Mortality at an Automotive Engine Foundry and Machining Complex

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Mortality was analyzed for an automotive engine foundry and machining complex, with process exposures derived from department assignments. Logistic regression models of mortality odds ratios (ORs) were calculated for 2546 deaths, and numbers of work-related deaths were estimated. Lung cancer mortality in the foundry was increased where cleaning and finishing of castings was performed (OR, 1.7; 95% CI, 1.15 to 2.4 [at mean exposure duration of exposed cases]) and in core-making after 1967 (OR, 1.5; 95% CI, 1.11 to 2.0). Black workers had excess lung cancer mortality in machining heat-treat operations (OR, 2.5, 95% CI, 1.4 to 4.3) and excess nonmalignant respiratory disease mortality in molding (OR, 2.5; 95% CI, 1.16 to 5.5) and core-making (OR, 2.7; 95% CI, 1.25 to 5.8). Stomach cancer mortality was elevated among workers with metalworking fluid exposures in precision grinding (OR, 2.4; 95% CI, 1.14 to 5.1). Heart disease mortality was increased among all workers in molding (OR, 1.6; 95% CI, 1.09 to 2.3), as was stroke mortality among workers exposed to metalworking fluids (OR, 1.8; 95% CI, 1.22 to 2.7). Malignant and nonmalignant liver disease mortality was elevated in assembly/testing and precision grinding. In this modern foundry, 11% of deaths were estimated to be work-related despite it's being largely in regulatory compliance over its 40-year existence. Machining plant exposures accounted for 3% or more of deaths there. (J Occup Environ Med. *2001;43:483–493*)

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This study resulted from concerns at an automobile and truck engine manufacturing complex near Cleveland, Ohio. In 1978, 15,000 workers at this ferrous (gray iron) foundry and two machining/assembly plants produced almost 1.9 million engines. A cancer cluster among crankshaft grinders was investigated by company staff in 1979, and analysis by the plant physician found elevated stomach and lung cancer mortality.* A cohort mortality study (1970 to 1987)[1] confirmed the stomach cancer findings and found that lung cancer mortality increased with duration of employment, but nested case-control studies identified no clear associations.[2] The present study added 8 years of follow-up and analyzed exposure associations for a broader group of mortality outcomes using different methods.

Foundry activities involved core-making; preparation of "green" sand molds; pouring molten iron; and drag-out, shake-out, cleaning, and finishing of castings. Excess lung cancer, nonmalignant respiratory disease (NMRD), and heart disease have been reported in foundry studies.[3–10] Machining of castings or forgings (eg, engine blocks, cylinder heads, crank shafts) was followed by precision grinding and honing. Metalworking fluids (MWF) were predominately "soluble oils." Other activities included heat treatment, assembly, testing, and repair. Elevated rates of digestive and respiratory tract cancers, and other diseases, have been observed in

* Williams A. Mortality in a gray iron foundry. Unpublished report for Ford Motor Co, 1985.

TABLE 1
Exposure Categories Characterizing Departments at Each Plant*

Foundry
1. *Foundry dust*: high-dust areas in production and maintenance/support.
2. *Foundry dust in maintenance*: maintenance/support departments only.
3. *Molding* (includes pouring and drag-out/shake-out): dusts, hot mold emissions.
4. *Core-making* (oil sand, hot-box, and cold-box): oil smoke, phenol-formaldehyde, triethylamine, isocyanates; mold release, cleaning, and core-repair materials.
5. *Melting*: dusts and fume, CO from cupolas, heat stress.
6. *Cleaning and finishing* (drag-out/shake-out and cleaning): silica dust, mold emissions, asbestos, and welding fume (casting repair).

Machining
7. *Dry machining*: metal/oxide, mist, smoke (surface oils and corrosion inhibitors).
8. *Machining or grinding using straight oils*: oil mist, splash, and associated smoke.
9. *Machining or grinding, using soluble MWF*: mist, splash from soluble oils, semisynthetics, and synthetics.
10. *Grinding with semisynthetic (or synthetic) MWF*: with probable nitrosamines.
11. *Inspection*: floor inspectors in machining production and assembly.
12. *Leak testing* (machining plants and foundry): corrosion inhibitors.
13. *Engine assembly/test*: adhesives, sealers, gaskets, lubricants, greases, solvents; burn-off and emissions from hot testing.
14. *Heat treat* (cam shafts, rocker arms, valve seats): quench oil mist, smoke.
15. *Packing and shipping* (parts and engines): corrosion inhibitors, engine-test emissions, industrial truck exhaust.
16. *Machining maintenance*: exposures in nonskilled and skilled trades maintenance.
17. *Tool grinding*: dust, including cemented tungsten carbide (cobalt), coolant mists.

* CO, carbon monoxide; MWF, metalworking fluids.

machining populations.[11–24] This study addressed the following prior hypotheses: (1) stomach cancer associated with water-based grinding fluids potentially containing nitrosamines[11,12,15,16,22]; (2) pancreas cancer associated with straight oil machining fluids[16,22] or other engine plant exposures[13,14,20,22,23]; (3) cirrhosis of the liver associated with MWF[15,16,19,22]; (4) lung cancer associated with foundry exposures,[3,5–10] silica,[25] core-making,[26] heat treatment,[7,15,16] and engine testing[13,22]; (5) NMRD associated with silica and other dust exposures in foundry cleaning and finishing operations,[4–10] core-making,[8] heat treatment,†[22] and engine testing[22]; (6) heart disease with carbon monoxide (CO) or other foundry exposures[3,6–10] and with unknown machining plant exposures[22]; and (7) diabetes[13,22] and stroke[15,22] with unknown machining plant exposures and diabetes with heat-treat exposures.†

† Park RM, Krebs JM, Mirer FE. Mortality in an automotive forging plant. Unpublished report for UAW/GM, Detroit, February 22, 1992.

Subjects and Methods

Study Population

The two machining/assembly plants opened in 1951 and 1955; the foundry began in 1952. Semiannual data tapes provided to the union since 1966 identified active members and their department, skill status, birth year, gender, and (usually) date of hire. From 20,959 workers identified between 1966 and 1983, the study analyzed those with at least 2 years of employment who died between January 1, 1968, and December 31, 1993. Pension-related information was available at the union for 92% of the population at risk. Deaths were identified by linkage to computerized death records of the State of Ohio (1968 to 1993). Ascertainment of vital status was largely complete except for non-Ohio deaths among those leaving employment some time before death. The underlying cause of death was nosologist-coded in the Ohio death registry in the International Classification of Diseases revision appropriate for the year of death. Smoking information was unavailable.

Employment History and Exposure Assessment

Hire and termination dates were obtained from pension records or the semiannual tapes, or they were estimated. Gaps in study plant employment were disregarded.[27] Processes and exposures were described in group interviews with experienced management and union personnel. Industrial hygiene records were examined, and one or two walk-throughs of each plant were conducted. Departments were characterized (indicator variable, 0,1) in 17 exposure categories (Table 1). The 70 foundry air samples in the period 1954 to 1972 responded mostly to complaints. Dust levels higher than 50 million particles per cubic foot (mppcf) were reported, but most were 5 to 20 mppcf. Molding areas had the highest dust levels, followed by core-making and cleaning/finishing. Dust silica content was highly variable (17% to 97%) and was probably higher in cleaning/finishing, but shake-out measurements (mold dust) dominated there. Two dust groupings were defined: (1) all departments with potentially important dust levels; and (2) the nonproduction subset, such as maintenance and sand management. The "oil-sand" core-making system was replaced, starting in 1960, with the phenol-formaldehyde ("hot-box") system, used widely after 1966. The triethylamine/isocyanate resin ("cold-box") system was introduced in the 1970s. Central coolant system records were obtained back to 1963; partial plant 2 records were available from 1955. These records identified coolants known or likely to contain nitrites, alkanolamines, and nitrosamines. For example, before 1980, coolants with the code "M3C4" were assumed to have these ingredients. Examples included Lusol (Anderson Oil and Chemical Co, Portland, CT) and Vantrol (Van Stratten, Chicago,

IL) products. In 24 air sampling investigations (1964 to 1986), machining plant oil mist concentrations were mostly in the range of 0.2 to 5.0 mg/m^3, but some exceeded 10 mg/m^3. CO levels were evaluated repeatedly in engine testing after 1957 and often exceeded 75 ppm (and sometimes 300 ppm) but declined to 10 to 50 ppm after 1975. Oil smoke, as total particulate, ranged from 1 to 3 mg/m^3 in engine hot-test areas. Evaluations of heat treatment (usually induction heating and oil quench) in 1967 and 1973 found oil smoke at the 0.5-to-1.5 mg/m^3 level.

Cumulative exposures were calculated by summing durations in departments with the specified process, using latency weighting to account for the delay between exposure and subsequent death, as described previously.[16,27] Long- and short-latency weighted and unweighted measures were analyzed. Workers' departments before 1966 (when history became available) were assumed to be the same as in 1966.

Analysis

Analyses were restricted to decedents because race, an important potential confounder, was unavailable for the surviving cohort. Proportional mortality ratios, calculated using US reference rates,[28,29] did not sufficiently account for duration of exposure, latency, bias caused by healthy worker and survivor effects, or multiple exposures; therefore, models of mortality ORs[30] using logistic regression comprised most analyses. The models combined gender/race groups and incorporated as an offset the expected mortality odds derived from age-, gender-, race-, and year-specific US reference rates.[28,31–33] This approach efficiently controls demographic confounding and was used in several earlier studies.[16,27,34,35] Controls were all deaths due to causes believed not work-related (ie, excluding cancers of the stomach, pancreas, lung, and bladder, stroke, diabetes, NMRD, and cirrhosis of the liver). Heart disease deaths were allowed as controls, despite small associations with some exposures, to increase statistical power. Pulmonary tuberculosis, known to be associated with silica exposure, was included with NMRD. The general model was:

$$\ln(p/1-p) = A + \ln(r/1-r_x) + B*X_1 + C*X_2 + D*X_3 + ,$$

where r = stratum and cause-specific proportional US mortality rate; r_x = corresponding combined rate for cause of interest and excluded causes of death; X_i = duration/cumulative exposures, or indicators [0,1] of cumulative exposures >0; and A = estimate of standardized mortality OR for $X_i = 0$. The predictors examined were those for which proportional mortality ratios showed elevations, tables of unadjusted odds suggested trends, or there were prior hypotheses. Overall employment duration (latency weighted) was included as a regression term to adjust for the healthy worker survivor bias,[27,36] and exposure opportunity (employment duration, related to age, may have differed between cases and controls). Some analyses distinguished exposure time after 1974, when energy conservation efforts may have degraded plant air quality. Formaldehyde exposures were assumed in core-making work after July 1968. Effect estimates for exposure (or employment duration) were expressed as predicted ORs at the mean exposure duration (or mean employment) of exposed cases. For goodness-of-fit, observed and predicted deaths were compared in intervals of cumulative exposure or employment duration.[37] Effect estimates judged to be causative on the basis of prior hypotheses, strength of exposure-response, and plausability were used

TABLE 2
Adjusted ORs for Respiratory Diseases*

Model†	*n*	OR	95% CI	*P*
Lung cancer				
Constant		1.07	0.76–1.50	0.693
Employment duration	291	0.84	0.63–1.11	0.212
Indicator: black men	66	0.79	0.55–1.12	0.187
Indicator: skilled trade	67	0.81	0.51–1.28	0.360
Indicator: skilled trade, foundry	40	2.29	1.26–4.16	0.007
Cleaning/finishing	36	1.66	1.16–2.38	0.006
Core-making after 1967	39	1.51	1.11–2.04	0.008
Molding, black men	12	1.73	0.94–3.20	0.078
Heat treat, black men	16	2.48	1.42–4.33	0.001
Indicator: packing and shipping	20	2.52	1.25–5.05	0.009
Packing and shipping		0.63	0.42–0.95	0.026
Indicator: machining plant after 1974	124	1.58	1.14–2.20	0.006
NMRD				
Constant		1.21	0.71–2.07	0.487
Employment duration	143	0.54	0.33–0.87	0.012
Indicator: black men	45	0.99	0.61–1.59	0.955
Indicator: skilled trade	27	1.03	0.64–1.66	0.888
Indicator: heat treat	21	1.49	0.87–2.55	0.151
Molding, black men	10	2.53	1.16–5.53	0.020
Core-making after 1967, black men	8	2.70	1.25–5.84	0.012
Dry machining, black men	7	2.34	1.03–5.31	0.043
Engine assembly/test	34	1.30	0.92–1.84	0.131

* OR, odds ratio; *n*, number of cases; CI, confidence interval; NMRD, nonmalignant respiratory disease.

† By logistic regression with offset term adjusting for age, gender, race, and year. For lung cancer, long-latency weighting of exposures and duration; for NMRD, unweighted exposures and duration. Indicator − indicator variable (0,1).

TABLE 3
Adjusted ORs for Selected Malignancies*

Model†	*n*	OR	95% CI	*P*
Stomach cancer				
Constant		1.21	0.52–2.80	0.661
Employment duration	32	0.92	0.40–2.13	0.848
Indicator: skilled trade	5	0.63	0.21–1.92	0.420
Grinding (semisynthetic)	7	2.40	1.14–5.07	0.022
Maintenance (machining plants)	9	1.83	0.98–3.40	0.057
Inspection	3	1.82	0.56–5.89	0.320
Prostate cancer				
Constant		1.26	0.53–2.97	0.597
Employment duration	63	0.86	0.41–1.83	0.697
Indicator: black men	30	0.87	0.36–2.06	0.745
Indicator: skilled trade	13	1.30	0.56–3.02	0.539
Indicator: maintenance (machining plants)	18	1.20	0.46–3.14	0.706
Indicator: maintenance (machining plants), black men	9	4.15	1.05–16.4	0.043
Foundry dust	27	0.51	0.23–1.11	0.091
Foundry dust, black men	16	4.24	1.22–14.7	0.023
Leukemia				
Constant		0.42	0.12–1.52	0.187
Employment duration	23	2.01	0.62–6.59	0.247
Molding: black men	3	10.5	2.82–39.0	<0.001
Indicator: cleaning/finishing, black men	4	6.25	2.03–19.2	0.001

* For definition of abbreviations, see Table 2.

† By logistic regression with offset term adjusting for age, gender, race, and year. For stomach and prostate cancers, long-latency weighting of exposures and duration; for leukemia, short-latency weighted exposures and duration. Indicator − indicator variable (0,1).

in logistic regression-derived prediction equations to compute work-related attributable risk fraction.[38]

Results

Of 2761 decedents identified, 2546 had 2 or more years of service, including 1852 white men, 660 black men, and 34 women. Seventy-nine percent of this study population were hired before 1960, at a mean age of 36. The foundry was the plant of longest duration (long-latency weighted) for 1113 of the 2546 study subjects (40 percent of white men and 54 percent of black men). Among the deceased nonskilled machining plant workers, 6.3 percent of white versus 11 percent of black workers had spent some time (after 1966) in the foundry.

Mortality ORs

Lung cancer and NMRD. Internal comparisons with logistic regression revealed a significant ($P = 0.006$) adjusted trend for lung cancer mortality with Foundry Dust: at the mean duration of dust exposure among exposed lung cancer decedents (147 months, latency weighted), the lung cancer OR was 1.52 (95% CI, 1.13 to 2.1) (data not shown). Cleaning/Finishing and Core-Making after 1967 each showed significant trends, and there was a greater than twofold excess for foundry skilled trades workers (vs machining plant skilled trades) (Table 2). Not restricting Core-Making to the period after 1967 (hot-box process) produced a weaker effect (OR, 1.39; 95% CI, 1.01 to 1.92), which was not observed for other foundry exposures. Among black men, there was a nonsignificant trend for lung cancer in Foundry Molding but a highly significant trend in machining plant Heat Treat (Table 2). Lung cancer risk more than doubled for all workers ever in Packing/Shipping but declined with cumulative exposure. Finally, lung cancer increased among workers active after 1974 in the machining plants but not in the foundry. NMRD mortality was elevated only among black men, in Molding, in Core-Making after 1967, and in Dry Machining (Table 2). There was no association with Cleaning/Finishing. When not restricted to time after 1967, the Core-Making effect was smaller (OR, 2.1; 95% CI, 0.98 to 4.6). Nonsignificant NMRD elevations were observed for all workers ever in Heat Treat and Engine Assembly/Test. The overall employment duration effect was negative and large (Table 2).

Selected other malignant diseases. There was a suggestion of excess stomach cancer mortality with Soluble Metalworking Fluids (OR, 1.9; 95% CI, 0.93 to 3.9), but for the subset Grinding with Semisynthetics the trend was stronger, along with a marginal trend in machining Maintenance and a nonsignificant trend in Inspection (Table 3). Prostate cancer mortality was elevated for black workers in Foundry Dust and machining Maintenance, and leukemia among black men showed a strong trend in Molding and for those ever in Cleaning/Finishing (Table 3). Four of the seven leukemia deaths were due to acute myeloid leukemia. Pancreas cancer was significantly elevated in Melting (OR, 4.5; 95% CI, 1.30 to 16; based on three deaths) and, for black men, in Core-Making (OR, 3.6; 95% CI, 1.21 to 10.4; four deaths) and Heat Treat (OR, 3.4; 95% CI, 0.99 to 12; three deaths) (data not shown). Non-Hodgkin lymphoma (International Classification of Diseases, 9th Revision [ICD-9], Code 202; combined with multiple myeloma, ICD-9, Code 203; in Monson's rates[28]) increased for those ever in Core-Making after 1967 (OR, 2.9; 95% CI, 1.00 to 8.2), and with Machining/Grinding in Soluble MWF (OR, 2.3; 95% CI, 1.10 to 4.9), also after 1967 (data not shown; without the 1967 restriction, the ORs were smaller.)

Ischemic heart disease, stroke, and diabetes. Heart disease mortality was elevated in the foundry and in Machining plant 2 (Table 4). The plant 2 excess was largely confined

TABLE 4
Adjusted ORs for Heart Disease and Stroke*

Model†	*n*	OR	95% CI	*P*
Heart disease				
Constant		1.12	0.80–1.58	0.500
Employment duration	794	1.34	0.99–1.81	0.058
Indicator: black men	154	0.93	0.71–1.21	0.596
Indicator: skilled trade	185	0.88	0.63–1.24	0.477
Indicator: skilled trade, foundry	94	1.89	1.19–2.99	0.007
Indicator: molding	71	1.58	1.09–2.28	0.015
Cleaning/finishing, black men	33	1.40	0.88–2.25	0.158
Indicator: machining plant 2	106	1.90	1.35–2.67	<0.001
Indicator: ICD-9 epoch (after 1978)	577	0.61	0.47–0.79	<0.001
Stroke				
Constant		1.34	0.75–2.41	0.320
Employment duration	126	0.77	0.45–1.34	0.358
Indicator: black men	26	0.41	0.24–0.69	<0.001
Indicator: skilled trade	23	0.90	0.54–1.48	0.669
Foundry dust (maintenance), black men	9	2.94	1.38–6.25	0.005
Machining/grinding (soluble)	34	1.82	1.22–2.69	0.003

* ICD-9, International Classification of Diseases, 9th Revision. For definition of other abbreviations, see Table 2.

† By logistic regression with offset term adjusting for age, gender, race, and year. For heart disease, short-latency weighting of exposures and duration; for stroke, unweighted exposures and duration. Indicator − indicator variable (0,1).

to Inspection (OR, 3.9; 95% CI, 1.82 to 8.4, $n = 30$) and Packing/Shipping (OR, 3.3; 95% CI, 0.74 to 14.7, $n = 8$). The foundry excesses were in Molding, skilled trades workers, and (possibly, for black men) in Cleaning/Finishing (Table 4). These findings were based on short-latency weighting; unweighted exposures yielded less precise estimates. Heart disease deaths were reduced after 1978 (the ICD-9 epoch). For stroke mortality, there was a positive trend in Machining/Grinding with Soluble MWF for all workers, and black men had an excess in Foundry Dust (Maintenance); otherwise, they had more than a twofold overall deficit in the combined plant populations (Table 4). Diabetes mellitis deaths showed nonsignificant trends in Heat Treat (OR, 2.0; 95% CI, 0.87 to 4.6), Foundry Dust (Maintenance) (OR, 1.81; 95% CI, 0.95 to 3.48), and machining Maintenance (OR, 1.80; 95% CI, 0.97 to 3.3) (data not shown).

Liver disease. Liver cancer mortality increased in Grinding with Semisynthetics (Table 5); the effect was smaller with soluble coolants generally, which include semisynthetics (OR, 1.4; 95% CI, 0.71 to 2.8). There was a strong association for those ever in Engine Assembly/Testing, but a weaker corresponding trend (OR, 1.8; 95% CI, 0.94 to 3.6; data not shown). For nonmalignant liver disease (cirrhosis), mortality increased in foundry Cleaning/Finishing and for workers ever in Engine Assembly/Testing and Grinding with Semisynthetics (Table 5). Those ever in any soluble coolants had a deficit of cirrhosis of the liver (OR, 0.89; 95% CI, 0.41 to 1.93; data not shown).

Goodness-of-Fit and Attributable Risk Fractions

On the basis of goodness-of-fit, the lung cancer regression model appeared to be well-specified for exposures (Table 6) and for employment duration (data not shown). Small numbers precluded statistical tests. Based on statistical strength, prior hypotheses, and both exposure and biological plausibility, I made judgments on work-relatedness and estimated the numbers of work-related deaths (Tables 6 and 7). The total number of lung cancer deaths attributable to Cleaning/Finishing, Core-Making, and Heat Treat was about 27, and approximately 20 were attributable to foundry skilled trades work (Table 7). Including the machining plant excess for working after 1974, there were 92 attributable lung cancer deaths. For stomach cancer, there were 3.5 attributable deaths in Grinding with Semisynthetics. Models also fit well for nonmalignant causes. Among black men, 5.6 heart disease deaths were attributable to Cleaning/Finishing; among all workers, 15 cardiac deaths were attributable to Molding and 23 to foundry skilled trades (Table 7). Thirteen stroke deaths were attributable to Machining/Grinding in Soluble MWF (Table 6), and 5.6 NMRD deaths among black men were attributable to Molding. Five of a total of about 13 attributable cirrhosis of the liver deaths were in Cleaning/Finishing (Table 6).

Deaths plausibly caused by foundry exposures numbered 117 (11%) of foundry deaths (Table 7). In the machining plants, the deaths plausibly related to MWF, heat treat, or engine testing numbered about 47 (3%) of machining plant deaths; about 29 (2%) were attributable specifically to MWF. About 90 (6%) of machining plant deaths were attributable to work if the general lung cancer excess after 1974 was included.

Discussion

Primary Findings

Several hypothesized exposure effects have been supported by the findings of this study. Mold emissions, associated with elevated lung cancer, heart disease, and leukemia mortality, deserve careful evaluation and better controls than exist in most foundries. The multiple excess mortality in core-making (lung diseases, [possibly] pancreas cancer and non-Hodgkin lymphoma) calls for review

TABLE 5
Adjusted ORs for Liver Diseases*

Model†	*n*	OR	95% CI	*P*
Liver cancer				
Constant		0.87	0.21–3.53	0.842
Employment duration	15	0.80	0.29–2.21	0.666
Indicator: black men	4	0.89	0.27–2.96	0.847
Indicator: skilled trade	3	1.96	0.45–8.56	0.371
Grinding (semisynthetics)	5	2.61	1.18–5.80	0.018
Indicator: engine assembly/test	6	4.08	1.28–13.0	0.018
Cirrhosis of liver				
Constant		0.39	0.16–1.00	0.050
Employment duration	44	1.07	0.50–2.30	0.864
Indicator: black men	10	0.68	0.32–1.47	0.329
Indicator: skilled trade	9	1.73	0.75–3.99	0.202
Cleaning and finishing	13	1.94	1.18–3.21	0.009
Indicator: grinding (semisynthetics)	8	2.12	0.93–4.84	0.075
Indicator: engine assembly/test	15	2.12	1.08–4.16	0.030

* For definition of abbreviations, see Table 2.
† By logistic regression with offset term adjusting for age, gender, race, and year. For liver cancer, long-latency weighting of exposures and duration; for nonmalignant liver disease, unweighted exposures and duration. Indicator – indicator variable (0,1).

of process exposures: formaldehyde, other core binder chemicals, epoxy resins in core repair, mold release and cleaning agents, and specialty core coatings. There was also support for several hypothesized MWF associations (stomach and liver cancer, cirrhosis of the liver, and stroke), but cancers of the esophagus, larynx, and rectum associated with MWF in previously studied populations[15,19,21,24] were not elevated. This may reflect lower statistical power in this study or the limited use of straight-oil machining fluids (more common in the past), which are subject to higher temperatures than soluble fluids. From the more certain foundry findings, about 11% of deaths could be plausibly attributable to exposures (Table 7), despite general compliance with regulations over the 40 years of this foundry's existence.

Among 95 industrial cohorts largely devoid of workplace risk factors, the mean all-noncancer and all-cancer SMRs were less than 1.0, indicating substantial healthy worker effects for both nonmalignant and malignant causes of death[39] (Table 8). The more than 25 plants of this employer, excluding foundry and machining operations, exhibited a similar pattern for all-noncancer and all-cancer SMRs in men and included unknown work-related mortality‡ (Table 8). For this engine plant complex, Rotimi et al[1] found noncancer and cancer SMRs during 1970 to 1987 to be 0.90 and 1.02, respectively, suggesting about 10% excess mortality (Table 8). In the present study, an estimate of 6% to 8% deaths attributable to past exposures (11% foundry, 3% to 6% machining) is consistent with the estimated 10% excess mortality.

The importance of race in many of the associations, particularly in the foundry, recalls the work of Lloyd in the US steel industry,[40] wherein black workers had the highest lung cancer rates and were subsequently found to have the highest exposures (coke ovens). The foundry was the plant of greatest exposure duration more often for black than white decedents (OR, 1.8), consistent with reports of black workers in the past being placed preferentially in the foundry by the central hiring office. Also, 30% of those ever in coremaking were black compared with 53% ever in molding (OR, 2.7), indicating that foundry nonskilled assignments also differed with race.

Lung cancer. Lung cancer in Cleaning/Finishing could be attributable to (1) gas and dust emissions from hot molds in the drag-out phase prior to cleaning, (2) silica dust from abrasive removal of sand adhesions, (3) formaldehyde from degradation of grinding wheels, (4) heat treatment of castings, or (5) repair welding (asbestos heat shields; there was a mesothelioma death in 1978 among welders). The increased lung cancer risk in Core-Making after 1967, when hot-box technology produced formaldehyde levels of 1 to 5 ppm, supports a previous suggestion of formaldehyde carcinogenicity in dusty environments[41] and implies significant risk with the current formaldehyde standard (0.75 ppm). Molding, with higher dust levels that include silica and polycyclic aromatic hydrocarbons,[42,43] had lung cancer excesses only for black workers, who were likely overrepresented in (high-dust) shake-out/drag-out and, possibly, pouring (mold emission) areas compared with core-setting and mold assembly. The lung cancer association with Heat Treat, although possibly confounded by MWF, was more plausibly related to heat treatment oil smoke, according to other studies.[15,16,18,19,22] Higher lung cancer mortality among workers active after 1974 in the machining/assembly plants suggests that plantwide risks increased after a reported reduction in exhaust ventilation to conserve energy in the winter (less an issue in the foundry because of surplus heat). These lung cancer findings contrast with the dearth of observations of Austin et al[2] in the same plants over a shorter period of follow-up (18 vs 26 years). In their matched case-control design, smoking was analyzed but latency and duration in exposure categories were not addressed (except as "ever/

‡ Data from Delzell E, Austin H, Macaluso M, Honda Y, Huang N. A study of mortality among UAW–Ford employees and an evaluation of a UAW–Ford mortality surveillance system. Unpublished report. Birmingham, Alabama, March 20, 1991.

TABLE 6
Goodness-of-Fit and Attributable Cases for Selected Outcomes From Logistic Regression Models of Mortality Odds Ratios

Malignant Diseases*	Cumulative Exposure (Duration) Strata† (months)								
	0	1–5	6–12	13–25	26–50	51–100	101–200	201+	Total
Lung cancer: cleaning/finishing									
Observed‡	255	2	6	5	0	4	6	13	291
Predicted‡	253.0	2.08	5.48	2.96	2.15	3.40	11.7	9.56	290.37
Attributed‡	0.00	0.02	0.17	0.19	0.28	0.80	4.70	5.15	11.3
Lung cancer: heat treat, black men									
Observed	275	1	2	1	3	2	3	4	291
Predicted	276.8	1.02	1.67	0.73	1.04	1.15	3.55	4.46	290.37
Attributed	0.00	0.02	0.09	0.09	0.21	0.44	2.19	3.28	6.3
Stomach cancer: grinding (semisynthetics)									
Observed	25	0	1	1	0	0	4	1	32
Predicted	25.6	0.26	0.32	0.33	0.18	0.80	2.97	1.55	32.00
Attributed	0.00	0.01	0.02	0.04	0.04	0.34	1.81	1.23	3.5
Stroke: machining/grinding (soluble)									
Observed	92	0	2	1	2	0	12	17	126
Predicted	90.1	0.02	2.81	1.55	1.94	1.51	10.4	17.7	125.99
Attributed	0.00	0.00	0.09	0.10	0.21	0.29	3.73	9.07	13.5
Cirrhosis of liver: cleaning/finishing									
Observed	31	4	0	2	1	2	2	2	44
Predicted	33.4	1.41	0.81	0.67	0.84	2.91	2.84	1.15	44.00
Attributed	0.00	0.07	0.08	0.11	0.25	1.54	2.03	0.91	5.0

* Based on final models: lung cancer, Table 2; stomach cancer, Table 3; stroke, Table 4; cirrhosis of the liver, Table 5.
† Intervals selected to reasonably span distribution of exposures; 0 = no cumulative exposure.
‡ Observed: number of specific deaths observed in stratum; predicted: number predicted by final model; attributed: number of predicted deaths attributable to exposure.

never" or "usual: yes/no"), and survivor bias was not considered. The classification of exposures relating to hot mold emissions has been unclear in most studies, unlike in cleaning/finishing, where associations with lung cancer have been highly consistent.[3,5–10] In a General Motors foundry, lung cancer increased among core-room workers after the introduction of formaldehyde-based resins.[10] A subsequent Poisson regression analysis[44] identified no formaldehyde association; however, latency for this relatively recent exposure and survivor bias[27,36] was not addressed.

NMRD. The lack of excess NMRD mortality in high-dust foundry areas using internal comparisons (except for black men in Molding and in Core-Making after 1967) was unexpected. Emphysema showed higher proportional mortality ratios in relation to foundry dust for all men, but the numbers were small; coremaking accounted for most of the excess. Other NMRD deaths plausibly related to foundry dust included silicosis ($n = 1$), broncholithiasis ($n = 3$), coalworkers' pneumoconiosis ($n = 3$), and tuberculosis ($n = 2$). Proportional mortality ratios for pneumonias were consistently low in all three plants for white men (about 0.5) and for black men in the foundry (0.7) (but not in the engine plants), indicating the strong underlying healthy worker effect expected for respiratory disease.[39] The possibility that symptomatic black workers moved from foundry to machining plant jobs before 1966 (first available work history) could not be evaluated. The relatively young age of this foundry may have allowed insufficient follow-up for silica-related respiratory mortality.[45–47] The nonsignificant excess NMRD in heat treat and engine testing gave modest support to prior hypotheses.

Heart disease. CO is a cardiovascular risk factor in foundries.[48] Elevated cardiac mortality has been observed in molding and pouring in several previous studies.[3,8,9,10] Acute exposure to CO was not responsible in this study because the relative risk was lower among current than retired molders. In tunnel and bridge officers, CO in the range of 35 to 50 ppm has been associated with arteriosclerotic heart disease mortality.[49] At one foundry, CO levels exceeded 100 ppm because of cupola and mold emissions.[50] Although carbon *disulfide*, which requires special laboratory procedures, has not been reported in foundry environments, carbonyl sulfide and sulfur dioxide have been identified in mold emissions.[42] A probable ischemic heart disease agent,[51–53] carbon disulfide would be produced in green sand molds by the pyrolysis of the powdered, high-sulfur sea coal added to limit molten metal penetration. A large foundry consumes more than 100 tons of sea coal (1 to 2 tons of sulfur) *per day*. Sulfur dioxide levels of 1 to 2 ppm, reported in the pouring and finishing areas of another large iron foundry, were attributed to sea

TABLE 7
Attributable Deaths Judged to be Possibly Work-Related

Cause of Death	Exposure	Attributable Deaths*	Attributable Fraction†
Foundry			
Lung cancer			
	Cleaning/finishing	11.3	0.31
	Core-making after 1967	9.8	0.25
	Skilled trades, foundry	19.9	0.50
Prostate cancer			
	Foundry dust, black men	11.3	0.71
Leukemia			
	Cleaning/finishing, black men	3.3	0.83
	Molding, black men	2.8	0.93
Heart disease			
	Molding	15.2	0.21
	Cleaning/finishing, black men	5.6	0.17
	Skilled trades, foundry	23.2	0.25
NMRD‡			
	Molding, black men	5.6	0.56
	Core-making after 1967, black men	4.4	0.55
Cirrhosis of liver			
	Cleaning/finishing	5.0	0.38
Total attributable deaths (foundry deaths = 1113)		117.5	0.105
Machining			
Stomach cancer			
	Grinding (semisynthetics)	3.5	0.50
Liver cancer			
	Grinding (semisynthetics)	2.2	0.44
	Engine assembly/test	4.5	0.75
Lung cancer			
	Heat treat, black men	6.3	0.39
	Active after 1974	44.5	0.36
Prostate cancer			
	Maintenance (machining), black men	6.3	0.70
NHL/MM§			
	Machining/grinding (Soluble)	3.3	0.47
Stroke			
	Machining/grinding (Soluble)	13.5	0.40
Cirrhosis of liver			
	Engine assembly/test	7.7	0.51
Total attributable deaths (machining deaths = 1433)		91.8	0.064

* Based on statistically significant findings judged to have exposure and biological plausibility from logistic regression models of mortality odds ratios (Tables 2–5).

† Attributable fraction among the exposed, for specific associations; attributable fraction among the full foundry or machining population, for total attributable deaths.

‡ NMRD, nonmalignant respiratory disease.

§ NHL/MM, non-Hodgkin lymphoma/multiple myeloma (results not included in Tables 2–5).

coal.§ Sulfur dioxide would form at the mold surface by the combustion of sulfur compounds generated in the reducing atmosphere within hot molds (resembling coke ovens, a commercial source of carbon disulfide). Heart disease in shipping and inspection at plant 2 may have been related to CO from industrial trucks and engine testing. CO levels in the 1950s and 1960s in engine testing routinely exceeded 50 ppm. However, it could not be ascertained if some jobs at plant 2 were preferred assignments for high-seniority workers or those with cardiac-related restrictions, which might also explain the excess. The general reduction in heart disease mortality after 1978 seen previously[20] appears to be an artifact of ICD coding changes or the result of an improved lifestyle in the autoworker population.

§ Merchant JA, Popendorf WJ, Burmeister LF, Miller E, Jones M, Calkins PJ. Unpublished addendum to the November 1993 draft final report. Saginaw Malleable Iron Foundry. UAW-GM respiratory surveillance project extension. March 1994.

Other Hypothesized or Exploratory Findings

The stomach cancer association with grinding in semisynthetic coolants likely containing nitrosamines supports a very specific prior hypothesis.[12,15,16,22,54,55] A crankshaft supervisor reported that grinding coolant in 1965 was almost exclusively Lusol, an early semisynthetic coolant, of which this employer was one of the first users (brochure, 1975 to 1980, Anderson Oil and Chemical). Straight oils and abrasive dusts[56] have been proposed for stomach cancer; however, no excess for Machining/Grinding in Straight Oils and a smaller effect for Soluble MWF generally favors the nitrosamine hypothesis, possibly with an abrasive cofactor.

Prostate cancer mortality, associated with foundry dust and machining maintenance here, was elevated in other metalworking studies[14,22] and with exposures to petroleum[57,58] and combustion[22,59] products. Excess acute myeloid leukemia in Molding and Cleaning/Finishing suggests exposure to benzene, another poorly described effluent from hot molds.[42,60] A population-based study of Danish foundry workers observed an excess of lymphopoietic cancer among 886 deaths (SMR, 26 ÷ 17.4 = 1.49; 95% CI, 0.97, 2.19), which included six deaths from acute myeloid leukemia, but no exposure classification was available.[61]

Excess mortality from liver (and stomach) cancer and cirrhosis of the liver in semisynthetic MWF, but not in soluble MWF generally, supports a prior hypothesis and suggests an etiology including nitrosamine while arguing against alcohol causation. Elevated liver cancer was also observed in a large General Motors MWF cohort.[18,19] Alternatively,

TABLE 8
Comparisons With General Industry and With the Nonmachining or Foundry Plants of This Employer

Population	Deaths	Noncancer SMR*	Noncancer 95% CI†	Cancer SMR	Cancer 95% CI
General industry‡	123,000	0.81	0.81–0.82	0.90	0.89–0.91
Nonmachining or foundry plants, this employer (men only)§	10,076	0.79	0.77–0.081	0.93	0.90–0.96
Engine foundry and machining complex, this employer (men only)‖	2,202	0.90	0.86–0.94	1.02	0.93–1.11
Relative risk: foundry and machining complex (men only)§‖ vs nonmachining or foundry plants, this employer		1.14		1.10	

* SMR, standardized mortality ratio.

† 95% confidence interval calculated using Gaussian approximation to Poisson distribution.

‡ 95 cohorts (from 95 published studies) selected to be largely free of work-related mortality[39]; includes men and women.

§ Delzell E, Austin H, Macaluso M, Honda Y, Huang N. A study of mortality among UAW–Ford employees and an evaluation of a UAW–Ford mortality surveillance system. Unpublished report, 1991. See also Delzell E, Macaluso M, Honda Y, Austin H Mortality patterns among men in the motor vehicle manufacturing industry. *Am J Ind Med. Med*. 1993;24:471–484.

‖ Rotimi et al.[1]

some other component of semisynthetic MWF may account for liver disease, such as diethanolamine (a liver toxin in rats[62]) or chlorinated oils. In the General Motors cohort,[19] cirrhosis of the liver mortality was elevated at lower cumulative exposures of water-based MWF (relative risk, 1.9 to 2.4), consistent with a healthy worker survivor effect,[27] but not in straight oil MWF.

Excess cirrhosis of the liver in foundry cleaning/finishing was unexpected, although solubilized silica can result in pathologic effects remote from the site of deposition.[63] Alternatively, dust at this foundry contained 2% to 20% organic pyrolysis products, which could include aromatic amines and polycyclic aromatic hydrocarbons.[42] Liver damage has been reported in coke-oven workers exposed to polycyclic aromatic hydrocarbons.[64] This and a previous engine plant study observed excess cirrhosis of the liver associated with exposures to combustion products.[22]

Lower stroke mortality in black workers, observed here and elsewhere,‖[22] may reflect the advantageous health status of these workers compared with the national black population. Elevated stroke mortality was observed in two bearing plants,[15,16] at another engine plant among workers exposed to soluble MWF,[22] and among those exposed to soluble MWF in the present study. Possible etiological agents in machining fluids might include biocides containing nitro-derivatives, and sodium nitrite,[65] which are methemoglobin formers, suspected to be risk factors for cardiovascular disease.

No deaths attributable to diabetes were found among workers employed less that 20 years at another engine plant (expected, 4.2), but there were more than expected after 20 years (observed, 5; expected, 3.3)[13] (also, personal communication, Dr John Vena, State University of New York, Buffalo). Another study found excess diabetes mortality among workers exposed to straight oil MWF.[22] Observing a weak association of diabetes deaths with Heat Treat in this study offered some support for a prior hypothesis.

‖ Silverstein M, Park R, Maizlish N, Mirer F. Mortality workers of automobile assembly plants. Report to the National Institute for Occupational Safety and Health. February 27, 1985:22. NIOSH Contract No. 210–81–5104.

Methodological Issues

Incomplete death ascertainment, estimated to be about 15% percent, was unlikely to depend jointly on outcome and exposure status and thus should not have biased ORs in internal comparisons. Rotimi et al,[1] with rigorous ascertainment, identified an average of 124 deaths per year. This study, in the same population beginning 2 years earlier and continuing 6 years later, identified 106 deaths per year ($124 - 106 \div 124 = 0.145$, or 15%). Exposure misclassification resulting from insufficient individual exposure characterization and unknown work history before 1966 would cause underestimation of effects, as would the movement of symptomatic workers between plants or away from high-exposure areas. Exclusions from the control group were based on prior evidence, not exposure status, so that bias resulting would be random. Unjustified exclusions (conditions that turned out to be non–work-related) would result mainly in loss of power owing to smaller control groups. The advantages and disadvantages of using decedents as controls has been debated without clear consensus.[66–69] Confounding by smoking does not introduce serious bias in most occupational studies that use internal comparisons.[70,71] Smoking was not controlled in this analysis; however, the decline in both lung cancer and NMRD mortality with employment duration (Table 2) suggests that smoking was a *negative confounder* of exposure-response. Moreover, internal comparisons largely addressed this issue. (Exposure groups were large enough so that sustained divergences in smoking prevalence over time would be unlikely to arise by chance.) Similarly, the confounding of stomach cancer estimates by ethnicity would

be minimized by internal comparisons. Attributable risk estimates were based on the more certain, but nonetheless imprecise, estimates of exposure effects and without rigorous control for nonwork risk factors. The aggregate attributable mortality (11% for the foundry) is a composite of multiple independent excesses and thus is more certain than the individual attributable fractions.

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