MORTALITY AMONG WORKERS EXPOSED TO CUTTING FLUIDS AND ABRASIVES

BEARING PLANT I

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A survey of mortality among United Automobile Union workers exposed to cutting fluids and abrasives was conducted. The cohort consisted of all hourly employees with 10 or more years of service who died from January 1, 1969 to July 31, 1982. A total of 702 death certificates and job histories of 768 employees were reviewed. Data was obtained from company and union records. Standardized proportional mortality ratios (PMR) were computed. Significant excesses of mortality from rectal cancer (PMR at 3.04), stomach cancer (PMR at 1.98), and stroke (PMR at 1.35) were found. A 25 percent elevation in mortality for lung cancer (PMR at 1.25) occurred among white males. Among females employed as grinders, the PMR for lung cancer was 2.8. A statistically significant correlation between stomach cancer and grinding exposure occurred. The grinding exposures consisted primarily of aerosols from soluble oil and oil based metal working fluids. The authors note that the pattern of stomach cancer suggests a correlation with water based cutting fluids. Possible correlations between lung cancer in males with forge and heat treatment exposures and females with grinding exposure are suggested. Further investigations are recommended to confirm these correlations.

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PREFACE

This is the second in a series of six mortality reports conducted under the terms of NIOSH contract #210-81-5104.

This study would not have been done without the initial concern and continued support of UAW Local Union 133 and UAW International Representative Richard Cardinal.

Preliminary work upon which this investigation rests was accomplished by David Wegman, MD and John Peters, MD with the assistance of Jung-der Wang, MD all of whom, at that time, were affiliated with the Harvard School of Public Health.

We appreciate the cooperation of the Bearing Company in providing some of the records used in this investigation and for reviewing a draft of this report.

We are particularly thankful for the indispensible and considerable clerical and secretarial efforts contributed by Carole Rogers and Margaret Auch.

MORTALITY AMONG WORKERS EXPOSED TO CUTTING FLUIDS AND ABRASIVES

BEARING PLANT I

NIOSH Contract #210-81-5104
International Union, United
Automobile Workers (UAW)

SUMMARY

Large numbers of workers are employed in plants where metal machining and grinding are major activities. Recent studies have identified digestive cancer excesses among workers in such plants. The chemical environment in these metal working facilities is complex. Potential exposures include cutting fluids, abrasive dusts, and oil smoke. Cutting fluids have evolved over time from acid-refined mineral oils to water-based emulsions and synthetics containing multiple additives. The suspect agents have included nitrosamines, polyaromatic hydrocarbons, and abrasive dusts.

Standardized proportional mortality analyses and case-control studies were carried out by the International Union, United Automobile Workers for a population exposed to cutting fluids and abrasives in the manufacture of ball bearings. Causes of death and job histories were obtained for 702 of 768 hourly employees with 10 or more years' service who died between 1/1/69 and 6/31/82. Work histories based on Union and Company records were used to define exposure measures.

The major findings were statistically significant excesses in proportional mortality from stomach cancer (PMR=1.98), rectal cancer (PMR=3.04), and stroke (PMR=1.35) among white men. There was a statistically significant association between stomach cancer and grinding exposures after control for age and country of birth. The grinding exposures had consisted primarily of aerosols from soluble oil metal working fluids (i.e. water emulsions), but some straight oils and synthetic cutting fluids were used as well. The pattern of stomach cancer suggests an association with the water-based cutting fluids.

Several other possible associations were noted which require additional confirmation: lung cancer among men with forge and heat treat jobs; lung cancer among women with grinding exposures; and an ill-defined group of disorders classified, probably incorrectly, as chronic alcoholism among grinders.

MORTALITY AMONG WORKERS EXPOSED TO CUTTING FLUIDS AND ABRASIVES

BEARING PLANT I

BACKGROUND

The finished production of manufactured goods from iron, steel and various specialty metals frequently requires lubricated machining and high speed abrasive grinding. Large numbers of workers in these metal working operations share potential exposure to metal working fluids and synthetic abrasives. The first NIOSH National Occupational Hazard Survey estimated that 1,229,000 workers are exposed to cutting fluids; 6,109,000 to any type of mineral oil; 2,137,000 to abrasives; and 1,221,000 to grinding wheel dust.[1] A considerable body of scientific literature has pointed to the possibility of digestive and/or respiratory cancer risk associated with exposure to these materials alone or in combination. The United Automobile Workers International Union (UAW) represents many thousands of workers in these metal working environments and has been concerned about the long-term health consequences of this work.

The Bearing Company has produced ball bearings in New Britain, Connecticut since 1911. The major metal working processes include high speed abrasive grinding with coolants, lubricated machining and hot forging. UAW Local Union 133 has represented most of the approximately 20,000 hourly employees who have worked at this plant since the 1930's.

In 1979 members of the Local Union bargaining committee became aware of an apparently unusual number of lung cancer deaths among their immediate friends and acquaintances in the plant. This was raised with management during collective bargaining and led to an agreement that the Company provide mortality information to investigators chosen by the Union for the purpose of an epidemiologic investigation. As a result, more than 600 death certificates were provided to Drs. David Wegman and John Peters of the Harvard School of Public Health who conducted a preliminary proportional mortality analysis. This analysis revealed substantial excesses of stomach and rectal cancer among white men, greatest among those with the longest duration of employment. Less pronounced excesses were also noted for respiratory cancer, malignant and non-malignant skin disease, and vascular disease of the central nervous system.

Based on concern with the potential carcinogenic effects of cutting fluids and other typical bearing plant exposures, and given the specific preliminary findings in this plant, the UAW decided to undertake a more comprehensive investigation. With the active participation of the local union and continuing Company cooperation, an expanded study population was defined, detailed information was developed on chemical use in the plant, and individual work histories were obtained.

LITERATURE REVIEW

Nature of Machining and Grinding Exposures

Metal products which have been roughly shaped by casting, stamping, and forging processes often require further removal and smoothing of metal to attain precise size and shape specifications. "Machining" refers to processes such as milling, turning and boring in which metal is sliced or cut from a workpiece by the application of a sharp, hard-tipped tool at relatively low speeds and temperatures (less than 1000°F)[2] "Grinding" removes fine particles of metal with abrasive wheels applied to metal surfaces at relatively high speeds producing temperatures as high as 2000°F. Both machining and grinding generally require the use of cutting fluids directed in a stream or mist to the working surface for lubrication, cooling and the removal of metal debris.

Metal working cutting fluids belong to three classes:

1. Cutting oils (also called insoluble or straight oils): These may be naphthenic or paraffinic mineral oils with added polar lubricants of animal and vegetable origin such as lard or sperm oil. Chlorine, sulfur or phosphorus based additives are frequently used to improve lubricating properties.

2. Soluble oils (also called water miscible or emulsified oils): These fluids contain emulsifying agents such as petroleum sulfonates or amine soaps to suspend oil droplets in water. Chlorine, sulfur and phosphorus based additives are often used to improve lubrication. Other additives include corrosion inhibitors (e.g. nitrites, polar organic compounds), chemical stabilizers (e.g. complex alcohols and non-ionic wetting agents) and biocides (e.g. triazines).

3. Synthetic fluids (also called chemical fluids): These are generally alkaline water solutions such as ethanolamines with added corrosion inhibitors (e.g. nitrates, nitrites, borates, phosphates), surfactants (e.g. complex alcohols and esters), non-mineral oil lubricants (e.g. silicones), and biocides (e.g. triazines).

Straight cutting oils have been used since the early days of the industrial revolution, whereas soluble oils and synthetic fluids have been used widely only since the 1940s. Straight oils are still used today in many machining and some grinding applications because of their lubricating qualities while soluble oils and synthetics are particularly useful for grinding because of their cooling properties. There is, however, considerable overlap in usage.

The use of cutting fluids invariably produces an aerosol both mechanically and by vaporization and condensation. Straight oils tend to produce larger and heavier droplets than the finer mists associated with synthetic fluids.[2] The present OSHA exposure limit for oil mist is 5 mg/m^3[3]. This standard was based on animal test data for exposure to straight mineral oils and was aimed

primarily at the prevention of lipoid pneumonia from gross exposures.[4] There is no specific standard for synthetic fluid mist.

Oil mist measurements in metal working plants in the past several years have typically been in the 0.5-5 mg/m^3 range. A NIOSH evaluation in an automotive transmission plant using soluble oils reported personal sampling exposures from 0.07 to 1.4 mg/m^3.[5]

Jarvholm reported area samples from a bearing plant similar to the one in this study.[6] Oil mist levels ranged from 0.3-2.3 mg/m^3 in the turning department (machining), 1.0-7.3 mg/m^3 in grinding, and 0.4-18.0 mg/m^3 in hardening (heat treat) with mean levels of 2.0, 3.2, and 2.9 respectively. Decoufle reported personal samples ranging from 0.4 to 1.67 mg/m^3 of mist in an engine plant using predominantly straight mineral and soluble fluids.[7] In this latter study the geometric mean droplet size of mineral oil mist in various samples ranged from 2.4 to 5.6 µm. Droplet sizes for aerosols of the soluble and synthetic fluids were not reported but were presumably smaller.

There are 4 specific groups of toxicologically suspect chemicals associated with these mists in metal working operations:

1. Polycyclic aromatic hydrocarbons, including benz(a)pyrene, have been measured in both new and used bulk samples of straight oils.[8] Solvent refining of these oils in recent years is thought to have reduced the level of these contaminants below that historically found in acid refined oils.[8,9] Recent measurements on bulk samples have found 0.5 to 150 pg/ml of benz(a)pyrene in new oils and higher levels in used oils.[10]

2. N-nitroso compounds including nitrosamines such as N-nitrosodiethanolamine have been detected in bulk samples of synthetic metal working fluids. These presumably result from the combination of ethanolamines and nitrite additives in solution. Fan has measured concentrations of N-nitrosodiethanolamine as high as 3% in new, bulk fluid concentrates.[11]

   A recent NIOSH survey found considerably lower levels (0.2 to 600 µg/ml) in a variety of new and used fluids.[12] Nitrosamines in fluids diluted for use were generally at the lower end of this scale. Only trace amounts were found in air samples, however the testing procedures were designed to measure nitrosamine vapors and not mist-borne nitrosamines. N-Nitrosodiethanolamine is known to be absorbed through human skin[13] and has been measured in the urine of metal grinders.[14]

   In 1976, NIOSH issued a "Current Intelligence Bulletin" to alert cutting fluid manufacturers, users and workers about the potential hazards of these exposures.[15] In January, 1984 the U.S. Environmental Protection Agency published a proposed rule which would prohibit the addition of nitrosating agents to the triethanolamine salt of tricarboxylic acid when used in metal working fluids.[16]

3. Biocides are typically added to all varieties of cutting fluids to reduce microbiologic growth. Those commonly used in water-based fluids have recently been reviewed and include a wide variety of organic compounds.(17) The most common are cyclic triazines which are condensates of formaldehyde with substituted alkylamines or ammonia. Although it is presumed that these act by releasing formaldehyde at microbial cell membranes and that little free formaldehyde is released under normal conditions in alkaline cutting fluids, very little effort has been made to examine this under field conditions.

4. Abrasives: Grinding wheels have been made predominantly from silicon carbide or aluminum oxide since the 1920s when these synthetic abrasives were introduced to reduce the silicosis hazard associated with sandstone. Several types of bonding agents including shellacs, rubbers and synthetic organic resins are used to hold the abrasive particles together. There is little information available about the chemical composition or size of airborne particles which are generated during dry or wet grinding or during "dressing" with diamond cutting devices.

There are other chemicals of toxicologic concern which have been found as components or contaminants of cutting fluids but which have received little systematic study. These include polychlorinated biphenyls which may be present when lubricating oils or hydraulic fluids contaminate cutting fluid systems and para-tert-butyl benzoic acid, a reproductive toxin in animal tests which has been used as an additive to soluble fluids.(18)

In addition to the machining and grinding operations, there are other metal working processes in bearing plants and similar facilities which may contribute hazardous exposures to the working environment. Smokes, oil mists and carbon monoxide may be generated in forging and heat treating operations when lubricating oils and cutting fluids are heated or burned. In forging, hot metal parts may be sprayed with heavy oil lubricants prior to pressing; in heat treating, red-hot parts may be quenched in heavy oil baths. Other potential exposures include chromic acid mists from electroplating operations and metal dusts such as beryllium or cadmium from grinding on specialty metals. Tool and die work in preparing the hard metal cutting tools includes grinding tungsten carbide and related special alloys. Cobalt and other exposures can result.

Health Effects

Animal Studies

Animal studies have demonstrated the carcinogenicity of several varieties of new and used cutting fluids as well as some of their specific components and contaminants.

A soluble and a straight cutting oil from a metal working plant where skin cancers among workers were reported [19] were both found to cause skin cancer when applied repeatedly to the skin of mice.[20] In another study, a number of base stocks used in the preparation of cutting oils were found to be carcinogenic to mouse skin.[9] Those oils conventionally refined with sulfuric acid were carcinogenic, whereas solvent refined oils did not produce tumors. However, in a later study, solvent-extracted cutting oils were found to be carcinogenic as well.[21] This study also reported more papillomas in mice painted with used oil than with the same oil before use, suggesting the formation of carcinogens during industrial use. Mulligan has reported significant excess numbers of hepatic nodules in female mice which were skin-painted with synthetic cutting fluids, both with and without measurable amounts of nitrosodiethanolamine.[22]

Both polycyclic aromatic hydrocarbons and nitrosamines have been shown to cause digestive[23,24] and respiratory[25] cancer in laboratory animals. N-nitrosodiethanolamine, the most frequently cited carcinogen found in synthetic cutting fluids, has potent effects in several animal species[26,27,28]. Nitrosodiethanolamine has also been found to be absorbed through rat skin when applied undiluted or in solution with water or cutting fluids.[29]

Female ICD-JCL mice exposed orally to triethanolamine developed a high incidence of malignant lymphomas in one study.[30] This has been questioned because of an unusually low rate of tumors in the control animals. Triethanolamine and diethanolamine are both currently being tested for carcinogenicity through the National Toxicology Program.[31] Formaldehyde has been shown in two studies to cause nasal cancers in rats.[32,33]

Neither aluminum oxide nor silicon carbide has been shown to induce tumors in animals. However, several respirable dusts, including sands, abrasives and metallic aerosols have co-carcinogenic properties when given together with benzo(a)pyrene or diethylnitrosamine. Stenback found that neither aluminum oxide nor diethylnitrosamine produced lung tumors when given alone to Syrian Golden hamsters, but that significant numbers of lung tumors developed among hamsters treated with subcutaneous diethylnitrosamine followed by intratracheal aluminum oxide.[34] However, similar tumors also developed when 0.9% NaCl, used as the vehicle for the dust, was given with diethylnitrosamine but without the aluminum oxide.

Silica, chromite sand, olivine sand, ferric oxide and aluminum silicate dusts have all been found co-carcinogenic for lung tumors in hamsters when given with benzo(a)pyrene.[35] Stenback was not able to demonstrate co-carcinogenicity of aluminum oxide given to hamsters together with benzo(a)pyrene although 2 of 48 animals developed lung adenomas which were not found when either material was administered alone.[36] Ferric oxide and titanium oxide had a marked co carcinogenic effect in the same study.

Human Epidemiology: Occupational Groups

Early concern for the health of workers exposed to metal working fluids and related chemicals focused on reports of dermatitis, skin cancer and non-malignant respiratory disease. Oil folliculitis has frequently been reported among those working with straight oils, while irritant and allergic contact dermatitis are the most frequent skin disorders associated with soluble and synthetic metal working fluids (reviewed by Taylor).[37] Proliferative skin diseases, including keratoses and malignant tumors, have been found among workers exposed to straight and soluble oils by direct skin contact or through oil impregnated clothing.[38] In Britain, this has been best documented for scrotal carcinomas and epitheliomas among workers exposed to straight oils and probably soluble oils (reviewed by Waterhouse).[39] A recent case-control study in Connecticut demonstrated a similar association between squamous cell carcinoma of the scrotum and employment on metal working jobs presumed to involve cutting fluid exposure (toolmakers, toolsetters, screw machine operators).[40]

Airborne mists from machining and grinding operations are known to contain droplets of respirable size (less than 5 μm). Lipoid pneumonia and impairment of respiratory function have been reported among workers exposed to such respirable mist.[41] There is disagreement, however, about the prevalence and severity of these effects and several investigators have failed to demonstrate respiratory dysfunction in coolant exposed workers.[42,43] In a recent cross-sectional evaluation of 164 bearing plant workers exposed to straight and soluble oil for a mean duration of 16 years, Jarvholm found a significant excess in respiratory symptoms relative to office worker controls.[6] He was unable to demonstrate differences in spirometric measures or x-rays.

Since 1970 a number of reports have linked cancers other than those of the skin with work in occupations or plants with cutting fluid exposures. In a population based case-control study Roush found a strong association between sino-nasal cancer mortality and jobs with presumed cutting fluid exposure.[44] In a similar study Silverman demonstrated a slight association between bladder cancer mortality and metal fabrication and machinist work.[45]

Several studies have found a relationship between cutting fluid mist exposure and cancers of the digestive and respiratory systems. Waterhouse reviewed 228 cases of scrotal cancer in oil mist exposed workers from the Birmingham Regional Cancer Registry and found significant excesses of subsequent primary malignancies of the skin, respiratory system and upper digestive tract.[46] Dubrow and Wegman examined death certificates for white men who died in Massachusetts between 1971-1973 using age standardized mortality odds ratios.[47] There were associations between stomach, colo-rectal, pancreas, respiratory and other cancers with work as machinists and in related occupations.

Decoufle calculated cause specific standardized mortality ratios among workers on metal machining jobs in an engine plant.[7] There was a slight excess of respiratory cancer (38 observed, 33.9 expected) and digestive cancer (59

observed, 49.6 expected) for white males employed 5 or more years in oil mist exposed jobs. For those first employed before 1938 and with at least 20 years latency there was a significant two-fold excess of cancer of the stomach and large intestine (15 observed, 7.6 expected). No dose response effect could be identified and although each of the three main classes of cutting fluids was used at the plant, no attempt was made to distinguish among exposures. Decoufle suggests that at least part of the digestive cancer excess might have been related to ethnic differences between the study group and the U. S. population, but this was not specifically investigated. An unpublished proportional mortality study of deaths among workers in an engine plant similar to that studied by Decoufle has also revealed significant excesses of lung and gastrointestinal cancers, particularly of the pancreas, among those with more than 20 years of employment.[48]

Of particular relevance is a Swedish cancer incidence study by Jarvholm in a bearing plant which is reportedly very similar to the plant in this investigation.[49] Among machining workers exposed to acid refined mineral oils, there were 4 scrotal cancer cases but no excess in lung or digestive cancer. Among grinders, however, who worked with soluble cutting fluids with nitrite additives and mineral oil contaminants, there was a statistically significant twofold excess in digestive system cancer (15 observed, 7.8 expected) among those with more than 5 years of service and 20 years latency. There were 6 stomach cancers in this group while 2.6 were expected. No excess of lung cancer was noted among grinders.

One study of a population exposed to oil mist reported no excess of cancer mortality,[42] however, the study group size and follow-up period were limited.

Exposures to synthetic abrasives which are potentially present in metal working plants have received far less epidemiologic attention than cutting fluids. Three reports, however, have recently appeared. First, Sparks found excess deaths from pancreatic cancer, stomach cancer, stomach ulcers and alcoholism in a proportional mortality analysis of 931 deaths among jewelry workers.[50] Other causes of death associated with alcoholism such as cirrhosis and accidents were not in excess. Stomach cancer and ulcers but not pancreatic cancer or alcoholism were elevated in the subgroup of polishers, presumed to experience exposure to abrasive dusts. Second, Wegman found excess digestive cancer and non-malignant respiratory disease deaths in a proportional mortality analysis of 968 deaths among workers in a synthetic abrasive product manufacturing plant.[51] Sites of excess cancer included stomach, esophagus, large intestine, rectum and liver. Conclusions could not be drawn about the association of cause specific mortality with specific jobs or exposure. Third, Jarvholm has reported a similar excess of digestive system cancer and non-malignant respiratory disease incidence in a group of workers employed in abrasive product manufacture.[52]

Human Epidemiology: Other Factors

Non-occupational factors in the epidemiology of stomach, colon and rectal cancer have received considerable attention. Stomach cancer incidence is strongly associated with ethnic background. Annual male age-adjusted incidence per 100,000 population ranges from the world's high of 91 among Japanese to a low of 10 among white U.S. men. Eastern European rates are in the mid-range, 3-4 times higher than U.S. rates.[53] Rates for Japanese migrants to the U.S. are intermediate between the rates for country of origin and destination, however the change in rate toward that of the host country is not fully expressed until the second generation.[54] Rates for first generation Polish migrants to the U. S. remained very close to the Polish rates and subsequent generations shifted somewhat toward the U.S. levels in migrant studies using data from the 1950s [55,56]. It has been argued that early life environmental factors might explain this pattern. Second generation family members would be more strongly affected by dietary changes which occured following migration if the dietary effects on cancer were strongly dependent on duration or initiation during childhood years. Some have speculated that intake of fresh fruits and vegetable is negatively associated with stomach cancer while intake of salted and pickled foods is positively associated.[56] The migrant findings have not been analyzed with respect to changes in occupational and other environmental factors.

Stomach cancer rates have also been associated with the use of nitrate fertilizers in Chilean communities in one study and with the nitrate concentrations in public water supplies in another.[56]

In the U.S. stomach cancer incidence is greater among men than women and greater among blacks and hispanics than whites.[57] Among white men in the 1970's stomach cancer incidence was somewhat higher in the Connecticut SEER area than in all the SEER areas combined (average annual age adjusted incidence rates per 100,000 population of 14.3 and 12.7 respectively).[57]

The highest rates worldwide for colon and rectal cancer are in the U.S., particularly in Connecticut, and lowest in Japan. Eastern European rates are close to the low Japanese levels. Schottenfeld[58] points out that colon cancer incidence in Connecticut has increased 165% among white men and 192% among white women over the past 30 years while rectal cancer incidence has remained stable. Colon cancer incidence is generally twice that of rectal cancer except in several Eastern European countries where the difference is much less marked and in some cases absent.[58]

The change in colon and rectal cancer incidence among migrants toward the rates of the country of destination is more marked for these sites than for stomach cancer. Staszewski[55] notes that this effect is strongly apparent among Polish migrants. Environmental factors, including those associated with changing occupational patterns, probably influence these national differences and migrant effects. There has been considerable speculation that dietary factors play a role, with high risk possibly associated with diets high in fats and animal protein and low in fiber.[58,59,60]

Patterns of cancer mortality are affected by alcohol consumption. Oropharyngeal, esophageal and liver cancer are clearly associated with heavy alcohol intake.[61] Associations between colo-rectal, pancreas and lung cancer have been reported, but the literature is inconsistent and the relationships are not considered well established.[62] Although one recent study found a strong, positive association between alcohol and stomach cancer mortality, most other studies have been negative or ambivalent.[63]

METHODS

Design

A standardized proportional mortality analysis was carried out for a study population of hourly workers with 10 or more years' service at a ball bearing manufacturing plant. Case-control studies within the deceased population were carried out to examine exposure, latency and duration effects.

A limited number of hypotheses for testing were selected based on published findings. The primary hypothesis considered was: Cancers of the stomach, colon, rectum and pancreas are associated with exposure to cutting fluids and abrasives, possibly distinguishable in terms of mineral oil vs. water-based emulsions or synthetic cutting fluids. A secondary hypothesis was that lung cancer is associated with cutting fluid exposures on grinding and machining jobs. Other possible associations were examined without prior hypotheses.

Study Site

The bearing manufacturer in this study has occupied five buildings near Hartford, Connecticut for varying periods since the 1930s. At present, it occupies four buildings, the most recent built in 1958. It is an independent supplier to the automotive, aircraft and other industries.

The work force has varied between 3 and 4 thousand from 1950-1970 and has consisted largely of caucasian workers, substantial numbers of whom are of Central European and Mediterranean descent. UAW Local Union 133 currently represents about 2,000 active and 2,400 retired workers out of approximately 20,000 hourly employees who have worked at this plant since the 1930s.

The major processes are forging; machining and grinding rings; and forming, grinding and honing balls for bearings. Other activities include heat treatment, abrasive tumbling, cleaning, plating and assembly operations.

A variety of cutting fluids have been used. Mineral oil cutting fluids have been used consistently for most machining operations, particularly the multi-spindle lathes which do high-volume production and account for most machining activity. Since the 1960s some subsidiary machining operations have been done with water-based fluids.

Water-based cutting fluids have predominated in grinding over the past 40 years. However selected grinding operations (usually race or "form" grinding) have not been adaptable to water-based fluids and have generally been done with oil. The distribution of oil grinding activity in the plant has varied over time; rarely is oil grinding the sole grinding activity of a department.

The water-based cutting fluids used have generally been emulsions of mineral oils with additives, but synthetic lubricants have occasionally been tried. One water-based cutting fluid used in the 1950s through mid-1960s was known by the trade name "Lusol". This material probably contained organic amines and potassium nitrite, based on industrial hygiene reports from a neighboring bearing manufacturer, referring to the same product in 1949. Some emulsified cutting fluids currently in use contain nitrite additives.

The steel used in this plant has generally contained 1% carbon and 1.5% chromium. Some heavy bearing rings have used .25% molybdenom/1.5% manganese steel. Special low volume jobs have required high chrome (18%) steel or beryllium-copper, but this has been infrequent.

Approximately 2% of the bearings in this plant have required plating. Of these, 75% are cadmium plated and 25% are copper-silver plated. Few workers have ever held plating jobs. Grinding or machining steel after plating has been infrequent.

Abrasive grinding wheels in this plant have typically consisted of vitreous (glass) bonded aluminum oxide particles. Smaller amounts of silicon carbide abrasives have also been used.

A limited number of industrial hygiene samples for airborne oil mist were taken by the employer's insurance carrier on 6 occasions between 1972 and 1980. These were largely short term breathing zone samples. There was no apparent effort to determine representative exposure levels for employees working on different operations or with various cutting fluids. Mist levels on jobs probably done with straight oils (lapping, race grinding, ball manufacture) ranged from 0.07 - 2.8 mg/m^3. Mist levels on other grinding jobs probably done with soluble fluids ranged from 0.6 - 7.16 mg/m^3.

Study Population: Definition

The study population was defined as all hourly employees with 10 or more years' service who died from January 1, 1969 to July 31, 1982. This was based largely on the nature and availability of employer pension and death benefit records. Ten years' service is the criterion for vesting of pension rights; workers terminating employment with less than 10 years' service would not qualify for retirement or death benefits and could not be identified through benefits-related records. Prior to the 1960s, the criteria for vesting was 15 years' service, so that deceased workers who had terminated employment in that period would not be identified for the study population unless they had 15 or more years' service. All active workers have had life insurance benefits so that their deaths are not likely to be missed.

Years of service was calculated by subtracting date of hire (seniority date) from date of termination. For those missing either date (5.7%), the date of first or last known department assignment, respectively, was used. These dates in most cases were employer-supplied and may encompass periods of layoff,

or multiple hirings. In some cases of multiple hirings only the last employment period may have been covered.

Most hourly employees were members of the United Automobile Workers (UAW). A small number belonged to the International Association of Machinists (IAM). Records for both groups were maintained in an equivalent fashion by the employer.

Study Population: Identification

The potential study population was assembled from 6 sources:

1. 636 death certificates with dates of employment were provided by the employer. These deaths occurred between 1969-1980 and had been the basis of the preliminary PMR analyses.

2. 175 additional death certificates for deaths occurring 1980-1982, without employment dates, were provided by the employer.

3. 17 additional deaths were identified from UAW local union pension claim records for the period 1979-1982. These constituted 6% of all deaths known in that period.

4. Employer files contained records of 35 more deaths of deferred vested employees (those leaving employment prior to retirement, with 10 or more year's service) but because these former employees had not qualified for death benefits the employer had not acquired death certificates.

5. 8 deaths were identified from a list of 331 deferred vested employees thought to be alive by the employer which was a) submitted to a commercial vendor which searched Social Security files, and b) matched against the State of Connecticut death registry for the years 1969-1978.

6. 26 deaths not otherwise identified in the first 5 sources were located in UAW local union-maintained annual pension summaries for the years 1981-82. These constituted 19% of all known deaths for that period and probably include deaths for which death benefit claims had not yet been filed.

768 of these 897 deaths met the study population criteria. Death certificates and job histories were obtained for 702 or 91%.

Causes of Death

Coding of underlying and contributing causes of death was performed by two coders: 1) A university based occupational physician experienced in ICD coding and not otherwise involved in the study did the first set of 638 deaths; 2) An NCHS nosologist performed the remainder. Previously, ICD coding by the occupational physician had been shown to be reliable: 1) in one study a 10% sample was recoded by this physician and only one out of 26 cases was coded sufficiently differently to result in a different cause-of-death category for calculation of PMRs; 2) in another study a comparison between the physician and the State of Michigan which uses the NCHS automated coding system, for a sample of 169 deaths, resulted in complete agreement (3 digit codes) for over 80% and major disagreements for less than 10%, primarily relating to heart disease and diabetes; only 3 disagreements involved cancer. All deaths were coded in the Eighth Revision of the International Classification of Diseases, corresponding to that used for the reference population's proportional mortality rates (which were based on the period 1968-1978).

Death certificates were also examined by the authors for deaths coded as stomach, rectal or lung cancer by the nosologists. No ambiguous cases which might be subject to variable nosology judgments were found.

All coding of death certificate information for data entry (including age, location of birth, race, etc.) was carried out by one person and verified by a different person.

Job Histories

Sources of Information

There were four major sources of workers' job history:

1. The most complete source was a 1967 UAW local union seniority list, giving department, job, and date of hire. This list was matched to the entire set of deaths identified, using last name, first initials and seniority date, and producing matches for 421 out of 755 (56%).

2. The second source was a UAW "Dues Check-Off" List for 1962, giving names and departments only. These employer-generated lists show all employed union members, for dues collection purposes. Matching to this list produced matches for 65% of the study population.

3. Dues cards with date of hire, last department worked and clock number for four year periods are maintained by the UAW local union. Some of these include Social Security Number. Incomplete and overlapping sets of dues cards were available for the periods 1952-1955, 1956-1959, 1964-1967, in addition to a file with the last dues card for all retired and terminated workers. Members of the study population were matched by hand to these card files.

4. Employer records of deferred vested employees gave sequences of departments worked but without dates other than hire and termination.

Using these records, department locations for large numbers of the study population were determined at the dates of hire and termination and at 5 points in time:

Jan. 1956	-	dues cards
Jan. 1960	-	dues cards
Sept. 1962	-	check-off list
Oct. 1967	-	seniority list
Jan. 1968	-	dues cards

For IAM workers, seniority lists were available for the years 1960, 1965, and 1973. IAM members worked exclusively in the tool and die departments.

Matches of the study population to job histories were sometimes ambiguous due to variations in spelling, duplicate names and missing information. To ensure that matching was accurate, several measures were taken utilizing additional information such as clock number, Social Security Number, date of initiation into union, and job classification, when available.

Job Categories and Exposure Variables

Approximately 200 departments which existed at one time or another were identified from old seniority lists and contracts. Most departments were characterized by a single major process which was used to assign that department to one of 35 Job Categories. This classification was based on: a) discussions with local union officials; b) meetings with the plant metallurgist, production chemist, engineers and physician; and c) an inspection of three of the plant's buildings.

Table 1 gives the Job Categories and Table 2 gives a sampling of the 200 departments with assigned Job Category Number. The "mixed" designation was used when both oil and water based grinding fluids were used in a department simultaneously or at different times, or when the fluid used was unknown.

For each member in the study population, a chronological sequence of department-based Job Categories was compiled. A summary Job Category was then defined as the last known Job Category held prior to 15 years before death (if unknown, the first known Job Category). The limited nature of the work histories available made it impossible to estimate cumulative exposure durations.

Job Categories were grouped according either to common processes or to common exposures in order to create two sets of work history variables for analysis. (Table 3)

The work history variables based on process are as follows:

1. Machining: includes all production machining job categories.

2. Grinding: includes all production grinding.

3. Tool and Die: includes tool and die workers and tool grinders.

4. Forge and Heat Treat: includes forge, cold heading, upsetting and heat treat for rings and balls.

5. Skilled Trades: includes electricians and maintenance machinists.

6. Maintenance: includes other skilled trades and janitors.

7. Assembly and Packaging: includes gauging, assembly, pressing, salvage and packing.

The work history variables based on character of exposures are as follows:

1. Mineral Oil Cutting Fluids, Comprehensive: A broad group of job categories sharing some potential exposure to mineral oils, including most machining, grinding with oil and mixed cutting fluid exposures, and material handlers supporting machining and grinding.

2. Mineral Oil Cutting Fluids, Restrictive: A less broadly defined set of job categories with more definite oil exposure and less likely water exposure, including most machining and a small number of grinders working only in oil.

3. Water-based Cutting Fluids, Comprehensive: includes almost all grinding, machining with water-based cutting fluids, as well as supporting material handlers in grinding.

4. Water-based Cutting Fluids, Restrictive: includes machining and grinding working only with water-based cutting fluids.

5. Mineral Oil in Grinding: includes all grinding with oil, or mixed cutting fluid exposures, and supporting material handlers (almost all of this group had exposure to water-based cutting fluids as well).

6. Inorganic Fiber, Dust or Pulmonary Irritant: includes shot blasting; plating; polishing; abrasive wheel making; tool grinding; tumbling and cleaning.

For analysis, then, a worker's work history was specified by those process- and exposure-based groupings of Job Categories which included the worker's summary Job Category.

These exposure groups are not mutually exclusive. The "restrictive" groups are subsets of their respective "comprehensive" versions. The two "comprehensive" groups overlap. However, the Water-Based Cutting Fluid, Restrictive group is mutually exclusive with both the Mineral Oil Cutting Fluid, Restrictive and the Mineral Oil in Grinding groups.

Analysis

Proportional Mortality

Standardized proportional mortality ratio analyses were carried out using Monson's PMR program.[64] The U.S. reference rates provided by Monson were updated through 1978.[65] Mantel-Haenszel based confidence intervals and X^2 statistics were computed. Poisson P-values were computed when expected numbers of cause-specific deaths were less than five.

Case-Control

In case-control studies, Fisher Exact statistics were computed for single 2 x 2 tables; Wolfe tests for homogeneity were computed for stratified 2 x 2 tables[66] and Mantel-Haenszel odds ratios computed where homogeneity was found.[67] In addition to stratification, an age-adjustment procedure was used to control for confounding by age at death, as described in Appendix I. A posteriori power calculations were done using the method of Fleiss[68] for unequal numbers of cases and controls.

Analysis of possible associations between cause-specific mortality and work related exposures was conducted by grouping plant jobs with common manufacturing processes and with common chemical use. The potential confounding risk factors analyzed were age at death and country of birth.

For each work history variable, a comparison "non-exposed" group was defined consisting of all other Job Categories except those known to be associated with the same health outcomes hypothesized for that exposure or known to share substantial exposures with that measure (Table 3). Thus for example, when the work history variable was Grinding in Mineral Oil, the comparison job categories excluded those involving machining in oil or other grinding. Exclusions were all based on prior knowledge or expectations except that the tool and die, hot/cold heading, forge, and heat treat Job Categories were excluded from comparison exposures for the various cutting fluid variables after their possible associations with rectal and lung cancer were found.

For each disease outcome, controls did not include other causes of death expected to be associated with the exposures under consideration. Thus,

specific gastrointestinal (GI) cancer cases were compared with controls excluding other GI cancer sites, lung cancer and non-malignant GI disease; controls for non-malignant digestive disease excluded GI and lung cancer. These exclusions were based on prior knowledge of exposure-associated health effects. Table 4 gives the outcome variables and their controls.

RESULTS

Study Population Characteristics

A total of 768 deaths met the study population criteria for date of death and years of service. Proportional mortality calculations were made for the 755 (98%) for whom cause of death was determined. Case-control analyses were limited to the 702 (91%) for whom both cause of death and job history (for at least one point in time) were available. (Figure 1) Ascertainment of cause of death and job history was 90% or better regardless of the source from which groups of decedents were identified.

The sex and race distribution of the study population is shown in Table 5.

For workers with job histories, most (88%) had a departmental assignment known at 2 or more points in time. The number of distinct Job Categories assigned to workers based on these known departmental assignments ranged from 1 to 4. Table 6 shows the distribution of the number of these distinct Job Categories. Most workers, including those whose department at multiple times was known, worked in only 1 or 2 Job Categories. For example, of the 444 workers with three or more department/time points, 418, or 94%, have one or two distinct Job Categories. In many cases in which an employee worked in two or more Job Categories, these were similar—e.g. different sub-categories of grinding.

The distribution of the study population in the major process and exposure categories is shown in Tables 7 and 8 respectively. Grinding makes up the largest process group, followed by Assembly and Packaging, Tool and Die, and Machining. The "Other" category includes a large number of inspectors, together with material handling and several smaller activities.

The distribution of deaths in the individual Job Categories making up the process and exposure variables relating to cutting fluids is shown in Table 9. There were very few deaths in the machining in mixed cutting fluids, machining in water, or grinding in oil categories. Therefore, for the purposes of this mortality study, machining was almost exclusively exposure to oil-based fluids, and grinding was exposure predominantly to water-based fluids together with, in some cases, straight oils.

Table 10 displays the study population's ages, duration of service and latency characteristics classified by race, sex and place of birth. 16% of white men and 11% of white women were Central European in origin. Central Europe includes Poland, Hungary and Czechoslovakia, but excludes Austria and Germany. The major constituents of the "all other" category are Canadians, particularly of French ancestry (n=22), Italians (n=56), and Germans, (n=9). Non-U.S. born decedents appear to enter this workforce later in life, leave employment slightly older and live to older ages.

The association of place of birth with the major process groups is shown in Table 11 for white men and women. Grinding is not associated with birthplace. White men born in the U.S. were overrepresented in Tool and Die and other skilled trades. 60% of Forge and Heat Treat workers were of Central European origin.

The distribution of age at hire, termination, and death, within the major process groups is shown in Table 12 In general, the distribution of age at death differed for the exposure measures in relation to their comparison groups, and thus control for confounding was required. For example there is a relatively large proportion dying below age 60 in those classified in Machining, compared with Grinding and others, despite similar ages of hire and termination.

Proportional Mortality Ratio Analysis

Overall analysis (Tables 13, 14)

- There was a 50% elevation in proportional mortality for gastrointestinal cancer among white men, largely attributable to stomach cancer (PMR=11/5.6=1.98, p=.02) and rectal cancer (PMR=11/3.6=3.04, p=.003). Among white women there were 12 gastrointestinal cancers compared with 9 expected.

- There was a 25% elevation in proportional mortality for lung cancer among white men (PMR=59/47.3=1.25, p=.08). Among white women there were 5 cases observed and 4.7 expected.

- There was a significant excess in deaths from stroke among white men (PMR=60/44.6=1.35, p=.01).

- There was a significant excess in deaths from an ill-defined group of neuropsychiatric disorders among white men (PMR=13/3.18=4.09, p=.001), 12/13 of which were attributed to variants of "chronic alcoholism" on death certificates.

- When the study population was restricted to those with 20 or more years' duration of employment, the overall pattern of PMRs for white men was essentially unchanged, however the PMR estimates for stomach cancer (PMR=2.23), rectal cancer (PMR=3.41) and "chronic alcoholism" (PMR=4.28) were all larger than in the full study population (representing 10 or more years' employment).

Gastrointestinal Diseases

The elevation in proportion of deaths due to gastrointestinal cancers among white men (PMR=55/35.3=1.56, p=.001) was mainly due to cancers of the stomach and rectum; cancer of the colon was only slightly elevated (Table 13). In white women, with much smaller numbers, no GI cancers were statistically significantly elevated. However there were 3 stomach cancers compared with one expected (Table 14).

The PMR findings in several sub-populations of white men are summarized in Table 15. In men born in Central Europe there was a non-significant elevation in cancer of the colon, rectum and stomach. Excluding those of Central European birth, stomach and rectal but not colon cancer were elevated.

Within exposure groupings, men in Grinding had excesses in proportional mortality due to GI malignancies, particularly cancer of the stomach (Table 16). The stomach cancer PMR is greatest among grinders with long latency, i.e. 30 or more years from date of hire until death (PMR=4/.91=4.4, p=.03). There were no significant excesses among the smaller group of white men assigned to Machining. Cancer of the rectum appeared to be elevated across all groups with the highest proportion dying of this cause in tool and die workers (PMR=2/.41=4.9), however this was not statistically significant.

Among white women there were gastrointestinal cancer proportional excesses, but numbers were too small for reliable inference even in the largest subgroups of grinders and assembly/packaging workers (Table 17).

In the non-white population there were 2 digestive cancers - stomach and colon - out of 22 deaths.

In none of the sub-populations of white men or women was non-malignant digestive disease elevated.

Respiratory Disease

Among white men the lung cancer PMR was 59/47.3 = 1.25 (p=.08). There was a significant deficit of pneumonia deaths (PMR=7/14.9=0.47), along with a slight excess of all other non-malignant respiratory deaths combined (PMR=33/28.1=1.17) although this excess was not statistically significant. There were no clear patterns according to place of birth or job category. There were no deaths attributed to lipoid pneumonia, pneumoconiosis or chronic interstitial lung diseases. In women, there was a cluster of lung cancer among grinders which accounts for all lung cancer deaths in women (PMR=5/1.77=2.8, p=.07) (Table 17). There were no nasal cancers.

"Chronic Alcoholism"

For white men there was a highly elevated proportion of deaths from "mental, psychoneurotic and personality disorders" (Table 13: PMR=13/3.2=4.06, p=.001). The actual causes of death were almost entirely (12/13) recorded as ICD 303, variants of chronic alcoholism. However, in every case the cause of death was assigned by a medical examiner rather than a physician attending the deceased prior to death. This group was characterized by relatively young ages of hire (29.3), termination (54.3), and death (57.3). Cirrhosis of the liver as the cause of death in the study population was slightly but not significantly elevated (PMR=18/14.1=1.27). There were fewer GI ulcers, accidents and suicides than expected. There was a cluster of these ill-defined diseases among grinders (PMR=7/1.05=6.66, p=.001) although not among the long latency grinders. There was a substantial excess both among those of Central European and non-Central European birth.

Other Findings

The PMR for stroke was significantly elevated only in the group of white men not of Central European birth (PMR=52/35.7=1.46, p=.004).

Skin cancer was the underlying cause of death in 4 white men. Three of the 4 were in machining or grinding, for whom the expected number was .94 giving a PMR of 3.2 (not statistically significant).

Case-Control Analysis

Stomach Cancer

Case-control analysis produced statistically significant crude odds ratios for the association of stomach cancer proportional mortality with Grinding and with Water-Based Cutting Fluids (Tables 18-20). For Grinding the odds ratio was 6.2 (p=.055), and for Water-Based Cutting Fluids, Comprehensive, the odds ratio = 6.5 (p=.043). These two exposure groups were composed largely of the same decedents, as there were no deaths in the grinding in oil Job Category, and only 3 in the machining in water (or mixed) categories (Table 9). For the Restrictive Water-Based Cutting Fluids group, the odds ratio was 4.7 (p=.23), a less stable estimate because there were only 2 exposed cases. For the Mineral Oil in Grinding exposure variable, composed of all grinding jobs believed likely to have had some oil mist exposure in addition to water-based mists, the odds ratio was 7.4 (p=.04); (Table 21).

In all these analyses, age at death is distributed differently among exposed and comparison groups and is thus considered a confounder. Stratification on the age at death for the Grinding group results in odds ratios of 4.9, 2.7 and

4.2 for the age intervals <60, 60-74, >74 respectively (0.5 added to arrays having empty cells). The Mantel-Haenszel summary odds ratio was 5.3 but was not statistically significant (p=.14). Because of loss of power with stratification, an age adjustment was calculated to correct for the confounding, based on national proportional mortality rates (see Appendix I). The correction factor for the odds ratio due to age at death confounding was .98, yielding an age-corrected odds ratio of 6.1.

The estimated stomach cancer odds ratio among grinders is quite unstable because of the small number of cases (7 exposed and 1 comparison). If the comparison group is expanded to include all other Job Categories except "production control" (which has significant grinding exposure) the resulting case-control analysis yields a crude odds ratio of 6.95 (p=.01), now with 2 cases in the comparison group. Age adjustment gave an odds ratio of 6.76 by the method described in Appendix I; the Mantel-Haenszel odds ratio was 6.77 (p=.02) and there was no significant inhomogeneity in the 3 strata.

Because there was no association between the exposures and origin of birth, there was no confounding. Second generation cultural factors might have been important confounders if a substantially larger proportion of grinders were second generation Central Europeans than the comparison group. However, only 1 out of 7 of the stomach cancer cases among grinders had a Central European name but U.S. origin of birth.

Cancer of the stomach in white men was not found to be associated with machining or tool and die work (there were no stomach cancer deaths) or with the other major process measures, however the power for these tests using case-control analysis was very small (.04-.08 for OR=4, α =.05). The power for detecting stomach cancer in women grinders was also very low (.06 for OR=4, α =.05).

Colon Cancer

No significant association between cancer of the colon and any of the exposure measures was found. In grinders, the crude odds ratio for colon cancer was 1.31 but this was not statistically significant. Moreover, this may be a slight overestimate because grinders died more often between the ages 60-74 than the comparison group and national colon cancer proportional mortality is highest in that age range (Table A-1, Appendix I). In grinding, the power for detecting colon cancer in the absence of confounding was .56 (for OR=4, α =.05).

Rectal Cancer

There were no statistically significant associations found between cancer of the rectum and any of the exposure groups, although the odds ratio for the Tool and Die process category was 2.3, based on 2 exposed cases. There were 2 other cases in the lapping Job Category out of 22 deaths there. When all

Job Categories where there was any type of abrasive grinding exposure, including tool and die, and lapping, were contrasted with all other categories, the crude odds ratio was 3.0, based on 7 exposed and 2 unexposed cases, but it was not statistically significant (p=.14).

Non-Malignant Digestive Disease

Case-control analyses for non-malignant digestive disease (ICD 8: 520-577) revealed no significant associations with the process or exposure groups.

Lung Cancer

The only association of lung cancer with the major processes or exposures, in white men, was with the Forge and Heat Treat category and this was not significant (Table 22: OR=3.6, p=.10). Age in this case is negatively confounding in that the forge and heat treat exposed workers are older at death than the comparison workers and the proportional mortality rates for lung cancer are declining above age 60.

When latency is examined for the forge and heat treat group, a large association is revealed in the subgroup of those dying between 15 and 30 years following hire (OR=9.3); all 3 lung cancers occured in this subgroup.

Lung cancer mortality in women occurred only among grinders. The odds ratio was large and significant. (Table 23: OR=19.3, p=.008) The level of risk was indistinguishable using the different grinding related exposure measures. However, if the two mutually exclusive cutting fluid exposure measures, Water-Based Restrictive and Mineral Oil in Grinding, are compared directly on proportion of lung cancer, a significant large odds ratio results (OR=20, p=.04), suggesting that the lung cancer is associated with the water-based cutting fluids in grinding (Table 24).

Age confounding in comparing grinders with non-grinders is small because women grinders were equally less likely to die below age 60 or above age 74 than were those in the comparison group, and the proportional mortality rates are consistently declining over the entire age range of 50-84. In the comparison of oil vs. water-based cutting fluids, the age-confounding is small because: 1) deaths in the two groups were similarly distributed, and 2) the decline in proportional mortality rates for lung cancer over the age range 50-84, by a factor of 4, is small compared with the observed odds ratio of 20.

Non-Malignant Respiratory Disease

Case-control investigations of non-malignant respiratory disease (ICD 460-519) among white men suggest an association with grinding and with water-based

cutting fluids. For grinders, there was a significant association only in those dying above age 74 (odds ratio = 5.3, p=.02), recognizing that the P-value must be viewed cautiously since the 3 age strata were examined for the highest association without a prior hypothesis concerning age. Within this age stratum associations with the related cutting fluids exposures are as follows:

Water-Based, Comprehensive - odds ratio = 4.2, p = .03;
Water-Based, Restrictive - odds ratio = 4.7, p = .10;
Mineral Oil in Grinding - odds ratio = 1.5, p = .19
Mineral Oil, Restrictive - No association.

For maintenance workers (including both janitors and skilled workers such as pipe fitters) there was a possible association (OR=1.6, p=.047), however only 2 cases were exposed.

There was no significant association of non-malignant respiratory disease with inorganic fiber/pulmonary irritant exposure, but only 1 case was exposed.

"Chronic Alcoholism"

Case control analysis of the deaths diagnosed by medical examiners as "chronic alcoholism" in relation to Grinding produced an odds ratio of 5.5 but it was not statistically significant. (Table 25).

Stroke

Stroke is elevated in white men (PMR=60/44.5=1.35, p=.01), however there were no significant associations with exposure measures. The highest proportion of stroke mortality is in the Other process category (20% of deaths). The Other category includes "ball inspectors", 7/42 of whom died of stroke (17%); "lapping", 3/22 (14%); "production control", 3/15 (20%) and "grey iron machining", 4/13 (31%). These departments do not share unique known exposures. One of the 10 forge/heat treat deaths was due to stroke.

DISCUSSION

The purpose of this study was: to test hypotheses concerning specific hazards believed present in metal working environments, and to generate hypotheses based on associations found but not expected a priori. For the latter, importance has been afforded to some findings such as lung cancer in forge and heat treat even though small numbers indicate that the association could well have arisen by chance. Findings which confirm prior hypotheses and add weight to the findings of previous research should lead to vigorous follow-up action. Findings of new associations not hypothesized should guide future investigation, but prudence also requires that some industrial hygiene and other steps be taken to assess and control exposure.

This investigation has limitations which affect the interpretation of results.

1. Findings are largely restricted to the white population. The number of non-white decedents (n=22) was too few for detecting all but very large effects, and among these there was no highly unusual concentration in causes of death. Therefore, this study provides no basis for concluding that the non-white population has or has not experienced a mortality pattern different from that of white workers..

2. The work histories which were available were incomplete. This limited the ability to evaluate latency and duration effects. However, the considerable stability observed in workers' assigned Job Categories over time provides some assurance that the exposure misclassification arising from incomplete job histories was not an important problem. In the case of the 7 grinders dying from cancer of the stomach, the average number of points in time where their department was known was 2.9 out of a possible 7. In every case, the summary Job Category falls within Grinding when based on each of two methods (the most frequently held Job Category, or the Job Category held 15 years prior to death). Misclassfication of exposure categories would tend to reduce the magnitudes of associations observed. Because no information was available regarding employment prior to that in the study plant, any relationship to mortality could not be assessed.

3. Proportional Mortality Ratios were calculated using national population reference rates. Differences between these national rates and Connecticut State and County rates might have affected the PMR calculations. For example, stomach cancer incidence and mortality were higher among white males in the Connecticut SEER area in the 1970's than other SEER areas combined[57]. National rates may thus have underestimated the expected number of stomach cancers and, consequently, over-estimated the stomach cancer PMR. The same effect might have artifactually exaggerated the PMR's for colon and rectal cancer which have high rates in Connecticut[58]. These State-National differences are small and therefore unlikely to have had a substantial impact on this study's findings or conclusions. For example, among

white men annual age adjusted stomach cancer mortality as a proportion of all cancer mortality in the 1970's was .051 in Connecticut and .045 in all SEER areas combined. For colon cancer the equivalent proportions are .107 and .102, while for rectal cancer they are .033 and .030.

4. Small numbers in many of the exposure limited the power of tests of association. Thus, for example, the forge and heat treat group was of interest because of unique worker exposures to oil smoke, but with only 10 decedents in this group possible associations could not be effectively studied. Similarly, some of the observed associations (such as the apparent high risk of lung cancer to women exposed to water based fluids relative to those exposed to oils) must be considered only tentative because of small numbers.

5. Standardized proportional mortality analysis, being conditional on death, gives no direct estimates of absolute risk, although PMR's have yielded reasonable approximations of Standardized Mortality Ratios (SMR's) obtained from the same populations of workers.[69,70] If the full at-risk population from which the decedents in this study were drawn had an all causes SMR substantially less than 1.0 and if this "healthy worker" effect reflected cardiovascular disease patterns, cancer PMR's could be elevated even if underlying cancer death rates were normal. In fact, the standardized PMR for all vascular disease in this population is 0.97 (for white men) which shows that an artifactual elevation in cancer PMRs could not have come from reduced cardiovascular disease in the study population at risk. Furthermore, such an elevation would be expected to be uniform across all cancers, aside from sampling fluctuations, and would not generate the specific pattern of GI cancer seen in this study. The associations between causes of death and exposures found in case-control analyses would not have been distorted by these effects.

Cancer of Stomach

The association of cancer of the stomach was found with Grinding but not with Machining or Tool and Die exposures. Using the proportional mortality rates of the U.S. population and the observed PMR for Grinding (3.88), the probability of observing no stomach cancer in each of Machining or Tool and Die, if they have the same risk as Grinding, is only .09 (Exp.=3.88x.62=2.4, Obs.=0; Poisson one-sided test). The failure to observe cancer of the stomach in either group provides moderate support for the conclusion that in this population the exposures experienced in these jobs have less risk of stomach cancer than grinding.

Therefore, the major finding of this study is that cancer of the stomach among white men is associated with grinding but not machining exposure in a bearing plant where grinding has been done predominantly with water-based cutting fluids. There is a nearly 4-fold increased proportional mortality ratio for

stomach cancer in grinders; when grinders are compared to those not exposed to cutting fluids, the odds ratio is 6.1. For the different cutting fluid exposure groups, within grinding, there is an apparent trend of increasing odds ratios toward groups using oil in addition to water-based fluids:

	Age Adj. OR	p
Water-Based CF, Restrictive	4.6	.22
Water-Based CF, Comprehensive	6.4	.04
Mineral Oil CF in Grinding	7.3	.04

However, due to the instability of these estimates, these comparisons are only suggestive. Furthermore differences in types of grinding may well confound the above comparisons: workers and plant personnel reported that mist generation and exposures are generally higher with bore and race (or form) grinding than with surface and outside diameter grinding, in part because the latter are less labor-intensive activities. Mineral oil cutting fluids tended to be used only for bore and race grinding, but these jobs would also experience the highest levels of water-based cutting fluid mists. Thus the suggestive trend in odds ratios is uninformative as to the type of cutting fluid more associated with stomach cancer.

Stomach cancer is associated with grinding but not machining. As machining was done primarily with oil while grinding used mostly water soluble fluids, we interpret the pattern of stomach cancer proportional mortality in this study as evidence that stomach cancer is associated with the use of water-based cutting fluids. The absence of stomach cancer in machining may reflect the small usage of water-based fluids in machining until relatively recently, in this plant. A second, less likely, interpretation is that stomach cancer is associated with straight oil cutting fluids, but only in grinding. This possibility requires that additional mechanisms be postulated to explain the absence of machining stomach cancer deaths, such as a) fluid temperatures are higher in grinding, b) abrasive dusts are an essential cofactor, or c) mist concentrations are higher or differ in other important characteristics. It may be that these factors are necessary for the expression of carcinogenic or co-carcinogenic properties of polycyclic aromatic hydrocarbons. Parsimony favors the first interpretation. Some previous reports have suggested that grinding with water soluble cutting fluids, containing nitrites, carries the greatest risk.[49] This suggestion is supported by the findings of this study, however further confirmation is needed because the job history data in this study did not permit a precise distinction between the different types of cutting fluids, and misclassifications were possible.

The stomach cancer associations could not be explained on the basis of confounding by age or place of birth. While stomach cancer incidence is quite elevated in Central Europe and also (but less so) for immigrants from Central Europe, there was no association in the study population of Grinding with Central European origin. Age at death confounding was present but estimated to be small in relation to the observed effect because: 1) a similar positive

association was observed in each age stratum; 2) proportional mortality due to stomach cancer in the U.S. population for white males varies little (.0085 to .0104) over the age range 50 - 84; and 3) age adjustment altered the odds ratio only by a factor of 0.97.

In view of these findings on stomach cancer the speculation of Correa et.al is worth note:[71] that erosion of the gastric surface by abrasive foods and surfactants predisposes the gastric mucosa to dysplasias and malignancy by the actions of nitrosamines or other mutagenic/carcinogenic agents. Although these authors were concerned with abrasives in the diet, this mechanism is equally plausible in precision grinding environments where abrasive, aerosolized particulates are endemic. Water-based cutting fluid mists presumably contain abrasives debris as well as metallic matter, low vapor pressure additives and in some cases, residual oils.[72] Inhaled mists are probably cleared in part from the upper respiratory passages and swallowed.

Nitrosamines are the key suspect carcinogen in water-based fluids, however this has not been thoroughly investigated. Soluble oil emulsions could also generate mineral oil-derived, polyaromatic hydrocarbons (PAH) however the levels would be expected to be lower than with the straight oils due to cooler operation with water-based fluids and lower oil concentrations.

Cancer of Lung: White Women

The finding of lung cancer in women grinders is potentially important, although it is based on only 5 cases and no smoking information was available. The high risk among women grinders using water based fluids in comparison with mineral oils merits further careful study, although the finding is based on only 3 exposed women (Table 30). Additionally, care in interpretation is necessary because the findings are somewhat inconsistent. If simply due to water-based fluids, there should be a higher observed proportion of cases in the Mineral Oil in Grinding category, because members of this exposure group also have at least partial exposure to water-based fluids. This suggests that other characteristics of the particular departmental activities are important beyond just grinding in water-based fluids, or that there has been misclassification of some Job Categories with respect to type of cutting fluid used.

The absence of lung cancer in the non-grinding population of women where 3.0 were expected indicates that the women in this study population were not heavy smokers overall.

No similar association was seen for men. This could reflect the generally greater smoking exposure in men, masking a smaller effect due to cutting fluid exposures. Or, the different kinds of grinding done by the two groups could explain the difference. Women generally work on the components of smaller bearings; included in their work are the so-called "precision products" which occasionally utilize more exotic metals including beryllium, nickel and chromium. There have also been organic material exposures in ring production arising from fabrication with epoxy resins and the molding of urethane to

rings. The hypothesis generated is that there are special activities associated with grinding in water-based cutting fluids, that have a high risk of lung cancer.

Cancer of Lung: White Men

Forging, upsetting, cold heading and heat treating operations were all included in the Forge and Heat Treat exposure measure. At this plant, these activities all generate smokes of pyrolyzed lubricants or quenching oils. Asbestos exposures were common in these kinds of jobs in the past, but probably at low levels. Thus the finding of lung cancer in 3 of 9 deaths in this group may have meaning despite the fact that the case-control associations were not significant (except when the latency interval was restricted, a posteriori, to the 15-30 year group). The hypothesis that forge, heat treat exposures are associated with lung cancer strongly merits a more definitive test.

Cancer of Rectum

Elevated proportional mortality from cancer of the rectum was among the most statistically significant findings for white men in the full population (PMR=3.04, p=.003), however no association with specific process or exposure groups was indicated except possibly for tool and die (PMR=2/.41=4.9, p=.12), or all abrasive grinding activities (OR=3.0, p=.14). Aside from machining and grinding operations, tool and die work involves the preparation, hardening and grinding of cutting tools made from a variety of tool steels and also from tungsten carbide. Tungsten carbide or "hard metal" contains a high proportion of cobalt whose likely role in "hard metal disease", a pulmonary disorder with features of interstitial fibrosis and granulomatous disease, has been described[73]; possible GI toxicity or carcinogenicity has not been investigated with this material. Lapping, the Job Category in which 2 of 22 deaths were due to rectal cancer, involves grinding of balls with pastes prepared with kerosene or other mineral oil fractions and abrasive powders.

"Chronic Alcoholism"

The finding of a markedly increased proportion of deaths coded as chronic alcoholism, was unexpected, although a subsequent rereading of the literature revealed a similar finding in a population of workers using synthetic abrasive materials.[50] This is particularly interesting in light of the association of these deaths with grinding jobs in this study.

These data do not support the conclusion that this group of workers in fact experienced excess mortality from chronic alcoholism. The alcoholism diagnoses were not made by attending physicians but by medical examiners who were unlikely to have been familiar with the decedents' medical conditions before death. There are no pathognomonic signs of alcoholism and these examiners may have been inclined to attach the specific diagnostic label of "alcoholism"

to cases with general findings of chronic, debilitating disease, particularly those with indications of liver damage. This possibility is supported by the fact that there were also somewhat more cirrhosis deaths than expected. However, several causes of death that are commonly elevated in groups with a high prevalence of alcoholism including accidents and oropharyngeal cancer were not elevated in this study. Esophageal cancer, which has been associated with alcohol intake, was proportionally increased among white men, although this was not statistically significant. The deficits in some alcoholism-associated conditions (e.g. suicide, GI ulcers), were not large enough to account for the alcoholism excess simply by reclassification, i.e. alcoholism-associated causes of death all being coded as alcoholism itself. The probable diagnostic misclassification, along with the association observed with Grinding, raises the possibility of an occupational exposure that leads to a chronic, debilitating disease with features of cirrhosis and alcoholism. Review of hospital and autopsy reports might be revealing in this regard.

Proportional Mortality for GI Disorders: Central European vs. U.S. Birthplace.

Central Europeans have elevated stomach cancer and reduced colon and rectal cancer incidence rates compared with U.S. born[53]. Assuming that proportional mortality for these disorders follows the same pattern and that there are strong migrant effects for colo-rectal but not stomach cancer, the observed PMR's are not what might be anticipated, i.e. a higher stomach cancer PMR among Central Europeans and no difference in colon or rectal cancer PMR's by country of birth. This may partly reflect the different age distributions in the study population: those deceased of Central European origin lived an average 7 years longer than U.S. born (and those of other origin lived 11 years longer), so that the indirect standardization underlying PMR analysis will produce non-comparable PMRs. More important, the age difference suggests different overall mortality rates so that the proportional mortalities are being strongly influenced by other causes of death with different incidence rates between the 2 populations. These differences may arise in part from a healthy worker effect that differs between the immigrant and indigenous population, which is additional grounds for controlling confounding by origin of birth. Finally, the incidence rates for the GI cancers may differ substantially from the cause-specific mortality rates, especially since conditions like colon cancer have a considerably more favorable prognosis than stomach cancer.

CONCLUSIONS AND RECOMMENDATIONS

1. This study finds a statistically significant increase in the proportions of deaths due to stomach cancer, rectal cancer, and stroke in a population of bearing plant workers. An association of increased proportional mortality from stomach cancer with grinding jobs was found among white men in this plant where abrasive grinding has been done predominantly with water soluble cutting fluids. It supports previous findings of excess gastrointestinal cancer among workers in plants where metal machining and grinding are major activities and confirms the previously reported association of stomach cancer with grinding jobs.

   Additional epidemiologic and toxicologic investigations are necessary to identify specific high-risk exposures more precisely. There is a particular need for thorough industrial hygiene evaluations in this industry so that epidemiologic associations between health outcomes and specific chemical exposures can be effectively evaluated. It is necessary that both the present and historic chemical characterization of these work environments be measured or reconstructed. Particular emphasis should be given to nitrosamines, polycyclic aromatic hydrocarbons, abrasive particulates, formaldehyde and other organic volatiles. Particle size and chemical composition of various grinding aerosols need evaluation. Work with soluble fluids, which in our experience is most common in the industry today and which is most convincingly associated with stomach cancer excesses, should receive priority attention in this regard.

2. Several other findings require confirmation and elucidation:

   a. An association of lung cancer in women with grinding, probably with water-based cutting fluids.

   b. An association among men of lung cancer with forge and heat treat operations.

   c. An association between grinding and a poorly described group of disorders probably misclassified as chronic alcoholism.

   d. An excess in proportional mortality from stroke and lung cancer among white men.

   e. A three-fold increase in proportional mortality due to cancer of the rectum in the full study population, but associated with no specific exposure group. There is a suggestion of an association with all jobs including any type of abrasive grinding.

3. Despite the need for further study, the present findings support the necessity of stringent measures to control exposures to cutting fluid and abrasive aerosols in this industry as a prudent public health measure. Design, installation and maintenance of local exhaust ventilation systems for grinding and machining operations need to be improved to capture

airborne particulates, mists and organic volatiles. Until such time as scientific investigations are able to distinguish risks associated with specific metal working processes, chemical exposures or levels of exposure, there is a need to reduce all cutting fluid mists and related aerosols to the lowest feasible level. Wheel dressing procedures (shaping grinding surfaces with diamond), which are typically done at the grinding machine, without lubricant, deserve special attention because of the unique potential for particulate abrasive exposure.

In addition to ventilation, other methods should be employed to reduce the generation and exposure to known and suspected carcinogens in cutting fluid environments. Splash guards should be installed whenever possible and maintained in good repair. The force and direction of coolant streams should be adjusted frequently to reduce mist generation. The speed and pressure at metal-tool interfaces should be maintained to reduce frictional heat and particle dispersion. A priority should also be placed on maintaining low levels of known and suspected carcinogens in circulating cutting fluids. Conditions for nitrosamine formation should be eliminated. Biocide use should be reduced in favor of other means to curtail microbiologic growth.

There is a particular need to reduce skin contact with cutting fluids, especially soluble and synthetic fluids, because of the ability of nitrosamines to penetrate human skin[13,14]. Biologic monitoring of workers exposed to cutting fluids deserves careful consideration, although such monitoring should not be construed as a substitute for primary methods of environmental control. Nitrosamines have been identified in the urine of exposed workers but dose-response and exposure-response relationships have not been determined. It might also be informative to test the urine of workers exposed to cutting fluids for mutagenicity, although clear interpretation of the test results, even if positive, might be difficult.

4. While further investigation is required to confirm the risk of lung cancer from heat treat or forge exposures, a preventive approach calls for more careful control of emissions from these sources. Both the major point sources (e.g. quench tanks and forge presses) and secondary sources (fugitive emissions from cooling forgings, heat treat conveyors or other points) need to be inventoried and controlled.

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Table 1

Job Categories Derived from
Production Activities of Departments

Job Category	Job Category No.
Unknown	00
Forge	01
Hot/Cold Heading	02
Machining (Oil CF)	03
Machining (Water CF)	04
Machining (Unknown or Mixed CF)	05
Machining (Second, OIL CF)	06
Machining, Grey Iron	07
Filing	08
Heat Treat, Rings	09
Heat Treat, Balls	10
Grinding (Oil CF)	11
Grinding (Water CF)	12
Grinding (Unknown or Mixed CF)	13
Grinding, Inspection	14
Plating	15
Lapping	16
Honing	17
Polishing	18
Stamping; Punching	19
Laboratory: Chem., Metall.	20
Wheelmaking	21
Tool Grinding	22
Assembly; Gaging; Pressing; Salvage	23
Packing	24
Shipping; Inventory; Storage; Mat. CtL; Misc.	25
Inspection, Balls, Final	26
Skilled Trades, Electricians (Maintenance)	27
Skilled Trades, Machinists (Maintenance)	28
Skilled Trades, Non-Elect, - Mach.; (Maintenace) (Includes Janitors)	29
Mill Supply	30
Production Control	31
Tumbling	32
Cleaner, Bearing Parts	33
Steel Handler	34
Tool & Die Makers	50

Table 2

A Sample of Departments with Activity and Assigned Job Category[1]

Department Name	Activity	Detail[2]	Major[2] Exposure	Assigned Job Category (No.)
H8	Machining	ASM	MOCF	03
P2	Machining	CPPD	WBCF	04
R3	Machining	ASM, Single	MOCF	03
05	Machining	2nd,TNC	MXCF	06
14	Grinding	OD, Bore	WBCF	12
A14	Grinding	OD, Bore, Face	MOCF, WBCF	11
L14	Grinding	OD, Bore, Face	WBCF	12
A16	Grinding	Bore	MXCF	13
H19	Grinding	Race	MXCF	13
M19	Grinding	Race	MXCF	13
M20	Grinding	Race	MXCF	13
S23	Grinding	Rough	MXCF	13
A25	Assembly			23
M25	Assembly			23
34	Tumbling		ABR	32
35	Heat Treat		PP	10
H40	Heat Treat		PP	09
A45	Inspection	Match & Grind	MXCF	14
M46	Inspection	OD, Face	MXCF	14
A48	Inspection		MXCF	14
D52	Inspection	Final		26
F52	Inspection	Final		26
L53	Packing			24
61	Tool & Die	Mach. Grind	ABR,PP,MOCF	50
N62	Maintenance	Non-E,-M		29
68	PDN Control		(MXCF)	31
M68	PDN Control		(MXCF)	31
N70	Maintenance	Mach.	(MOCF)	28
N71	Tool Grinders		ABR,DST	22
73	Steel Handlers		(MOCF)	34

1. Sample of 194 Departments known to have existed.

2. Explanations:
ASM - Multi-spindle Lathes, Automatic
CPPD - Precision Products (Small Bearings)
TNC - Numerically Controlled Lathes
OD - Outside Diameter
MOCF - Mineral Oil Cutting Fluids
WBCF - Water-Based Cutting Fluids
MXCF - Mixed Cutting Fluids
ABR - Abrasives
PP - Pyrolysis Products
DST - Dusts

Table 3

Exposure Measures and Component Job Categories for Case-Control Analysis

	Job Categories	
Process	Exposed Group	Comparison Group
Machining	3-6	16-21, 23-27, 30, 32-33
Grinding	11-14	16-21, 23-28, 30, 32-33
Tool and Die	22,50	16-21, 23-27, 30, 32-33
Forge and Heat Treat	1,2,9,10	16-21, 23-28, 30,32-33
Skilled Trades	27-28	14, 16-17, 19-20, 23-26, 30, 33
Maintenance	29	16-21, 23-28, 30, 32-33
Assembly and Packaging	23,24	16-21, 25-28, 30, 32-33
Type of Exposure		
Mineral Oil CF, Comprehensive	3,5-8,11,13,31,34	16-21, 23-27, 30, 32-33
Mineral Oil CF, Restrictive	3,6,11	16-21, 23-27, 30, 32-33
Water-Based CF, Comprehensive	4,12-14,31	16-21, 23-28, 30, 32-33
Water-Based CF, Restrictive	4,12	16-21, 23-28, 30, 32-33
Mineral Oil in Grinding	11,13,14,31	16-21, 23-27, 30, 32-33
Inorganic Dusts and Fibers	1,9,15,18,21,22,32,33	14, 16-17, 19-20, 23-28, 30, 34

Table 4

Outcomes and Component Causes of Death for Case-Control Analyses

Outcome	Cause of Death (coded according to ICD 8)	
	Cases	Controls
Cancer of GI Tract	150-159	1-139, 170-519, 580-999
Cancer of Stomach	151	1-139, 170-519, 580-999
Cancer of Colon	153	1-139, 170-519, 580-999
Cancer of Rectum	154	1-139, 170-519, 580-999
Non-Malignant Digestive Disease	520-577	1-139, 170-519, 580-999
Cancer of Lung	162	1-139, 170-999
Cancer of Upper Resp. Tract	140-149, 160, 161, 163	1-139, 170-999
Non-Malignant Respiratory Disease	460-519	1-149, 170-459, 520-999
Chronic Obstructive Lung Disease	519.3	1.0-149.9, 170.0-438.9, 520.0-999.9
Cancer of Lymphopoietic System	200-209	1-139, 240-999
Cancer of Lymphopoietic: Other	202-208	1-139, 240-999
Chronic Alcoholism	303.2	1.0-139.0, 170.0-302.0, 305.0-999.9
Stroke	430-438	1-139, 170-429, 439-999

Table 5

Study Population: Race and Sex Distribution

	Study Population	Study Population with Cause of Death	%	Study Population with Cause of Death and Job History	%
White Men	616	610	99	565	92
White Women	124	123	99	116	94
Total	740	733	99	681	92
Non-White Men	20	20	100	20	100
Non-White Women	2	2	100	1	50
Total	22	22	100	21	95
Unknown	6	0	0	0	0
TOTAL	768	755	98	702	91

Table 6

Distribution of Number of Points in Time Where Department is Known, and Number of Distinct Job Categories Over Study Population[1]

		No. of Department/Time Points in In Worker Job History							
		1	2	3	4	5	6	7	Total
No. of Cases		79	179	230	182	29	2	1	702
Cum. %		100.	88.7	63.2	30.4	4.5	.4	.2	
No. of Distinct Job Categories in a Worker's Job History	1	79	130	150	93	15	0	0	467
	2		49	70	75	14	1	0	209
	3			10	13	0	1	0	24
	4				1	0	0	1	2
	5					0	0	0	0

1. Only those with cause of death and work history; Job Categories defined in Table 1.

Table 7

Distribution of Study Population Over Major Process Categories[1]

		Machining	Grinding	Tool & Die	Forge & Heat Treat	Skilled Trades	Maint-enance	Assembly and Packaging	All[2] Other	Total
White Men	n	68	194	68	10	8	21	71	124	565
	%	12.0	34.3	12.0	1.7	1.4	3.7	12.5	21.9	100
White Women	n	2	38	6	0	0	2	25	43	116
	%	1.7	32.7	5.2	0	0	1.7	21.6	37.1	100
Total	n	70	232	74	10	8	23	96	167	681
	%	10.2	34.1	10.9	1.4	1.2	3.4	14.1	24.5	100

1. For those with cause of death and work history.

2. Includes: Plating, polishing, inspection, mill supply, etc.

Table 8

Distribution of Study Population in Cutting Fluid Exposure Groups[1]

	Mineral Oil Cutting Fluids		Water-Based Cutting Fluid		Mineral Oil in Grinding
	Compreh.	Restrict	Compreh.	Restrict.	
White Men					
Exposed Group	200	65	210	67	143
Comparison Group	159	159	161	161	159
White Women					
Exposed Group	25	3	40	4	37
Comparison Group	58	58	58	58	58

1. See Table 3 for composition of exposure groups; these exposure groups are not mutually exclusive.

Table 9

Distribution of Study Population in Selected Job Categories Involving Cutting Fluid Exposures[1]

Job Category	White Men	White Women
3 - Machining, Oil	46	0
4 - Machining, Water	1	0
5 - Machining, Mixed	2	0
6 - Machining, Oil (Second)	19	2
7 - Machining, Grey Iron	13	0
8 - Filing (Ball Shop)	5	0
11 - Grinding, Oil	0	1
12 - Grinding, Water	66	4
13 - Grinding, Mixed	96	19
14 - Grinding, Inspection	32	14
31 - Production Control	15	3
34 - Steel Handling	4	0

1. Based on last department worked in at 15 years prior to death, or if unknown, first known department.

Table 10

Relation of Ages of Hire, Termination and Death, Duration and Latency to Origin of Birth, in Study Population (Means)[1]

	Birth	n	%	Age at: Hire	Term.	Death	Duration[2] (mos.)	Latency[3] (mos.)
White Men								
	U.S.	409	67.2	33.7	58.8	64.0	300.5	363.6
	Central Europe	100	16.4	41.6	61.8	71.1	242.8	354.1
	Other[4]	100	16.4	41.3	63.8	75.5	269.8	409.9
	Total	609	100.					
White Women								
	U.S.	101	82.1	38.4	60.0	67.9	259.5	354.8
	Central Europe	14	11.4	43.3	64.3	75.8	251.8	389.3
	Other	8	6.5	37.6	58.9	70.2	256.0	391.5
	Total	123	100.					

1. For those with known cause of death; 1 case missing origin of birth information.

2 Interval between date of hire or first known department and date of termination or last known department.

3. Interval between date of hire or first known department and date of death.

4. Major constituents of Other are: Canada, Italy and Germany.

Table 11

Association of Origin of Birth with Major Process Groups, in White Men and Women[1]

	Machining		Grinding		Tool & Die		Forge and Heat Treat		Skilled Trades		Maintenance		Assembly & Packaging		Total	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
White Men																
U.S.	49	72.1	131	67.5	57	83.8	4	40.0	7	87.5	15	71.4	45	64.3	392	69.5
Central Europe	6	8.8	30	15.5	2	2.9	6	60.0	1	12.5	2	9.5	10	14.3	81	14.4
Other	13	19.1	33	17.0	9	13.2					4	19.0	15	21.4	91	16.1
Total[2]	68	100.	194	100.	68	100.	10	100.	8	100.	21	100.	70	100.	564	100.
White Women																
U.S.	1	50.	34	89.5	6	100.					2	100.	18	71.	96	81.8
Central Europe			3	7.9									5	20.	12	10.3
Other	1	50.	1	2.6									2	8.	8	6.9
Total	2	100.	38	100.	6	100.					2	100.	25	100.	116	100.

1. Excludes All Other processes: n=125 men, 48 women.

2. 1 case missing origin of birth information.

Table 12

Distribution and Mean for Ages of Hire, Termination and Death in Major Process Categories for White Men[1]

		Machining		Grinding		Tool & Die		Forge & and Heat		Skilled Trades		Maint-enance		Assembly and Packaging		Total	
		n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Age at Hire	15-	24	35.	66	34.	20	29.	1	10.	0	0.	6	29.	26	37.	178	32.
	30-	40	58.	112	58.	41	60.	6	60.	6	75.	11	52.	38	54.	330	58.
	50+	4	6.	16	8.	7	0.	3	30.	2	25.	4	19.	7	10.	57	10.
Age at Term-ination	25-	16	24.	36	19.	10	15.	1	10.	1	13.	5	24.	12	17.	110	20.
	55-	22	32.	47	24.	10	15.	1	10.	3	38.	4	19.	18	25.	131	23.
	60-	20	29.	79	41.	23	34.	5	50.	1	13.	3	14.	30	42.	209	37.
	65+	10	15.	32	17.	25	37.	3	30.	3	38.	9	43.	11	16.	115	20.
Age of Death	30-	29	43.	47	24.	15	22.	2	20.	3	38.	7	33.	17	24.	160	28.
	60-	28	41.	117	60.	19	41.	3	30.	4	50.	5	24.	33	47.	263	47.
	75+	11	16.	30	16.	34	50.	5	50.	1	13.	9	43.	21	30.	142	25.
Total		68		194		68		10		8		21		70		565	
Mean Age at Hire		34.1		34.3		36.8		39.7		45.7		40.0		35.5		35.9	
Mean Age at Term.		58.8		59.9		61.7		61.5		60.9		60.8		59.7		59.8	
Mean Age at Death		63.5		65.4		70.3		71.1		64.9		69.7		67.8		66.4	

1. Excludes All Other processes: n=125.

Table 13

Standardized Proportional Mortality Ratios for Selected Causes of Death in White Men with 10 or more Years Duration of Employment

	OBS./EXP.	PMR	95% CI	X^2	p(1)
ALL MALIGNANT NEOPLASMS	157/135.20	1.16	1.01,1.33	4.56	.03
Cancer of Buccal Cavity and Pharynx	2/4.09	0.49	0.13,1.89	1.07	—
Cancer of Digestive Organs and Peritoneum	55/35.32	1.56	1.21,2.01	11.66	.001
Cancer of Esophagus	6/3.19	1.88	0.86,4.12	2.48	—
Cancer of Stomach	11/5.55	1.98	1.11,3.53	5.42	.02
Cancer of Large Intestine	15/12.82	1.17	0.71,1.93	0.38	—
Cancer of Rectum	11/3.62	3.04	1.74,5.32	15.14	.003
Cancer of Liver	3/2.05	1.46	0.48,4.49	0.44	—
Cancer of Pancreas	8/7.35	1.09	0.55,2.17	0.06	—
Cancer of Respiratory System	60/49.61	1.21	0.95,1.54	2.40	—
Cancer of Lung - Primary and Secondary	59/47.29	1.25	0.98,1.59	3.18	.08
Cancer of Skin	4/2.11	1.90	0.72,4.96	1.70	--
Mental, Psychoneurotic and Personality Disorders[2]	13/3.18	4.09	2.48,6.74	30.53	.001
All Diseases of Nervous System and Sense Organs	7/4.48	1.56	0.75,3.25	1.42	—
All Diseases of Circulatory System	323/331.83	0.97	0.90,1.05	0.52	—
All Vascular Lesions of CNS	60/44.51	1.35	1.06,1.71	5.92	.01
All Respiratory Diseases	40/43.15	0.93	0.69,1.25	0.25	—
All Pneumonia	7/14.86	0.47	0.23,0.98	4.28	.04
Emphysema	8/9.98	0.80	0.40,1.59	0.40	—
All Diseases of Digestive System	24/26.14	0.92	0.62,1.36	0.18	—
All Gastric and Duodenal Ulcer	0/2.71	0.00	0.00,0.00	2.73	—
Cirrhosis of Liver	18/14.12	1.27	0.81,2.00	1.10	—
All Accidents	12/20.95	0.57	0.33,0.99	4.04	.05
Suicide	8/8.40	0.95	0.48,1.89	0.02	—
All Other Causes	26/36.67				
Total Deaths	610				

1. Based on 2-tailed test of significance (Mantel-Haenszel X (Sq) used unless EXP less than 5.0; then Poisson-P used).

2. 12/13 deaths coded as "chronic alcoholism".

Table 14

Standardized Proportional Mortality Ratios, for Selected Causes of Death in White Women, with 10 or More Years Duration of Employment

	OBS./EXP.	PMR	95% CI	X^2	p(1)
ALL MALIGNANT NEOPLASMS	36/33.73	1.07	0.81,1.40	0.22	—
Cancer of Buccal Cavity and Pharynx	0/0.51	0.00	0.00,0.00	0.51	—
Cancer of Digestive Organs and Peritoneum	12/8.99	1.34	0.78,2.30	0.09	—
Cancer of Esophagus	0/0.35	0.00	0.00,0.00	0.35	—
Cancer of Stomach	3/1.00	2.99	1.02,8.75	4.02	.16
Cancer of Large Intestine	6/4.13	1.45	0.66,3.17	0.87	—
Cancer of Rectum	1/0.80	1.25	0.18,8.79	0.05	—
All Cancer of Liver	1/0.70	1.43	0.21,10.02	0.13	—
Cancer of Pancreas	0/1.79	0.00	0.00,0.00	1.82	—
Cancer of Respiratory System	5/4.83	1.03	0.44,2.43	0.01	—
All Cancer of Lung - Primary and Secondary	5/4.67	1.07	0.46,2.51	0.02	—
Cancer of Skin	1/0.41	2.44	0.37,16.17	0.85	—
Cancer of Breast	9/6.84	1.32	0.70,2.46	0.73	—
Cancer of All Genital Organs	3/4.60	0.65	0.22,1.95	0.58	—
Cancer of Other Lymphopoietic Tissue	3/1.06	2.83	0.96,8.30	3.57	.18
Mental, Psychoneurotic and Personality Disorders	1/0.42	2.37	0.36,15.80	0.80	—
All Diseases of Nervous System and Sense Organs	0/1.17	0.00	0.00,0.00	1.18	—
All Diseases of Circulatory System	64/63.36	1.01	0.86,1.19	0.01	—
All Vascular Lesions of CNS	11/12.97	0.85	0.49,1.48	0.34	—
All Respiratory Diseases	4/6.18	0.65	0.25,1.67	0.81	—
All Pneumonia	3/2.65	1.13	0.37,3.46	0.05	—
All Diseases of Digestive System	4/4.82	0.83	0.32,2.16	0.15	—
All Gastric and Duodenal Ulcer	0/0.40	0.00	0.00,0.00	0.41	—
Cirrhosis of Liver	2/2.08	0.96	0.25,3.78	0.00	—
All Accidents	6/3.03	1.98	0.91,4.29	3.00	—
All Other Causes	8/10.29				
Total Deaths	123				

1. Based on 2-tailed test of significance (Mantel-Haenszel X (Sq) used unless EXP less than 5.0; then Poisson-P used).

Table 15

Standardized Proportional Mortality Ratios for Selected Causes of Death in Sub-Populations of White Men

Underlying Cause of Death	All White Men n=610			Cen. Eur. Birth n=100			Non-Cen. Eur. Birth n=509		
Cancer	Obs/Exp	PMR	p[3]	Obs/Exp	PMR	p	Obs/Exp	PMR	p
All G.I.	55/35.3	1.56	.001	14/5.5	2.54	.001	41/29.8	1.38	.04
Stomach	11/5.6	1.98	.02	2/.89	2.25	—	9/4.6	1.94	.09
Colon	15/12.8	1.17	—	5/2.05	2.44	.11	10/10.8	.93	—
Rectum	11/3.62	3.04	.003	2/.58	3.47	—	9/3.0	2.96	.007
Lung	59/47.3	1.25	.07	8/6.6	1.22	—	51/40.6	1.26	.10
Nonmalignant Disease									
NMRD[1]	40/43.2	.93	—	6/7.6	.79	—	34/35.5	.96	—
"Mental" Disorders[2]	13/3.2	4.09	.001	4/.45	8.96	.002	9/2.72	3.30	.004
Stroke	60/44.5	1.35	.016	8/8.7	.91	—	52/35.7	1.46	.004

1. All nonmalignant respiratory disease (ICD 8: 460-519)

2. Mental, psychoneurotic and personality disorders - primarily chronic alcoholism (ICD 8: 290-317)

3. Based on 2-tailed significance test; Poisson P-value computed for Exp. less than 5.

Table 16

Standardized proportional Mortality Ratios for Selected Causes of Death in Sub-Populations of White Men

Underlying Cause of Death	Grinding n=194			Grinding (Latency > 30 yr.) n=98[4]			Machining n=68			Tool & Die n=68		
Cancer	Obs/Exp	PMR	p[3]	Obs/Exp	PMR	p	Obs/Exp	PMR	p	Obs/Exp	PMR	p
All G.I.	23/11.8	1.96	.001	10/5.9	1.69	.15	3/3.9	.75	—	8/3.8	2.11	.08
Stomach	7/1.8	3.88	.005	4/.91	4.39	.03	0/.62	.00	—	0/.63	.00	—
Colon	6/4.21	1.43	—	3/2.14	1.40	—	2/1.4	1.40	—	2/1.4	1.42	—
Rectum	3/1.9	2.52	.10	1/.60	1.65	—	1/.40	2.50	—	2/.41	4.95	.12
Lung	21/16.8	1.25	—	6/8.2	.73	—	6/5.7	1.05	—	4/4.5	.87	—
Nonmalignant Disease												
NMRD[1]	16/13.4	1.19	—	11/7.1	1.54	—	1/4.4	.23	—	5/5.1	.97	—
"Mental" Disorders[2]	7/1.05	6.66	.001	2/.48	4.19	.16	1/.41	2.40	—	1/.3	3.38	—
Stroke	16/12.4	1.29	—	9/6.8	1.33	—	5/4.2	1.19	—	6/5.9	1.00	—

1. All nonmalignant respiratory disease (ICD 8: 460-519)

2. Mental, psychoneurotic and personality disorders - primarily chronic alcoholism (ICD 8: 290-317).

3. Based on 2-tailed significance test; Poisson P-value computed for Exp. less than 5.

4. Latency (employment): interval from date of hire to date of death.

Table 17

Standardized Proportional Mortality Ratios for Selected Causes of Death in Sub-Populations of White Women

Underlying Cause of Death	All White Women n=123			Grinding n=38			Assembly & Packaging n=25		
Cancer	Obs/Exp	PMR	p[3]	Obs/Exp	PMR	p	Obs/Exp	PMR	p
All GI	12/8.9	1.34	—	3/3.0	.99	—	3/1.8	1.68	—
Stomach	3/1.0	3.00	.16	1/.32	3.10	—	1/.20	5.06	—
Colon	6/4.1	1.45	—	0/1.4	.00	—	1/.83	1.20	—
Rectum	1/.80	1.25	—	0/.26	.00	—	1/.16	6.45	—
Lung	5/4.7	1.07	—	5/1.8	2.82	.07	0/.96	.00	—
Other Lympho-poietic	3/1.1	2.83	.18	2/.38	5.28	.11	0/.22	.00	—
Nonmalignant Disease									
NMRD[1]	4/6.2	.65	—	0/1.9	.00	—	2/1.28	1.57	—
"Mental" Disorders[2]	1/.42	2.37	—	0/.13	.00	—	1/.09	10.70	—
Stroke	11/12.9	.85	—	3/3.5	.87	—	3/2.7	1.12	—

1. All nonmalignant respiratory disease (ICD 8: 460-519).

2. Mental, psychoneurotic and personality disorders - primarily chronic alcoholism (ICD 8: 290-317).

3. Based on 2-tailed significance test; Poisson P-value computed for Exp. less than 5.

Table 18 a

Association of Cancer of Stomach with Grinding Exposure in White Men

		Underlying Cause of Death		
		Stomach Cancer	Other[1]	Total
Comparison Exposure	n	1	124	125
	%	0.8	99.2	100.
Grinding Exposure	n	7	141	148
	%	4.7	95.3	100.

Crude Odds Ratio = 6.21, p=.055[2]
Age Adjusted Odds Ratio = 6.08
Mantel-Haenszel Odds Ratio = 5.31, p = .14

Table 18 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	86	19	19	32	55	38	125
	%	69.4	15.3	15.3	25.6	44.0	30.4	100.
Grinding Exposure	n	97	22	29	35	85	28	148
	%	65.5	14.9	19.6	23.6	57.4	18.9	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. 1 case missing origin of birth.

Table 19 a

Association of Cancer of Stomach with Water-Based Cutting Fluids, Comprehensive, in White Men

		Underlying Cause of Death		
		Cancer	Other[1]	Total
Comparison Exposure	n	1	124	125
	%	0.8	99.2	100.
Cutting Fluid Exposure	n	8	153	161
	%	5.0	95.0	100.

Odds Ratio = 6.48, p=.043[2]
Age Adjusted Odds Ratio = 6.36

Table 19 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	86	19	19	32	55	38	125
	%	69.4	15.3	15.3	25.6	44.0	30.4	100.
Cutting Fluid Exposure	n	105	25	31	39	90	32	161
	%	65.2	15.5	19.3	24.2	55.9	19.9	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. 1 case missing origin of birth.

Table 20 a

Association of Cancer of Stomach with Water-Based Cutting Fluids, Restrictive, in White Men

		Underlying Cause of Death		
		Cancer	Other[1]	Total
Comparison Exposure	n	1	124	125
	%	0.8	99.2	100.
Cutting Fluid Exposure	n	2	53	55
	%	3.6	96.4	100.

Crude Odds Ratio = 4.68, p = .22[2]
Age Adjusted Odds Ratio = 4.59

Table 20 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	86	19	19	32	55	38	125
	%	69.4	15.3	15.3	25.6	44.0	30.4	100.
Cutting Fluid Exposure	n	32	10	13	12	32	11	55
	%	58.2	18.2	23.6	21.8	58.2	20.0	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. 1 case missing origin of birth.

Table 21 a

Association of Cancer of Stomach with Grinding in Mineral Oil Cutting Fluids, in White Men

		Underlying Cause of Death		
		Stomach Cancer	Other[1]	Total
Comparison Exposure	n	1	123	106
	%	0.8	99.2	100.
Grinding Exposure	n	6	100	106
	%	5.7	94.3	100.

Crude Odds Ratio = 7.38, p=.038
Age Adjusted Odds Ratio = 7.25

Table 21 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	85	19	19	32	54	38	124
	%	69.1	15.4	15.4	25.8	43.5	30.6	100.
Grinding Exposure	n	73	15	18	27	58	21	106
	%	68.9	14.2	17.0	25.5	54.7	19.8	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. 1 case missing origin of birth.

Table 22 a

Association of Lung Cancer with Forge and Heat Treat Exposure, in White Men

		Underlying Cause of Death		
		Lung Cancer	Other[1]	Total
Comparison Exposure	n	18	131	149
	%	12.0	87.9	100.
Forge/Heat Treat Exposure	n	3	6	9
	%	33.3	66.6	100.

Crude Odds Ratio = 3.6, p=.10

Table 22 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	104	22	22	43	65	41	149
	%	70.3	14.9	14.9	28.9	43.6	27.5	100.
Forge/Heat Treat Exposure	n	4	5	0	2	3	4	9
	%	44.4	55.6	0	22.2	33.3	44.4	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. 1 case missing origin of birth.

Table 23 a

Association of Lung Cancer with Grinding Exposure in White Women

		Underlying Cause of Death		
		Lung Cancer	Other[1]	Total
Comparison Exposure	n	0	53	53
	%	0.0	100.	100.
Grinding Exposure	n	5	30	35
	%	14.3	85.7	100.

Crude Odds Ratio = 19.3, p=.008[2]

Table 23 b

Association of Potential Confounders with Exposure

		Origin of Birth			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	44	6	3	13	27	13	53
	%	83.0	11.3	5.7	24.5	50.9	24.5	100.
Grinding Exposure	n	31	3	1	5	25	5	35
	%	88.6	8.6	2.9	14.3	71.4	14.3	100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability; 0.5 added to each cell for calculation of odds ratio, due to zero value.

Table 24 a

Association of Lung Cancer with Water-Based vs. Mineral Oil Cutting Fluids in Women in Grinding

		Underlying Cause of Death		
		Lung Cancer	Other[1]	Total
Oil CF[2] Exposure	n	3	31	34
	%	8.8	91.2	100.
Water CF Exposure	n	2	1	3
	%	66.6	33.3	100.

Crude Odds Ratio 20.3, p=.04[2]

Table 24 b

Association of Potential Confounders with Exposure

		Origin of Birth			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Oil CF Exposure	n	30	3	1	6	22	6	34
	%	88.2	8.8	2.9	17.6	64.7	17.6	100.
Water CF Exposure	n	3	0	0	0	3	0	3
	%	100.				100.		100.

1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Oil CF - Mineral Oil in Grinding; Water CF - Water-Based Cutting Fluid, Restrictive, limited to grinding jobs only.

3. Fisher Exact Probability.

Table 25 a

Association of "Chronic Alcoholism" Deaths with Grinding Exposure in White Men

		Underlying Cause of Death		
		Chronic Alcholism	Other[1]	Total
Comparison Exposure	n	1	130	131
	%	0.7	99.3	100.
Grinding Exposure	n	6	142	148
	%	4.0	96.0	100.

Crude Odds Ratio = 5.5, p=.082

Table 25 b

Association of Potential Confounders with Exposure

		Origin of Birth[3]			Age at Death			Total
		U.S.	C.Eur.	Other	<60	60-74	75+	
Comparison Exposure	n	91	19	20	36	57	38	131
	%	70.0	14.6	15.4	27.5	43.4	29.0	100.
Grinding Exposure	n	98	21	29	36	85	27	148
	%	66.2	14.2	19.6	24.3	51.4	18.2	100.

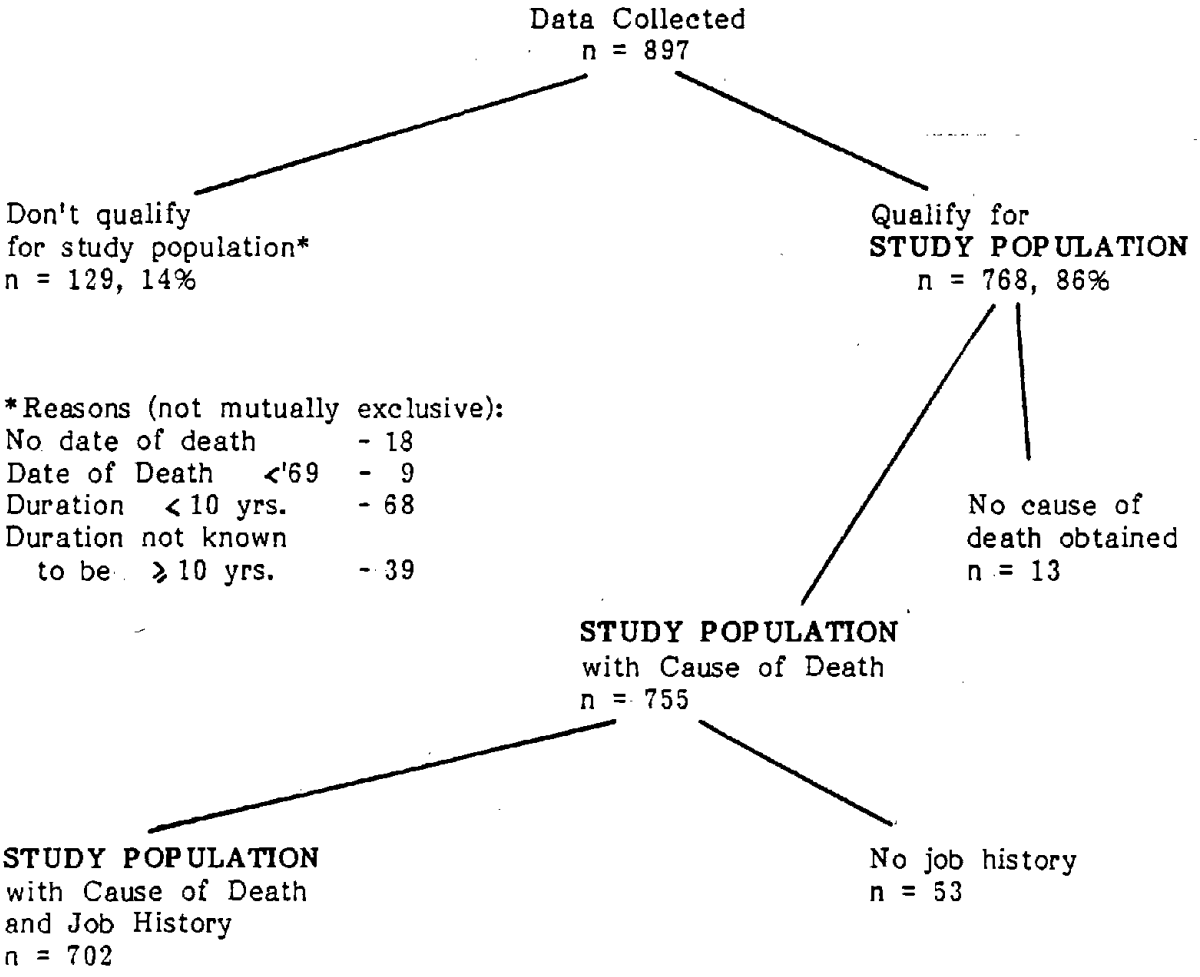
1. Other Causes of Death exclude specific causes also associated with the exposure; Comparison Exposures exclude exposures also associated with cause of death of interest; see Methods.

2. Fisher Exact Probability.

3. cases missing origin of birth.

Figure 1

Study Population Derivation



APPENDIX I

Adjustment of Odds Ratio for Age at Death Confounding in Unmatched Case-Control Analysis

The following adjustment procedure is intended for use when there is confounding in a case-control analysis of proportional mortality and the numbers are too small for meaningful stratification. The proportion of cases vs. controls in the comparison group of a case-control study can be adjusted to reflect the age at death distribution of the exposed group, using national proportional mortality reference rates, provided 2 assumptions are reasonably satisfied: 1) the study population is a stratified sampling from the reference population (by age, race, etc.) except for some overall selection feature, such as the healthy worker effect, and 2) the overall selection has operated uniformly across the age at death strata. In this investigation, the selection is expected to involve 2 mechanisms: 1) relatively healthier workers are hired and 2) relatively heathier workers remain in employment long enough to satisfy the 10 year service requirement of this study. For these reasons, the study population at risk would be expected to be generally healthier at all ages of death, compared with the reference population.

Suppose the case-control study array is:

	Case	Control
Exposed	a	b
Comparison	c	d

The odds ratio is given by ad/bc. If the c and d are to be adjusted to yield c', d' (where c'+d' = c+d, the total comparison group remaining the same size) then the adjusted odds ratio is ad'/bc', and the odds ratio correction factor is

$$\frac{d'/c'}{d/c} = \frac{d'c}{c'd} = A_{or}.$$

If there have been no cause-of-death exclusions, then the expected number of cases in the comparison group, based on proportional mortality rates of the reference population (Table A-1), is given by

$$E(c) = \sum n_i r_i$$

where r_i is the cause- and age-specific proportional mortality rate and n_i the number of the comparison group in the i^{th} age stratum; and, $\sum n_i = c + d$.

Let $c = k\,E(c) = k \sum n_i r_i$, where k is the realization of a random variable representing both the sampling fluctuations in the number of observed cases in the comparison group, and the effects of selection with respect to the reference population.

Adjust the age distribution of the comparison group to match the exposure group: i.e. create m_i such that $m_i = e\, l_i$, where e is a constant, and l_i are the age strata sizes for the exposed, and thus, $e = \frac{\sum m_i}{\sum l_i} = \frac{\sum n_i}{\sum l_i}$, where:

	Age Strata	Total
Exposed	$l_1, l_2 \ldots l_p$	a + b
Comparison	$n_1, n_2, \ldots n_p$	c + d
Adj. Comparison	$m_1, m_2, \ldots m_p$	c + d

Then the expected number of cases in the adjusted comparison group is:

$$\mathcal{E}(c') = \sum m_i \lambda_i$$

and the adjusted observed value of c is $c' = k \sum m_i \lambda_i$, where k has the same value as observed in $c = k \sum n_i \lambda_i$. Therefore,

$$k = \frac{c}{\sum n_i \lambda_i} \quad , \quad c' = c \frac{\sum m_i \lambda_i}{\sum n_i \lambda_i} \; .$$

Strictly speaking, because k is determined both by the selection with respect to the underlying population and by random sampling effects, the estimate of c' is biased in a manner that cannot be described without knowing the underlying selection. However, for small adjustments (i.e. less than 50%), this bias will be negligible.

The resulting adjustment factor is:

$$A_{or} = \frac{d'c}{c'd} = \frac{(c+d-c')c}{dc'} = \frac{c\left(c+d-c\left(\frac{\sum m_i \lambda_i}{\sum n_i \lambda_i}\right)\right)}{cd\,\frac{\sum m_i \lambda_i}{\sum n_i \lambda_i}} = \frac{c+d}{d}\left(\frac{\sum n_i \lambda_i}{\sum m_i \lambda_i}\right) - \frac{c}{d}$$

When exclusions are made based on determinants (exposures) there is no effect on this procedure. When exclusions are made based on outcomes i.e. certain causes of death excluded from the cases and controls, then the adjustment procedure must use revised proportional mortality rates corrected by a factor

$$\left(1 - \sum_{j \in E} \lambda_{ij}\right)^{-1}$$

where r_{ij} is the proportional mortality of age stratum i due to cause of death j and E is set of causes excluded. This correction simply insures that

$$\sum_{j \in S} \lambda^*_{ij} = 1$$

where S is the set of causes of death included in the case and control categories and where

$$\lambda^*_{ij} = \lambda_{ij}\left(1 - \sum_{j \in E} r_{ij}\right)^{-1} .$$

Table A-1

Proportional Mortality Rates for GI and Lung Cancers in U.S. White Men and Women[1]

	Cancer							
	Stomach		Colon		Rectum		Lung	
Age at Death	1970-74	1975-78	1970-74	1975-78	1970-74	1975-78	1970-74	1975-78
White Men								
45-49	.00740	.00727	.01325	.01596	.00440	.00392	.07022	.08224
50-54	.00874	.00878	.01643	.01896	.00576	.00541	.08276	.09931
55-59	.00938	.00901	.01845	.02101	.00652	.00587	.08975	.10579
60-64	.01000	.00971	.01993	.02329	.00685	.00622	.09056	.10675
65-69	.01039	.00966	.02119	.02463	.00718	.00643	.08293	.09806
70-74	.01035	.00941	.02157	.02461	.00684	.00633	.06670	.08194
75-79	.01010	.00921	.02085	.02306	.00621	.00568	.04602	.05894
80-84	.00924	.00851	.01822	.02090	.00586	.00533	.02794	.03715
85-89	.00685	.00621	.01286	.01520	.00453	.00448	.01243	.01692
White Women								
45-49							.05085	.06921
50-54							.05357	.07458
55-59							.05149	.07272
60-64							.04134	.06262
65-69							.02874	.04623
70-74							.01815	.02884
75-79							.01130	.01721
80-84							.00711	.00998
85-89							.00381	.00504

1. From rates prepared by Richard Monson (ref. 64) and updated through 1978 by Neil Maizlish (see Appendix II).