FAST TRACK ARTICLE

Long-term Health Experience of Jet Engine Manufacturing Workers: II. Total and Cause-Specific Mortality Excluding Central Nervous System Neoplasms

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Objective: *As part of an exploratory investigation of an unusual occurrence of glioblastoma at one jet engine manufacturing facility located in North Haven, Connecticut (CT), we examined total and cause-specific (excluding central nervous system neoplasms) mortality rates at eight of the company's CT facilities.* ***Methods:*** *Subjects were 223,894 workers ever employed in one or more of the manufacturing facilities from 1952 to 2001. Vital status was determined through 2004 for 99% of subjects and cause of death for 95% of 68,701 deaths. We computed standardized mortality ratios (SMRs) based on US and CT state rates and modeled internal cohort rates.* ***Results:*** *We observed overall deficits in deaths based on national and state comparisons from all causes, all cancers and most of the cause of death categories examined. State comparisons revealed statistically significant excesses in deaths greater than 25% for kidney cancer (68 deaths, SMR = 1.30, CI = 1.01–1.65) and "other non-malignant respiratory disease" (291 deaths, SMR = 1.27, CI = 1.13–1.42) among subjects employed only at North Haven, and for bronchitis (713 deaths, SMR = 1.28, CI = 1.18–1.37) among all hourly workers. These excesses occurred mainly among short-term workers and hourly workers.* ***Conclusions:*** *We found no evidence of elevated mortality risks for all causes combined, all cancers combined and most of the causes of death categories examined. The pattern of findings for kidney cancer, bronchitis and other non-malignant respiratory disease, based on currently available data, suggests these excesses may be due to non-occupational risk factors or to external occupational factors. We will investigate these excesses further when detailed work history and exposure data from the companion exposure assessment project become available.* (J Occup Environ Med. *2008;50:1117–1129*)

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In 2002, in response to the perception of an unusual occurrence of glioblastoma at one jet engine manufacturing facility, we began work on a multipart epidemiology study to determine whether mortality or incidence rates from central nervous system (CNS) neoplasms were elevated at the index site or seven other of the company's Connecticut (CT)-based facilities. The epidemiology and biostatistical component of the study, conducted by the University of Pittsburgh (UPitt), Department of Biostatistics, includes historical cohort mortality and cancer incidence studies, a nested case-control study of malignant and benign neoplasms of the brain and other CNS cancer sites (termed CNS neoplasms) and a genetics study based on tissue specimens obtained from cases of malignant brain neoplasms. A companion exposure assessment project, conducted independently by the University of Illinois at Chicago (UIC), Division of Environmental and Occupational Health Sciences, is characterizing the historical work practices and exposures that occurred in each Pratt & Whitney (P&W) study plant. Ultimately, UPitt will link the work history and exposure information to examine the relationship between mortality and incidence from CNS neoplasms and past working environments of the P&W study plants.

We report here the results of our first analysis of data from our histor-

ical cohort mortality study with focus on total and cause-specific mortality patterns, excluding those from CNS neoplasms. Our detailed evaluation of mortality from CNS neoplasms is reported elsewhere.[1] The main objective of the current analysis was to determine whether mortality rates from all causes combined or from any malignant or non-malignant cause of death category (excluding CNS neoplasms) were elevated among the P&W workforce. For cause of death categories with elevated mortality rates, we also sought to identify possible occupational associations using the currently available data on workplace factors. We considered this epidemiological investigation exploratory in nature and did attempt to test any a priori etiologic hypotheses.

Materials and Methods

Details on the methods and results of our cohort enumeration, mortality tracing and cause of death ascertainment, and our statistical analysis methods, are reported elsewhere.[1] Following is a summary of these methods and results.

Cohort Enumeration

The study cohort includes all persons ever employed at one or more of the eight CT study plants from 1952 through 2001, where 1952 is the start-up date of the North Haven (NH) plant where the "index" cases arose. Plant locations and start-up dates of the other plants are: East Hartford–1930, Manchester Foundry–1943, Southington Aircraft Road–1943, Middletown–1966, Southington Newell Street–1967, Rocky Hill–1967, and Cheshire–1990. From 1957 to 1965 the Middletown site was a government-owned nuclear research laboratory, the Connecticut Advanced Nuclear Engineering Laboratory (CANEL), operated by P&W. P&W acquired the site in 1966 and began jet engine production. Work histories were collected for CANEL employees that continued to hold P&W jobs in 1966 after the acquisition; however, exposures related to CANEL jobs will not be characterized since the activities do not truly correspond to those in jet engine manufacturing and work histories for employees who did not transfer to P&W were unavailable. We enumerated the study cohort from multiple sources identified through discussion with company human resources personnel and exhaustive searches of the company's archives and facilities. The cohort enumeration was verified to be complete using internal and external data sources. The study cohort includes 223,894 subjects who contributed 7,713,434 person-years of observation during the 1952–2004 study period. Detailed characteristics of the study cohort are presented elsewhere.[1]

Vital Status Tracing and Cause of Death Ascertainment

We used our standard two-stage vital status tracing protocol to identify deaths among cohort members with unconfirmed vital status (not known from company-held records to be alive as of the study end date).[2] Deaths were coded to the underlying cause of death using the International Classification of Diseases (ICD) rules in effect at time of death. The Appendix (available online at www.joem.org) provides the cause of death categories and revision-specific ICD codes used in the general mortality analysis. We identified 68,701 subjects or 30.7% of the cohort as deceased and cause of death was obtained for 65,272 or 95.0%. Only 2066 subjects, or 0.9% of the cohort, were lost to follow-up. We were able to confirm that 138,087 subjects or 61.6% of the cohort were alive as of the study end date (12/31/2004); the remainder (6.8%) was assumed to be alive per our vital status tracing protocol.

Statistical Methods

We computed standardized mortality ratios (SMRs) based on U.S. and CT state rates using a modified life table procedure from the Occupational Cohort Mortality Program.[3] Population-weighted CT state rates were obtained from the Mortality and Population Data System maintained by UPitt (Due to Mortality and Population Data System data limitations [mortality rates for all causes combined and non-malignant causes limited to 1962–2004], expected numbers of deaths for these cause of death categories during the years 1952–61 were based on standard rates for 1962–64.).[4] County-specific rates were not used as the contiguous counties surrounding the eight study plants comprised seven of the eight counties in CT. Using the proportional allocation module in Occupational Cohort Mortality Program, person-years and observed deaths for subjects of unknown race were assigned to white or non-white categories in proportion to the factor-specific distributions of study members with known race. Statistically significant deviations of the SMRs below and above 1.00 were identified using Poisson probabilities.[5]

We used relative risk (RR) regression modeling to investigate the dependence of the internal cohort mortality rates for selected cause of death categories (modeled as time to death) on each of the study factors considered.[3,6,7] Because no non-CNS neoplasm category was of a priori interest, we selected categories for more detailed external and internal cohort rate analysis if the results of the external mortality comparisons revealed a statistically significant excess in deaths greater than 25%. We assessed the statistical significance of each main effect (expressed as a global *P*-value) with a likelihood ratio statistic and, for duration of employment (DOE) and time since first employment (TSFE), also conducted a test for linear trend (expressed as a trend *P*-value).

We computed corresponding SMRs and RRs for selected subgroups defined by worker type (short-term [<1 year] versus long-term [1+ years]), age and year of

hire, DOE and TSFE. Results were not stratified by race due to the large number of unknown values. DOE was considered as a surrogate measure of a subject's overall experience or "exposure" in the working environments of the study sites; TSFE was used to account for the combined induction and latency periods of chronic diseases, which for some diseases such as cancer, can equal 20 or more years.[8] Based upon P&W clock number data, we also assigned "payroll type" to each study member as hourly, salary or mixed. This variable was used as a surrogate measure of socioeconomic status (SES) and, if warranted, to control for potential confounding by SES in some mortality comparisons. Using currently available data, we also created a "study plant group" variable to distinguish subjects who had exclusive or partial employment at the "index" NH plant from subjects never employed at NH or whose plant affiliation is currently not known for all jobs (unspecified) (Mortality analyses of the P&W cohort are planned that will enable stratification by each of the eight study plants.). SMRs and RRs based on small numbers of observed or expected deaths were (and should be) interpreted with appropriate caution due to their relative imprecision (ie, wide confidence intervals). All tests on SMRs and RRs were done at the 0.05 significance level and no formal adjustments were made for multiple comparisons.

Results

Table 1 shows, for the total cohort during the entire 1952–2004 study period, observed deaths and SMRs for selected cause of death categories based on both US and CT state death rates. The all causes and all cancers categories are shown with and without all CNS neoplasms and all malignant CNS neoplasms, respectively (All "CNS neoplasms" and "malignant CNS neoplasms" are special cause of death categories developed for our companion paper on CNS mortality. Refer to Marsh G, Buchanich J, Youk A, et al, for corresponding ICD codes.[1]). Because of the large number of observed deaths associated with many categories, some very small deficits or excesses in deaths were statistically significant (indicated here by confidence interval on SMRs not including 1.00). The US and CT comparisons revealed similar, statistically significant overall deficits in deaths for total mortality and most of the malignant and non-malignant cause of death categories examined. The exclusion of the special CNS neoplasm categories had negligible effect on the SMRs for total or all cancer mortality. The state comparisons revealed excesses in deaths compared with US rate-based deficits for three cause of death categories: a not statistically significant 4% excess in deaths from "all other respiratory system cancer" (compared with a 24% deficit), a 39% not statistically significant excess in cancer of the eye (compared with a 9% deficit) and a statistically significant 9% excess in deaths from "bronchitis, emphysema and asthma" (compared with a 20% deficit).

Observed deaths and state rate-based SMRs during 1952–2004 are shown in Table 2 by study plant group. About two-thirds (n = 46,092) of the deaths observed in the cohort were among subjects who never worked at the NH plant, and 11,354 (16.5%) deaths were observed among subjects who worked only or partly at NH. For subjects in the only NH group, the all causes combined and many cause-specific SMRs were larger than those in the other plant groups, but remained as deficits or not statistically significant excesses in deaths. The exclusion of the special CNS neoplasm categories had little or no effect on the SMRs for total or all cancer mortality. Elevated, statistically significant SMRs were observed in the only NH group for kidney cancer (68 deaths, SMR = 1.30, CI = 1.01–1.65) and "other non-malignant respiratory disease" (O-NMRD) (291 deaths, SMR = 1.27, CI = 1.13–1.42). The overall 4% state rate-based excess for "all other respiratory system cancer" (Table 1) concentrated in the never NH group, yielding a not statistically significant 30% excess based on 41 deaths. The overall excesses in mortality from bronchitis, emphysema and asthma (Table 1) was due to a not statistically significant 9% excess in the only NH group and a statistically significant 15% excess (1117 deaths, CI = 1.08–1.22) in the never NH group.

Observed deaths and state rate-based SMRs during 1952–2004 are shown in Table 3 by payroll type. About 78% (53,516) of the total deaths occurred among hourly workers. Only 3428 (5.0%) deaths were observed among workers with mixed payroll type. For most of the cause of death categories examined, deficits in deaths were observed for workers of each payroll type and most of the deficits were statistically significant. For hourly workers, SMRs were generally larger than those observed for salaried or mixed payroll workers and for several cause of death categories we observed statistically significant excesses in deaths. These excesses included respiratory system cancer (4995 deaths, SMR = 1.09, CI = 1.06–1.12), and the subcategory, cancer of the bronchus, trachea, and lung (4761 deaths, SMR = 1.10, CI = 1.07–1.13); bronchitis, emphysema, asthma (1258 deaths, SMR = 1.22, CI = 1.16–1.29), and the subcategories bronchitis (713 deaths, SMR = 1.28, CI = 1.18–1.37), and emphysema (475 deaths, SMR = 1.22, CI = 1.11–1.33). The excesses noted in the only NH group for kidney cancer and O-NMRD (Table 2) occurred as not statistically significant 10% and 5% excesses, respectively, among the hourly workers only.

Tables 4–6 show observed deaths, SMRs (CT state comparison) and RRs by selected study factors for the three cause of death categories in Tables 2 and 3 that revealed a statistically significant, greater than 25% excess

TABLE 1
Observed Deaths and SMRs for Selected Causes of Death, US and Connecticut State Comparisons, All Workers, 1952–2004

		US		Connecticut	
Cause of Death*	Obs	SMR	95% CI†	SMR	95% CI†
All causes (including all CNS neoplasms‡)	68701	0.81	0.80–0.81	0.89	0.88–0.90
All causes (excluding all CNS neoplasms‡)	68095	0.81	0.80–0.81	0.89	0.88–0.89
All cancer (including malignant CNS neoplasms‡)	18641	0.87	0.86–0.88	0.89	0.88–0.90
All cancer (excluding malignant CNS neoplasms‡)	18179	0.87	0.86–0.89	0.89	0.87–0.90
Buccal cavity and pharynx	415	0.89	0.81–0.98	0.80	0.73–0.88
Digestive organs and peritoneum	4624	0.89	0.86–0.91	0.83	0.81–0.85
Esophagus	551	0.97	0.90–1.06	0.83	0.77–0.91
Stomach	580	0.88	0.81–0.95	0.72	0.66–0.78
Large intestine	1647	0.90	0.86–0.95	0.89	0.84–0.93
Rectum	321	0.83	0.74–0.93	0.73	0.65–0.82
Biliary passages and liver primary	429	0.82	0.74–0.90	0.84	0.76–0.92
Pancreas	953	0.89	0.83–0.95	0.85	0.79–0.90
All other digestive	143	0.82	0.69–0.96	0.84	0.71–0.99
Respiratory system	6208	0.87	0.84–0.89	0.98	0.95–1.00
Larynx	186	0.80	0.69–0.92	0.79	0.68–0.91
Bronchus, trachea, lung	5972	0.87	0.85–0.89	0.98	0.96–1.01
All other respiratory	50	0.76	0.57–1.01	1.04	0.77–1.37
Breast	561	0.79	0.73–0.86	0.75	0.69–0.82
All uterine (female only)	117	0.59	0.49–0.71	0.70	0.58–0.84
Cervix (female only)	50	0.50	0.37–0.66	0.70	0.52–0.92
Other female genital organs (female only)	167	0.72	0.62–0.84	0.73	0.62–0.85
Prostate (male only)	1341	0.88	0.84–0.93	0.89	0.85–0.94
Testis and other male genital organs (male only)	39	0.63	0.45–0.87	0.65	0.46–0.89
Kidney	492	0.96	0.88–1.05	0.99	0.90–1.08
Bladder and other urinary organs	524	1.01	0.92–1.10	0.92	0.85–1.01
Malignant melanoma of skin	269	0.81	0.72–0.91	0.83	0.73–0.94
Eye	11	0.91	0.45–1.62	1.39	0.69–2.49
Thyroid gland and other endocrine glands and related structures	48	0.72	0.53–0.96	0.70	0.52–0.93
Bone	36	0.67	0.47–0.92	0.82	0.58–1.14
All lymphatic and hematopoietic tissue	1766	0.85	0.82–0.90	0.88	0.84–0.92
Hodgkins disease	91	0.75	0.60–0.92	0.67	0.54–0.82
Non-hodgkins lymphoma	678	0.87	0.80–0.94	0.89	0.82–0.95
Leukemia and aleukemia	663	0.84	0.78–0.91	0.88	0.82–0.95
All other lymphopoietic tissue	334	0.89	0.80–0.99	0.95	0.85–1.06
All other malignant neoplasms§	1564	0.92	0.87–0.96	0.87	0.82–0.91
Benign neoplasms‖	184	0.83	0.72–0.96	0.72	0.62–0.83
Diabetes mellitus	1379	0.74	0.70–0.78	0.88	0.83–0.93
Cerebrovascular disease	3433	0.71	0.68–0.73	0.83	0.80–0.85
All heart disease	22407	0.77	0.76–0.78	0.84	0.83–0.86
Rheumatic	197	0.52	0.45–0.60	0.54	0.47–0.62
Ischemic	16698	0.75	0.74–0.77	0.87	0.85–0.88
Chronic disease of endocardium and other myocardial insufficiency	726	0.68	0.63–0.73	0.82	0.76–0.88
Hypertension with heart disease	574	0.65	0.59–0.71	0.80	0.74–0.87
All other heart disease	4212	0.91	0.88–0.93	0.79	0.77–0.82
Hypertension w/o heart disease	295	0.79	0.70–0.88	0.81	0.72–0.91
Nonmalignant respiratory disease	5319	0.78	0.76–0.81	0.91	0.89–0.94
Influenza and pneumonia	1474	0.69	0.66–0.73	0.73	0.69–0.76
Bronchitis, emphysema, asthma	1608	0.80	0.76–0.84	1.09	1.04–1.15
Bronchitis	915	0.86	0.81–0.92	1.10	1.03–1.17
Emphysema	598	0.74	0.68–0.80	1.13	1.04–1.22
Asthma	95	0.65	0.52–0.79	0.87	0.70–1.06
Other nonmalignant respiratory	2237	0.85	0.82–0.89	0.97	0.93–1.01
Ulcer of stomach and duodenum	169	0.60	0.51–0.69	0.82	0.70–0.96
Cirrhosis of liver	1254	0.71	0.67–0.75	0.74	0.70–0.78
Nephritis and nephrosis	670	0.77	0.72–0.84	0.77	0.72–0.83
All external causes of death	3727	0.52	0.51–0.54	0.74	0.72–0.77

(Continued)

TABLE 1
(Continued)

Cause of Death*	Obs	US SMR	US 95% CI†	Connecticut SMR	Connecticut 95% CI†
Accidents	2302	0.51	0.49–0.53	0.72	0.69–0.75
Motor vehicle accidents	1052	0.49	0.46–0.52	0.79	0.74–0.84
All other accidents	1250	0.53	0.50–0.56	0.67	0.63–0.71
Suicides	1068	0.65	0.61–0.69	0.87	0.82–0.93
Homicides and other external	357	0.34	0.31–0.38	0.61	0.55–0.67
All other causes§	7425	0.75	0.73–0.77	0.79	0.77–0.81
Acquired immunodeficiency syndrome (AIDS) (from 1987)	329	0.62	0.55–0.69	0.58	0.52–0.65
Unknown causes	3429				

*See online Appendix for listing of corresponding ICD codes by ICD revision.

†SMRs are statistically significant at 5% level, if the 95% confidence interval does not include 1.00.

‡"All CNS neoplasms" and "malignant CNS neoplasms" are special cause of death categories developed for our companion paper on CNS mortality. See Marsh G, Buchanich J, Youk A, et al,[1] for corresponding ICD codes.

§Does not include the CNS neoplasm category shown in online Appendix.

‖Includes benign CNS neoplasms (see online Appendix for ICD codes).

in deaths (kidney cancer, O-NMRD, and bronchitis). The analysis is limited to the main subgroup where the excesses occurred and covers the entire 1952–2004 study period. For kidney cancer among only NH workers (Table 4), external mortality comparisons revealed elevated and statistically significant SMRs for hourly workers (66/68 deaths, SMR = 1.38, CI = 1.07–1.76) and workers hired after 1959 (42 deaths, SMR = 1.57, CI = 1.13–2.12). Internal comparisons revealed that payroll type was a statistically significant predictor of mortality risk (global *P*-value = 0.047) due to a 96% excess in deaths among hourly workers (66/68 deaths, RR = 1.96, CI = 0.48–8.02).

For O-NMRD among only NH workers (Table 5), SMRs were elevated and statistically significant for many subcategories of the study factors considered, including short-term workers (140 deaths, SMR = 1.45, CI = 1.22–1.72) and hourly workers (278 deaths, SMR = 1.32, CI = 1.17–1.49). Internal comparisons found payroll type as a statistically significant predictor of mortality risk (global *P*-value = 0.009) due mostly to a 52% excess in deaths among hourly workers (278 deaths, RR = 1.52, CI = 0.81–2.89), and no evidence of an association with DOE or TSFE with or without the predictor payroll type in the model (results with predictor included not shown).

For bronchitis among all hourly workers (Table 6), SMRs were elevated and statistically significant for many subcategories of the study factors considered, including short-term workers (290 deaths, SMR = 1.47, CI = 1.31–1.65), workers in the never NH plant group (524 deaths, SMR = 1.35, CI = 1.24–1.47), age at hire less than 30 years old (315 deaths, SMR = 1.43, CI = 1.27–1.59), year of hire 1960–1969 (203 deaths, SMR = 1.45, CI = 1.26–1.67) and workers followed 30–39 years (195 deaths, SMR = 1.51, CI = 1.31–1.74) and 40+ years (461 deaths, SMR = 1.31, CI = 1.19–1.43). Internal comparisons found study plant group as a borderline statistically significant predictor of bronchitis mortality risk (global *P*-value = 0.056) due partly to the excess among never NH workers. RRs were similarly elevated in all non-baseline categories of TSFE and for 30–39 years was statistically significant (RR = 2.49, CI = 1.14–5.44). For each of the causes of death examined in Tables 4–6, internal comparisons revealed no evidence of increasing risk with increasing DOE or TSFE with or without the predictor payroll type in the model (model with predictor included not shown).

Discussion

At the total cohort level, our findings indicated a statistically significantly reduced mortality risk from all causes combined and all cancer sites combined with and without the inclusion of deaths from the special CNS neoplasm cause of death categories used in our companion paper of CNS neoplasm mortality.[1] We also found statistically significant deficits in deaths from most of the other malignant and non-malignant disease categories examined. With relatively few exceptions, these overall mortality patterns were maintained within subgroups of workers defined by study plant groups and payroll type. These favorable mortality patterns, particularly those for the long-term chronic diseases examined, are probably influenced in part by the "healthy worker effect," a relative absence of deleterious health risks in relation to employment, and the effects of continuing employment with its many benefits, such as improved health care and quality of life.

SES differences between the study population and the general US and CT state comparison populations may also have influenced the overall

TABLE 2
Observed Deaths and SMRs for Selected Causes of Death, Connecticut State Comparison, All Workers, 1952–2004, by Study Plant Group

	Only North Haven		Partially North Haven		Never North Haven		Unspecified Plant	
Cause of Death[a]	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
All causes (including all CNS neoplasms[b])	7868	0.95** (0.93–0.97)	3486	0.79** (0.77–0.82)	46092	0.90** (0.89–0.91)	11255	0.83** (0.81–0.85)
All causes (excluding all CNS neoplasms[b])	7797	0.95** (0.93–0.97)	3448	0.79** (0.77–0.82)	45691	0.90** (0.89–0.91)	11159	0.83** (0.81–0.85)
All cancer (including malignant CNS neoplasms[b])	2196	0.99 (0.95–1.03)	987	0.82** (0.77–0.87)	12470	0.90** (0.88–0.92)	2988	0.80** (0.78–0.83)
All cancer (excluding malignant CNS neoplasms[b])	2139	0.99 (0.94–1.03)	957	0.86** (0.81–0.92)	12164	0.90** (0.89–0.92)	2919	0.80** (0.78–0.83)
Buccal cavity and pharynx	63	1.03 (0.79–1.32)	16	0.54** (0.31–0.87)	287	0.85** (0.75–0.95)	49	0.56** (0.41–0.74)
Digestive organs and peritoneum	558	0.94 (0.86–1.02)	253	0.79** (0.70–0.90)	3088	0.84** (0.81–0.87)	725	0.74** (0.69–0.80)
Esophagus	60	0.73* (0.56–0.94)	32	0.85 (0.58–1.20)	369	0.85** (0.77–0.94)	90	0.83 (0.66–1.01)
Stomach	82	0.96 (0.77–1.20)	39	0.86 (0.61–1.18)	374	0.70** (0.63–0.77)	85	0.61** (0.49–0.76)
Large intestine	197	1.05 (0.91–1.20)	87	0.82 (0.65–1.01)	1123	0.91** (0.86–0.97)	240	0.72* (0.63–0.82)
Rectum	40	0.93 (0.66–1.27)	15	0.60* (0.33–0.98)	206	0.70** (0.61–0.81)	60	0.77* (0.59–0.99)
Biliary passages and liver primary	56	0.97 (0.73–1.25)	26	0.87 (0.57–1.28)	281	0.83** (0.74–0.94)	66	0.74* (0.57–0.94)
Pancreas	108	0.89 (0.73–1.08)	42	0.65** (0.47–0.88)	646	0.87** (0.81–0.94)	157	0.79** (0.67–0.92)
All other digestive	15	0.84 (0.47–1.38)	12	1.23 (0.64–2.16)	89	0.79* (0.64–0.98)	27	0.90 (0.59–1.31)
Respiratory system	727	1.06 (0.99–1.14)	325	0.87* (0.78–0.97)	4185	1.00 (0.97–1.03)	971	0.87** (0.82–0.93)
Larynx	24	0.87 (0.56–1.29)	8	0.59 (0.26–1.17)	127	0.82* (0.69–0.98)	27	0.68* (0.45–0.98)
Bronchus, trachea, lung	702	1.08 (1.00–1.16)	315	0.89* (0.79–0.99)	4017	1.00 (0.97–1.04)	938	0.88** (0.82–0.94)
All other respiratory	1	—	2	0.73 (0.09–2.62)	41	1.30 (0.93–1.76)	6	0.72** (0.27–1.58)
Breast	51	0.70** (0.52–0.92)	30	0.78 (0.52–1.11)	360	0.76** (0.68–0.84)	120	0.76** (0.63–0.90)
All uterine (female only)	16	0.95 (0.54–1.54)	1	—	87	0.82 (0.66–1.01)	13	0.36** (0.19–0.62)
Cervix (female only)	9	1.18 (0.54–2.24)	1	—	38	0.84 (0.59–1.15)	2	0.14** (0.02–0.50)
Other female genital organs (female only)	13	0.63 (0.33–1.07)	5	0.43 (0.14–1.00)	118	0.81* (0.67–0.97)	31	0.61** (0.42–0.87)
Prostate (male only)	147	0.99 (0.83–1.16)	77	0.90 (0.71–1.13)	886	0.88** (0.82–0.94)	231	0.89 (0.78–1.01)
Testis & other male genital organs (male only)	6	0.92 (0.34–2.00)	1	—	24	0.59** (0.38–0.88)	8	0.85 (0.37–1.67)
Kidney	68	1.30* (1.01–1.65)	25	0.86 (0.56–1.27)	323	0.98 (0.88–1.10)	76	0.87 (0.69–1.09)
Bladder and other urinary organs	57	1.07 (0.81–1.38)	24	0.72 (0.46–1.08)	372	0.98 (0.88–1.08)	71	0.70** (0.55–0.89)
Malignant melanoma of skin	32	0.97 (0.66–1.37)	16	0.85 (0.48–1.38)	169	0.78** (0.67–0.91)	52	0.93 (0.70–1.22)
Eye	2	2.33 (0.28–8.42)	2	4.50 (0.54–16.26)	5	0.96 (0.31–2.25)	2	1.40 (0.17–5.06)
Thyroid gland and other endocrine glands and related structures	2	0.28 (0.03–1.03)	2	0.50 (0.06–1.81)	35	0.78 (0.54–1.09)	9	0.73 (0.34–1.39)
Bone	7	1.41 (0.57–2.90)	2	0.83 (0.10–2.98)	21	0.72 (0.45–1.10)	6	0.82 (0.30–1.79)
All lymphatic and hematopoietic tissue	182	0.85* (0.73–0.99)	90	0.78* (0.63–0.96)	1193	0.90** (0.85–0.95)	301	0.86 (0.77–0.96)
Hodgkins disease	15	0.97 (0.54–1.61)	2	0.26* (0.03–0.92)	57	0.63** (0.48–0.81)	17	0.75 (0.44–1.20)
Non-hodgkins lymphoma	66	0.83 (0.64–1.05)	33	0.74 (0.51–1.04)	463	0.91* (0.83–0.99)	116	0.86 (0.71–1.03)
Leukemia and aleukemia	57	0.72* (0.54–0.93)	40	0.92 (0.66–1.26)	460	0.92 (0.84–1.01)	106	0.81* (0.66–0.98)
All other lymphopoietic tissue	44	1.14 (0.83–1.53)	15	0.76 (0.42–1.25)	213	0.92 (0.80–1.06)	62	1.01 (0.77–1.29)
All other malignant neoplasms[c]	208	1.05 (0.91–1.21)	88	0.85 (0.68–1.04)	1014	0.86** (0.80–0.91)	254	0.80** (0.71–0.91)
Benign neoplasms[d]	23	0.88 (0.56–1.32)	14	0.96 (0.53–1.61)	115	0.68** (0.56–0.81)	32	0.70* (0.48–0.98)
Diabetes mellitus	156	0.88 (0.75–1.03)	85	0.96 (0.77–1.19)	907	0.88** (0.83–0.94)	231	0.84** (0.73–0.95)
Cerebrovascular disease	350	0.86** (0.77–0.95)	168	0.74** (0.63–0.86)	2325	0.84** (0.81–0.88)	590	0.79** (0.73–0.86)
All heart disease	2486	0.94** (0.90–0.97)	1154	0.76** (0.72–0.81)	14949	0.85** (0.83–0.86)	3818	0.81** (0.78–0.83)
Rheumatic	16	0.47** (0.27–0.76)	13	0.62 (0.33–1.05)	132	0.55** (0.46–0.66)	36	0.53** (0.37–0.73)
Ischemic	1764	0.96 (0.91–1.00)	855	0.77** (0.72–0.83)	11217	0.87** (0.85–0.89)	2862	0.83** (0.80–0.86)
Chronic disease of endocardium and other myocardial insufficiency	82	0.93 (0.74–1.15)	39	0.78 (0.55–1.07)	469	0.80** (0.73–0.88)	136	0.83* (0.70–0.98)
Hypertension with heart disease	68	0.76* (0.59–0.96)	37	0.96 (0.67–1.32)	355	0.76** (0.68–0.84)	114	0.95 (0.78–1.14)
All other heart disease	556	0.93 (0.86–1.01)	210	0.69** (0.60–0.79)	2776	0.80** (0.77–0.83)	670	0.72** (0.66–0.77)
Hypertension w/o heart disease	43	1.02 (0.73–1.37)	11	0.55* (0.28–0.99)	195	0.82** (0.71–0.95)	46	0.73* (0.54–0.98)

(Continued)

TABLE 2
(Continued)

Cause of Death[a]	Only North Haven Obs	Only North Haven SMR (95% CI)	Partially North Haven Obs	Partially North Haven SMR (95% CI)	Never North Haven Obs	Never North Haven SMR (95% CI)	Unspecified Plant Obs	Unspecified Plant SMR (95% CI)
Nonmalignant respiratory disease	591	1.03 (0.95–1.11)	264	0.79** (0.70–0.90)	3597	0.93** (0.90–0.96)	867	0.82** (0.77–0.88)
Influenza and pneumonia	143	0.71** (0.60–0.84)	71	0.63** (0.49–0.79)	1003	0.74** (0.70–0.79)	257	0.70** (0.62–0.79)
Bronchitis, emphysema, asthma	157	1.09 (0.92–1.27)	79	0.91 (0.72–1.14)	1117	1.15** (1.08–1.22)	255	0.95 (0.84–1.08)
Bronchitis	97	1.18 (0.96–1.44)	47	0.94 (0.69–1.25)	634	1.16** (1.07–1.25)	137	0.89 (0.75–1.06)
Emphysema	50	1.04 (0.77–1.37)	25	0.82 (0.53–1.21)	416	1.17** (1.06–1.29)	107	1.12 (0.92–1.35)
Asthma	10	0.72 (0.34–1.32)	7	1.16 (0.47–2.40)	67	0.94 (0.73–1.20)	11	0.59 (0.29–1.05)
Other nonmalignant respiratory disease	291	1.27** (1.13–1.42)	114	0.86 (0.71–1.03)	1477	0.96 (0.91–1.01)	355	0.84** (0.76–0.93)
Ulcer of stomach and duodenum	18	0.91 (0.54–1.44)	5	0.43 (0.14–1.01)	103	0.75** (0.61–0.91)	43	1.17 (0.85–1.58)
Cirrhosis of liver	161	0.79** (0.67–0.92)	67	0.69** (0.53–0.87)	865	0.78** (0.73–0.83)	161	0.56** (0.48–0.66)
Nephritis and nephrosis	91	0.90 (0.72–1.10)	35	0.72* (0.50–0.99)	450	0.80** (0.72–0.87)	94	0.62** (0.50–0.76)
All external causes of death	512	0.76** (0.69–0.83)	148	0.54** (0.46–0.63)	2532	0.77** (0.74–0.80)	535	0.70** (0.64–0.76)
Accidents	302	0.73** (0.65–0.82)	95	0.54** (0.44–0.66)	1569	0.74** (0.70–0.78)	336	0.68** (0.61–0.75)
Motor vehicle accidents	145	0.81** (0.68–0.95)	41	0.57** (0.41–0.77)	731	0.82** (0.77–0.89)	135	0.68** (0.57–0.80)
All other accidents	157	0.67** (0.57–0.78)	54	0.52** (0.39–0.68)	838	0.68** (0.63–0.72)	201	0.68** (0.59–0.78)
Suicides	140	0.98 (0.83–1.16)	42	0.61** (0.44–0.83)	731	0.89** (0.83–0.96)	155	0.80** (0.68–0.94)
Homicides and other external	70	0.59** (0.46–0.74)	11	0.37** (0.19–0.66)	232	0.64** (0.56–0.73)	44	0.57** (0.41–0.76)
All other causes[c]	877	0.83** (0.78–0.89)	379	0.72** (0.65–0.80)	4910	0.80** (0.77–0.82)	1259	0.77** (0.73–0.82)
Acquired immunodeficiency syndrome (AIDS) (from 1987)	85	0.66** (0.53–0.82)	5	0.18** (0.06–0.42)	215	0.62** (0.54–0.71)	24	0.39** (0.25–0.59)
Unknown causes	274		163		2,430		562	
Number at Risk	26,801		11,898		153,130		32,065	

*$P < 0.05$; ** $P < 0.01$.

[a]See online Appendix for listing of corresponding ICD codes by ICD revision.

[b]"All CNS neoplasms" and "malignant CNS neoplasms" are special cause of death categories developed for our companion paper on CNS mortality. See Marsh G, Buchanich J, Youk A, et al,[1] for corresponding ICD codes.

[c]Does not include the CNS neoplasm category shown in online Appendix.

[d]Includes benign CNS neoplasms (see online Appendix for ICD codes).

favorable patterns. Due to the technological nature of the jet engine manufacturing industry, the P&W cohort, unlike many other industrial cohorts, included a relatively large number of highly skilled and highly paid managers, scientists and operators, who would be of relatively high SES in comparison with the general population. As such, payroll type (hourly, salary, mixed) in this cohort may not correspond as well to SES as in other occupational cohorts due to the high skill level, and correspondingly high pay, required of hourly operators. Within our study, however, we observed generally more favorable mortality patterns among the salaried and mixed payroll type workers compared with the hourly workers. This could be due to the fact that of the three components which comprise SES, education, income and occupation, education has been shown to be most strongly correlated with mortality.[9–11] It is possible that while hourly operators in skilled positions have relatively high incomes, they do not have correspondingly higher levels of education. Also, while higher SES is generally assumed to confer better health status and decreased mortality rates than lower SES,[12] the relationship between total and cause-specific mortality and SES is complex. SES appears to have different effects by gender[13] and by race.[14] For all-cause mortality, women show slightly less of an effect than do men.[13] The effect of SES appears to be stronger in blacks than in whites for both men and women.[15]

Our analysis of total and cause-specific mortality by study plant group and payroll type revealed statistically significant excesses in deaths for only four main cause of death categories that were not of a priori interest—kidney cancer and the residual category "all O-NMRD" in the only NH plant group, "bronchitis, emphysema and asthma" in the never NH group and among hourly workers and respiratory system cancer among hourly workers. Kidney cancer is not generally considered to be occupationally related,[16] although some occupational exposures have been implicated, including asbestos[17,18] cadmium,[19] trichloroethylene,[20] lead[19,21] chromium[19,22] and polychlorinated biphenyls.[23] Non-occupational risk factors for kidney cancer include obesity, hypertension and smoking, including a dose-response relationship with cigarette smoking.[24] The internal comparisons done as part of our detailed evaluation of kidney cancer mortality in the only NH group revealed greater risks for short-term workers, hourly workers

TABLE 3
Observed Deaths and SMRs for Selected Causes of Death, Connecticut State Comparison, All Workers, 1952–2004, by Payroll Type

	Salary		Hourly		Mixed	
Cause of Death[a]	Obs	SMR (95% CI)	Obs	SMR (95% CI)	Obs	SMR (95% CI)
All causes (including all CNS neoplasms[b])	11,757	0.72** (0.71–0.73)	53,516	0.95** (0.94–0.95)	3428	0.73** (0.70–0.75)
All causes (excluding all CNS neoplasms[b])	11,605	0.72** (0.71–0.73)	53,096	0.95** (0.94–0.96)	3394	0.73** (0.70–0.75)
All cancer (including malignant CNS neoplasms[b])	3526	0.73** (0.71–0.76)	14,105	0.95** (0.93–0.96)	1010	0.76** (0.72–0.81)
All cancer (excluding malignant CNS neoplasms[b])	3407	0.72** (0.70–0.75)	13,786	0.95** (0.94–0.97)	986	0.76** (0.72–0.81)
Buccal cavity and pharynx	58	0.57** (0.44–0.74)	344	0.88* (0.79–0.98)	13	0.42** (0.22–0.72)
Digestive organs and peritoneum	875	0.74** (0.69–0.79)	3517	0.87** (0.84–0.89)	232	0.67** (0.59–0.76)
Esophagus	63	0.49** (0.38–0.63)	468	0.93 (0.85–1.02)	20	0.50** (0.30–0.77)
Stomach	86	0.55** (0.44–0.68)	470	0.77** (0.70–0.84)	24	0.50** (0.32–0.74)
Large intestine	341	0.84** (0.75–0.93)	1223	0.91** (0.86–0.97)	83	0.72** (0.57–0.89)
Rectum	51	0.56** (0.42–0.73)	253	0.79** (0.69–0.89)	17	0.63 (0.31–1.01)
Biliary passages and liver primary	84	0.74** (0.59–0.92)	319	0.87** (0.77–0.97)	26	0.79 (0.51–1.15)
Pancreas	219	0.87** (0.75–0.99)	676	0.84** (0.78–0.91)	58	0.81 (0.62–1.05)
All other digestive	31	0.83 (0.56–1.18)	108	0.88 (0.72–1.06)	4	0.39* (0.11–0.99)
Respiratory system	936	0.66** (0.61–0.70)	4995	1.09** (1.06–1.12)	317	0.78** (0.69–0.87)
Larynx	22	0.48** (0.30–0.73)	155	0.87 (0.74–1.02)	9	0.63 (0.29–1.19)
Bronchus, trachea, lung	907	0.66** (0.62–0.71)	4761	1.10** (1.07–1.13)	304	0.78** (0.69–0.87)
All other respiratory	7	0.70 (0.28–1.44)	39	1.10 (0.78–1.50)	4	1.38 (0.38–3.53)
Breast	257	0.83** (0.73–0.94)	269	0.69** (0.61–0.78)	35	0.82 (0.57–1.13)
All uterine (female only)	40	0.64** (0.46–0.87)	74	0.78* (0.61–0.98)	3	0.33* (0.07–0.96)
Cervix (female only)	17	0.61* (0.36–0.98)	33	0.83 (0.57–1.17)	0	—
Other female genital organs (female only)	80	0.82 (0.65–1.02)	76	0.64** (0.51–0.80)	11	0.80 (0.40–1.44)
Prostate (male only)	229	0.83* (0.74–0.96)	1023	0.89** (0.84–0.95)	89	0.97 (0.78–1.20)
Testis and other male genital organs (male only)	0	—	36	0.81 (0.57–1.13)	3	0.76 (0.16–2.22)
Kidney	73	0.66** (0.52–0.83)	389	1.10 (0.99–1.22)	30	0.93 (0.63–1.33)
Bladder and other urinary organs	78	0.68* (0.53–0.84)	420	1.01 (0.92–1.11)	26	0.72 (0.47–1.06)
Malignant melanoma of skin	79	0.97 (0.77–1.21)	173	0.80** (0.68–0.92)	17	0.78 (0.45–1.25)
Eye	3	1.75 (0.36–5.12)	7	1.22 (0.49–2.50)	1	2.09 (0.05–11.63)
Thyroid gland and other endocrine glands and related structures	14	0.85 (0.46–1.42)	31	0.65* (0.44–0.93)	3	0.69 (0.14–2.02)
Bone	3	0.32* (0.07–0.95)	29	0.89 (0.60–1.28)	4	1.55 (0.42–3.97)
All lymphatic and hematopoietic tissue	393	0.85** (0.77–0.94)	1265	0.89** (0.84–0.94)	108	0.84 (0.69–1.01)
Hodgkins disease	18	0.57* (0.34–0.91)	67	0.68** (0.53–0.86)	6	0.71 (0.26–1.54)
Non-hodgkins lymphoma	148	0.82* (0.69–0.96)	486	0.91* (0.83–0.99)	44	0.87 (0.63–1.17)
Leukemia and aleukemia	145	0.84* (0.71–0.99)	479	0.90* (0.82–0.99)	39	0.81 (0.57–1.10)
All other lymphopoietic tissue	82	1.07 (0.85–1.32)	233	0.92 (0.81–1.05)	19	0.87 (0.52–1.36)
All other malignant neoplasms[c]	291	0.71** (0.63–0.80)	1179	0.92** (0.87–0.97)	94	0.82 (0.66–1.00)
Benign neoplasms[d]	44	0.79 (0.57–1.06)	125	0.68** (0.56–0.81)	15	0.95 (0.53–1.57)
Diabetes mellitus	195	0.57** (0.49–0.66)	1110	0.97 (0.91–1.03)	84	0.88 (0.70–1.09)
Cerebrovascular disease	567	0.70** (0.64–0.76)	2682	0.86** (0.83–0.90)	184	0.77** (0.66–0.89)
All heart disease	3581	0.68** (0.66–0.70)	17,761	0.90** (0.88–0.91)	1155	0.72** (0.68–0.76)
Rheumatic	38	0.50** (0.35–0.68)	146	0.55** (0.47–0.65)	13	0.60 (0.32–1.03)
Ischemic	2626	0.71** (0.68–0.73)	13,219	0.92** (0.90–0.93)	853	0.74** (0.69–0.79)
Chronic disease of endocardium and other myocardial insufficiency	127	0.69** (0.58–0.82)	564	0.86** (0.79–0.94)	35	0.66** (0.46–0.91)
Hypertension with heart disease	110	0.82** (0.68–0.99)	424	0.77** (0.70–0.84)	40	1.01 (0.72–1.38)
All other heart disease	680	0.59** (0.55–0.64)	3318	0.86** (0.83–0.89)	214	0.64** (0.56–0.73)
Hypertension w/o heart disease	48	0.65** (0.48–0.87)	234	0.86* (0.75–0.98)	13	0.62 (0.33–1.06)
Nonmalignant respiratory disease	879	0.71** (0.67–0.76)	4181	0.99 (0.96–1.02)	259	0.72** (0.64–0.82)
Influenza and pneumonia	251	0.64** (0.57–0.73)	1166	0.76** (0.72–0.81)	57	0.49** (0.37–0.63)
Bronchitis, emphysema, asthma	266	0.77** (0.68–0.87)	1258	1.22** (1.16–1.29)	84	0.87 (0.69–1.07)
Bronchitis	154	0.72** (0.61–0.84)	713	1.28** (1.18–1.37)	48	0.82 (0.60–1.09)
Emphysema	89	0.85 (0.68–1.04)	475	1.22** (1.11–1.33)	34	1.06 (0.73–1.47)

(Continued)

TABLE 3
(Continued)

Cause of Death[a]	Salary Obs	Salary SMR (95% CI)	Hourly Obs	Hourly SMR (95% CI)	Mixed Obs	Mixed SMR (95% CI)
Asthma	23	0.87 (0.55–1.31)	70	0.90 (0.70–1.13)	2	0.32 (0.04–1.17)
Other nonmalignant respiratory disease	362	0.73** (0.66–0.81)	1757	1.05 (0.99–1.10)	118	0.81* (0.67–0.97)
Ulcer of stomach and duodenum	27	0.72 (0.48–1.05)	136	0.87 (0.73–1.03)	6	0.51 (0.19–1.11)
Cirrhosis of liver	196	0.54** (0.47–0.62)	1002	0.81** (0.76–0.86)	56	0.54** (0.41–0.70)
Nephritis and nephrosis	113	0.62** (0.51–0.74)	522	0.82** (0.75–0.89)	35	0.66* (0.46–0.92)
All external causes of death	592	0.55** (0.50–0.59)	3000	0.82** (0.79–0.85)	135	0.45** (0.37–0.53)
Accidents	338	0.49** (0.44–0.55)	1872	0.80** (0.76–0.83)	92	0.47** (0.38–0.58)
Motor vehicle accidents	143	0.49** (0.41–0.57)	876	0.91** (0.85–0.97)	3	0.40** (0.27–0.56)
All other accidents	195	0.50** (0.43–0.57)	996	0.72** (0.68–0.77)	59	0.52** (0.40–0.68)
Suicides	202	0.72** (0.62–0.82)	834	0.97 (0.91–1.04)	32	0.41** (0.28–0.58)
Homicides and other external	52	0.46** (0.35–0.61)	294	0.63* (0.56–0.70)	11	0.37** (0.18–0.66)
All other causes[c]	1390	0.66** (0.66–0.73)	5682	0.83** (0.81–0.85)	353	0.62** (0.56–0.69)
Acquired immunodeficiency syndrome (AIDS) (from 1987)	28	0.28** (0.18–0.40)	294	0.65** (0.58–0.73)	7	0.26** (0.10–0.53)
Unknown causes		569		2,745		115
Number at Risk		64,718		146,693		12,483

*$P < 0.05$; ** $P < 0.01$.
[a]See online Appendix for listing of corresponding ICD codes by ICD revision.
[b]"All CNS neoplasms" and "malignant CNS neoplasms" are special cause of death categories developed for our companion paper on CNS mortality. See Marsh G, Buchanich J, Youk A, et al,[1] for corresponding ICD codes.
[c]Does not include the CNS neoplasm category shown in online Appendix.
[d]Includes benign CNS neoplasms (see online Appendix for ICD codes).

TABLE 4
Observed Deaths, Standardized Mortality Ratios (SMR-CT State Comparison) and Relative Risks[a.] (RR) for Kidney Cancer, Workers in Only North Haven Plant Group, 1952–2004, by Selected Study Factors

Study Factor	External Rate Comparison Deaths	SMR	(95% CI)	Internal Rate Comparison Non-cases[b]	RR (95% CI)	P[c]
Worker Type						
Short-term (<1 yrs)	36	1.38	0.97–1.92	155,500	1.00	0.639
Long-term (1+ yrs)	32	1.23	0.84–1.74	104,097	0.89 (0.54–1.46)	
Payroll Type						
Salary	2	0.73	0.09–2.65	12,876	1.00	
Hourly	66	1.38*	1.07–1.76	239,215	1.96 (0.48–8.02)	
Mixed	0	—	—	7506	—	0.047
Age at hire						
<30 yr	31	1.31	0.89–1.86	177,673	1.00	0.989
30+	37	1.29	0.91–1.77	81,924	1.01 (0.42–2.41)	
Year of hire						
<1960	26	1.01	0.66–1.48	87,270	1.00	
1960+	42	1.57**	1.13–2.12	172,327	1.42 (0.81–2.50)	0.225
Duration of employment (DOE) (yrs)						
<1	36	1.38	0.96–1.90	155,500	1.00	0.570 (0.312)
1–4	18	1.60	0.95–2.52	56,954	1.11 (0.62–1.96)	
5–19	7	0.85	0.34–1.75	24,793	0.65 (0.28–1.51)	
20+	7	1.16	0.47–2.38	22,350	0.72 (0.31–1.67)	
Time since first employment (TSFE) (yrs)						
<20	12	1.15	0.59–2.00	36,901	1.00	0.885 (0.578)
20–29	18	1.33	0.79–2.10	66,099	0.72 (0.26–1.95)	
30–39	23	1.19	0.76–1.79	120,829	0.62 (0.20–1.94)	
40+	15	1.69	0.95–2.79	35,768	0.65 (0.17–2.46)	

*$P < 0.05$;** $P < 0.01$.
[a]Risksets were matched on exact age, gender and date of birth (using a 1 yr caliper).
[b]The number of persons in decedents' risk sets used in calculation of RR.
[c]*P*-value: global, (trend).

TABLE 5
Observed Deaths, Standardized Mortality Ratios (SMR-CT State Comparison) and Relative Risks[a] (RR) for Other Nonmalignant Respiratory Disease (O-NMRD)[b], Workers in Only North Haven Plant Group, 1952–2004, by Selected Study Factors

	External Rate Comparison			Internal Rate Comparison		
Study Factor	Deaths	SMR	(95% CI)	Non-Cases[c]	RR (95% CI)	*P*[d]
Worker type						
Short-term (<1 yrs)	140	1.45**	1.22–1.72	332,096	1.00	0.091
Long-term (1+ yrs)	151	1.16	0.98–1.36	305,952	0.81 (0.61–1.03)	
Payroll type						
Salary	10	0.87	0.42–1.61	31,650	1.00	
Hourly	278	1.32**	1.17–1.49	582,930	1.52 (0.81–2.89)	
Mixed	3	0.42	0.09–1.22	23,468	0.42 (0.12–1.54)	0.009
Age at hire						
<30 yr	79	1.30*	1.03–1.62	297,548	1.00	0.955
30+	212	1.26**	1.10–1.45	340,500	1.01 (0.68–1.51)	
Year of hire						
<1960	176	1.36**	1.17–1.58	307,644	1.00	0.497 (0.506)
1960–69	98	1.15	0.93–1.40	294,021	0.86 (0.66–1.12)	
1970+	17	1.29	0.75–2.07	36,383	1.04 (0.60–1.79)	
Duration of employment (DOE) (yrs)						
<1	140	1.45**	1.22–1.71	332,096	1.00	0.278 (0.170)
1–4	56	1.14	0.86–1.48	135,484	0.77 (0.56–1.47)	
5–19	57	1.25	0.95–1.62	81,376	0.91 (0.66–1.26)	
20+	38	1.14	0.80–1.56	89,092	0.76 (0.53–1.11)	
Time since first employment (TSFE) (yrs)						
<20	37	1.13	0.80–1.56	71,646	1.00	0.345 (0.900)
20–29	76	1.32*	1.04–1.66	154,603	0.90 (0.55–1.47)	
30–39	104	1.25*	1.02–1.52	280,488	0.74 (0.44–1.26)	
40+	74	1.33*	1.05–1.67	131,311	0.98 (0.55–1.78)	

*$P < 0.05$; ** $P < 0.01$.
[a]Risksets were matched on exact age, gender and date of birth (using a 1 yr caliper).
[b]Excludes influenza, bronchitis, pneumonia, asthma, emphysema.
[c]The number of persons in decedents' risk sets used in calculation of RR.
[d]*P*-value: global, (trend).

and workers hired after 1959 and no evidence of an association with DOE or TSFE. This pattern of findings suggests that the kidney cancer excess may be a reflection of nonoccupational risk factors, many of which correlate with the lower SES of hourly workers and the less healthy behavior and lifestyle often found among short-term workers.[8] Also, because the only NH plant group had the largest percentages of hourly workers (91.0%) and short-term workers (57.1%),[1] subjects had ample opportunity for employment and exposures outside of P&W.

The O-NMRD category, which excludes influenza, pneumonia, bronchitis, emphysema and asthma, included, for these subjects, deaths from occupationally related diseases not likely related to P&W employment, such as silicosis and coal workers' pneumoconiosis, and deaths from diseases possibly related to employment including pneumonitis due to solids and liquids, post-inflammatory pulmonary fibrosis and chronic airways obstruction, not elsewhere classified. We know from available death certificate information that following P&W employment some men worked in the coal mines of Pennsylvania and West Virginia where some of the pneumoconioses may have developed; however, this does not appear to explain the entire O-NMRD excess. In a case-control study of jet engine repair workers, Kilburn[25] found excessive respiratory symptoms attributable to welding stainless steel combined with cigarette smoking and concluded that manganese exposure was the responsible exposure agent. Nevertheless, our detailed internal comparisons of O-NMRD mortality in the only NH plant group found greater risks for short-term workers and hourly workers and no evidence of an association with DOE or TSFE. This pattern of findings and the short-term workers' greater opportunity for other employment suggest that the O-NMRD excess may reflect the role of external occupational factors including the known histories of coal mining work after employment at P&W.

Our finding of statistically significantly elevated bronchitis mortality among all hourly workers was similar to findings in the never NH plant

TABLE 6
Observed Deaths, Standardized Mortality Ratios (SMR-CT State Comparison) and Relative Risks[a] (RR) for Bronchitis, Hourly Workers, 1952–2004, by Selected Study Factors

	External Rate Comparison			Internal Rate Comparison		
Study Factor	**Deaths**	**SMR**	**(95% CI)**	**Non-cases[b]**	**RR (95% CI)**	***P*[c]**
Worker type						
Short-term (<1 yrs)	290	1.47**	1.31–1.65	788,924	1.00	0.188
Long-term (1+ yrs)	423	1.18**	1.07–1.30	904,794	0.90 (0.77–1.05)	
Study plant group						
Never North Haven	524	1.35**	1.24–1.47	1,137,636	1.00	0.056
Only North Haven	90	1.21	0.97–1.49	268,767	0.84 (0.67–1.05)	
Partially North Haven	28	1.09	0.73–1.58	74,183	0.80 (0.55–1.17)	
Unknown Plant	71	1.00	0.78–1.27	213,132	0.76* (0.59–0.97)	
Age at hire						
<30 yr	315	1.43**	1.27–1.59	1,097,684	1.00	0.709
30+	398	1.17**	1.05–1.29	596,034	1.04 (0.86–1.26)	
Year of hire						
<1960	475	1.19**	1.09–1.31	939,520	1.00	0.721 (0.573)
1960–69	203	1.45**	1.26–1.67	653,301	1.08 (0.90–1.30)	
1970+	35	1.36	0.95–1.35	100,897	1.02 (0.71–1.46)	
Duration of employment (DOE) (yrs)						
<1	290	1.47**	1.31–1.65	788,924	1.00	0.542 (0.341)
1–4	156	1.21*	1.03–1.42	415,999	0.87 (0.72–1.06)	
5–19	151	1.11	0.94–1.30	250,940	0.94 (0.77–1.16)	
20+	116	1.23*	1.01–1.47	237,855	0.89 (0.72–1.11)	
Time since first employment (TSFE) (yrs)						
<20	12	0.34*	0.18–0.60	40,306	1.00	0.070 (0.905)
20–29	45	0.99	0.72–1.33	109,441	2.12 (0.99–4.53)	
30–39	195	1.51**	1.31–1.74	581,139	2.49* (1.14–5.44)	
40+	461	1.31**	1.19–1.43	962,832	2.19 (0.99–4.78)	

*$P < 0.05$; ** $P < 0.01$.
[a]Risksets were matched on exact age, gender and date of birth (using a 1 yr caliper).
[b]The number of persons in decedents' risk sets used in calculation of RR.
[c]*P*-value: global, (trend).

group, which included the bulk of the bronchitis deaths among hourly workers. Bronchitis was included as part of a larger cause of death category of chronic obstructive pulmonary diseases ("bronchitis, emphysema and asthma") the first two of which are caused mainly by cigarette smoking.[26] As with kidney cancer, the pattern of findings for bronchitis suggest the role of non-occupational risk factors, in particular higher rates of smoking, that correlate with the lower SES of hourly workers and the less healthy behavior and lifestyle often found among short-term workers. This same pattern may explain our finding of elevated mortality risks for respiratory system cancer among all hourly workers. We will investigate these malignant and non-malignant respiratory disease excesses further when detailed work history and exposure data become available in later phases of the overall investigation.

The results of this evaluation of total and cause-specific mortality must be interpreted with consideration given to the exploratory, hypothesis generating nature of the statistical analysis, especially considering that the overall investigation began in response to an unusual occurrence of glioblastoma at the NH facility. To minimize false positive results stemming from the multiple statistical comparisons inherent in exploratory analyses, we examined a relatively small number of available study factors and limited the number of corresponding subcategories. Study factors were categorized a priori to the extent possible and recategorized only to evenly distribute or ensure sufficient numbers of observed deaths used in risk estimate calculations. Despite these precautions, the likelihood remains that some comparisons were deemed statistically significant by chance factors alone.

Although large size and high statistical power are major strengths of this study, many very small excesses or deficits were detected as being statistically significant. Recognizing that very small excesses or deficits derived from observational epidemiology studies may not be meaningful indicators of the presence or absence of mortality risks, we placed little importance on statistical significance in these situations and focused primarily on the magnitude, patterns and consistency of our risk estimates

across the subgroups of the study population examined. Despite the large overall size of the study, we observed relatively small numbers of deaths in some subcategories of the rarer diseases examined resulting in low precision in estimating mortality risks via SMRs or RRs and low statistical power to detect possibly important mortality excesses.

The current analysis has other important limitations. Due to ongoing efforts to finalize the work histories and exposure assessment component of the investigation, we were limited here to evaluating only basic work history factors, such as study plant group, payroll type, year of hire, age at hire, DOE and TSFE. Refinements made to subjects' work histories in the ongoing exposure assessment component of this investigation will allow us to evaluate findings of interest at the detailed job level and with respect to potential exposures to classes of chemical and physical agents instead of the basic or surrogate work history factors analyzed here. It will also permit us to assign subjects to the current plant groups more completely and to perform detailed analyses of the remaining seven study plants rather than only NH.

Our historical cohort study has many unique features and methodological strengths. It is among the largest and most comprehensive historical cohort studies ever undertaken in the occupational setting. Our cohort of 233,894 subjects contributed more than 7.7 million person-years of observation, of which about 1.4 million or 18% were among workers employed more than 5 years and followed for 20 or more years from first employment. This led to the observation of 68,701 deaths through 2004, including 18,641 deaths from all cancers combined. Other major strengths of the study include: long (up to 53 years) observation periods; nearly complete cohort enumeration; internal and external validation of cohort completeness; excellent rates of vital status ascertainment and cause of death determination; the use of national and state external mortality comparisons and robust statistical modeling of internal cohort rates.

Our study is also the first large-scale, comprehensive epidemiological study of workers in the jet engine manufacturing and repair industry. Only a limited number of health studies have been conducted on allied professions or subareas of this industry including cancer incidence studies of pilots and flight attendants[27–29] several noise and hearing studies for airport personnel[30–33] toxicological studies on jet fuel, engine exhaust, and jet engine lubricating oils[34–39] and one study looking at respiratory, rheumatic and neurobehavioral symptoms among jet engine repair workers.[25] Larger studies have been conducted on aircraft maintenance workers[40–42] and aircraft manufacturing workers,[43,44] each of which found non-significant excesses of lymphatic and hematopoietic cancers, in particular non-Hodgkins lymphoma, among workers exposed to solvents, a pattern which was not seen in this cohort.

Conclusions

Our cohort mortality analysis of total and cause-specific mortality, excluding CNS neoplasms, resulted generally in very favorable mortality comparisons with the US and State of CT. We found no evidence among cohort members of elevated mortality risks for all causes combined, all cancers combined and most of the specific cause of death categories examined. Although not a priori categories of interest, we observed some isolated findings of elevated mortality risks for kidney cancer, bronchitis and a category of non-malignant respiratory disease that excluded influenza, pneumonia, bronchitis, emphysema and asthma (O-NMRD). The pattern of findings based on currently available data suggests that these excesses may be due non-occupational risk factors or to external occupational factors. We will investigate these excesses further when detailed work history and exposure data from the companion exposure assessment project become available.

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References

1. Marsh G, Buchanich J, Youk A, et al. Long-term health experience of jet engine manufacturing workers: I. Mortality from central nervous system neoplasms. *J Occup Environ Med.* 2008;50:1099–1116.
2. Buchanich J, Dolan D, Marsh G, Madrigano J. Under ascertainment of deaths using social security records: a recommended solution to a little-known problem. *Am J Epidemiol*. 2005;162:193–194.
3. Marsh G, Youk A, Stone R, Sefcik S, Alcorn C. OCMAP-PLUS, a new program for the comprehensive analysis of occupational cohort data. *J Occup Environ Med.* 1998;40:351–362.
4. Marsh G, Youk A, Sefcik S. *Mortality and Population Data System (MPDS)*. University of Pittsburgh, Department of Biostatistics Technical Report; 2005.

5. Breslow NE, Day NE. *Statistical Methods in Cancer Research*. IARC Scientific Publications Lyon: Oxford University Press; 1987.
6. StataCorp. *Stata Statistical Software: Release 9.0*. College Station, TX: StataCorp; 2004.
7. Cytel S. *LogXact 8: Discrete Regression Software Featuring Exact Methods*. Cambridge, MA: Cytel Software; 2007.
8. Checkoway H, Pearce N, Kriebel D. *Research Methods in Occupational Epidemiology*. Oxford: Oxford University Press; 2004.
9. Winkleby M, Jatulis D, Frank E, Fortmann S. Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *Am J Public Health*. 1992;82:816–820.
10. Ross CE, Wu C-L. The links between education and health. *Am Sociol Rev*. 1995;60:719–745.
11. Elo I, Preston H. Educational differences in mortality: United States, 1979–85. *Soc Sci Med*. 1996;42:47–57.
12. Gerhardt U. Ideas About Illness: An Intellectual and Political History of Medical Sociology. London: MacMillan Educational Ltd; 1989.
13. Mackenbach JP, Kunst AE, Groenhof F, et al. Socioeconomic inequalities in mortality among women and among men: An international study. *Am J Public Health*. 1999;89:1800–1806.
14. Howard G, Anderson RT, Russell G, Howard V, Burke GL. Race, socioeconomic status, and cause-specific mortality. *Ann Epidemiol*. 2000;10:214–223.
15. Anderson RT, Sorlie P, Backlund E, Johnson N, Kaplan GA. Mortality effects of community socioeconomic status. *Epidemiology*. 1997;8:42–47.
16. McLaughlin JK, Blot WJ, Devesa SS, Fraumeni JF. *Renal Cancer*. New York: Oxford University Press; 2006.
17. Selikoff IJ, Hammond EC, Seidman H. Mortality experience of insulation workers in the United States and Canada, 1943–1976. *Ann N Y Acad Sci*. 1979;330: 91–116.
18. Enterline PE, Hartley J, Henderson V. Asbestos and cancer: a cohort followup to death. *Am J Ind Med*. 1987;44:396–401.
19. Pesch B, Haerting J, Ranft U, Klimpel A, Oelschlagel B, Schill W. Occupational risk factors for renal cell carcinoma: agent-specific results from a case-control study in Germany. *Int J Epidemiol*. 2000; 29:1014–1024.
20. Bruning T, Pesch B, Wiesenhutter B. Renal cell cancer risk and occupational exposure to trichloroethylene: results from a consecutive case-control study in Arnsberg, Germany. *Am J Ind Med*. 2003;43:274–285.
21. Fu H, Boffetta P. Cancer and occupational exposure to inorganic lead compounds: a meta-analysis of published data. *Occup Environ Med*. 1995;52:73–81.
22. Parent M-E, Hua Y, Siemiatycki J. Occupational risk factors for renal cell carcinoma in Montreal. *Am J Ind Med*. 2000;38:609–618.
23. Shalat SC, True TD, Fleming LE. Kidney cancer in utility workers exposed to polychlorinated biphenyls (PCBs). *Br J Ind Med*. 1989;46:823–824.
24. McLaughlin JK, Hrubec Z, Heineman EF, Blot WJ, Fraumeni JF Jr. Renal cancer and cigarette smoking in a 26-year followup of U.S. veterans. *Public Health Rep*. 1990;105:535–537.
25. Kilburn KH. Neurobehavioral and respiratory findings in jet engine repair workers: a comparison of exposed and unexposed volunteers. *Environ Res*. 1999;80:244–252.
26. Ingram R. Chronic bronchitis, emphysema, and airways obstruction. In: Wilson JD, Isselbacher KJ, eds. *Harrison's Principles of Internal Medicine*. 13th ed. New York: McGraw-Hill; 1994:1197–1205.
27. Band PR, Le ND, Fang R. Cohort study of Air Canada pilots: mortality, cancer incidence, and leukemia risk. *Am J Epidemiol*. 1996;143:137–143.
28. Rafnsson V, Hrafnkelsson J, Tulinius H. Incidence of cancer among commercial airline pilots. *Occup Environ Med*. 2000; 57:175–179.
29. Buja A, Lange JH, Perissinotto E, et al. Cancer incidence among male military and civil pilots and flight attendants: an analysis on published data. *Toxicol Ind Health*. 2005;21:273–282.
30. Becker AB, Warm JS, Dember WN, Hancock PA. Effects of jet engine noise and performance feedback on perceived workload in a monitoring task. *Int J Aviat Psychol*. 1995;5:49–62.
31. Frendi A, Dorland WD, Maung T, Nesman T, Wang TS. A jet engine noise measurement and prediction tool. *J Acoust Soc Am*. 2002;112:2036–2042.
32. McNeil EL. The effect of jet engine harmonics on conscious and subconscious repetitive recall of music. *Aviat Space Environ Med*. 1988;59:386–387.
33. Warner RL, Bender RA. Thresholds of discomfort for complex stimuli: acoustic and sound-quality predictors. *J Speech Lang Hear Res*. 2002;45:1016–1026.
34. Tessereaux I. Risk factors of jet fuel combustion products. *Toxicol Lett*. 2004; 149:295–300.
35. van Netten C, Leung V. Comparison of the constituents of two jet engine lubricating oils and their volatile pyrolytic degradation products. *Appl Occup Environ Hyg*. 2000;15:277–283.
36. Kobayashi A, Kikukawa A. Increased formaldehyde in jet engine exhaust and changes to JP-8, lower temperature, and lower humidity irritates eyes and respiratory tract. *Aviat Space Environ Med*. 2000;71:396–399.
37. van Netten C, Leung V. Hydraulic fluids and jet engine oil: pyrolysis and aircraft air quality. *Arch Environ Health*. 2001; 56:181–186.
38. Mackerer CR, Barth ML, Krueger AJ, Chawla B, Roy TA. Comparison of neurotoxic effects and potential risks from oral administration or ingestion of tricresyl phosphate and jet engine oil containing tricresyl phosphate. *J Toxicol Environ Health*. 1999;57:293–328.
39. Miyamoto Y. Eye and respiratory irritants in jet engine exhaust. *Aviat Space Environ Med*. 1986;57:1104–1108.
40 Spirtas R, Stewart PA, Lee JS, et al. Retrospective cohort mortality study of workers at an aircraft maintenance facility. I. Epidemiological results *Br J Ind Med*. 1991;48:515–530.
41. Stewart PA, Lee JS, Marano DE. Retrospective cohort mortality study of workers at an aircraft maintenance facility: II. Exposures and their assessment. *Br J Ind Med*. 1991;48:531–537.
42. Blair A, Hartge P, Stewart PA, McAdams M, Lubin J. Mortality and cancer incidence of aircraft maintenance workers exposed to trichloroethylene and other organic solvents and chemicals: extended follow-up. *Occup Environ Med*. 1998;55: 161–171.
43. Garabrant D, Held J, Langholz B, Bernstein L. Mortality of aircraft manufacturing workers in southern California. *Am J Ind Med*. 1988;13:683–693.
44. Boice J, Marano D, Fryzek J, Sadler C, McLaughlin J. Mortality among aircraft manufacturing workers. *Occup Environ Med*. 1999;56:581–597.