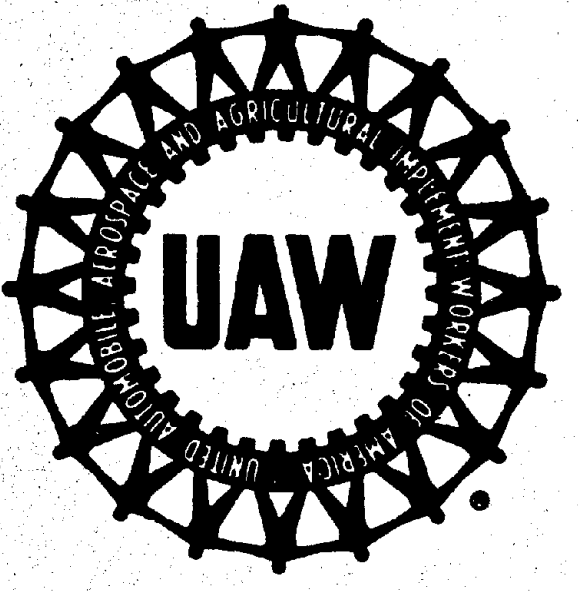
MORTALITY IN WORKERS OF AUTOMOBILE ASSEMBLY PLANTS



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16. Abstract (Limit: 200 words)

A survey of mortality among automobile assembly workers (SIC-3711) was conducted. The cohort consisted of all hourly workers with 10 or more years of service in four Ford assembly factories who died between January 1, 1970 and December 31, 1981. Union records, company pension records, the Michigan death registry, and other records were reviewed. Standardized proportionate mortality ratios were calculated and analyzed according to sex, race, hourly status, latency, and employment duration. A total of 1,808 deaths were identified. Of these, 1,483 satisfied the cohort criteria. A total of 1,217 deaths were white males. An excess proportion of deaths due to lymphoreticulosarcomas, stomach cancer, and nonmalignant diseases of the central nervous system occurred in white males. Among 159 black males, excess mortality due to cancer of the pancreas and colon occurred. Mortality due to stroke was significantly reduced. Lung cancer mortality was elevated only in white males who died 10 to 15 years after initial employment. The authors note that the results contradict previously reported increases in lung cancer mortality among automobile assembly workers. The observed decrease in stroke as a cause of death among black assembly workers should be investigated further.

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TABLE OF CONTENTS

PREFACE

This is the third 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 initiative and assistance of Gary McCalvy, Health and Safety Representative, UAW Local 879 (Twin Cities), and Earl E. Wilson, Health and Safety Representative, UAW Local 249 (Kansas City), and the support of Eli Kovacich, Ford Department, UAW International Union.

We appreciate the cooperation of the Ford Motor Company in jointly defining the data base required for this study, based on Ford employee and pension records, and in providing a file extract. In particular, the efforts of Dr. David S. Sugano, Corporate Epidemiologist, and Stephen J. Kuritz, Biostatistician, were essential for completion of this study.

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

MORTALITY IN WORKERS OF AUTOMOBILE ASSEMBLY PLANTS

SUMMARY

Vehicle assembly plants employ 0.3% of all U.S. workers in a variety of jobs, many of which involve exposure to potentially toxic agents. Besides relatively well characterized exposures associated with welding, painting, and lead soldering, there are many others arising from proprietary formulations such as anti-corrosion treatments, glues, adhesives, sealers and plastics. Previously published findings indicate elevated proportional mortality due to cancer of the lungs and lymphopoietic tissue.

A standardized proportional mortality analysis of 1400 deaths was carried out by the UAW International Union for a population that had been employed at four assembly plants. The major statistically significant findings were: increased proportions of deaths due to cancer of the pancreas and colon and a substantially reduced proportion of deaths due to stroke in black men; an increased proportion of deaths due to lympho/reticulo sarcomas and stomach cancer in white men. Increased proportional mortality due to lung cancer was found only in the group of white men whose time of death was within 10 to 15 years of the date of hire.

BACKGROUND

Motor vehicles, including cars, trucks, buses, farm and construction equipment, are assembled from thousands of manufactured parts in a process of enormous intricacy and scale. Nearly 7 million cars, trucks and buses were manufactured and assembled in the U.S. in 1982, down from a historic high in 1978 of nearly 13 million.[1] This accounted for 15% of the nation's entire steel consumption, 60% of its lead, 45% of its malleable iron, 20% of its zinc, 75% of its natural rubber and 4% of its plastic.[1]

In 1983 more than 835,000[2] hourly and salaried workers were employed by the U.S. motor vehicle industry (SIC 371*). About 397,500[2] of these worked in 90[3] car, truck and bus assembly plants (SIC 3711**). Nearly 300,000 were production workers represented by the International Union, United Automobile Workers.

Assembly plants have generally been considered substantially less hazardous working environments than many of the plants from which they receive manufactured parts and supplies, such as foundries, machining plants, battery and tire plants. Despite this view the UAW decided to undertake a review of assembly plant worker mortality. Three considerations contributed to this decision: First, within the total assembly environment there are departments where large numbers of workers may be exposed to known and potential chemical hazards; second, a recent proportional mortality study revealed excess cancer mortality among a group of assembly plant workers[4,5]; and third, UAW members have expressed concern about assembly plant risks and a preliminary review of death records gathered by one local union suggested possible excesses of cancer. This mortality study was proposed for inclusion in work under a NIOSH contract to the UAW and was subsequently peer-reviewed by NIOSH and approved.

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* SIC Group No. 371, "Motor Vehicles and Motor Vehicle Equipment", includes the manufacture of passenger cars, trucks, buses, truck trailers and a large group of parts and accessories for these vehicles. It does not include farm and construction vehicles, such as tractors and bulldozers, or motorcycles, aircraft and tanks.

** SIC Industry No. 3711, "Motor Vehicles and Passenger Car Bodies", is limited to establishments engaged in assembly of complete passenger cars, trucks and buses.

Overview of Automobile Assembly and Associated Potential Hazards

In automotive assembly plants, completed cars are assembled from a variety of components ranging from single pieces of stamped steel, glass or molded plastic to preassembled engines, transmissions and axles. Assembly is generally organized so that workers at fixed stations install and adjust parts as the partially assembled vehicle passes by them on a moving line. Many phases of this work expose workers to potentially toxic agents.

Body Department. Spot welding (a form of resistance welding) is used to join together many steel parts of vehicle bodies during assembly. Potential exposures include ozone, oxides of nitrogen, iron oxide and other metal fumes. These exposures are common to many welding operations but are thought to be less serious with spot welding than with arc welding which requires higher temperatures and a wider range of materials in welding rods and fluxes.[6] However, levels of copper fume exceeding the permissible exposure limit (PEL) have been found in breathing zone samples for resistance welding operations in a bus assembly plant.[7]

During the fabrication of the car body, materials consisting of resins, foaming agents, solvents and other organic components are applied or injected onto future metal-to-metal seams. Sheet metal arrives precoated with oil films. Subsequent resistance welding of seams causes the release of smoke and fume from the pyrolysis of adjacent organic substances, as well as the release of metal fume. Arc welding of sheet metal and heavier frame components releases welding emissions (fume, ozone, nitrogen oxides) and organic debris from pyrolysis. Measurements of copper and zinc fume associated with MIG arc welding using copper-coated wire in one Ford assembly plant have identified exposures near the PEL for copper, and exceeding the PEL for zinc.[8] In another Ford plant, where metal fume fever symptoms were reported by a group of welders, copper and zinc composite levels exceeded the PEL.[9] Thus unique and poorly characterized welding exposures are possible in the assembly environment.

Welding areas of assembly plants tend to fill with visible haze over the course of a working day unless dilution ventilation is exceptionally good. Over the past 10 years worker exposures have been reduced more through automation and introduction of robots than through control of chemical emissions at the source.[10] Exposures from some of these operations may be particularly important when workers are situated in pits beneath production lines or when doing numerous welds in confined areas.

Metal finishing operations for producing smooth surfaces generate lead dust from grinding on soldered seams. In the past, the oil film sprayed on the metal during solder operations to prevent excessive heating generated an oil smoke. Inorganic lead exposure has been a recognized problem in vehicle assembly since the 1930s when serious outbreaks of poisoning resulted in hospitalizations and deaths.[11] At that time lead solder was applied in large amounts to fill seams between steel parts and workers were required to smooth the soldered seams using powered hand grinders without ventilation or

respiratory protection. Protective measures have been taken so that today lead solder grinders work in enclosed booths with air-supplied respirators. Within the past 10 years maximum blood lead levels of solder grinders have dropped below 50 µg/100ml with average values below 40µg/100ml.[12] Several other jobs near the solder grind booths in the body shop area still involve exposures to measurable amounts of airborne lead dust, but air levels are generally below 50 µg/m^3 and blood levels are generally below 40 µg/100 ml.[12]

Paint Department. Metal preparation prior to painting typically involves the passage of car bodies through a series of heated dip tanks for cleaning, sealing and priming. These tanks are largely enclosed and ventilated. A small group of employees may be exposed to emissions during operation and maintenance activities. Materials of concern include caustic soda, surfactants, glycol ether solvents, lead and chromate pigments. In one such proprietary process, called Bonderite, sodium nitrite is a routine ingredient while aliphatic amines are periodically used for microbiological control in the same sequence of dip tanks.[13] This poses a potential for nitrosamine formation in the heated dip tanks.

At several points in the assembly sequence, materials may be sprayed on sheet metal or other surfaces: adhesives, undercoating, sound deadening compound, primer, anti-corrosion agents and paint. Organic mists, dusts and vapors result. Spray painting of vehicle bodies is typically done by workers with hand held spray guns inside booths fitted with local exhaust ventilation. It has not been standard practice to fit spray painters with respirators.[14] (Spray painting with urethane paints in recent years is an exception). They are therefore potentially exposed to an overspray of paint mist which may include pigments containing heavy metals such as lead and chromium, along with organic binders such as traditional paint and enamel resins and acrylics. Painters are also exposed to potentially high solvent levels. Solvents used in the past include aliphatic and aromatic hydrocarbons and ketones. Solvents in widespread use today include toluene, xylene and various glycol ethers. The amount of exposure is a function of the type of spray (more exposure with air spray, less with electrostatic spray) the size and shape of part, and the adequacy of exhaust ventilation. Area samples in a number of Ford assembly paint departments show levels of glycol ethers in the range 1-5 ppm (TWA) at many work stations, particularly in paint mixing.[15] Sanding between intermediate paint coats generates dusts of primer and paint components.

Trim Department. Large quantities of plastic parts, foam cushions and other components made from synthetic materials (e.g. vinyl sheet, fiber glass) are processed daily by material handlers and the assembly workers involved in "trim" operations. These workers are thereby exposed to organic dusts and vapors and sometimes have direct contact with the solvents, resin monomers, catalysts, plasticizers and other additives contained in these materials. However, these exposures are expected to be small.

Final Assembly. At the end of the production line, pyrolysis products are generated during engine testing from gasoline and diesel exhaust emissions, burning machine oil, and paint on hot surfaces. Carbon monoxide is another exposure of concern in engine test and drive-off areas. Carbon monoxide

measurements in the drive-off area at one Ford plant included 4 out of 21 exceeding 30 ppm (TWA); 2 exceeded the NIOSH-recommended PEL (35 ppm).[16] Repair workers may be exposed to any of the assembly line exposures including welding, lead and paint in performing repairs during the production sequence.

Maintenance and Skilled Trades. Maintenance and skilled trades workers are exposed to many of the assembly exposures as well as special exposures, for example when welding on production line fixtures that are coated with paint or other drippings, when working in areas with accumulated dusts such as lead, or when using solvents. As with maintenance workers generally, asbestos exposure may be experienced by assembly maintenance workers during plant renovations, rearrangements and repairs.

Preliminary Investigation by Local Union Representatives

In 1981 the UAW local union health and safety representative in one of the study plants collected 267 death certificates from the Local Union Death Benefit Fund for the years 1970-1980. His unstandardized calculations indicated that 41% of 59 cancer deaths were from lung cancer, compared with a state proportion of 24% obtained from public health department documents. The local union requested assistance in investigating this potential problem further.

Epidemiologic Studies

Chiazze has conducted the only published general mortality study of vehicle assembly workers. This was a proportional mortality study of 4,215 deaths occurring between 1970-1976 among employees at ten different automotive and farm machinery assembly plants. The study was intended to determine mortality patterns among chromate spray painters. No differences were reported between workers who had ever been spray painters compared with all others.[4] However, there were a number of significant differences between all the assembly workers studied and both the U.S. population as well as comparison populations drawn from communities surrounding each study plant.[5] Among white males, significant excess proportional mortality was found for the following: all cancers (PMR=1.17), respiratory cancers (PMR=1.29), lymphomas (PMR=1.60) and other and unspecified digestive cancers (PMR=1.48). Among non-white males there were significant excesses for: all cancers (PMR=1.18), liver cancer (PMR=4.17) and respiratory cancer (PMR=1.40). Except for the limited information concerning spray painters, the analyses did not take into account length of employment, years between hire and death, or job histories.

There have been several epidemiologic investigations of workers in other industries who are exposed to environments or chemicals like those found within vehicle assembly plants:[17-30]

- Paints and Pigments: In a proportional mortality study Dalager found 21 respiratory cancers among zinc chromate spray painters compared with 11.4 expected (PMR=1.84, $p < .01$)[17] Chiazze's study of assembly plant spray painters described above had insufficient statistical power to detect a 50% elevation in lung cancer in spray painters (power less than .8). No other spray paint studies have been reported, but Morgan published results of a cohort mortality study of 2,633 deaths among 18,000 workers employed for at least a year since 1946 in one of 32 paint or coating production plants.[18] Statistically significant increases in SMRs for colon cancer (SMR=1.38) and rectal cancer (SMR=1.39) were found for the group as a whole. Several other site specific cancer excesses were noted among subgroups exposed to solvents, lacquers, or pigments. However, the value of the study was limited because there was no analysis of subgroups of varying duration of employment or latency. Two additional studies of workers in plants manufacturing chromate pigments for paints have revealed excess lung cancer mortality.[19,20] A substantial number of additional animal and epidemiologic studies have linked cancer to other chemicals potentially used in various paint spray operations - isocyanates, glycidyl ethers, talc, chlorinated hydrocarbon solvents - but none of these are directly applicable to the spray painting environment in auto assembly plants.

- Welding: Stern has recently summarized results from 18 mortality studies which include data for lung cancer among welders.[21] Statistically significant excess relative risk for lung cancer among welders was found in 8 of these. Five others revealed similar but non-significant excess risks. Most of the estimated relative risks in these 13 studies were between 1.25 and 2.50 but in one case-control study the odds ratio was 7.7 with 14 cases among welders. Of the positive studies, the one which most thoroughly took latency and cumulative exposure into account found a strongly positive relation between lung cancer among welders and the length of time since initial exposure.[22]

The etiologic agent responsible for lung cancer among welders has not been clearly established. Beaumont and Weiss point out that many of the positive studies above included shipyard workers and suggest that asbestos exposure may have played a contributing role.[22] Stern suggests that chromate exposures experienced during welding on stainless steel may be the main factor.[21] None of the published welder mortality studies has been able to examine lung cancer risks among welders working only with mild steel (without nickel or chrome) and unexposed to asbestos. NIOSH is currently conducting a study of welders of mild steel. Spot welders such as those in the current study plants have not been previously studied.

- Lead: Multiple acute and chronic toxic effects of inorganic lead exposure have been exhaustively documented, but little attention has been devoted to examining mortality patterns among cohorts of workers exposed to this metal in various industries. Dingwall-Fordyce and Lane followed 425 pensioned battery plant workers and reported an excess of deaths from cerebrovascular accidents among the most heavily exposed workers.[23,24] More recently Cooper and Gaffey studied 7031 men who had been employed in a group of lead smelters and battery plants.[25] 1267 death certificates were obtained and standardized mortality ratios were calculated. There were more deaths than expected from "other hypertensive disease" and "chronic nephritis"

although there was no excess found for cerebrovascular diseases. A significant excess of deaths was found for smelter workers for all cancers (SMR=1.33). Site specific excesses were noted for digestive organ and respiratory system cancers among both battery and smelter workers, but the findings were not statistically significant (SMRs ranging between 1.23-1.50). Inorganic lead exposure has been associated with renal tumors in rats and mice in several studies, but similar associations have not been demonstrated in clinical series or epidemiologic studies among humans.[26]

- Miscellaneous: In a population based case-control study of bladder cancer Silverman found a slightly increased relative risk (OR=1.4, 95% CI=0.9-2.0) for those whose usual employment was in the auto manufacturing industry.[27] Within the auto manufacturing group the highest relative risks were for machinists (OR=2.6), machine operators (OR=1.7) and materials handlers (OR=1.5) but none of these were statistically significant. Three other bladder cancer studies among automobile workers have found no consistent associations.[28,29,30] The only one of these specifically reporting results from an assembly plant observed 2 bladder cancer deaths compared with 2.6 expected.[28] Chiazze found 34 bladder cancer deaths among his population of white deceased automobile assembly workers. This was a significant excess compared with 22 cases predicted from national statistics but not significant when compared to 25 cases predicted from local community statistics.[5]

METHODS

Design

A proportional mortality analysis was c ried out for a population of deceased workers with 10 or more years servic for the Ford Motor Company, who last worked at any of 4 assembly plants. e deaths comprising the study population were identified by first specifying the cohort at risk and then performing vital status determination. (A col t follow-up analysis was not performed because date of birth and race formation was not generally available except on death certificates.)

Study Sites

Three large automobile assembly plants d one smaller adjoining truck assembly plant belonging to the Ford Motor ompany were chosen. Plant choice was based on availability of death ce ificates collected at 2 local unions and access to the state death registry r the remaining 2 plants. At one of the plants a cursory analysis had been arried out by both company and local union representatives. Plants from a ingle employer were selected to facilitate records retrieval.

The plants were located as follows: Minneapolis-St. Paul (Twin Cities), Minnesota (car), 2) Kansas City, Missou (car), and 3) Wayne, Michigan (car and truck). The Minnesota plant prior to 950 was also engaged in glass production. The Kansas City operation made craft in the 1940s and early 1950s. The Michigan plants, unlike the other o, were near numerous other plants of Ford and thus experienced considerab more intra-company transfer of employees.

Study Population: Definit n

The study population was defined as:

> all hourly workers with 10 or mc years service (in any Ford plant) who died between January 1970 and December 31, 1981 and whose last Ford employment w at one of the four assembly plants included in study.

This definition, formulated to allow use of computer-based employer pension records, excludes workers who transfere from any of the study plants to other Ford facilities. It includes employees t nsfering into the study plants and remaining until ending Ford employment, it with no minimum service requirement in that plant because years of se ice at individual plants was not recorded in the pension file. Years of servic was defined as years credited for pension benefits and is not an exact, cumul ve measure of time actually

employed in Ford plants, due to special provisions covering layoffs, leaves of absence or other gaps in employment. Deceased employees known to have ever been hourly ("ever-hourly"), or not known to have never been hourly ("possibly-hourly") were included in the study population.

Study Population: Identification

The identification of the study population proceeded by specifying a candidate population, determining vital status and employment status (whether or not employee was ever an hourly employee) and ultimately identifying those who satisfy the study criteria. The candidate population was derived from the Employee Masterfile of the employer's pension system, covering the period 1966 - 1981, by selecting all employees with 10 or more years service, employed last at one of the 4 study sites, not known to be dead on January 1, 1966 and not known to be alive on December 31, 1981. This candidate population was then matched by the employer (on social security number, SSN) to the following files for vital status and death information:

1) The set of death certificates collected by 2 of the UAW local unions;

2) Retiree Extract, a Ford pension system file, providing additional information on employment and indicating deaths of retirees;

3) Work History, a file created by Ford for occupational epidemiologic surveillance;

4) Mortality, a Ford file containing information for epidemiologic use;

5) The Social Security Administration's Death Master File (all deaths known to SSA 1965 - 1980), of which Ford has a copy;

6). The claim registers of the life-insurance carrier for this workforce for the purpose of both vital status determination and death certificate retrieval;

7). The data file created for a previous study of spray painters which included deaths from 2 of the 4 study plants, for the period 1971 - 1976, as identified from life insurance claims[4,5];

8). The Michigan death registry for the years 1970 - 1982.

Death certificates were sought from State Vital Records Offices for study members for whom death information was not available from the existing files of the employer or from the local union sources.

The date of termination of employment from an assembly plant was not uniformly available for the study population, based on employer files. An estimated date was calculated using the date of hire and years of service, but adjusted whenever the resulting date exceeded the date of death or the date at which age 65 was attained. When a hire date was missing, and a termination date was available, the latter was used. The final date of hire was assigned

as the date of termination minus the years of service. Based on these dates, employment duration and latency interval (from date of hire until date of death) were computed. In three cases where improbable dates of hire or termination were computed, due to unlikely dates in the source files, dates were taken from job history files (which were available for half of the study population).

ICD Coding of Underlying Cause of Death

Several sources were utilized for ICD coding of death certificates, all in the Eighth Revision of the International Classification of Disease, or in the Ninth Revision followed by translation to the Eighth for some deaths occuring after 1978.[31] An ICD-coded underlying cause of death was available from more than one source for approximately half of the study population. In comparing cause of death information from multiple sources, only 25 cases were found where the disagreement affected the first 3 digits of the ICD. For several of these, the disagreements arose from illegibility; for the remaining cases, a final arbitration was obtained by submitting those death certificates to a Michigan Department of Public Health (MDPH) nosologist. For multiple ICD codes involving disagreements only in the fourth digit, the preferred one was selected according to the following precedence: 1) coding in ICD8 by an NCHS nosologist; 2) coding in ICD8 or ICD9 translated to ICD8 depending on the Revision current at death, by MDPH nosologists (translation by program), 3) ICD codes obtained from the Michigan death registry (also coded by MDPH nosologists) 4) ICD codes contained in Ford mortality file (also done by MDPH nosologists) and 5) ICD codes used in previous study of spray painters.

Analysis

Standardized proportional mortality ratios (SPMR) were computed for the study population classified on sex, race, hourly status, employment latency (years from hire by Ford until death) and employment duration. Monson's program [32] was used with U.S. national reference rates current through 1975 for black men, and through 1978 for white men[33]. There were insufficient women for PMR analysis. Two-sided Poisson P-values were computed for causes of death where 5 or fewer were expected.

Two prior hypotheses were to be tested: the proportion of deaths due to 1) lung cancer and 2) lymphomas are elevated in assembly plant workers when compared with the U.S. national population. One-tailed tests of significance were used when evaluating prior hypotheses.

RESULTS

Vital Status Determination

The employer pension system generated a candidate study population of 2,905 former employees known to be deceased or not known to be alive. Of 1,808 deaths identified within the candidate study population, the employer pension system identified 1,645, or 91%, and the life insurance claim register identified 1,499 or 83%. Table 1 shows the vital status revealed jointly by the pension and life insurance sources; 164 deaths were known only to the pension system (9%) and 18 were known only to life insurance (1%). Pension and life insurance together identified 92% of the known deceased.

Of the 145 deaths not identified in either the pension or life insurance systems, 46 were identified by the Social Security search, 11 were found in the Michigan death registry search (for the 2 Michigan plants), and finally 88 were known to the employer's mortality surveillance file (Table 2).

Of the 1,808 deaths identified in the candidate population, 1,483 ultimately satisfied the study population criteria (on date of death and hourly status) and 1,417 of these also had cause of death information collected, constituting the final analysis population. Tables 1 and 2 show the vital status determination for this population.

Death Certificate Sources

Cause of death information was obtained for 1,417 or 95.5% of the study population. The actual source of cause of death information for the final analysis population is shown in Table 3. The Michigan death registry was the unique source for 171 cases, the spray painter study for 205 cases, and another 195 were available from both sources; 207 cases were available from both the Michigan death registry and the employer mortality file. For the 329 remaining cases, 301 death certificates were collected from either local union sources or State health departments directly, and 28 certificates were obtained from the life insurance company. In addition, another roughly 300 death certificates were available from local union sources for cases also having death information from the file sources above.

The ascertainment rates for cause of death by plant and employment status are given in Table 4. For ever-hourly employees, the rates exceed 96% in all plants but the smaller, Michigan Truck, plant.

Study Population Characteristics

The numbers of ever-hourly workers in the final analysis population are roughly similar for the 3 auto assembly plants but smaller for the truck plant

(Table 5). The possibly-hourly group for the Wayne plant, however, is disproportionately large because job history files were not examined for this plant and the personnel staff was unable to exclude ever-hourly status for a large proportion of those employees terminating in salaried status. Possibly ever-hourly employees were included in the study population.

Table 5 also indicates that the final analysis population was composed almost entirely of white men at the Kansas City and Twin Cities plants while at the Wayne and Michigan Truck plants, 30% of the every-hourly workforce were black men.

Age of hire and duration of employment are shown in Table 6. Deceased white men terminating from the Michigan truck plant have roughly 8 fewer years service than those from the other assembly plants, possibly reflecting the known higher variability in total workforce over time at this plant. Compared with white men, black men generally had fewer years of service, averaging 20 years of Ford service. Age of death and employment latency (period from Ford hire to death), also shown in Table 6, indicate lower means for the Michigan truck employees, again possibly reflecting the shorter years of service and younger age structure for the group at risk.

The joint distribution of ever hourly men in the final study population over categories of employment duration and latency is given in Table 7. There were 382 white men (41.%) with 30 or more years service; only 14 (9%) of black men had 30 or more years service. Of white men with 30 or more years latency, only 20 or 3% have less than 20 years service; for black men the numbers were 9 or 16%.

In order to assess the possible importance of more recent exposures (i.e. those of the 1960s and 1970s) the distribution of the study population with respect to termination date was obtained (Table 8). Thirty-seven percent of the ever-hourly white men ended employment prior to 1966; 20.6% terminated in 1974 to 1981.

Standardized Proportional Mortality Analysis

Black Men

For 159 ever-hourly black men in the final analysis population, the proportion of all malignancies was elevated: PMR = 50/34.1 = 1.46, p = .002 (Table 9). The sites contributing more than expected proportions of deaths and more than 5 cases were lung (PMR=17/11.4=1.49, p=.14), colon (PMR=7/2.18=3.22, p=.02), pancreas (PMR=6/1.86=3.22, p=.02) and prostate (PMR=7/3.54=1.98, p=.14). The sites with elevations that are statistically significant were colon and pancreas.

Proportional mortality due to arteriosclerotic heart disease is close to expected for black men in the analysis population. The proportion of deaths

due to strokes is quite significantly reduced in this population compared with the U.S. population: 4 observed, 14.7 expected (p=.005).

Table 10 displays the PMRs for selected sites of malignancy in groups of ever-hourly black men classified by latency (interval from hire date until death) and duration of employment, and Table 11 displays the same for heart disease and stroke. The overall elevation in malignancies remains statistically significant only in the group with more than 30 years latency but with less than 30 years duration, however most of the other groups are small and there is insufficient power for detecting an elevation or reduction. The lung, colon, and pancreas cancers occur indistinguishably across latency intervals.

White Men

For all 1217 white men in the final analysis population, including both ever-hourly and possibly-hourly, there were statistically significant elevations only for stomach cancer (PMR=18/10.8=1.66, p=.04), all non-malignant diseases of the central nervous system (PMR=15/8.91=1.68, p=.04), and lympho- and reticulosarcomas (PMR=8/3.89=2.06, p=.09) (Table 12). The one-tailed P-value for the sarcomas (which had been hypothesized), was .045 in the ever- and possibly-hourly group; in the ever-hourly only group, the sarcomas were similarly elevated but not statistically significant (PMR=6/3.15=1.90, p=.10, one-tailed) (Table 13).

Other cancer sites with more than the expected numbers were esophagus, lung and brain. Of the 8 esophageal cancer deaths observed in hourly workers, 6 occured in the two (adjacent) Michigan plants and 1 occured at each of the non-Michigan plants. The PMRs for the ever-hourly group were similar to those of the possibly- and ever-hourly group but none were statistically significant (Table 13).

PMRs for selected malignancies in latency categories are shown in Table 14, and for selected non-malignant causes of death, in Table 15. Lung cancer appears to be more concentrated in the lower latency groups, and is statistically significant only for latencies of 10-15 years. Stomach cancer is statistically significant only in the 30 or more years latency group of possibly and ever-hourly workers. Both malignant and non-malignant brain disease indicate elevated PMRs for the groups with 30 or more years latency. Small numbers preclude inferences concerning the shorter latency groups. The 15 deaths due to non-malignant diseases of the brain in white men, where 8.9 were expected, included 7 cases of paralysis agitans (Parkinson's Disease), 4 cases of "other diseases of the brain or motor neurones", and 2 cases of cerebral atrophy.

The proportion of deaths due to arteriosclerotic heart disease for the white men of this study is close to that expected (PMR range: .95 - 1.03) and indicates no apparent trend with latency (Table 15). Non-malignant respiratory disease constitutes a significantly reduced proportion of deaths (PMR=46/65.0=.71, p=.01 for ever-hourly white men) (Table 13). About two thirds of this deficit is accounted for by a deficit in pneumonia deaths.

Table 16 shows the PMRs for ever-hourly white men who died within 15 years of hire, with a statistically significant elevation in the proportion

dying of lung cancer. Also revealed is the high proportion (25%), observed and expected, of those dying of external causes in this relatively young age-at-death group.

Table 17 shows proportional mortality ratios for the ever-hourly white men grouped according to date of termination of employment. These groups differ in their age distributions as indicated in Table 8. There is an apparent increase in the proportion of malignancies of the lung and stomach comparing those terminating after 1965 and those before, however the differences are not statistically significant and non-comparability of age at death between the 2 groups limits inference.

Because lung cancer was one of the original concerns, and there had been an indication of elevated lung cancer at one of the plants, PMR analyses were done for each of the plants, for ever-hourly white men. Only the plants located in Michigan had a PMR for lung cancer that was greater than 1.0; the other two plants both had lung cancer PMRs of 0.96, a slightly lower proportion of deaths due to lung cancer than expected from national rates. In the Michigan plants, together, there were 32 lung cancers where 23.9 were expected (PMR=1.34, p=.13).

DISCUSSION

Ascertainment of Study Population

There were 1808 deaths identified in the candidate study population of 2905. The 1097 not known to be deceased are believed to consist of the following:

1. Workers employed for 10 or more years who subsequently terminated employment prior to retirement age (i.e. deferred vested workers) and who did not reach retirement age by the end of the study period (1982). Although some of those workers may have died and their death not been known to the Social Security system, the employer, or (in the case of Michigan) the state death registry, the vast majority of these workers were likely to be alive in 1982.

2. Workers who were in deferred vested status and who reached retirement age before 1982, but who did not claim pension benefits, and who subsequently died. The economic incentives make this group likely to be very small, although the incentives operating are probably less effective than in the case of retiring active employers.

The overall completeness of vital status determination is unknown, however, an indication can be inferred from the observation that 11 additional deaths were identified from the state of Michigan death registry (covering roughly 41% of the study population). The identification of deaths in Michigan is believed to be quite complete (i.e. > 95%) because social security numbers were available for the entire candidate study population and because matching to the state death registry was done both by social security number (when one was present in the death registry) and by name (and date of birth). If analogous state registries had been utilized for the other plants, perhaps 11/.41 = 26 deaths would have been identified. The estimated 26 deaths constitute 1.4% of the known deceased and probably consist almost entirely of deaths occuring following termination but prior to retirement age so that they are often not known to the social security system or to the employer pension system. Still missed would be those dying in a different state from that of their last plant; those tend to be a minority in industrial populations (in UAW experience). However, in the specific category of deferred vested employers, the population dying out-of-state may be considerably higher. Assuming 75% of deferred vested employees die in a different state, the estimated 26 + 11 deaths identifiable from death registries should be increased to 37/.25 = 148. Therefore, we estimate that vital status ascertainment efficiency in the candidate study population is in the range 90-95%.

The identification of deceased members of the candidate study population, relying on a pension system alone, produced 91% of those ultimately known to be dead. Insurance sources alone did not do as well - 83% were identified.

Proportional Mortality of Black Men in Ever-Hourly Workforce

The statistically significant excess proportion of deaths due to all cancers in black men is similar to Chiazze's findings. Although there was overlap between the Chiazze study population and the present one, the group in common comprised less than one third of the present study and less than 12% of the larger Chiazze population. Site-specific excesses were found in both this study population and Chiazze's for lung, liver and pancreas, although the lung and liver excesses were significant only in Chiazze and the pancreas only in the present study. The colon cancer excess in this study has not been previously reported.

The observed statistically significant elevated proportions of deaths due to malignancies of the colon and pancreas, aside from possibly being random clusters, may have been caused by occupational exposures. Alternatively, the excess could reflect special selection features of this population: black workers employed in relatively high paying jobs with attendant life style differences compared with the rest of the black U.S. population. In an unpublished study of several large plants where machining was a major activity, standardized mortality ratios (SMRs) were calculated for a population of 822 black male workers[34]. Compared with the national reference population, this group of black workers had SMRs substantially lower than 1.0 for all causes (.60), for all malignancies (.71), for digestive malignancy (.57), for all cardiovascular disease (.62), for stroke (.61) and for accidental death (.29). Although the individual work histories were unavailable, the black workers constituted more than 25% of the plant populations and thus were likely to have worked in a variety of job classifications including the major ones. There was no evidence of increased colon and pancreas cancer in black workers in this study (for all digestive cancer there were 9 deaths observed, 15.8 expected), however the expected numbers were small.

All but 1 of the 6 pancreas cancer cases in this study occured at the Michigan plants, which is where most of the black workers in the study population were. However, comparing age-adjusted death rates for pancreas cancer[35] in black men, for the Detroit SEER area vs. all U.S. (SEER) areas, the Detroit area, which encompasses the 2 Michigan plants, has a lower rate, by 9%. Thus differences in regional rates do not account for the cluster of pancreas cancers.

The colon cancer cases in black workers all occured in the Michigan plants. The age-adjusted death rate for colon cancer in the Detroit SEER area is about 2% higher than in the rest of the U.S., for black men, thus not accounting for the observed proportional mortality excess.

The significantly reduced PMR for stroke (PMR=4/14.7=.27, p=.005) has not been reported before and is not explained. In the SMR study of machining plants[34] noted above, the stroke SMR (.61) among black men was indistinguishable from the all causes SMR (.60) and thus the standardized proportional mortality ratio (PMR) for stroke, in that population, should be close to unity. Arteriosclerotic heart disease proportional mortality is not

reduced in the present study, making it unlikely that the favorable stroke experience simply reflects a low overall cardiovascular risk in this group.

Hypertension is a well established risk factor for stroke and it is possible that hypertension is less prevalent in this population or is under better control than in the general population of black men. However available data from hypertension detection and control programs in similar populations is insufficient to resolve this question. To the extent that information is available it appears that mere availability of comprehensive health insurance benefits such as were available in the study plants (including broad coverage for hospitalization, laboratory tests and medications but not, in most cases, outpatient physician fees) does not reduce the prevalence of uncontrolled hypertension.[36,37]

Foote and Erfurt[36] have reported results of a worksite hypertension control project conducted at 4 plants in the Detroit area owned by the same employer as the plants in this investigation. Although these plants were not assembly but stamping, transmission and plastic fabrication plants, their hiring patterns and demographic makeup (percent male range 71-95%, percent white range 81-89%, mean age range 33-41) are close to that of the plants in this study. The prevalence of hypertension in these 4 plants prior to introduction of a control program ranged from 13-19%. Among those with hypertension the percent adequately controlled ranged from 13-26%. These figures were not adjusted for sex, race and age. They are not markedly different from those reported from large community screening programs although precise comparisons cannot be made. Stamler,[37] for example, reported hypertension prevalence ranging from 4% among those age 20-29 to 17% among those age 60-64 in a large screened population; 22% of hypertensives under the age of 40 were adequately controlled, while 45% of hypertensives of all ages were under control.

Other risk factors which are possibly, although less convincingly, associated with stroke mortality include elevated serum lipids, diabetes and obesity.[38] Cigarette smoking, while associated with arteriosclerotic cardivascular disease is probably not a risk factor for stoke.[38] There is no evidence that any of these factors have a different prevalence in the study plants than in the general population of black men, although the relatively high income and job stability of auto assembly workers might plausibly have an impact on them through the mediating effects of lifestyle difference such as diet.

Ostfeld[38] points out that death certificate diagnoses of stroke were incorrect 40% of the time in the Framingham Study and that "differences in application of medical terminology and differences in social views" could affect reporting. Most of the black deaths in this study were from the two Wayne, Michigan plants and it is possible, although speculative, that under-reporting of stroke mortality in this area could have led to an artifactually low PMR.

Stroke mortality in the U.S. has been decreasing substantially since 1973-74.[38] Since the comparison rates for stroke in this study are from 1970-1975, the expected number of stroke deaths for 1975-80 were probably overestimated. This could have led to a falsely low stroke PMR. However such an effect should have been approximately the same for both black and whites.

Proportional Mortality of White Men in Ever-Hourly Workforce

Although many of the possibly-hourly members of the study population may once have been hourly, the large group from the Wayne and Michigan truck plants probably includes substantial numbers of never-hourly employees. For this reason the discussion will focus largely on the ever-hourly workforce even though the PMR results are in fact quite similar when the possibly-hourly group is included.

For the group of all white men with ever-hourly status, taken as a whole, it was not possible to confirm the previously reported excess proportional mortality due to all cancers, lung cancer or lymphomas. However, in the larger group of possibly- and ever-hourly white men (using a one-tailed test of significance for testing a prior hypothesis), the proportion of deaths due to lympho- and reticulosarcoma, a subset of Chiazze's lymphoma group, is significantly elevated (p=.045). The power for detecting a lung cancer proportional excess in the ever-hourly group (PMR=1.25, α =.05) was only 60% (Poisson exact).

The excess cases of stomach and esophageal cancer in white men occured only in the Michigan plants. Age-adjusted death rates for esophageal and stomach cancer in the Detroit SEER area are somewhat elevated compared with all SEER areas combined:[35] the esophagus cancer rate by 18%, the stomach cancer rate by 12%. However, these rate differences do not account for the observed clusters.

The sarcomas observed in hourly workers occured primarily at the non-Michigan plants (5 of 6). Using the Iowa SEER area as a surrogate for Kansas, and the Seattle SEER area for Minnesota, the expected rates for the Kansas City plant are about 8% higher than for all U.S. SEER areas combined, and for the Minnesota plant, about 5% lower.[35]

The clusters of lung cancer, lympho/reticulo sarcoma and stomach cancer seen for certain latency groups constitute exploratory findings which need to be investigated further with work histories. The ever-hourly workers with a significantly elevated proportional mortality due to lung cancer were those with less than 15 years employment latency. This group, by virtue of the selection on latency, differs from the rest of the study population by having a younger age of death (44.5 vs 67.7). Short latency would also be expected to be associated with older age at hire, however the age of hire of the short latency group is indistinguishable from that of the whole study population (mean =31.7). This is perhaps a consequence of the high proportion of accidental deaths expected and observed in the short latency group, and the likely association of young age of hire with accidental death. Furthermore, the mean age of hire for short latency lung cancer deaths is substantially higher than for other short latency deaths: 39.4 compared with 30.4 for the non-lung cancer cases.

The 7 lung cancers observed in the short latency group could merely reflect chance, non-occupational exposures, or prior employment: the average age of hire of the lung cancer cases is 7 years older than the mean for the

study population. Another possible explanation is exposure to relatively virulent and currently unidentified carcinogens resulting in short latency disease. In the Chiazze study of spray painters,[4] a doubling of lung cancer proportional mortality (not statistically significant) was seen in the group whose first spray painting experience came after 1956: PMR=8/3.8=2.06. This too would be a relatively short latency group with respect to spray painting exposure since all exposures would have begun less than 20 years prior to death (deaths occured 1972-1976).

The malignant and non-malignant brain disease mortality deserves work history analysis particularly in view of the probable use of chlorinated hydrocarbon solvents and exposures to lead. Elevated brain cancer in some industrial populations has been attributed to relatively superior medical diagnostic access for those populations in recent times compared with that available to the population as a whole.[38] The recent dissemination of brain scan technology might be one such diagnostic biasing factor. However, the dates of death for the brain cancers observed were roughly evenly scattered over the 12 year study period; 5 of 12 brain cancer deaths (42%) occured in the first 5 years 1970-1975, where 47% of all study deaths occured. Thus a time-trend suggesting changing diagnostic sensitivity was not observed for these brain cancer deaths.

Arteriosclerotic heart disease in the ever-hourly white men was not proportionally low in this study population as is often observed in a healthy industrial population. However all non-malignant respiratory disease was significantly reduced (PMR=.71, p=.02). This observation has no obvious explanation but one hypothesis suggested is that levels of ambient carbon monoxide below the OSHA standard may predispose to heart disease deaths. Carbon monoxide is produced by industrial trucks, welding, new car start-up and possibly paint ovens in assembly plants.

CONCLUSIONS AND RECOMMENDATIONS

Major Findings

1. Statistically significant elevations of proportions of deaths due to cancer of the colon and pancreas, as well as all cancers combined, were observed in black men who were ever-hourly employees in auto assembly plants.

2. A significant reduction in the proportion of deaths due to stroke was found among black men.

3. The previously reported increase in proportion of deaths due to lung cancer was not confirmed in white men ever employed as hourly workers in auto assembly plants, except in one subpopulation - those whose company employment began within 15 years of death. The previously reported excess of lymphoma was confirmed only in so far as lymphosarcomas and reticulosarcomas were in excess in the possibly- and ever-hourly group of white men. There was also a significant excess of stomach cancer, in this group.

Recommended Follow-up

1. The findings of elevated proportions of deaths due to cancer of the colon and pancreas in black assembly workers and lung cancer, stomach cancer and lymphomas in white workers should be examined in relation to work histories to determine if there are exposures or other factors associated with these outcomes. Case-control studies of proportional mortality (i.e. within the current study population of deceased workers) would be appropriate, using broad exposure classifications and stratifying on age. Confirmatory hypothesis testing should be carried out for two major outcomes - lung cancer and lympho/reticulo sarcomas. The suspect exposures for case-control analyses are 1) pyrolysis products, 2) mixed organic sources and 3) asbestos related work. Additional exploratory analyses should be conducted for the purpose of identifying associations between other particular outcomes and exposures such as stomach cancer in white men and colon and pancreas in black men. These latter analyses are likely to be limited by low power.

2. The substantial reduction in stroke as cause of death, among black assembly workers, should be investigated further to determine, if confirmed in similar populations, whether the deficit is associated with: 1) access to better health care, including hypertension control programs, 2) higher compliance with hypertension control regimens, or 3) some factor which reduces the prevalence of hypertension (such as secure employment).

3. Although the observed elevations in proportional mortality are not as large as some that have been reported for more specific exposures in other industrial settings, the diversity of exposures and low power for detecting many specific effects should be cause for continuing and increasing efforts at control of hazards in the environment of assembly plants.

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Table 1

Vital Status Determination from Employer Pension and Life Insurance Records

Known Deceased Members of Candidate Study Population (% of Total)

Life Insurance Vital Status	Pension Vital Status: Not Known Deceased	Pension Vital Status: Known Deceased	Total
Not Known Deceased	145 7.9%	164 9.0%	309 17%
Known Deceased	18 1.0%	1,481 82%	1,499 83%
Total	163 9.0%	1,645 91%	1,808 100%

Final Analysis Population (% of Total)

Life Insurance Vital Status	Pension Vital Status: Not Known Deceased	Pension Vital Status: Known Deceased	Total
Not Known Deceased	12 .85%	46 3.2%	58 4.1%
Known Deceased	15 1.1%	1,344 95%	1,359 96%
Total	27 1.9%	1,390 98%	1,417 100%

Table 2

Vital Status Sources for Deaths not Identified in Employer Pension or Insurance Records[1]

Candidate Study Population Deaths (n = 1,808)	
Social Security	46
Michigan Death Registry	11
Employer Mortality File	88[2]
Spray Painter Study	0
Total	145
Final Analysis Population (n = 1,417)	
Social Security	0
Michigan Death Registry	11
Employer Mortality	1[3]
Spray Painter Study	0
	12

1. Mutually exclusive categories: each line displays number found of those not identified from preceding sources listed.

2. Includes 87 deaths occuring in 1982 and thus excluded from study population.

3. A death missed in manual search of Insurance claim registry.

Table 3

Cause of Death Information Sources[1] for Final Analysis Population

Michigan Death Registry (MDR) Alone	171
Spray Painter Study (SPS) Alone	205
Mortality File Alone	310
Both MDR and SPS	195
Both MDR and Mortality File	207
Death Certificates from insurance company	28
Total from employer or other systematic sources	1116
Death Certificates from Local Unions or State health departments	301
Total	1417

1. Mutually exclusive categories: each line displays number of population with death information that was not available from preceding sources.

Table 4

Rates of Ascertainment of Cause of Death for Known Deceased by Plant and Employment Status (%)

	Plant			
	Kansas City	Twin Cities	Wayne	Mich. Truck
Study Population: Ever-Hourly Status (n=1141)	97.6	98.6	96.1	86.5
Possibly-Hourly (n=342)	100.0	94.0	89.2	100.

Table 5

Characteristics of Final Analysis Population: Distribution by Plant, Employment Status, Sex and Race

	Plant[1]				
	KC	TC	WA	MT	Total
Employment status					
Ever-Hourly	365	279	415	45	1104
%	33.1	25.3	37.6	4.1	100%
Possibly-Hourly	48	47	214	4	313
%	15.3	15.0	68.4	1.3	100%

	KC		TC		WA		MT		Total	
Sex/Race: Ever-Hourly [2]		%		%		%		%		%
White Men	343	94.0	275	98.6	276	66.5	32	71.1	926	84.2
Black Men	19	5.2	3	1.0	125	30.1	12	26.6	159	14.5
White Women	1	0.3	0	0.0	10	2.4	1	2.2	12	1.1
Black Women	0	0.0	0	0.0	2	.48	0	0.0	2	.2
Total									1099	100.

	KC		TC		WA		MT		Total	
Sex/Race: Possibly-Hourly		%		%		%		%		%
White Men	47	97.9	46	97.8	194	90.6	4	100.	291	93.0
Black Men	1	2.1	0	0.0	17	7.9	0	0.0	18	5.7
White Women	0	0.0	1	1.2	2	.93	0	0.0	3	.96
Black Women	0	0.0	0	0.0	1	.46	0	0.0	1	.32
Total									313	100.

1. KC - Kansas City
   TC - Twin Cities
   WA - Wayne
   MT - Michigan Truck

2. 5 cases omitted because race was not white or black, or was unknown.

Table 6

Final Analysis Population: Age of Hire, Duration of Employment, Age at Death and Employment Latency[1], for Ever-Hourly White Men and Black Men

		White Men				Black Men			
	Plant[2]:	KC	TC	WA	MT	KC	TC	WA	MT
Total	n	343	275	276	32	19	3	125	12
Age of Hire (Yrs.)	Mean	31.2	31.8	31.6	33.3	32.2	38.3	34.3	33.2
	(SD)	(8.3)	(7.3)	(8.9)	(9.9)	(6.4)	(15)	(8.5)	(6.1)
Duration (Mos.)	Mean	316	338	322	217	215	260	253	200
	(SD)	(108)	(91)	(109)	(75)	(60)	(124)	(83)	(72)
Age at Death (Yrs.)	Mean	64.8	69.5	67.1	53.9	54.5	61.3	61.7	51.8
	(SD)	(12)	(12)	(12)	(10)	(11)	(9.1)	(11)	(11)
Latency (Mos.)	Mean	404	455	427	248	268	275	330	224
	(SD)	(149)	(144)	(142)	(83)	(83)	(140)	(98)	(90)

1. Time interval from date of hire until date of death.

2. KC - Kansas City, TC - Twin Cities, WA - Wayne, MT - Michigan Truck

Table 7

Employment Duration and Latency for Ever Hourly Employees in the Final Analysis Population

White Men

		Duration (Yr.)				
		< 20	20-29	30-39	≥ 40	Total (%)
Latency (Yrs.)	< 20	107				107 (12)
	20-29	80	155			235 (25)
	30-39	18	131	103		252 (27)
	≥ 40	2	51	219	60	332 (36)
	Total (%)	207 (22)	337 (36)	322 (35)	60 (6.4)	926

Black Men

		Duration (Yr.)				
		< 20	20-29	30-39	≥ 40	Total (%)
Latency (Yrs.)	< 20	36				36 (23)
	20-29	24	45			69 (43)
	30-39	9	30	11		50 (31)
	≥ 40	0	1	2	1	4 (2.5)
	Total (%)	69 (43)	76 (48)	13 (8.1)	1 (0.6)	159

Table 8

Year of Termination for Ever Hourly White Men in Final Analysis Population

Age at Death	Year of Termination: Before 1950	1950-57	1958-65	1966-73	1974-81	Total
< 40	—	--	--	4	20	24
40-49	—	1	1	21	43	66
50-59	—	1	8	86	73	168
60-69	—	1	39	184	53	277
70-79	—	5	147	94	2	248
80-89	1	50	82	1	—	134
90-99	2	7	—	—	—	9
Total	3	65	277	390	191	926
%	.32	7.0	30.	42.	20.6	100.

Table 9 **Standardized Proportional Mortality Ratios For Selected Causes of Death in Ever Hourly Black Men for Selected Causes of Death**

	OBS./EXP.	PMR	95% CI	X^2	P(1)
All Malignant Neoplasms	50/34.1	1.46	1.15,1.87	9.49	.002
Cancer of Buccal Cavity and Pharynx	0/1.35				—
Cancer of Digestive Organs, Peritoneum	18/10.0	1.80	1.16,2.79	6.81	.030
Cancer of Esophagus	1/2.18	0.46	0.07,3.05	0.65	—
Cancer of Stomach	1/2.25	0.44	0.07,2.94	0.71	—
Cancer of Large Intestine	7/2.18	3.22	1.28,6.62		.016
Cancer of Rectum	2/0.62	2.94	0.79,11.0	2.57	—
All Cancer of Liver	1/0.69	1.44	0.21,10.1	0.14	—
Cancer of Pancreas	6/1.86	3.22	1.18,7.02		.024
Cancer of Respiratory System	17/12.1	1.40	0.89,2.20	2.12	—
All Cancer of Lung	17/11.4	1.49	0.95,2.35	2.99	.144
Cancer of Prostate	7/3.54	1.98	0.79,4.08		.137
Cancer of Bladder	1/0.63	1.59	0.23,11.1	0.22	—
Cancer of Brain and Other CNS	2/0.41	4.89	0.59,17.6		.128
All Lymphopoietic Cancer	3/2.21	1.36	0.44,4.16	0.28	—
Leukemia and Aleukemia	2/0.77	2.59	0.58,9.81	1.96	—
All Diseases of Nervous System and Sense Organs	2/1.31	1.53	0.39,6.02	0.37	—
Arteriosclerotic Heart Disease, Including CHD	46/43.2	1.06	0.83,1.36	0.25	—
All Vascular Lesions of CNS	4/14.7	0.27	0.11,0.65	8.69	.005
All Respiratory Diseases	4/8.84	0.45	0.18,1.14	2.80	—
All Diseases of Digestive System	6/8.71	0.69	0.32,1.48	0.91	—
Cirrhosis of Liver	4/5.21	0.77	0.30,1.99	0.29	—
All Diseases of Genito-Urinary System	4/3.46	1.16	0.44,3.05	0.09	—
Chronic Nephritis	1/1.27	0.78	0.11,5.50	0.06	—
All Diseases of Skin, Cellular Tissue	1/0.22	4.53	0.76,27.0	2.76	—
All Diseases of the Bones, Organs of Movement	1/0.27	3.65	0.59,22.7	1.93	—
All External Causes of Death	21/16.2	1.30	0.88,1.91	1.77	—
Total Deaths	159				

1. P-value for 2-sided test, all causes of death treated independently; when EXP. is less than 5.0 and a P-value is presented, the P-value and 95% CI are based on the Poisson distribution; otherwise the P-values and 95% CIs are based on the Chi-Squared distribution. Large P-values are not presented.

Table 10

Standardized Proportional Mortality Ratios for Selected Malignant Causes of Death in Ever Hourly Black Men Classified According to Latency[1]

Latency (Yrs.)	n		All	Lung	Colon	Rectum	Pancreas	Lympho-poietic	Brain
All	159	PMR	50/34.1=1.46	17/11.4=1.48	7/2.18=3.22	2/.68=2.94	6/1.86=3.22	3/2.21=1.36	2/.41=4.89
(⩾10)		p[2]	.002	.14	.016	--	.024	—	--
10-14	22	PMR	4/3.61=1.11	2/1.34=1.50	1/.19=5.38	0/.06=0	1/.17=5.77	0/.28=0	0/.07=0
		p	--	--	.04	--	.03	--	--
15-29	83	PMR	25/18.3=1.36	10/6.48=1.54	3/1.09=2.75	0/.35=0	4/1.01=3.97	1/1.17=.86	2/.24=8.36
		p	.08	.24	.19	—	.04	—	.05
⩾30	40	PMR	17/8.84=1.92	4/2.5=1.60	2/.67=2.98	2/.20=9.86	1/.49=2.04	1/.56=1.79	0/.07=0
Dur <30		p	.02	--	--	.04	--	--	--
⩾30	14	PMR	4/3.37=1.19	1/1.08=.92	1/.23=4.33	0/.07=0	0/.19=0	1/.21=4.74	0/.03=0
Dur ⩾30		p	--	--	--	--	--	--	--

1. Based on Proportional Mortality Rates for U.S. Non-White Men 1970-1975.

2. P-value for 2 sided test, all causes of death treated independently; Poisson P-value computed for Exp. for less than 5.

Table 11

Standardized Proportional Mortality Ratios for Selected Non-Malignant Causes of Death in Ever-Hourly Black Men Classified According to Latency[1]

Latency (Yrs.)	n		Stroke	Arteriosclerotic Heart Disease
All	159	PMR	4/14.7=.27	46/43.2=1.06
(⩾ 10)		p[(2)]	.005	—
10-14	22	PMR	1/1.31=.76	5/4.27=1.17
		p	—	—
15-29	83	PMR	1/7.26=.14	26/22.3=1.17
		p	.01	—
⩾ 30	40	PMR	1/4.70=.21	12/12.5=.96
Dur <30		p	.10	—
⩾ 30	14	PMR	1/1.45=.69	3/4.15=.72
Dur ⩾30		p	—	—

1. Based on Proportional Mortality Rates for U.S. Non-White Men 1970-1975.

2. P-value for 2-sided test, all causes of death treated independently; Poisson P-value computed for Exp. for less than 5.

Table 12 **Standardized Proportional Mortality Ratios for Selected Causes of Death in All White Men (Possibly and Ever-Hourly)**

	OBS./EXP.	PMR	95% CI	X^2	P(1)
All Malignant Neoplasms	277/253.03	1.09	0.99,1.21	2.90	—
Cancer of Buccal Cavity and Pharynx	6/7.08	0.85	0.38,1.88	0.16	—
Cancer of Digestive Organs, Peritoneum	75/67.34	1.11	0.89,1.39	0.92	—
Cancer of Esophagus	8/5.57	1.44	0.72,2.86	1.06	—
Cancer of Stomach	18/10.84	1.66	1.05,2.62	4.77	.04
Cancer of Large Intestine	26/24.95	1.04	0.71,1.52	0.05	—
Cancer of Rectum	5/7.01	0.71	0.30,1.70	0.58	—
Cancer of Liver	2/3.87	0.52	0.13,2.01	0.91	—
Cancer of Pancreas	11/13.70	0.80	0.45,1.44	0.54	—
Cancer of Respiratory System	98/86.99	1.13	0.93,1.38	1.52	—
Cancer of Larynx	4/3.46	1.16	0.43,3.07	0.08	—
All Cancer of Lung	94/82.94	1.13	0.93,1.38	1.61	—
Cancer of Testis (Other Genital Organs)	2/0.75	2.66	0.70,10.1	2.07	—
Cancer of Bladder	7/8.84	0.79	0.38,1.65	0.39	—
Cancer of Kidney	2/5.92	0.34	0.09,1.26	2.61	—
Cancer of Brain and Other CNS	10/5.76	1.73	0.94,3.19	3.24	.14
All Lymphopoietic Cancer	27/22.86	1.18	0.81,1.72	0.77	—
Lymphosarcoma and Reticulosarcoma	8/3.89	2.06	0.87,4.05		.09
All Diseases of Nervous System and Sense Organs	15/8.91	1.68	1.02,2.77	4.20	.04
Arteriosclerotic Heart Disease, Incl. CHD	481/477.27	1.01	0.94,1.08	0.05	—
All Vascular Lesions of CNS	97/100.29	0.97	0.80,1.17	0.12	—
All Respiratory Diseases	66/89.27	0.74	0.59,0.93	6.59	.01
Emphysema	20/20.33	0.98	0.64,1.52	0.01	—
All Diseases of Digestive System	44/48.42	0.91	0.68,1.21	0.42	—
Cirrhosis of Liver	22/24.37	0.90	0.60,1.36	0.24	—
All Diseases of Genito-Urinary System	8/15.72	0.51	0.26,1.00	3.85	.05
Chronic Nephritis	1/3.46	0.29	0.05,1.81	1.76	—
All Diseases of Skin, Cellular Tissue	1/0.89	1.12	0.16,7.95	0.01	—
All Diseases of Bones, Organs of Movement	3/2.04	1.47	0.48,4.52	0.45	—
All External Causes of Death	69/71.33	0.97	0.78,1.20	0.09	—
Total Deaths	1217				

1. P-value for 2-sided test, all causes of death treated independently; when EXP. is less than 5.0 and a P-value is presented, the P-value and 95% CI are based on the Poisson distribution; otherwise the P-values and 95% CIs are based on the Chi-Squared distribution. Large P-values are not presented.

Table 13

Standardized Proportional Mortality Ratios for Selected Causes of Death in Ever Hourly White Men

	OBS./EXP.	PMR	95% CI	X^2	p(1)
All Malignant Neoplasms	217/200.	1.08	0.96,1.22	1.81	—
Cancer of Buccal Cavity and Pharynx	6/5.86	1.02	0.46,2.27	0.00	—
Cancer of Digestive Organs, Peritoneum	57/52.2	1.09	0.85,1.41	0.48	—
Cancer of Esophagus	8/4.55	1.76	0.89,3.47	2.62	—
Cancer of Stomach	13/8.23	1.58	0.92,2.70	2.79	.10
Cancer of Large Intestine	20/19.1	1.05	0.68,1.62	0.04	—
Cancer of Rectum	3/5.34	0.56	0.18,1.71	1.03	—
All Cancer of Liver	1/3.02	0.33	0.05,2.13	1.36	—
Cancer of Pancreas	8/10.8	0.74	0.37,1.47	0.74	—
Cancer of Respiratory System	79/71.9	1.10	0.89,1.35	0.75	—
Cancer of Larynx	4/2.84	1.41	0.53,3.73	0.48	—
All Cancer of Lung	75/68.7	1.09	0.88,1.36	0.64	—
Cancer of Testis (Other Genital Organs)	2/0.63	3.18	0.86,11.8	2.99	—
Cancer of Bladder	5/6.43	0.78	0.33,1.86	0.32	—
Cancer of Kidney	2/4.85	0.41	0.11,1.57	1.68	—
Cancer of Brain and Other CNS	9/5.09	1.77	0.93,3.36	3.03	.15
All Lymphopoietic Cancer	20/18.1	1.11	0.72,1.17	0.21	—
Lympho- and Reticulosarcoma	6/3.15	1.90	0.70,4.15		.20
All Diseases of Nervous System and Sense Organs	12/7.11	1.68	0.97,2.95	3.40	.11
Arteriosclerotic Heart Disease, Incl. CHD	368/358.	1.03	0.95,1.11	0.40	—
All Vascular Lesions of CNS	67/67.8	0.99	0.79,1.24	0.01	—
All Respiratory Diseases	46/65.0	0.71	0.54,0.93	6.04	.01
Emphysema	14/15.0	0.93	0.55,1.56	0.08	—
All Diseases of Digestive System	33/39.9	0.83	0.59,1.15	1.27	—
Cirrhosis of Liver	19/21.7	0.88	0.56,1.36	0.35	—
All Diseases of Genito-Urinary System	6/10.9	0.55	0.25,1.20	2.24	—
Chronic Nephritis	1/2.59	0.39	0.06,2.55	0.98	—
All Diseases of Skin, Cellular Tissue	1/0.66	1.53	0.22,10.7	0.18	—
All Diseases of Bones, Organs of Movement	2/1.61	1.24	0.31,4.95	0.09	—
All External Causes of Death	62/61.0	1.02	0.81,1.27	0.02	—
Total Deaths	926				

1. P-value for 2-sided test, all causes of death treated independently; when EXP. is less than 5.0 and a P-value is presented, the P-value and 95% CI are based on the Poisson distribution; otherwise the P-values and 95% CIs are based on the Chi-Squared distribution. Large P-values are not presented.

Table 14

Standardized Proportional Mortality Ratios for Selected Malignant Causes of Death in White Men Classified According to Latency[1]

Latency: (Yrs.)	n		All	Lung	Stomach	Lympho/ Reticulo Sarcoma	Brain
			Possibly and Ever Hourly				
All ($\geq$10)	1217	PMR	277/253.=1.09	94/82.9=1.13	18/10.8=1.66	8/3.89=2.05	10/5.76=1.73
		p[2]	—	—	.04	.09	.14
10-14	49	PMR	13/8.67=1.50	7/2.79=2.51	0/.3=0	0/.21=0	1/.56=1.77
		p	.10	.04	—	—	—
15-29	345	PMR	96/78.9=1.22	37/29.3=1.26	5/3.1=1.61	3/1.40=2.15	3/2.57=1.17
		p	.03	—	—	—	—
$\geq$ 30	823	PMR	168/165.=1.02	50/50.8=.98	13/7.44=1.75	5/2.29=2.19	6/2.63=2.28
		p	—	—	.04	.16	.10
			Ever Hourly Only				
All ($\geq$10)	926	PMR	217/200.=1.08	75/68.7=1.09	13/8.23=1.58	6/3.15=1.90	9/5.09=1.77
		p	—	—	.10	.20	.15
10-14	46	PMR	11/8.24=1.34	7/2.68=2.61	0/.28=0	0/.20=0	0/.53=0
		p	—	.04	—	—	—
15-29	296	PMR	83/68.6=1.21	31/25.9=1.19	5/2.64=1.89	3/1.23=2.44	3/2.36=1.27
		p	.05	—	—	—	—
$\geq$ 30	584	PMR	123/123.=1.00	37/40.0=.92	8/5.31=1.51	3/1.72=1.74	6/2.20=2.73
		p	—	—	—	—	.05

1. Based on Proportional Mortality Rates for U.S. White Men 1970-1978.

2. P-value for 2-sided test, all causes of death treated independently; Poisson P-value computed for Exp. less than 5.

Table 15

Standardized Proportional Mortality Ratios for Selected Non-Malignant Causes of Death in White Men Classified According to Latency[1]

Latency: (Yrs.)	n		Arteriosclerotic Heart Disease	Emphysema	CNS (ICD: 320-389)
			Possibly and Ever-Hourly		
All (⩾ 10)	1217	PMR	481/477.=1.01	20/20.3=.98	15/8.91=1.68
		p[2]	—	—	.04
10-14	49	PMR	11/12.2=.90	0/.24=0	2/.62=3.22
		p	—	—	—
15-29	345	PMR	127/131.=.96	8/5.11=1.57	3/2.91=1.03
		p	—	—	—
⩾ 30	823	PMR	343/334.=1.03	12/14.9=.80	10/5.38=1.86
		p	—	—	.10
			Ever-Hourly Only		
All (⩾ 10)	926	PMR	368/358.=1.03	14/15.12=.93	12/7.11=1.69
		p	—	—	.11
10-14	46	PMR	11/11.6=.95	0/.24=0	2/.58=3.47
		p	—	—	.23
15-29	296	PMR	109/111.5=.98	7/4.15=1.69	2/2.55=.78
		p	—	—	—
⩾ 30	584	PMR	248/236.=1.05	7/10.7=.66	8/3.98=2.01
		p	—	—	.10

1. Based on Proportional Mortality P-value Rates for U.S. White Men 1970-1978.

2. P-value for 2 sided test, all causes of death treated independently; Poisson P-value computed for Exp. less than 5.

Table 16

Standardized Proportional Mortality Ratios for Ever Hourly White Men with Less than 15 Years Latency

	OBS./EXP.	PMR	95% CI	X^2	p(1)
All Malignant Neoplasms	11/8.24	1.34	0.79,2.26	1.15	—
Cancer of Buccal Cavity and Pharynx	0/0.24				—
Cancer of Digestive Organs and Peritoneum	2/1.79	1.12	0.29,4.32	0.03	—
Cancer of Esophagus	1/0.16	6.25	1.14,34.4	4.44	—
Cancer of Stomach	0/0.28				—
Cancer of Large Intestine	1/0.64	1.56	0.23,10.8	0.21	—
Cancer of Rectum	0/0.16				—
Cancer of Liver	0/0.10				—
Cancer of Pancreas	0/0.38				—
Cancer of Respiratory System	7/2.79	2.51	1.01,5.17		.05
Cancer of Larynx	0/0.09				—
All Cancer of Lung	7/2.68	2.61	1.05,5.38		.04
Cancer of Testis (Other Genital Organs)	0/0.15				—
Cancer of Bladder	0/0.10				—
Cancer of Kidney	0/0.24				—
Cancer of Brain and Other CNS	0/0.53				—
All Lymphopoietic Cancer	1/1.17	0.86	0.12,5.92	0.02	—
Lympho- and Reticulosarcoma	0/0.20				—
All Diseases of Nervous System and Sense Organs	2/0.58	3.47	0.42,12.4		.23
Arteriosclerotic Heart Disease, Including CHD	11/11.6	0.95	0.58,1.55	0.04	—
All Vascular Lesions of CNS	2/1.43	1.40	0.36,5.41	0.23	—
All Respiratory Diseases	1/1.55	0.65	0.10,4.36	0.20	—
Emphysema	0/0.24				—
All Diseases of Digestive System	3/3.20	0.94	0.31,2.78	0.01	—
Cirrhosis of Liver	2/2.25	0.89	0.23,3.42	0.03	—
All Diseases of Genito-Urinary System	0/0.33				—
Chronic Nephritis	0/0.13				—
All Diseases of Skin, and Cellular Tissue	0/0.03				—
All Diseases of Bones, Organs of Movement	0/0.09				—
All External Causes of Death	12/13.4	0.89	0.58,1.38	0.26	—
Total Deaths	46				

1. P-value for 2-sided test, all causes of death treated independently; when EXP. is less than 5.0 and a P-value is presented, the P-value and 95% CI are based on the Poisson distribution; otherwise the P-values and 95% CIs are based on the Chi-Squared distribution. Large P-values are not presented.

Table 17

Standardized Proportional Mortality Ratios for Selected Causes of Death in Ever Hourly White Men, Classified According to Date of Termination[1]

Malignant Disease

Year of Termination	n		All	Lung	Stomach	Lympho-poietic	Brain
Before 1965	264	PMR	48/46.2=1.04	8/11.9=.67	3/2.25=1.33	8/4.2=1.91	2/.50=3.96
		p[2]	--	--	--	.12	.18
1965-1981	662	PMR	169/154.=1.10	67/56.8=1.18	10/5.98=1.67	12/13.8=.87	7/4.58=1.53
		p	--	--	--	--	--

Non-Malignant Disease

			Arteriosclerotic Heart Disease	Emphysema	CNS (ICD: 320-339)
Before 1965	264	PMR	110/108.=1.01	2/4.51=.44	3/1.56=1.93
		p[2]	--	--	--
1965-1981	662	PMR	258/250.=1.03	12/10.6=1.14	9/5.53=1.62
		p	--	--	--

1. Based on Proportional Mortality Rates for U .S. White Men 1970-1978.

2. P-value for 2-sided test, all causes of death treated independently; Poisson P-value computed for Exp. less than 5.