The potential toxicity of the occupational CO exposure was corroborated by the fact that a number of the examiners tested had post-shift COHb levels in excess of 5%.

In studying the health effects resulting from CO exposure, the major potential confounder is cigarette smoking. Our data (see RESULTS—carboxyhemoglobin shifts) indicate that smokers start with a higher amount of CO in their lungs than do nonsmokers, but their proportional increase in COHb levels is lower than that of nonsmokers, which is consistent with Cohen et al.'s[12] study. Unfortunately, smoking history information for this cohort was not available from employment records nor was the smoking experience of all current workers assessed. Sterling and Weinham[19] analyzed cigarette smoking by type of employment and showed that white male service workers had approximately the same smoking experience as that of the general U.S. white male population. If it is assumed that the motor vehicle examiners in this study had a similar smoking experience, then it does not appear that cigarette smoking independently would have played a major role in the findings of the study. Furthermore, if smoking patterns were a major factor, an excess of lung cancer mortality among the study population would have been expected, when, in fact, a deficit was observed. However, studies by Buchwald,[20] Jones,[21] and Ayres,[22] as well as this study, have shown that environmental CO exposures and smoking independently increase COHb levels. Therefore, even if one assumes no unusual smoking patterns in this cohort, the possibility still exists that, among smokers an additive effect of CO exposure from both smoking and automotive exhaust may have contributed to the excess cardiovascular mortality observed following employment as a motor vehicle examiner.

The use of national mortality rates as a standard instead of local rates may have introduced a potential bias in the cancer results because New Jersey white male cancer rates have been consistently higher than those noted in the national statistics. Since New Jersey cancer death rates for white males were not available for all years of the study, it was not possible to generate expected deaths. Therefore, an age- and cause-specific comparison was made between

Table 8.—Cancer Mortality by Underlying Cause of Death of a Cohort of White Male New Jersey Motor Vehicle Examiners with More Than 30 yr Latency*

Case	Underlying Cause of Death	Age at Death (yr)	Total Exposure† (yr)	Latency Period (yr)
1	Adenocarcinoma of lung	73	21	30
2	Adenocarcinoma of lung	69	27	32
3	Carcinoma of lung	74	22	33
4	Carcinoma of lung	75	20	30
5	Carcinoma of prostate	84	16	30
6	Carcinoma of prostate	68	32	35
7	Carcinoma of colon	79	22	35
8	Carcinoma of liver	67	26	33
9	Carcinoma of kidney	69	29	32
10	Carcinoma of bladder	77	26	32
11	Carcinoma of esophagus	76	12	34
12	Carcinoma of bone	64	31	31
13	Carcinomatosis	70	27	33

*All examiners employed at least 6 months.
†Duration of employment.

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the cancer death rates of New Jersey and those of the United States population for the 1970 census year. The New Jersey cancer death rates averaged 5% higher than those of the United States. Yet, this 5% difference could not explain the 89% cancer deaths increase observed for motor vehicle examiners with 30 yr latency. This excess cancer risk may have resulted from the variety of toxic substances, other than CO, found in automotive exhaust. Polynuclear aromatic (PNA) and long chain aliphatic hydrocarbons have been isolated from automotive exhaust by various scientists, and at least nine are reported to be carcinogenic.[23] One such hydrocarbon, benzo(a)pyrene (BaP), is present in automotive exhaust in large amounts.[23] Tetraethyl lead (TEL) and tetramethyl lead (TML) are also components found in automotive exhaust. While most studies to date have not indicated a carcinogenic risk from exposure to lead particulates, Epstein and Mantel[24] have observed an increased incidence of lymphomas in adult female mice injected with TEL.

The statistically significant deficit of accidental deaths in this study was surprising, because previous studies of CO exposure have suggested behavioral effects, including impaired time estimation and distance discrimination at COHb levels less than 10%.[25, 26]

Perhaps the safety-oriented work of motor vehicle examiners may have prompted them to follow safety work practices, and thereby decreased their risk of fatal accidents.

SUMMARY AND CONCLUSIONS

A non-statistically significant, but highly unusual, excess of cardiovascular disease mortality was found within the first 10 yr following employment. It is hypothesized that this finding may have been at least partially due to occupational CO exposure.

Cancer deaths in the cohort were found to be higher than expected when compared to United States statistics, and were significantly increased for that group of workers achieving 30 yr latency. It is speculated that components of automotive exhaust other than CO may have contributed to this excess.

A statistically significant deficit of accidental mortality was seen in the study, a finding which is contrary to the known behavioral effects of CO. The reason for this outcome is not clear.

This study is suggestive that exposure to automotive exhaust may lead to excess mortality, but additional studies are required to substantiate this hypothesis. One such study is currently under way at NIOSH.

* * * * * * * * * *

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The Incidence and Severity of Acute Respiratory Illness in Families Exposed to Different Levels of Air Pollution, New York Metropolitan Area, 1971-1972

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ABSTRACT. The incidence and severity of acute respiratory disease was studied in families in three New York communities with different ambient levels of SO_2 and particulate air pollution. Upper, lower, and total respiratory disease rates in fathers, mothers, and school children tended to be higher in the communities with higher pollution levels. Similar higher rates, however, were not observed among preschool children. Regression analyses were used to adjust rates for socioeconomic status, parental smoking, chronic bronchitis in parents, and possible indoor pollution resulting from the use of a gas stove for cooking. After these adjustments the community differences were still significant ($P < .01$), for schoolchildren. The indoor pollution related to gas stoves was a significant covariate among children. The effects of smoking were inconsistent. It was not possible to attribute the higher rates observed to any specific pollutant, since both SO_2 and particulate matter levels were higher in the high pollution communities, nor was it possible to attribute the excesses to current levels of exposure or to a residual effect of previous higher exposure concentrations. The fact that young children did not follow the pattern suggests the latter. It was concluded, however, that current or previous exposures to the complexity of air pollutants in New York City was at least partially responsible for increased incidences of acute respiratory disease.

THE INCIDENCE AND SEVERITY of acute respiratory disease was studied in New York families exposed to different concentrations of air pollution during the 1971-1972 school year. The study concentrated on respiratory infections because their high incidence and associated disability ranked them as a major public health problem.[1] A number of studies in different parts of the world have shown an association between the incidence of acute