**SUPPLEMENTAL FILE**

**Additional details on state of residence**

Multiple sources of address information were considered. Workers were assumed to have resided in the states where the plants were located while employed. Some addresses during the period of employment (starting as early as 1939 when the Massachusetts plant opened) were available from plant personnel records. Various tracing efforts from previous studies of this cohort [Prince, et al. 2006; Ruder, et al. 2006; Silver, et al. 2009] included the Internal Revenue Service, Post Office, and credit services. Together, these sources provided address information for 22,249 eligible workers (97%). Finally, eligible workers were also matched to LexisNexis® (a private vendor of residential information) in 2011 using first and last name, last known address, date of birth and Social Security Number (SSN); this provided additional address information for 19,235 eligible workers (84%).

Since only changes in the state of residence were relevant, these sources of address information were combined to create a residence history for each worker. State of residence was estimated for time periods with no known address information by dividing the gap at the midpoint and assigning the earlier state to the first half of the gap and the later state to the second half. For a given follow-up year, the worker was considered to be in the registry catchment if known to be living in at least one state associated with the catchment in that year.

**SIR sensitivity analyses for prostate cancer**

The primary analysis used data from the nine cancer registries to identify cases and the corresponding states to define the catchment. To evaluate the decision to expand the cancer registries beyond the states where the plants were located, life-table analyses were repeated using just the cancer registries for the three study states (and defining the catchment to be New York 1976-1981; New York and Massachusetts 1982-1986; and New York, Massachusetts, and Indiana 1987-2007).

Because state cancer registries generally will not release information about tumors only known to them through other state registries, we evaluated the potential under-ascertainment of incident cases by repeating life-table analyses additionally including prostate cancer deaths identified from our earlier mortality study that occurred in any of the nine cancer registry states [Ruder, et al. 2014] that may not have been included as cases in the primary analysis. For these, we estimated an approximate diagnosis date as seven years prior to the death date [Antonarakis, et al. 2007] and required the estimated diagnosis date to be in the catchment.

The primary analysis was limited to person-time in the first (initial) risk period; however, since others have considered disjoint risk periods when estimating SIRs [Bender, et al. 2007] we performed additional life-table analyses that considered all person-time while residing in the catchment.

In the absence of complete residential histories, Bender et al. [Bender, et al. 2006] recommended conducting uncertainty analyses to understand the limitations of the available residential history information. Our primary analysis assigned states of residence to gaps in the residential history by splitting the gap at the midpoint. To evaluate this decision, we repeated the life-table analyses assigning the entire gap to the earlier state. Next we repeated the life-table analyses assigning the entire gap to the later state.

Since the date last observed was updated based on cancer registry information for nine workers previously thought to be dead (n=1) or lost to follow-up (n=8) we repeated the life-table analyses excluding these workers because other workers lost to follow-up not known to have been diagnosed with cancer were not similarly brought forward.

**External analyses for prostate cancer**

Plant-specific prostate cancer SIRs were compared using Poisson regression models (SAS 9.2 GENMOD procedure, SAS Institute Inc., Cary, NC): the dependent variable was the number of cases (assumed to follow a Poisson distribution); the independent variables included plant indicator variables, and an offset term (with parameter fixed at 1.0) reflected the expected number of prostate cancer cases in each age and calendar-year stratum. Model parameters reflected the ratios of SIRs and can be interpreted as standardized rate ratios in the absence of a plant-age interaction [Armstrong 1995]. Similar methods were used to compare prostate cancer SIRs between short-term (<90 days of employment) and long-term workers.

**Internal analyses for prostate cancer**

Directly standardized prostate cancer incidence rates among workers with higher cumulative exposure were compared to rates among workers in the lowest cumulative exposure category. SRR 95% CIs were estimated using approximate methods [Rothman and Greenland 1998] and tests of linear trend for cumulative exposure using methods described by Rothman [Rothman 1986]. To account for potential latency, we considered exposure lag periods of 0, 10, 20, and 30 years.

Cox regression was used to estimate prostate cancer hazard ratios for workers with higher compared with lower cumulative exposure. In these analyses, age was specified as the time variable, cumulative exposure was time-dependent, and controls were matched to cases within risk sets on race and attained age. All eligible controls were included and the resulting matched risk sets were analyzed using conditional logistic regression (SAS 9.2 PHREG procedure, ibid.), equivalent to a Cox proportional hazards model stratified on race. Various transformations of cumulative exposure (continuous variable) were evaluated including square root, natural log, and restricted cubic splines. Categorical models used quintiles of the exposure distribution among cases. Confounding was evaluated for birth and calendar year. Exposure lag periods of 0 to 30 years were evaluated; the best-fitting lag period was selected based on model fit (AIC, Akaike’s Information Criterion). Cutpoints partitioning exposure into three windows by levels of hormonal activity--exposure accrued before age 23, from age 23 to age 49, and at 50 years or older – were also considered [Agalliu, et al. 2005]. Effect modification was evaluated for plant using the likelihood ratio test for interaction. To evaluate the effect of changes in prostate cancer screening and guidelines in the late 1980s, we tested for interaction between cumulative exposure and calendar year. The proportional hazards assumption was evaluated by the likelihood ratio test for interaction between age and cumulative exposure.

We repeated internal analyses (SRRs and Cox regression) after excluding short-term workers because a large percentage of the cohort had worked fewer than 90 days [Ruder, et al. 2014].

**SUPPLEMENTAL RESULTS**

**Cancer diagnoses and ascertainment**

Diagnosis dates of included cancers were based on year only for 28 matches, month and year only for 2106 matches, and complete for 1946 matches. The cancer registries were unable to provide a diagnosis year for four matches; for these, a diagnosis date was imputed as the date of death minus the approximate duration with the disease, when available (n=2), or as the midpoint of the years for which the registry was in operation (n=2).

Matching the cohort to the cancer registries led to our extending date last observed for 23 workers previously thought to be lost to follow-up and two workers previously thought to be deceased. After excluding ineligible workers who had died (n=1306) or were otherwise lost to follow-up (n=656) before their respective cancer registry was in operation, 22,903 workers were eligible for the primary cancer incidence analysis (10,993 male workers were eligible for the prostate cancer analysis). Through 2007, 7006 (31%) of the eligible workers died and 6055 (86%) of these deaths occurred in one of the nine registry states (Indiana, 559; Massachusetts, 2735; New York, 1867; California, 166; Connecticut, 77; Florida, 463; North Carolina, 57; Rhode Island, 77; and Texas, 54); the remaining deaths occurred in other states, U.S territories, or the District of Columbia (n=872), or at unknown locations (n=79) (Supplemental Table S1).

**Results of internal analyses for prostate cancer**

In separate Cox regression models, both birth year and calendar year were confounders. Results were adjusted for calendar year since prostate cancer incidence increased dramatically starting in the late 1980s when prostate-specific antigen screening began [Etzioni, et al. 1999], and continuing in 1992 when screening was recommended for asymptomatic men over 50 [American Cancer Society 2012]. Results (not shown) were similar when exposure lag periods of 10, 20, and 30 years were applied; results are presented based on a 20-year lag period which was best-fitting in an earlier analysis of prostate cancer mortality [Ruder, et al. 2014]. In simple models, prostate cancer incidence was not significantly associated with cumulative exposure (Supplemental Table S6, Models 1 and 3). Adjusting for calendar year improved model fit, but associations remained null (Supplemental Table S6, Models 2 and 4). Associations remained null (data not shown) for models that excluded short-term workers, that evaluated transformations of cumulative exposure (log, square root, and restricted cubic spline), and that evaluated exposure age windows. Plant and calendar year were evaluated and determined not to be effect modifiers. Including terms for time since last exposure did not improve model fit and the adjusted association remained null. The assumption of proportional hazards was not violated (data not shown).

**Supplemental Table S1: Cancer registries, ascertainment, and cohort deaths among 22,903 PCB cohort members eligible for the cancer incidence study**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| State | PCB plant  located  here | Complete  ascertainment  from | Cohort  deaths  through 2007 | |
| California | No | 1988 | 166 | 2.4% |
| Connecticut | No | 1973 | 77 | 1.1% |
| Indiana | Yes | 1987 | 559 | 8.0% |
| Florida | No | 1997 | 463 | 6.6% |
| Massachusetts | Yes | 1982 | 2735 | 39.0% |
| New York | Yes | 1976 | 1867 | 26.6% |
| North Carolina | No | 1999 | 57 | 0.8% |
| Rhode Island | No | 1986 | 77 | 1.1% |
| Texas | No | 1995 | 54 | 0.8% |
| Total in registry states |  |  | 6055 | 86.4% |
| Other states, territories, or District of Columbia | No |  | 872 | 12.4% |
| Unknown | No |  | 79 | 1.1% |
| Total |  |  | 7006 | 100% |

**Supplemental Table S2: Recode from ICD-O-3 codes reported by cancer incidence registries to diagnostic minor codes used in the NIOSH Lifetable Analysis System (LTAS.NET) 1**

| Major Category | Minor | Minor Category | ICD-10 Codes | ICD-O-3 Site Codes | ICD-O-3 Histology Codes |
| --- | --- | --- | --- | --- | --- |
| MN of buccal cavity and pharynx | 1 | MN of lip | C00 | C000-C009 | All excluding 9140, 9050-9055, and 9590-9989 |
| 2 | MN of tongue | C01, C02 | C019-C029 |
| 3 | MN of other buccal cavity | C03-C08 | C039-C069, C079-C089 |
| 4 | MN of pharynx | C09-C14 | C090- C119, C129-C148 |
| MN of colon and rectum | 5 | MN of colon | C18 | C180-C189 |
| 6 | MN of rectum | C19, C20 | C199, C209 |
| MN of other digestive organs and peritoneum | 7 | MN of esophagus | C15 | C150- C159 |
| 8 | MN of stomach | C16 | C160-C169 |
| 9 | MN of small intestine | C17 | C170-C179 |
| 10 | MN of biliary, liver, gall bladder | C22-C24 | C220, C221, C239-C249 |
| 11 | MN of pancreas | C25 | C250-C259 |
| 12 | MN of anus, peritoneum, other, and unspecified digestive | C21, C26, C48 | C210-C212, C218, C260, C268, C269, C422, C480-C482, C488 |
| MN of respiratory and intrathoracic organs | 13 | MN of larynx | C32 | C320-C329 |
| 14 | MN of trachea, bronchus, and lung | C33, C34 | C339-C349 |
| 15 | MN of pleura | C38.4 | C384 |
| 16 | MN of other respiratory and intrathoracic organs | C30, C31, C37, C38.0-C38.3, C38.8, C39 | C300,C301, C310-C319, C379, C380-C383, C388, C390, C398, C399 |
| MN of breast | 17 | MN of breast | C50 | C500-C509 |
| MN of female genital organs | 18 | MN of cervix uteri | C53 | C530-C539 |
| 19 | MN of other and unspecified parts of uterus | C54, C55, C58 | C540-C549, C559, C589 |
| 20 | MN of ovary, fallopian tube, and broad ligament | C56, 57.0-C57.4, C57.8 | C569-C574, C578 |
| 21 | MN of other and unspecified female genital organs | C51, C52, C57.7, C57.9 | C510-C519, C529, C577, C579 |
| MN of male genital organs | 22 | MN of prostate | C61 | C619 |
| 23 | MN of testes | C62 | C620-C629 |
| 24 | MN of other and unspecified male genital organs | C60, C63 | C600-C609, C630-C639 |
| MN of urinary organs | 25 | MN of kidney | C64-C66 | C649, C659, C669 |
| 26 | MN of bladder and other urinary organs | C67, C68, D09.0 2 | C670-C689 |
| MN of thyroid and other endocrine glands | 27 | MN of thyroid gland | C73 | C739 |
| 28 | MN of other endocrine glands | C74, C75 | C740-C749, C750-C759 |
| MN of other solid cancers | 29 | MN of bone | C40, C41 | C400-C419 |
| 30 | Malignant melanoma of skin | C43 | C440-C449 | 8720-8790 |
| 31 | Kaposi sarcoma | C46 | Not used | 9140 |
| 32 | Mesothelioma | C45 | Not used | 9050-9055 |
| 33 | MN of connective tissue | C49 | C490-C499 | All excluding 9140, 9050-9055, and 9590-9989 |
| 34 | MN brain and other parts of nervous system | C47, C70-C72 | C470-C479, C700-C729 |
| 35 | MN eye | C69 | C690-C699 |
| Malignant neoplasms of lymphatic and hematopoietic tissue | 36 | Hodgkin lymphoma | C81 | Not used | 9650- 9667 |
| 37 | Non-Hodgkin lymphoma | C82-C85, C88.0, C88.3, C91.4, C96.0-C96.3, C96.7 | Not used | 9590, 9591, 9596, 9670, 9671, 9673, 9675, 9678- 9680, 9684, 9687, 9688, 9689-9691, 9695, 9698-9702, 9705, 9708, 9709, 9712, 9714-9719, 9724-9729, 9735, 9737, 9738, 9740, 9750, 9754-9759, 9761, 9764, 9940 |
| 38 | Multiple myeloma | C90 | Not used | 9731-9734 |
| 39 | Leukemia and aleukemia | C91.0-C91.3, C91.5, C91.7, C91.9, C92-C95 | Not used | 9742, 9800, 9801, 9805, 9820, 9823, 9826, 9827, 9831-9837, 9840, 9860, 9861, 9863, 9866, 9867, 9870-9876, 9891, 9895-9897, 9910, 9920, 9930, 9931, 9945, 9946, 9948, 9963 |
| 40 | Other lymphatic and hematopoietic neoplasms | C88.2, C88.7, C88.9, C96.9, D45, D46.1-D46.4, D46.7, D46.9, D47.1, D47.3, D47.7 | Not used | 9751, 9760, 9762, 9950, 9960-9962, 9970, 9975, 9980, 9982-9987, 9989 |
| Ill-specified and residual | 41 | MN of Ill-specified and residual sites | C44, C76, C77, C80, C97 | C440-C449 | All excluding 8720-8790, 9140, 9050-9055, and 9590-9989 |
| C760-C768, C809, C420-C424, C770-C779 | All excluding 9140, 9050-9055, and 9590-9989 |

Abbreviations: ICD-O-3, International Classification of Diseases for Oncology, 3rd Edition; ICD-10, International Classification of Diseases, 10th Revision; MN, malignancy; SEER, Surveillance, Epidemiology, and End Results Program

1 Table adapted from supplemental table S5 in Daniels RD, Kubale TL, Yiin JH, Dahm MM, Hales TR, Baris D, Zahm SH, Beaumont JJ, Waters KM, Pinkerton LE. Mortality and cancer incidence in a pooled cohort of U.S. firefighters from San Francisco, Chicago and Philadelphia. Occupational and Environmental Medicine 2014;71:388-97.

2 Urinary bladder incidence cases originally coded in situ (Behavior=2) were recoded to invasive (Behavior=3) per SEER protocol.

**Supplemental Table S3: Prostate cancer standardized incidence ratios**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Analysis 1 | No.  workers 2 | PYAR | OBS | EXP | SIR | 95% CI |
| All workers | 9905 | 193,960.3 | 454 | 515.6 | 0.88 | 0.80‑0.97 |
| By plant |  |  |  |  |  |  |
| Indiana | 2208 | 37,631.0 | 99 | 123.1 | 0.80 | 0.65-0.98 |
| Massachusetts | 3319 | 64,341.4 | 167 | 180.6 | 0.92 | 0.79-1.08 |
| New York | 4378 | 91,987.8 | 188 | 211.9 | 0.89 | 0.77-1.02 |
| By employment duration |  |  |  |  |  |  |
| Short-term workers (< 90 days) | 2638 | 51,462.5 | 96 | 108.9 | 0.88 | 0.71-1.08 |
| Long-term workers (90+ days) | 7267 | 142,497.8 | 358 | 406.7 | 0.88 | 0.79-0.98 |

Abbreviations: PYAR – person-years at risk, OBS – observed number of cases, EXP – expected number of cases based on SEER rates, SIR – standardized incidence ratio, CI – confidence interval

1 The analysis included prostate cancer cases identified using the nine state cancer registries (CT, NY, MA, RI, IN, CA, TX, FL, and NC); split any gaps in the residence history at the midpoint and assigned the first half of the gap to the earlier state and the second half of the gap to the later state; and limited person-time at risk to the initial risk period (i.e., person-time at risk was censored at the date the worker was first known to be living outside the catchment).

2 The number of workers (9905) and prostate cancer cases (454) differs slightly from those reported in table 2 (9891 and 432, respectively) because the prostate cancer analysis only excluded workers with a prostate cancer diagnosis before the cancer registry begin date whereas the analysis of first primary cancer excluded workers with any cancer diagnosis before the cancer registry begin date.

**Supplemental Table S4: Prostate cancer standardized incidence ratios for the sensitivity analyses**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Sensitivity analyses | No.  workers | PYAR | OBS | EXP | SIR | 95% CI |
| S1: Included only cases from the IN, MA, and NY cancer registries 1 | 9134 | 181,478.6 | 396 | 464.3 | 0.85 | 0.77-0.94 |
| S2: Included (a) all cancer-registry identified cases (primary) and (b) death-certificate identified cases who resided in any of the nine registry states 2 | 9899 | 193,902.5 | 465 | 515.1 | 0.90 | 0.82-0.99 |
| S3: Included all risk periods 3 | 9898 | 200,632.2 | 473 | 541.7 | 0.87 | 0.80-0.96 |
| S4: Assigned entire gap to earlier state 4 | 10549 | 215,287.0 | 454 | 531.2 | 0.85 | 0.78-0.94 |
| S5: Assigned entire gap to later state 5 | 9492 | 194,584.6 | 470 | 535.5 | 0.88 | 0.80-0.96 |
| S6: Excluded “lost and found” workers 6 | 9896 | 193,821.6 | 451 | 515.1 | 0.88 | 0.80-0.96 |

Abbreviations: PYAR – person-years at risk, OBS – observed number of cases, EXP – expected number of cases based on SEER rates, SIR – standardized incidence ratio, CI – confidence interval

The primary analysis included cases identified using the nine state cancer registries (CT, NY, MA, RI, IN, CA, TX, FL, and NC); split any gaps in the residence history at the midpoint and assigned the first half of the gap to the earlier state and the second half of the gap to the later state; and limited person-time at risk to the initial risk period (i.e., person-time at risk was censored at the date the worker was first known to be living outside the catchment).

1 S1 was like the primary analysis except that it defined the catchment to be the states where the plants were located (NY, MA, and IN) and limited cases to those identified using the cancer registries affiliated with these three states.

2 S2 was like the primary analysis except that it additionally included cases from the nine registry states who were identified using death certificates.

3 S3 was like the primary analysis except that all risk periods were included (i.e., all person-time at risk in the catchment contributed to the denominator).

4 S4 was like the primary analysis except that gaps in the residence history were assigned to the earlier state.

5 S5 was like the primary analysis except that gaps in the residence history were assigned to the later state.

6 S6 was like the primary analysis except that nine “lost and found” workers were excluded.\

**Supplemental Table S5: Observed and expected numbers of incident prostate cancers, standardized incidence ratios, and directly standardized rate ratios, by exposure category** 1

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Cumulative exposure category (unit-years) 2 | PYAR | OBS | EXP | SIR | 95% CI |  | SRR | 95% CI |
| Unlagged |  |  |  |  |  |  |  |  |
| 1: <23 | 42,320.7 | 90 | 102.5 | 0.88 | 0.71-1.08 |  | 1 | (referent) |
| 2: 23-<99 | 42,019.1 | 90 | 100.1 | 0.90 | 0.72-1.11 |  | 1.06 | 0.78-1.42 |
| 3: 99-<330 | 42,457.4 | 90 | 104.9 | 0.86 | 0.69-1.05 |  | 1.03 | 0.76-1.38 |
| 4: 330-<1100 | 35,085.5 | 89 | 90.7 | 0.98 | 0.79-1.21 |  | 1.16 | 0.86-1.56 |
| 5: 1100+ | 30,220.4 | 88 | 108.8 | 0.81 | 0.65-0.997 |  | 1.00 | 0.73-1.38 |
|  |  |  |  |  |  |  |  | ptrend=0.99 |
| 20 year lag |  |  |  |  |  |  |  |  |
| 1: <23 | 74,696.8 | 90 | 107.9 | 0.83 | 0.67-1.02 |  | 1 | (referent) |
| 2: 23-<86 | 30,924.1 | 91 | 89.9 | 1.01 | 0.82-1.24 |  | 1.25 | 0.93-1.68 |
| 3: 86-<320 | 36,098.5 | 90 | 111.6 | 0.81 | 0.65-0.99 |  | 1.02 | 0.76-1.37 |
| 4: 320-<1100 | 27,792.0 | 89 | 92.0 | 0.97 | 0.78-1.19 |  | 1.18 | 0.88-1.58 |
| 5: 1100+ | 22,591.9 | 87 | 105.6 | 0.82 | 0.66-1.02 |  | 1.07 | 0.78-1.47 |
|  |  |  |  |  |  |  |  | ptrend=0.90 |

Abbreviations: PYAR – person-years at risk, OBS – observed number of cases, EXP – expected number of cases based on SEER rates, SIR – standardized incidence ratio, CI – confidence interval, SRR – standardized rate ratio, ptrend – p-value for linear trend test

1 Results exclude seven cases and 1857 PYAR with unknown cumulative exposure.

2 Categories of cumulative exposure based on the quintiles of the lag-specific case distribution.

**Supplemental Table S6: Cox regression models for prostate cancer incidence with estimated cumulative exposure (lagged by 20 years) 1**

|  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
| Model term | Model 1 | |  | Model 2 | |  | Model 3 | |  | Model 4 | |
| HR | 95% CI | HR | 95% CI | HR | 95% CI | HR | 95% CI |
| Cumulative exposure |  |  |  |  |  |  |  |  |  |  |  |
| At 1000 unit-years | 0.965 | 0.913-1.013 |  | 0.980 | 0.929-1.028 |  |  |  |  |  |  |
| Category (unit-years) |  |  |  |  |  |  |  |  |  |  |  |
| 1: <23 |  |  |  |  |  |  | 1 | (reference) |  | 1 | (reference) |
| 2: 23-<86 |  |  |  |  |  |  | 1.23 | 0.92-1.65 |  | 1.20 | 0.90-1.62 |
| 3: 86-<320 |  |  |  |  |  |  | 1.00 | 0.74-1.33 |  | 1.00 | 0.74-1.34 |
| 4: 320-<1100 |  |  |  |  |  |  | 1.16 | 0.86-1.56 |  | 1.14 | 0.85-1.54 |
| 5: 1100+ |  |  |  |  |  |  | 0.95 | 0.70-1.27 |  | 1.04 | 0.77-1.40 |
| Calendar year |  |  |  |  |  |  |  |  |  |  |  |
| <1990 |  |  |  | 1 | (reference) |  |  |  |  | 1 | (reference) |
| 1990-1994 |  |  |  | 2.73 | 1.79-4.26 |  |  |  |  | 2.71 | 1.77-4.23 |
| 1995-1999 |  |  |  | 3.42 | 2.32-5.21 |  |  |  |  | 3.41 | 2.31-5.20 |
| 2000+ |  |  |  | 3.42 | 2.38-5.12 |  |  |  |  | 3.43 | 2.38-5.12 |
|  |  |  |  |  |  |  |  |  |  |  |  |
| Likelihood ratio test for exposure |  |  |  |  |  |  |  |  |  |  |  |
| Degrees of freedom | 1 | |  | 1 | |  | 4 | |  | 4 | |
| Chi‑square | 1.98 | |  | 0.62 | |  | 4.40 | |  | 2.56 | |
| P-value | 0.16 | |  | 0.43 | |  | 0.35 | |  | 0.63 | |
|  |  |  |  |  |  |  |  |  |  |  |  |
| Model fit |  |  |  |  |  |  |  |  |  |  |  |
| -2 log likelihood | 7036.58 | |  | 6977.98 | |  | 7034.16 | |  | 6796.04 | |
| Akaike’s information criterion | 7038.58 | |  | 6985.98 | |  | 7042.16 | |  | 6990.04 | |
|  |  |  |  |  |  |  |  |  |  |  |  |

Abbreviations: HR – hazard ratio, CI – profile likelihood based confidence interval

1 For all models, controls were matched to cases within risk sets on race in addition to attained age and all eligible controls were included. Cumulative exposure (lagged by 20 years) was evaluated within risk sets at the case’s failure age and treated as a continuous variable in models 1 and 2 and as a categorical variable in models 3 and 4. The effect of cumulative exposure is adjusted for age at diagnosis in models 1 and 3 and for age at diagnosis and calendar year in models 2 and 4.

**SUPPLEMENTAL REFERENCES**

Agalliu I, Kriebel D, Quinn MM, Wegman DH, Eisen EA. 2005. Prostate cancer incidence in relation to time windows of exposure to metalworking fluids in the auto industry. Epidemiology 16(5):664-71.

American Cancer Society. 2012. Chronological history of ACS recommendations for the early detection of cancer in asymptomatic people.

Antonarakis ES, Blackford AL, Garrett-Mayer E, Eisenberger MA. 2007. Survival in men with nonmetastatic prostate cancer treated with hormone therapy: a quantitative systematic review. J Clin Oncol 25(31):4998-5008.

Armstrong BG. 1995. Comparing standardized mortality ratios. Ann Epidemiol 5(1):60-4.

Bender TJ, Beall C, Cheng H, Herrick RF, Kahn AR, Matthews R, Sathiakumar N, Schymura MJ, Stewart JH, Delzell E. 2006. Methodologic issues in follow-up studies of cancer incidence among occupational groups in the United States. Ann Epidemiol 16(3):170-9.

Bender TJ, Beall C, Cheng H, Herrick RF, Kahn AR, Matthews R, Sathiakumar N, Schymura MJ, Stewart JH, Delzell E. 2007. Cancer incidence among semiconductor and electronic storage device workers. Occup Environ Med 64(1):30-6.

Etzioni R, Legler JM, Feuer EJ, Merrill RM, Cronin KA, Hankey BF. 1999. Cancer surveillance series: interpreting trends in prostate cancer--part III: Quantifying the link between population prostate-specific antigen testing and recent declines in prostate cancer mortality. J Natl Cancer Inst 91(12):1033-9.

Prince MM, Ruder AM, Hein MJ, Waters MA, Whelan EA, Nilsen N, Ward EM, Schnorr TM, Laber PA, Davis-King KE. 2006. Mortality and exposure response among 14,458 electrical capacitor manufacturing workers exposed to polychlorinated biphenyls (PCBs). Environ Health Perspect 114(10):1508-1514.

Rothman KJ. 1986. Modern Epidemiology. Boston, MA: Little, Brown and Company.

Rothman KJ, Greenland S. 1998. Modern Epidemiology. Philadelphia, PA: Lippincott.

Ruder AM, Hein MJ, Hopf NB, Waters MA. 2014. Mortality among 24,865 workers exposed to polychlorinated biphenyls (PCBs) in three electrical capacitor manufacturing plants: A ten-year update. Int J Hyg Environ Health 217(2-3):176-187.

Ruder AM, Hein MJ, Nilsen N, Waters MA, Laber P, Davis-King K, Prince MM, Whelan E. 2006. Mortality among workers exposed to polychlorinated biphenyls (PCBs) in an electrical capacitor manufacturing plant in Indiana: an update. Environ Health Perspect 114(1):18-23.

Silver SR, Whelan EA, Deddens JA, Steenland NK, Hopf NB, Waters MA, Ruder AM, Prince MM, Yong LC, Hein MJ and others. 2009. Occupational exposure to polychlorinated biphenyls and risk of breast cancer Environ Health Perspect 117(2):276-82.