

Mortality Among Workers Exposed to Cutting Oil Mist: Update of Previous Reports

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Background Earlier reports of the mortality experience of this cohort of automotive workers followed from 1938 to 1967 who were exposed to cutting oil mist noted an excess of gastrointestinal cancer. The present report describes the mortality experience of these workers followed for mortality through 1980.

Methods Cause-specific standardized mortality ratios were calculated by comparing the observed number of deaths to the expected numbers based on rates for the U.S. male population.

Results The SMRs for liver and biliary tract, and testicular cancers were significantly elevated. Among the subset of workers with heavy oil mist exposure, SMRs were significantly elevated for cancers of the lung and testis, and for Hodgkin's disease. The risk of death due to lung cancer was greatest among workers with heavy exposure to oil mist employed for 15 or more years. Mortality due to stomach cancer was in excess among workers with heavy exposure to oil mist who were employed for 5 or more years. There were significant excesses of deaths due to asthma and emphysema.

Conclusions Further studies with information on the presence of contaminants and additives in oil mists will help elucidate the relationship between oil mist exposure and cancer. *Am. J. Ind. Med.* 38:410–416, 2000. Published 2000 Wiley-Liss, Inc.†

KEY WORDS: cohort study; cutting oil mist; automotive workers; lung cancer; liver and biliary tract cancer; Hodgkin's disease; testicular cancer; asthma; emphysema

INTRODUCTION

Decouffé [1976, 1978] found increased mortality due to cancers of the stomach and large intestine combined among white male automotive workers who were exposed to cutting oil mists for at least 5 years prior to 1938, and followed for at least 20 years through 1967. This study could not account for exposure past 1968 since exposure records

ended at that time. Cruickshank and Squire [1950] described skin lesions including scrotal cancer among workers exposed to mineral oil. Further case reports and epidemiologic studies established the association between lubricating oil exposure and skin cancer, particularly of the scrotum [IARC, 1984; Järholm and Easton, 1990]. Stomach, pancreas, and bladder cancer excesses have been observed in other studies of workers exposed to mineral oil [Tolbert, 1997]. In addition, Eisen et al. [1992] found an association between asthma and exposure to cutting oils. There have also been clinical reports of occupational asthma [Robertson et al., 1988] and respiratory symptoms [Järholm et al., 1982] in workers exposed to oil mist. According to NIOSH, exposure to metalworking fluids aerosols has been associated with excess risk for nonmalignant respiratory disease and skin diseases [NIOSH, 1998].

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In 1974, a survey of the study facility was carried out to determine oil mist concentrations in the breathing zone of workers engaged in machining and other associated operations. The cutting oils used were (a) straight mineral oils, (b) proprietary compounded oils consisting of mineral oils, synthetic wetting agents and emulsifiers, bactericides and rust inhibitors, diluted in water to 2–4% original material, and (c) synthetic coolants, containing substitute materials such as special soaps, alkanolamides and glycols in place of mineral oils [Decouffé, 1978]. For the industry in general, straight mineral oils were most frequently used for machining operations in the early decades, but their use began to decline in the 1940s. Straight mineral oils still account for approximately 45% of metal cutting fluids, soluble oils account for another 45%, and synthetic fluids account for the remaining 10% [Rothman and Emmett, 1988].

Mineral oils are mutagenic [Pasquini et al., 1985] or have clastogenic properties [Stram et al., 1985] in humans. Concerns have also been raised about the formation of nitrosamines and polycyclic aromatic hydrocarbons (PAH) in soluble and synthetic oils [Järholm et al., 1991; Keefer et al., 1990], as these compounds have been associated with a number of cancers in humans [Mirvish, 1995; Boffetta et al., 1997]. In addition, ethanalamines may be used as additives in cutting oil fluid to balance Ph or inhibit corrosion [NIOSH, 1998]. It has been reported that triethanolamine may cause occupational asthma and may be an animal carcinogen [Savonius et al., 1994]. The National Toxicology Program (NTP) has reported excess liver cancer incidence in a 2-year chronic dermal study of diethanolamine in male mice [1994].

To expand on the findings of Decouffé (1976, 1978), the follow-up of this cohort was extended for an additional 13 years through 1980. The present study includes short-term workers and both whites and nonwhites.

MATERIALS AND METHODS

From employment records of a single U.S. automobile plant, 23,698 men who had worked for at least 1 year in manufacturing jobs between January 1, 1938 and December 31, 1967 were identified. Subjects were traced for vital status through December 31, 1980, using records of the Social Security Administration, the motor vehicle administration in the state where the plant was located, and credit bureaus. Death certificates were obtained from state vital records offices for deceased individuals. Men whose vital status was reported unknown by all of the tracing sources and those presumed deceased but for whom a death certificate was not received were treated as lost to follow-up (7.9%). Race was determined for all but 28 men (0.1%). Underlying cause of death for deceased subjects was coded by a qualified nosologist according to the rules in effect at

the time of death, using 7th Revision ICD [World Health Organization, 1957] codes.

The present report was limited to the 11,838 men who had worked at least 1 month in Building B and were exposed to cutting oil mist. Person-years at risk for each study subject were accumulated beginning with the later of the date the subject attained 1 year of employment in Building B, or January 1, 1938; and ending when he died, was lost to follow-up, or the study period ended (December 31, 1980). In the analysis, person-years for those lost to follow-up were counted from the date of entry into the study until the day the individual was last known to be alive. Cause-specific expected numbers of death were calculated by applying age–race–calendar year-specific death rates for U.S. men to the corresponding person-years at risk in the study cohort. Standardized Mortality Ratios (SMRs) were calculated as the ratio of observed deaths to the total expected for each cause. Confidence intervals for the SMR were calculated, assuming an underlying Poisson distribution [Liddell, 1984]. Analyses were performed for all races combined including the individuals for whom race was unknown, and for whites alone. The number of nonwhite cohort members, 7.8% of subjects (3% of the person-years), was too small for separate analyses.

Jobs with exposure to oil mists were located primarily in the plant's metal machining shop (Building B) where boring, broaching, lapping, grinding, drilling, milling and cutting operations took place. Mineral oils were used for cooling and lubricating equipment and parts during the performance of these tasks. Levels of exposure were based on the likelihood of contact with oil mist as determined by company personnel. Jobs that directly involved metal machining tasks were classified as having "heavy" exposure and all other jobs in Building B were assigned "moderate" or "low" exposure. Complete work histories of subjects through 1967 were abstracted from plant personnel records, and each subject was categorized into an oil mist exposure category according to his job with the highest classification; however, all jobs involving oil mist exposure were considered in calculating total years exposed and for determining the date of first exposure.

RESULTS

As presented in Table I, 35.4% of the exposed cohort were deceased. The majority of the cohort were white (92%). Sixty-four percent of the cohort were classified as having had heavy oil mist exposure. Seventy-one percent of the subjects who entered the study were between the ages of 20 and 39. Follow-up of the 11,838 subjects resulted in a total of 327,357 person years, averaging 27.7 person years per subject.

The number of deaths from all causes among men exposed to cutting oil mist in Building B was as expected

TABLE I. Characteristics of Building B Male Automotive Workers Exposed to Cutting Oil Mist, 1938–1980

	Number	Percent
Study Subjects	11,838	100.0
Alive	6,707	56.7
Deceased	4,185	35.4
Lost-to-follow-up	945	8.0
Ethnicity		
White	10,908	92.2
Nonwhite	923	7.8
Unknown	6.0	0.1
Level of Exposure		
Low	2,671	22.6
Moderate	1,583	13.4
Heavy	7,583	64.1
Age at Entry		
< 20	936	7.9
20–39	8,428	71.2
40+	2,473	20.9

(Table II). There were significant excesses of deaths due to asthma, pneumonia, emphysema, and cirrhosis of the liver among subjects ever exposed to oil mist. Among subjects who had heavy exposure to oil mist, there were significant excesses of deaths due to asthma and emphysema.

Site-specific cancer mortality among the entire study cohort and among those with heavy exposure is shown in

Table III. Among all exposed workers, mortality from all cancers of the digestive tract combined was nearly the same as expected; however, the SMR for cancers of the biliary tract and liver was significantly elevated. Mortality due to testicular cancer was also in excess.

Among men who had heavy exposure, SMRs were significantly elevated for cancers of the biliary tract and liver, lung, testis, and for Hodgkin’s disease. Whereas, men with low/moderate exposure to cutting oil mist had no significant excess of cancer.

Analyses by duration of exposure among men who had heavy exposure to cutting oil mist are shown in Table IV. Excesses of stomach cancer were observed for workers employed for greater than 5 years in jobs with oil mist exposure (SMR = 1.42, 95% CI = 0.87–2.19), but the results were not statistically significant. Workers exposed for 5 or more years experienced a significant excess of lung cancer deaths (SMR = 1.33, 95% CI = 1.04–1.68). There was no evidence that duration of employment was associated with increased mortality from any other cancers among these workers.

Workers with heavy exposure to oil mists for less than 5 years had a fivefold increase in the risk of asthma 5–14 years after initial exposure (SMR = 5.17, 95% CI = 1.89–11.26), and a threefold increase in risk 15–24 years after initial exposure (SMR = 3.42, 95% CI = 1.10–7.99). No excess was observed 25 or more years after initial exposure. Excess mortality due to emphysema was seen among men with less than 5 years of exposure 25 years after initial exposure (SMR = 1.94, 95% CI = 1.30–2.78).

TABLE II. Standardized Mortality Ratios (SMR) for Selected Non-cancer Causes of Death Among Male Automotive Workers Exposed to Oil Mists, 1938–1980

Underlying cause of death (7th Revision, ICD)	Ever exposed			Ever heavy exposure		
	O	SMR	95% CI	O	SMR	95% CI
All Causes (001–998)	4185	0.96	0.93–0.99	2695	0.97	0.93–1.01
Infectious & Parasitic Disease (001–138)	53	0.60	0.45–0.79	34	0.60	0.41–0.84
Allergic, Endocrine, Metabolic, Nutritional Disease (240–289)	84	1.11	0.89–1.38	58	1.20	0.91–1.56
Asthma (241)	18	1.71	1.01–2.71	14	2.09	1.10–3.39
Circulatory disease (400–468)	1911	0.96	0.92–1.01	1226	0.97	0.92–1.03
Arteriosclerotic heart disease (420)	1446	0.94	0.89–0.99	925	0.95	0.89–1.01
Respiratory disease (470–527)	247	1.00	0.88–1.13	161	1.01	0.88–1.20
All pneumonia (490–493)	120	1.21	1.00–1.45	79	1.25	0.99–1.55
Emphysema (527)	85	1.43	1.14–1.77	55	1.47	1.11–1.91
Digestive disease (530–587)	223	1.06	0.92–1.20	139	1.03	0.86–1.21
Cirrhosis (581)	127	1.28	1.06–1.52	75	1.18	0.92–1.47
Genitourinary disease (590–639)	49	0.64	0.48–0.85	37	0.76	0.54–1.05
Diseases of the skin and cellular tissue (690–716)	5	1.61	0.52–3.76	5	2.52	0.81–5.88
All external causes of death (800–998)	357	0.93	0.84–1.03	233	0.93	0.82–1.061
All accidents (800–949)	227	0.85	0.74–0.96	145	0.82	0.70–0.97

O, observed; CI, confidence interval.

TABLE III. Standardized Mortality Ratios (SMR) for Selected Causes of Cancer Death Among Male Automotive Workers Exposed to Oil Mists, 1938–1980

Underlying cause of death (7th Revision, ICD)	Exposed to oil mist			Low/moderate exposure			Heavy exposure		
	O	SMR	95% CI	O	SMR	95% CI	O	SMR	95% CI
Cancer (140–205)	855	1.02	0.96–1.10	304	1.00	0.89–1.12	551	1.04	0.96–1.13
Oropharyngeal (140–148)	21	0.77	0.48–1.18	7	0.71	0.28–1.45	14	0.81	0.44–1.36
All digestive (150–159)	262	1.05	0.93–1.19	105	1.15	0.94–1.39	157	0.99	0.84–1.16
Esophagus (150)	22	1.10	0.69–1.66	9	1.23	0.56–2.33	13	1.02	0.54–1.75
Stomach (151)	53	1.01	0.76–1.32	17	0.89	0.52–1.42	36	1.08	0.76–1.50
Large intestine (153)	74	0.96	0.75–1.21	34	1.21	0.84–1.69	40	0.82	0.58–1.11
Rectum (154)	26	0.90	0.59–1.31	13	1.23	0.65–2.10	13	0.71	0.38–1.21
Biliary Tract & Liver (155,156)	30	1.60	1.08–2.28	10	1.45	0.70–2.67	20	1.68	1.02–2.59
Pancreas (157)	49	1.07	0.79–1.42	20	1.20	0.73–1.85	29	1.00	0.67–1.44
Cancer of Respiratory System	289	1.08	0.96–1.21	89	0.91	0.73–1.11	200	1.18	1.02–1.35
Larynx (161)	6	0.47	0.17–1.02	1	0.21	0–1.18	5	0.61	0.20–1.43
Lung (162–163)	283	1.12	0.99–1.26	88	0.95	0.76–1.17	195	1.22	1.05–1.40
Prostate (177)	56	0.96	0.72–1.24	22	1.02	0.64–1.55	34	0.92	0.64–1.29
Testis (178,179)	10	2.10	1.01–3.87	2	1.22	0.14–4.41	8	2.57	1.11–5.06
Kidney (180)	18	0.87	0.52–1.38	7	0.93	0.37–1.92	11	0.84	0.42–1.50
Bladder (181)	26	0.98	0.64–1.44	9	0.93	0.42–1.76	17	1.02	0.59–1.63
Brain (193)	25	1.05	0.68–1.56	9	1.06	0.48–2.01	16	1.05	0.60–1.71
Lymphatic & hematopoietic (200–205)	93	1.18	0.95–1.45	33	1.16	0.80–1.63	60	1.19	0.91–1.54
Kidney (180)	18	0.87	0.52–1.38	7	0.93	0.37–1.92	11	0.84	0.42–1.50
Non-Hodgkin's lymphoma (200,202,203,205)	41	1.18	0.85–1.60	14	1.11	0.61–1.86	27	1.21	0.80–1.77
Hodgkin's disease (201)	16	1.59	0.91–2.58	2	0.57	0.06–2.04	14	2.14	1.17–3.60
Multiple myeloma (203)	12	1.20	0.62–2.10	6	1.64	0.60–3.57	6	0.95	0.35–2.06
Leukemia (204)	36	1.09	0.76–1.51	17	1.42	0.83–2.28	19	0.90	0.54–1.41

O, observed; CI, confidence interval.

TABLE IV. Standardized Mortality Ratios (SMR) for Selected Causes by Duration of Total Oil Exposure, Among Male Automotive Workers with Heavy Oil Mist Exposure, 1938–1980

Underlying cause of death (7th Revision, ICD)	< 5 Years PY = 162084			5+ Years PY = 57999		
	O	E	SMR	O	E	SMR
Cancer of:						
Stomach (151)	16	19.57	0.82	20	14.08	1.42
Large Intestine (153)	19	30.31	0.63	21	18.79	1.12
Liver [all] (155,156)	16	7.16	2.23 ^a	4	4.85	0.82
Lung (162,163)	123	106.40	1.16	72	54.03	1.33 ^a
Testis (178,179)	6	2.32	2.59	2	0.89	2.25
Hodgkin's disease (201)	11	4.67	2.36 ^a	3	2.00	1.50
Asthma (241)	11	3.99	2.76 ^a	3	2.77	1.08
Emphysema (527)	40	22.78	1.76 ^a	15	14.64	1.02
Cirrhosis (581)	55	44.92	1.22	20	19.40	1.03

^a*P* < 0.05.

O, observed; E, expected; PY, total person-years.

DISCUSSION

This study of 11,838 workers included over 327,357 person-years of follow-up. Among all subjects, asthma, pneumonia, emphysema, cirrhosis of the liver, and cancers of the biliary tract and liver, and testicular cancer were in excess. For heavily exposed workers, elevations were also noted for asthma, emphysema, biliary tract and liver cancer, testicular cancer, lung cancer, and Hodgkin's disease. A nonsignificant excess of stomach cancer was found for workers with longer duration of exposure.

In the first follow-up of this cohort, excess mortality due to stomach cancer was found among white men employed for 5 or more years in jobs involving heavy exposure to oil mists [Decoufflé, 1978] and a number of other studies suggested that workers exposed to cutting oils may be at increased risk for this disease [Mallin et al., 1986; Park et al., 1988, 1994, 1996; Silverstein et al., 1988; Rotimi et al., 1993]. Some studies suggested that grinding operations, which can entail exposure to either soluble oil-based or ethanalamine-based fluids, may increase the risk of stomach cancer. This association may be due to possible contaminants such as polycyclic aromatic hydrocarbons (PAH) which are formed in higher concentrations in fluids used for grinding as a result of pyrolysis in these operations [Tolbert, 1997]. A number of previous studies did not find an association between mineral oil exposure and stomach cancer [Siemiatycki et al., 1987; Delzell et al., 1993; Chow et al., 1994; Leon et al., 1994]. The only digestive cancer deaths in excess in the present study was due to biliary tract

and liver cancer. In a study of engine plant workers at another facility, an excess of liver cancer deaths was observed for workers with 20 or more years of employment [Vena et al., 1985].

Our results suggested a relationship between lung cancer and heavy exposure to oil mists. Mortality from respiratory cancer was not elevated in the previous follow-up of this cohort [Decoufflé, 1976, 1978] and findings from other studies have been inconsistent [Coggon et al., 1984; Siemiatycki et al., 1987; Eisen et al., 1992; Tolbert et al., 1992; Acquavella et al., 1993; Delzell et al., 1993; Rotimi et al., 1993; Park et al., 1994]. In a recent study by Schroeder et al. [1997], exposure to machining fluids was not associated with an increased risk of lung cancer mortality in automotive workers. On the other hand, results from several case–controls studies have indicated a statistically significant association between lung cancer mortality and metal working. In a number of studies, lung cancer has been associated with occupational and environmental exposure to PAH [Boffetta et al., 1997]. PAH are produced from cutting oils during heating [Rothman and Emmett, 1988] and may have contributed to excess lung cancer risk in our study, but specific measures of PAH were not made in this investigation. The results for Hodgkin's disease and testicular cancer are new findings. These results were based on small numbers and although statistically significant, may be due to chance. Some cutting oils, additives, and contaminants have mutagenic [Pasquini et al., 1985] and carcinogenic properties [NTP, 1994; Savonius et al., 1994; Mirvish, 1995; Boffetta et al., 1997]; however, we lack

specific information in this study on exposure to these compounds.

Our study suggested that workers exposed to cutting oil mist, and particularly those with heavy exposure, have an increased risk of mortality due to asthma and emphysema. This finding is consistent with previous studies [Järholm et al., 1982; Robertson et al., 1988; Eisen et al., 1992]. However, mortality may not be a good indicator of occupationally related asthma because few asthmatics die of this condition. Also, for a disease such as asthma, the healthy worker effect is a concern; workers who develop occupationally related asthma are likely to transfer out of jobs involving such exposures. Eisen et al. [1996] reported a significantly elevated incidence of asthma related to exposure to metal working fluids when exposure was assessed at the time of the onset of asthma. A number of studies have shown adverse effects of oil mist on the respiratory system. Massin et al. [1996] showed that workers exposed to soluble oil mist in the metal industry have significantly more airway hyper-responsiveness and chronic respiratory symptoms. Järholm et al. [1982] found a greater frequency of respiratory symptoms in oil mist-exposed workers compared to office workers. Additionally, based upon serial peak expiratory flow recordings, Robertson et al. [1988] determined that more than half of a group of 25 oil mist-exposed workers had work-related asthma. It should be noted that in our study, under-ascertainment of asthma and emphysema could have occurred since we did not have information available on contributory causes of death. Further study of morbidity due to respiratory disease is needed to confirm the associations reported here.

This is one of the largest cohorts of workers exposed to oil mists that has been studied. The cohort was followed for more than 40 years and was adequate for studying mortality from cancers with a long latency period. Certain limitations must be taken into account when interpreting our findings. Information on possible confounders such as smoking, alcohol consumption, previous occupational exposures, and diet was not available. We also did not have information regarding exposure levels on the type of cutting oil, additives or contaminants in the oil used in the plant. In addition, mortality may not be a good indicator of the extent of occupationally related disease, particularly for non-cancer outcomes.

In summary, we found significant excesses of lung cancer among long-term workers exposed to oil mist, but tobacco use could not be taken into account; excesses of biliary tract and liver cancer and non significant excesses of stomach cancer were also found. Studies with more direct exposure measurements and more specific information regarding the composition and contaminants of oils used are necessary to clarify health risks associated with exposure to oil mist.

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