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Mortality Among Sheet Metal Workers Participating in a Respiratory Screening Program

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Background *The Sheet Metal Occupational Health Institute Trust (SMOHIT) established a screening program in 1985 to examine the health hazards of the sheet metal industry in the U.S. and Canada.*

Methods *17,345 individuals with over 20 years in the trade and who participated in the program were followed for causes of death between 1986 and 2010. Both SMRs and Cox proportional hazards models investigated predictors of death due to lung cancer, mesothelioma, and chronic obstructive pulmonary disease (COPD).*

Results *Significant excess mortality was seen for mesothelioma and asbestosis. Controlling for smoking, a strong trend for increasing lung cancer risk with increasing chest x-ray profusion >0/0 was observed. With an profusion score <1/0, FEV1 /FVC <80% was associated with lung cancer risk. COPD risk increased with increasing profusion score.*

Conclusions *This study demonstrates asbestos-related diseases among workers with largely indirect exposures and an increased lung cancer risk with low ILO scores.* Am. J. Ind. Med. 58:378–391, 2015. © 2015 Wiley Periodicals, Inc.

KEY WORDS: sheet metal worker; lung cancer; mortality; construction; asbestos

INTRODUCTION

Numerous studies have documented the health effects of occupational exposure to asbestos [Becklake, 1976; Selikoff et al., 1978; Nicholson et al., 1982; American Thoracic Society, 2004; IARC, 2009]. Based on the results of studies undertaken in the 1980s [Zoloth and Michaels, 1985; Selikoff and Lilis, 1991], the Sheet Metal Workers International Association (SMWIA) and the Sheet Metal and Air Conditioning National Association formed The Sheet Metal Occupational Health Institute Trust (SMOHIT) to examine the health impact of asbestos exposure in the sheet metal industry. This investigation updates prior reports on findings concerning mortality patterns among screening program participants, and further investigates predictors of increased mortality due to lung cancer, mesothelioma, and chronic obstructive pulmonary disease (COPD).

Sheet metal work involves fabrication or installation of metal products, such as ventilation systems, metal roofing, and metal facades, as well as large-scale production of metal products, such as refrigerators and air conditioners. Sheet metal workers are primarily employed in the construction industry but have also worked in the railroad industry and shipyards, as well as in specialized sheet metal production shops. The craft of sheet metal work handled asbestos-containing materials through the use of gaskets, but at the same time for many years sheet metal workers in construction were exposed to asbestos while working in close proximity to insulation workers applying asbestos containing materials for fireproofing and insulation, by working on or around beams that had been previously fireproofed with asbestos, and by renovating asbestos-insulated metal ventilation systems [Williams et al., 2007]. Very high levels of airborne asbestos fibers were measured during spray application of asbestos before 1973 [Paik et al., 1973], when this application method was banned.

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Prior analyses have examined the prevalence of asbestos-related diseases among this cohort of sheet metal workers [Welch et al., 1991, 1994, 2007]. Welch and colleagues [2007] reported that among 18,211 individuals 10.4% had radiographic findings consistent with asbestosis and 21.7% had pleural scarring; the prevalence of asbestos-related radiographic change increased with years worked and with any shipyard work. In a study of mortality in this same cohort [Dement et al., 2009] statistically significant excess mortality was observed for pleural cancers, mesothelioma, and asbestosis. Standardized mortality ratios for both lung cancer and COPD increased consistently and strongly with increasing ILO profusion score on the radiograph. Here we report an extended follow-up of this same group of workers.

MATERIALS AND METHODS

Cohort Definition

We have previously presented mortality results for this cohort with follow-up through 2004 [Dement et al., 2009]. The current analyses extended the mortality follow-up of this cohort through 2010 and readers are referred to the prior publication for details of the cohort and follow-up methods. Briefly, individuals who were members of the Sheet Metal Workers International Union for 20 years or more as of January 1, 1986 were invited to participate in a medical examination program provided by the Sheet Metal Occupational Health Institute Trust (SMOHIT). Previous reports on this population describe the methods in more detail and present the prevalence of asbestos-related disease on chest radiographs [Welch et al., 1991, 1994, 2007]. Components of the screening program included: (i) completion of an occupational and medical questionnaire; (ii) a limited physical examination (blood pressure determination, examination of the heart and lungs, and examination for digit clubbing); (iii) spirometry, performed according to American Thoracic Society (ATS) guidelines [American Thoracic Society, 1987, 1995]; and (iv) postero-anterior (PA) and lateral chest radiograph, interpreted using the International Labour Office (ILO) classification for pneumoconiosis [International Labour Office, 1980]. Each chest x-ray was classified by one reader who was an A-reader, a B-reader, or a physician with proficiency in the use of the ILO classification but who was neither an A nor a B reader [Welch et al., 2007]. For the current analyses, percent predicted forced vital capacity (FVC) and forced expiratory volume in one second/forced vital capacity (FEV1/FVC) ratio were calculated based on the prediction equations of Hankinson et al. [1999].

The current study cohort included 17,345 individuals who were screened for asbestos related disease by chest x-ray at 62 sites nationwide, between 1986 and 2004. We excluded 1,582 workers lacking sufficient demographic data to determine vital status. Workers may have repeated examinations; however, the current study was based on each worker's initial examination.

Two sources of information were used to obtain vital status and cause of death data: (i) records in the Sheet Metal National Pension Fund (SMNPF), and (ii) the National Death Index (NDI). Records of the SMNPF were searched, and members covered by this plan and those receiving pension benefits or still actively contributing toward their pension as of December 31, 2010 were considered alive. Workers with unknown vital status by match with the SMNPF or not identified as deceased via a previous record linkage with the NDI were followed to identify deaths and causes of deaths through December 31, 2010 using the NDI Plus system [Bilgrad, 1995], maintained by the National Center for Health Statistics (NCHS). Record linkage with the NDI was accomplished using probabilistic scores assigned by the NDI and recommended cut-off scores by class for records without a perfect match [Horm, 1996; NCHS, 2013]. We assumed that workers not identified as deceased by the NDI were still alive as of December 31, 2010 as the NDI provides virtually complete ascertainment of deaths [Stampfer et al., 1984; Boyle and Decouflė, 1990].

The study was conducted in accord with the recommendations of the Helsinki Declaration (World Medical Association, 1975) and was approved by the Institutional Review Board of Center for Construction Research and Training (CPWR). Each participant signed an informed consent to permit this use of data collected in the examinations.

Cohort Mortality Analyses

The Life Table Analysis System (LTAS.Net Version 3.0.3) developed by the National Institute for Occupational Safety and Health (NIOSH) [Steenland et al., 1990; Robinson et al., 2006] was utilized to compute cause-specific Standardized Mortality Ratios (SMRs), comparing the mortality experience of the cohort to that of the U.S. national population for 119 causes of death, adjusting for age, race, sex, and calendar year. The chest x-ray date was selected as the starting point for person-years accumulation for each cohort member and person-years accumulated until death or the study cut-off date of December 31, 2010. SMRs were calculated as the ratio of observed to expected deaths, and 95% confidence intervals for SMRs were computed under the assumption that the observed number of deaths in the cohort is a Poisson random variable. The NIOSH LTAS uses the Byar approximation when the number of cases is six or more and the exact Poisson confidence interval when the number of cases is five or fewer [Rothman and Boise, 1979; NIOSH, 2011]

In addition to overall results for the entire cohort, we investigated mortality for selected causes by time since entry into the sheet metal trade, ILO parenchymal profusion category, and presence or absence of pleural changes. Parenchymal profusion categories were grouped into four categories as was done by Cullen et al. [2005]. A pleural abnormality was defined as bilateral pleural thickening or plaques, with or without calcification [Cullen et al., 2005].

Multivariate Modeling of Lung Cancer, Mesothelioma, and COPD Mortality Predictors

Within the overall cohort, further analyses were undertaken to examine the association between chest x-ray readings, spirometry, work history, and smoking; and mortality due to lung cancer, mesothelioma, and COPD. Analyses of the relationship between chest x-ray image changes and lung cancer mortality were restricted to White males with 20 or more years of work in the sheet metal trade for whom data was available for other covariates considered in the models. Other race groups and females were excluded from these analyses due to small numbers. In the main analyses, only workers with spirometry meeting 1994 ATS criteria for repeatability between maneuvers were included. Other investigators have suggested that exclusion of workers not meeting reproducibility criteria could introduce bias through exclusion of workers with accelerated loss of lung function [Eisen et al., 1984]. To investigate this issue, we conducted additional sensitivity analyses to determine effects of eliminating workers with spirometry not meeting ATS criteria.

Stratified Cox models were fitted using the SAS PHREG procedure with age as the time axis and model strata defined by smoking status at examination (never, past, and current) and birth cohort decade [Pencina et. al., 2007]. The stratified Cox models thus adjusted for age and calendar time period (birth cohort) and allowed the form of the underlying hazard function to vary across levels of stratification variables. Covariates considered in the baseline models for each disease included pack-years of smoking (0, 1–19, 20–39, or $\geq$40 pack-years), time since last work in the sheet metal trade ($<$5, 5–9, or $\geq$10 years), and years of sheet metal work (20–24, 25–29, 30–34, or $\geq$35 years). Pack-years and years of sheet metal work were modeled as grouped linear variables, constructed by assigning ordinal scores to categories based on category median values, and fitted as continuous variables in a manner similar to the study by Cullen et al. [2005]. Asbestos related chest radiographic predictors (presence of pleural abnormalities, and ILO parenchymal profusion category) and spirometry classifications for percent predicted FVC, percent predicted FEV_1, and FEV1/FVC ratio ($\geq$80, 70–79, 60–69, and $<$60%) were investigated in the models as categorical variables, and tests for trends across covariate category were performed by entering the covariate in the model as a grouped linear variable [Rothman and Greenland, 1998].

After assessing the magnitude of the effect of each single variable on outcome, covariates that were significant in a univariate model (likelihood ratio P-values $<$0.1) were considered candidate variables for inclusion in multivariate models for each disease. Covariates were retained in the final models if statistically significant based on their Wald Type 3 chi-square or if their inclusion in the multivariate models changed the parameter estimates for pleural changes, parenchymal changes, or spirometry more than 10 percent. For some complex models such as spirometry and chest x-ray predictors of COPD mortality, we did not seek parsimony but rather present full and reduced models to allow readers to better judge predictors of mortality. We restricted our analyses of spirometry predictors of lung cancer mortality to a sub-cohort with spirometry and an ILO profusion score $<$1/0. For analyses of mesothelioma risk, cases included mesotheliomas (ICD-10) and pleural cancers (ICD-9), and parenchymal profusion was dichotomized into $<$1/0 and $\geq$1/0 due to small numbers in higher profusion categories. Analyses of COPD risks were restricted to the sub-cohort with spirometry, and parenchymal profusion was dichotomized into $<$1/0 and $\geq$1/0 due to small numbers in higher profusion categories.

Cox proportional hazards models were fit using PROC PHREG in SAS Version 9.3 [SAS, 2011]. The EXACT method of handing ties in PROC PHREG was used and the assumption of proportional hazards over the follow-up period was assessed with time-dependent covariates (the product of log-transformed time and the factor of interest). The ASSESS option for testing the proportional hazard assumption available in SAS Version 9.3 was also used for this purpose.

RESULTS

There were 6,636 deaths as of December 31, 2010 among the 17,345 workers in the cohort (Table I). The cohort was almost entirely male and White with a mean age of 57.4 years at intake exam. Twenty-six percent of the cohort had never smoked cigarettes, and 25.4% were still smoking at the time of their intake exam. Radiographic parenchymal changes (profusion $>$1/0) were observed in 10.4% of workers, and 21.7% had radiographic pleural changes. There were 808 deaths from lung cancer, 85 deaths from mesothelioma with an additional 11 deaths coded to malignant neoplasm of the pleura, and 461 deaths from COPD.

Standardized mortality ratio analyses showed a significant deficit for all causes of death (Table II). The SMR for

TABLE I. Sheet Metal Worker Cohort Demographic Characteristics and Vital Status

Characteristic	Total cohort	Lung cancer deaths
Number of Workers	17,345	808
Total Deaths, December 31, 2010	6,636	—
Percent Male	99.8%	100%
Percent White	99.2%	99.4
Age at Intake Exam (Mean, (SD))	57.4(8.7)	61.2 (7.7)
Smoking Status at Intake Exam (No. (%))[1]		
Never Smoked	26.0%	6.6%
Past Smoker	48.6%	42.7%
Current Smoker	25.4%	50.7%
Smoking Pack-Years for Ever Smoked (Mean, (SD))	32.3 (22.4)	44.3 (24.4)
Years of Sheet Metal Trade Work (Mean, (SD))	31.9 (7.2)	33.5 (7.7)
Prevalence of Radiographic Parenchymal Changes[2]	10.4%	19.7%
Prevalence of Radiographic Pleural Changes[3]	21.7%	26.1%

[1]24 workers missing smoking data.
[2]ILO profusion of small irregular shadows in the lung parenchyma $\geq$ category 1/0.
[3]Any notations of positive findings in sections 3A-D of the NIOSH ILO coding form.

malignant neoplasm of the trachea, bronchus, and lung was 1.03 which was not significantly elevated. The SMR for mesothelioma and for malignant neoplasms of the pleura were both significantly elevated. The SMR was significantly decreased for a number of causes of death, including heart disease and diseases of the respiratory system, with the exception of a significantly elevated SMR of 11.74 for asbestosis.

Table III displays mortality for selected causes of death by time since entry into the sheet metal trade, a variable that reflects both duration of exposure and latency. The SMR for lung cancer did not increase with time since entry into the trade. The vast majority of deaths from asbestosis, mesothelioma and malignant neoplasm of the pleura occurred after 40 years from first exposure. COPD mortality was of borderline statistical significance among workers 50 + years of age.

Table IV displays mortality by increasing amount of parenchymal disease on chest x-ray. The SMRs for lung cancer and asbestosis increased significantly with increasing profusion of parenchymal abnormalities. Table V shows that deaths from asbestosis were significantly higher among workers with pleural changes, but pleural changes were not associated with a higher SMR for other conditions. COPD mortality also increased significantly with increasing profusion category and was significantly elevated among workers in profusion categories 1/0–1/2 and 2/1–2/3. Only one death occurred among workers with higher profusion, resulting in unstable risk estimates.

Mortality from selected causes by chest x-ray pleural category is presented in Table V. While mortality risk for mesothelioma and asbestosis was significantly elevated among workers without pleural changes, risk was further increased among those with pleural changes. Workers with pleural changes also were at significantly increased risk of death due to COPD.

Tables VI Tables VI–IX present results of the Cox models for lung cancer, mesothelioma, and COPD. The hazard ratio for lung cancer death increased significantly as the profusion category on chest x-ray increased, and also increased with age, smoking, years in sheet metal trade beyond 20 years, and years since last sheet metal trade work at exam. A test for linear trend for lung cancer risk by profusion category was highly significant ($P < 0.001$). There was no significant relationship between lung cancer deaths and the presence or absence of pleural abnormalities after parenchymal changes, smoking and other covariates were entered into the model. Our Cox model for lung cancer controlled for smoking through stratification on smoking status and inclusion of pack-years of smoking as a model covariate. We ran an additional model (not shown) restricted to workers who reported having never smoked (4,323 workers and 50 lung cancers) and obtained comparable results. In this reduced model parenchymal changes were significantly associated with lung cancer risk ($P < 0.001$). Non-smokers with profusion $\geq$1/0 were at significant risk of lung cancer (HR = 2.19, 95% CI = 1.01–4.72) and pleural changes alone did not increase the risk of lung cancer.

Among workers with an ILO profusion score <1/0, an FEV1/FVC ratio less than 80% was associated with an increased risk for lung cancer and the relative risk increased as the ratio decreased. Although some evidence of increased risk was observed among those with FVC or FEV1 less than 80% of predicted, the increased risks associated with these

TABLE II. Sheet Metal Worker Overall Mortality

Cause of death	Obs.	Exp.	SMR	95% CI lower	upper
All Causes	6636	7972.54	0.83**	0.81	0.85
All Cancers	2224	2271.73	0.98	0.94	1.02
MN buccal & pharynx	31	38.33	0.81	0.55	1.15
MN lip	1	0.45	2.23	0.06	12.45
MN tongue	6	8.92	0.67	0.25	1.46
MN other buccal	12	9.77	1.23	0.63	2.14
MN pharynx	12	19.19	0.63	0.32	1.09
MN digestive & peritoneum	475	540.23	0.88**	0.80	0.96
MN esophagus	60	73.81	0.81	0.62	1.05
MN stomach	51	51.82	0.98	0.73	1.29
MN intestine	139	184.96	0.75**	0.63	0.89
MN rectum	28	37.04	0.76	0.50	1.09
MN biliary, liver, gall bladder	58	68.52	0.85	0.64	1.09
MN pancreas	131	117.79	1.11	0.93	1.32
MN peritoneum, other & unspecified sites	8	6.29	1.27	0.55	2.51
MN respiratory	839	812.78	1.03	0.96	1.10
MN larynx	15	23.21	0.65	0.36	1.07
MN trachea, bronchus, lung	808	784.86	1.03	0.96	1.10
MN pleura	11	1.48	7.42**	3.70	13.27
MN other respiratory	5	3.23	1.55	0.50	3.62
MN breast	5	3.07	1.63	0.53	3.80
MN female genital organs	0	0.20	0.00	0.00	18.28
MN male genital organs	179	225.30	0.79**	0.68	0.92
MN urinary	110	132.48	0.83	0.68	1.00
MN kidney	49	60.02	0.82	0.60	1.08
MN bladder & other urinary site	61	72.46	0.84	0.64	1.08
MN other & unspecified sites	350	291.14	1.20**	1.08	1.33
MN bone	2	3.38	0.59	0.07	2.14
MN melanoma	30	37.20	0.81	0.54	1.15
MN other skin	14	13.22	1.06	0.58	1.78
MN mesothelioma	85	11.58	7.34**	5.86	9.08
MN connective tissues	7	11.57	0.60	0.24	1.25
MN brain & other nervous	48	48.47	0.99	0.73	1.31
MN eye	1	1.02	0.98	0.02	5.45
MN thyroid	3	4.34	0.69	0.14	2.02
MN other & unspecified sites	160	160.35	1.00	0.85	1.17
MN lymphatic & hematopoietic	235	228.19	1.03	0.90	1.17
Hodgkin's disease	5	4.21	1.19	0.39	2.77
Non-Hodgkin's lymphoma	97	92.40	1.05	0.85	1.28
Multiple myeloma	43	42.73	1.01	0.73	1.36
Leukemia	90	88.86	1.01	0.81	1.24
Benign & unspecified nature neoplasms	18	27.05	0.67	0.39	1.05
Diseases blood & blood-forming organs	31	39.17	0.79	0.54	1.12
Diabetes mellitus	112	220.61	0.51**	0.42	0.61
Mental & psychiatric disorders	107	136.79	0.78**	0.64	0.95
Alcoholism	13	22.64	0.57*	0.31	0.98
Other mental disorders	94	114.15	0.82	0.67	1.01
Nervous system disorders	249	263.28	0.95	0.83	1.07
Heart diseases	1871	2486.51	0.75**	0.72	0.79

(*Continued*)

TABLE II. (*Continued.*)

Cause of death	Obs.	Exp.	SMR	95% CI lower	upper
Other diseases of the circulatory system	509	645.79	0.79**	0.72	0.86
Diseases respiratory system	743	839.70	0.88**	0.82	0.95
Acute resp. infection, except. flu, pneumonia	3	1.22	2.46	0.51	7.19
Influenza	3	2.76	1.09	0.22	3.18
Pneumonia	129	196.57	0.66**	0.55	0.78
COPD	461	485.42	0.95	0.86	1.04
Asthma	5	7.28	0.69	0.22	1.60
Asbestosis	48	4.11	11.68**	8.61	15.48
Silicosis	0	0.68	0.00	0.00	5.44
Other pneumoconiosis	1	4.00	0.25	0.01	1.39
Other respiratory diseases	93	137.67	0.68**	0.55	0.83
Diseases digestive system	194	267.43	0.73**	0.63	0.84
Diseases skin & subcutaneous	5	8.21	0.61	0.20	1.42
Diseases musculoskeletal & connective	14	23.02	0.61	0.33	1.02
Diseases genito-urinary system	109	162.66	0.67**	0.55	0.81
Symptoms & ill-defined conditions	39	59.32	0.66**	0.47	0.90
Transportation injuries	62	71.80	0.86	0.66	1.11
Falls	63	56.42	1.12	0.86	1.43
Other injury	56	75.67	0.74*	0.56	0.96
Violence	65	90.62	0.72**	0.55	0.91
Other & unspecified causes	164	206.96	0.79**	0.68	0.92

Obs., observed; Exp., expected; MN, malignant neoplasm.

TABLE III. Sheet Metal Worker Mortality by Time Since Entry into Sheet Metal Trade

Disease category	Time since trade entry (Years)	Obs.	Exp.	SMR	95% Confidence limits lower	upper
Lung Cancer	20–29	31	28.18	1.10	0.75	1.56
	30–39	142	144.32	0.98	0.83	1.16
	40–49	309	307.80	1.00	0.90	1.12
	50+	326	304.47	1.07	0.96	1.19
MN Pleura	20–29	1	0.08	12.15	0.31	67.71
	30–39	1	0.32	3.10	0.08	17.25
	40–49	6	0.62	9.73**	3.57	21.17
	50+	3	0.46	6.51*	1.34	19.02
Mesothelioma	20–29	1	0.13	7.54	0.19	42.02
	30–39	7	1.09	6.43**	2.58	13.24
	40–49	36	3.66	9.84**	6.89	13.63
	50+	41	6.70	6.12**	4.39	8.31
COPD	20–29	7	11.67	0.60	0.24	1.24
	30–39	29	53.80	0.54**	0.36	0.77
	40–49	131	157.35	0.83**	0.70	0.99
	50+	294	262.54	1.12	1.00	1.26
Asbestosis	20–29	2	0.08	24.83**	3.01	89.69
	30–39	4	0.36	11.13**	3.03	28.50
	40–49	11	1.19	9.23**	4.60	16.52
	50+	31	2.48	12.51**	8.50	17.75

Obs., observed; Exp., expected; MN, malignant neoplasm.

TABLE IV. Sheet Metal Worker Mortality by Chest X-Ray Parenchymal Category

Disease category	Parenchymal change category	Obs.	Exp.	SMR	95% Confidence limits lower	upper
Lung Cancer	0/- to 0/1	649	693.46	0.94	0.87	1.01
	1/0 to 1/2	143	86.85	1.65**	1.39	1.94
	2/1 to 2/3	14	4.28	3.27**	1.79	5.48
	3/2 to 3/+	2	0.26	7.58	0.92	27.38
MN Pleura	0/- to 0/1	10	1.27	7.89**	3.78	14.51
	1/0 to 1/2	1	0.20	4.92	0.12	27.40
	2/1 to 2/3	0	0.01	0.00	0.00	300.99
	3/2 to 3/+	0	<0.01	0.00	0.00	6087.99
Mesothelioma	0/- to 0/1	69	10.37	6.65**	5.18	8.42
	1/0 to 1/2	16	1.16	13.85**	7.91	22.49
	2/1 to 2/3	0	0.04	0.00	0.00	82.29
	3/2 to 3/+	0	<0.01	0.00	0.00	1050.38
COPD	0/- to 0/1	356	423.03	0.84**	0.76	0.93
	1/0 to 1/2	92	58.90	1.56**	1.26	1.92
	2/1 to 2/3	12	3.31	3.63**	1.87	6.33
	3/2 to 3/+	1	0.18	5.52	0.14	30.74
Asbestosis	0/- to 0/1	27	3.58	7.54**	4.96	10.96
	1/0 to 1/2	18	0.50	35.96**	21.30	56.84
	2/1 to 2/3	3	0.03	116.75**	24.08	341.20
	3/2 to 3/+	0	<0.01	0.00	0.00	2446.02

Obs. observed; Exp. expected.
*Two-Sided $P < 0.05$.
**Two-Sided $P < 0.01$.

measures did not achieve statistical significance ($P = 0.07$ for FEV1 and 0.31 for FVC).

Parenchymal profusion changes $\geq 1/0$ and pleural changes were associated with increased risk of mesothelioma mortality (Table VIII), with the risk associated with pleural changes reaching statistical significance ($P = 0.0116$). The risk associated with a profusion score of $\geq 1/0$ was of borderline statistical significance ($P = 0.0879$). No spirometry measure was predictive of mesothelioma mortality ($P > 0.23$).

Table IX shows that death from COPD was predicted by FVC below 70% of predicted, FEV1 below 70% predicted, an FEV1 /FVC ratio below 70%, and pack-years of smoking. Chest x-ray parenchymal changes were of borderline statistical significance ($P = 0.07$). The presence of increased profusion scores was significantly correlated

TABLE V. Sheet Metal Worker Mortality by Chest X-Ray Pleural Category

Disease category	Pleural changes	Obs.	Exp.	SMR	95% Confidence limits lower	upper
Lung Cancer	No	597	585.39	1.02	0.94	1.11
	Yes	211	199.47	1.06	0.92	1.21
MN Pleura	No	7	1.05	6.66**	2.68	13.73
	Yes	4	0.43	9.25**	2.52	23.68
Mesothelioma	No	53	8.72	6.08**	4.55	7.95
	Yes	32	2.86	11.19**	7.65	15.79
COPD	No	287	349.36	0.82**	0.73	0.92
	Yes	174	136.06	1.28**	1.10	1.48
Asbestosis	No	21	2.94	7.15**	4.42	10.93
	Yes	27	1.17	23.03**	15.17	33.51

Obs. observed; Exp. expected.
*Two-Sided $P < 0.05$.
**Two-Sided $P < 0.01$.

TABLE VI. Cox Model Chest Radiograph Predictors of Lung Cancer Mortality[1] in Sheet Metal Workers

Risk predictor	Number in model	No. of cancer cases	Hazard ratio[2]	95% Confidence limits lower	upper
Profusion Categories[4]					
0/–0/0	13066	526	1.00	Ref	Ref
0/1	1341	78	1.08	0.85	1.37
1/0–1/2	1559	131	1.45	1.19	1.76
2/1–2/3	95	12	2.72	1.52	4.86
3/2–3/+	7	2	3.97	0.97	16.26
Pleural Abnormalities					
Negative	13997	635	1.00	Ref	Ref
Positive	2071	114	0.90	0.74	1.11
Years Since Last Sheet Metal Trade Work at Exam					
<1	9880	366	1.00	Ref	Ref
1–4	2942	189	1.21	1.00	1.46
5–9	1884	102	1.09	0.85	1.41
≥10	1362	92	1.71	1.27	2.29
Smoking Pack-Years[3]	16068	749	1.025	1.019	1.030
Years in Sheet Metal Trade Beyond 20 Year[3]	16068	749	1.012	1.001	1.027

[1]Cox proportional hazard analyses based on 16068 White males with 20 or more years in sheet metal trade and having data on other model covariates.
[2]Stratified Cox model with strata defined by smoking status (never, past, and current) and birth cohort decade with adjustment for pack-years of smoking (0, 1–19, 20–39, or ≥40 pack-years), time since last work in the sheet metal trade at start of follow-up (<1, 1–4, 5–9, or ≥10 years), years of sheet metal work beyond 20 years (20–24, 25–29, 30–34, or ≥35 years), presence of pleural abnormalities, and profusion category. A pleural abnormality was defined as bilateral pleural thickening or plaques, with or without calcification.
[3]Pack-years of smoking, and years of sheet metal work entered as grouped continuous variables.
[4]Test for trend across profusions categories, $P < 0.0001$.

with spirometry changes; therefore, we ran an additional Cox model for COPD omitting the spirometry parameters. This model is shown in Table X and demonstrated a strong association between presence of parenchymal changes ≥1/0 and risk of COPD mortality after adjusting for smoking and other model covariates. In a separate model (not shown) COPD risks associated with spirometry measures were relatively unchanged when the parameters for pleural and parenchymal chest x-ray changes were omitted from the model.

We tested potential effects of reader type by the ILO system (A-reader, B-reader, and other or missing reader type) in the final lung cancer, mesothelioma, and COPD Cox models by addition of an indicator variable. Among workers included the Cox models, 82.9% of chest images were read by B-readers, 11.9% by A-readers, and 5.2% were missing reader type or were read by a physician with proficiency in the use of the ILO classification but who was neither an A nor a B reader. The parameter for reader type was not significant ($P > 0.26$) and did not change the parameter estimates for other model covariates in any meaningful way in the lung cancer and mesothelioma models. In the COPD model, inclusion of reader type in the model increased the risk estimate for a profusion score ≥1/0 (RR = 1.35, 95% CI = 1.00–1.84), and all other parameter estimates were essentially unchanged.

DISCUSSION

Sheet metal workers who participated in this nationwide screening program had a reduced SMR overall compared to the US population, consistent with a healthy worker effect. In the SMR analysis, no overall increase in lung cancer mortality was observed among this cohort when compared to the US population; however, SMR analyses revealed excess mortality for mesothelioma, malignant neoplasm of the pleura, and asbestosis. Additionally, the SMR analyses demonstrated significant excess risk for lung cancer and COPD among workers with parenchymal changes ≥1/0 in profusion. The SMR was significantly elevated for pleural cancers, mesothelioma, and asbestosis among workers who did not have parenchymal changes >1/0 in profusion. Cox proportional hazards models controlling for smoking confirmed the excess risk of lung cancer among workers with a profusion score ≥1/0 and provided additional evidence for excess lung cancer risk among workers with parenchymal profusion scores <1/0 on the ILO scale.

In addition to smoking and abnormal pulmonary function, which are known risk factors for COPD mortality, increased interstitial markings on chest x-ray and years in the sheet metal trade were also predictive of death from COPD. These findings suggest a relationship between asbestos exposure and death from COPD. Previous research has

TABLE VIII. Cox Model Predictors of Mesothelioma[1] Mortality in Sheet Metal Workers

Risk predictor	Number in model	No. of cancer cases	Hazard ratio[2]	95% Confidence limits lower	upper
Profusion Categories					
<1/0	13773	67	1.00	Ref	Ref
≥1/0	1582	15	1.67	0.93	3.02
Pleural Abnormalities[3]					
Negative	13997	67	1.00	Ref	Ref
Positive	2071	22	1.95	1.16	3.27
FVC Percent Predicted[4]					
>=80	12005	68	1.00	Ref	Ref
70–79	1992	5	0.36	0.13	0.99
60–69	862	6	0.90	0.27	2.92
<60	496	3	0.88	0.17	4.49
FEV_1 Percent Predicted[4]					
>=80	11454	62	1.00	Ref	Ref
70–79	1839	7	1.06	0.41	2.75
60–69	976	7	1.79	0.56	5.70
<60	1086	6	1.37	0.26	7.16
FEV_1/FVC[4]					
>=80	5134	32	1.00	Ref	Ref
70–79	6788	32	0.67	0.40	1.11
60–69	2250	10	0.60	0.27	1.35
<60	1183	8	0.99	0.29	3.34
Years Since Last Sheet Metal Trade Work at Exam					
<1	9510	44	1.00	Ref	Ref
1–4	2793	20	1.05	0.59	1.86
5–9	1776	7	0.48	0.19	1.16
≥10	1276	11	1.13	0.49	2.60
Smoking Pack-Years[3]	15355	82	0.992	0.980	1.004
Years in Sheet Metal Trade Beyond 20 Years[3]	15355	82	0.979	0.937	1.022

[1]Mesothelioma included ICD-9 code 163 and ICD codes C38.4, C45#, C45.0-C45.2, C45.7, and C45.9.
[2]Cox proportional hazard analyses based on 15355 White males with 20 or more years in sheet metal trade and having data on other model covariates. Stratified Cox model with strata defined by smoking status (never, past, and current) and birth cohort decade with adjustment for pack-years of smoking (0, 1–19, 20–39, or ≥40 pack-years), time since last work in the sheet metal trade at start of follow-up (<1, 1–4, 5–9, or ≥10 years), years of sheet metal work beyond 20 years (20–24, 25–29, 30–34, or ≥35 years), spirometry categories, presence of pleural abnormalities, and profusion category. A pleural abnormality was defined as bilateral pleural thickening or plaques, with or without calcification.
[3]An alternate model with pleural abnormalities defined as any indication of pleural change on the ILO recording form resulted in slightly greater risk (RR = 2.10, 95% CI = 1.32–3.33).

shown that asbestos exposure is associated with obstructive disease on lung function testing [ATS, 2004, Dement et al., 2010]. Exposure to dust, fumes, gases, and vapors is now recognized as a cause of COPD [Omland et al., 2014]. A recent study by Toren et al. [2013] investigated cause of death among a cohort of 354,718 male construction workers. Exposure to inorganic dust, wood dust, vapors, fumes and gases, and irritants was based on a job-exposure matrix with a focus on exposure in the mid-1970 s. Construction workers with any occupational exposure to vapors, gases, fumes and dust showed an increased mortality due to COPD (RR = 1.32, 95% confidence interval (CI) 1.18–1.47), and there was significantly increased mortality due to COPD among those exposed specifically to fumes (RR 1.20, 95% CI 1.07–1.36) and to inorganic dust (RR 1.19, 95% CI 1.07–1.33). This is consistent with prior research [Bergdahl, 2004]. Among sheet metal workers, asbestos is an integral part of dust exposure before 1980, and some members of this cohort were likely to have had significant exposure to welding fume as well.

It is well accepted that workers with exposure to asbestos sufficient to cause radiographic changes consistent with clinical asbestosis are at extremely high risk of lung cancer [Huuskonen, 1978; Liddell and McDonald, 1980; Berry, 1981; Finkelstein et al., 1981; Cookson et al., 1985; Coutts et al., 1987; Roggli, 1990; Oksa et al., 1997;

TABLE VIII. Cox Model Predictors of Mesothelioma[1] Mortality in Sheet Metal Workers

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Positive	2071	22	1.95	1.16	3.27
FVC Percent Predicted[4]					
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70–79	1992	5	0.36	0.13	0.99
60–69	862	6	0.90	0.27	2.92
<60	496	3	0.88	0.17	4.49
FEV_1 Percent Predicted[4]					
>=80	11454	62	1.00	Ref	Ref
70–79	1839	7	1.06	0.41	2.75
60–69	976	7	1.79	0.56	5.70
<60	1086	6	1.37	0.26	7.16
FEV_1/FVC[4]					
>=80	5134	32	1.00	Ref	Ref
70–79	6788	32	0.67	0.40	1.11
60–69	2250	10	0.60	0.27	1.35
<60	1183	8	0.99	0.29	3.34
Years Since Last Sheet Metal Trade Work at Exam					
<1	9510	44	1.00	Ref	Ref
1–4	2793	20	1.05	0.59	1.86
5–9	1776	7	0.48	0.19	1.16
≥10	1276	11	1.13	0.49	2.60
Smoking Pack-Years[3]	15355	82	0.992	0.980	1.004
Years in Sheet Metal Trade Beyond 20 Years[3]	15355	82	0.979	0.937	1.022

[1]Mesothelioma included ICD-9 code 163 and ICD codes C38.4, C45#, C45.0-C45.2, C45.7, and C45.9.
[2]Cox proportional hazard analyses based on 15355 White males with 20 or more years in sheet metal trade and having data on other model covariates. Stratified Cox model with strata defined by smoking status (never, past, and current) and birth cohort decade with adjustment for pack-years of smoking (0, 1–19, 20–39, or ≥40 pack-years), time since last work in the sheet metal trade at start of follow-up (<1, 1–4, 5–9, or ≥10 years), years of sheet metal work beyond 20 years (20–24, 25–29, 30–34, or ≥35 years), spirometry categories, presence of pleural abnormalities, and profusion category. A pleural abnormality was defined as bilateral pleural thickening or plaques, with or without calcification.
[3]An alternate model with pleural abnormalities defined as any indication of pleural change on the ILO recording form resulted in slightly greater risk (RR = 2.10, 95% CI = 1.32–3.33).

Karjalainen et al., 1999; Reid et al., 2005; Reid 2006; Markowitz et al., 2013]. Consistent with the current study, increased lung cancer risk also has been observed among workers without radiological evidence of asbestosis [Anttila et al., 1993; Wilkinson et al., 1995; Cullen et al., 2005; Reid, 2006; Finkelstein, 2010; Markowitz et al., 2013]. The most recent study to report on this issue comes from an updated analysis of the North American insulator cohort [Markowitz et al., 2013]. The authors reported that asbestos exposure without radiographic evidence of asbestosis in non-smokers raised the risk of lung cancer by 3.6 fold, and that asbestosis further doubled the lung cancer mortality risk.

Although prior research had found a relationship between the presence of pleural plaque and lung cancer mortality [Loomis et al., 1989; Hillerdal, 1994; Karjalainen et al., 1999; Cullen et al., 2005; Ameile et al., 2011], our study did not find an excess lung cancer risk among workers with pleural changes after adjustment for other model covariates including duration of sheet metal trade work, smoking, presence of parenchymal changes, and time since last sheet metal work at exam. The prevalence of pleural changes increased markedly with age in our cohort with a low of 9.5% among workers less than 55 years of age to 43.8% among workers older than 70 years. A logistic model

TABLE IX. Cox Model Predictors of COPD Mortality in Sheet Metal Workers—All Parameters[1]

Risk predictor	Number in model	No. of COPD cases	Hazard ratio[2]	95% Confidence limits lower	upper
Profusion Categories					
<1/0	9224	187	1.00	Ref	Ref
≥1/0	1032	59	1.33	0.98	1.80
Pleural Abnormalities					
Negative	8931	193	1.00	Ref	Ref
Positive	1325	43	0.96	0.70	1.32
FVC Percent Predicted[4]					
>=80	7979	102	1.00	Ref	Ref
70–79	1353	50	1.20	0.78	1.83
60–69	597	48	1.76	1.07	2.87
<60	327	46	1.72	1.02	2.91
FEV1 Percent Predicted[4]					
>=80	7678	58	1.00	Ref	Ref
70–79	1208	28	1.50	0.91	2.47
60–69	674	36	2.01	1.18	3.42
<60	696	124	3.24	1.69	6.25
FEV_1/FVC[4]					
>=80	3512	14	1.00	Ref	Ref
70–79	4542	45	1.48	0.81	2.73
60–69	1471	54	3.09	1.63	5.86
<60	731	133	8.94	4.48	17.86
Years Since Last Sheet Metal Trade Work at Exam					
<1	6588	77	1.00	Ref	Ref
1–4	1723	61	1.21	0.84	1.74
5–9	1152	61	1.14	0.74	1.74
≥10	746	47	1.08	0.68	1.73
Smoking Pack-Years[3]	10256	246	1.019	1.009	1.029
Years in Sheet Metal Trade Beyond 20 Years[3]	10256	246	1.019	0.992	1.046

[1]Cox proportional hazard analyses based on 10256 White males with 20 or more years in sheet metal trade, spirometry, and having data on other model covariates. Only workers with spirometry meeting ATS reproducibility were included.
[2]Stratified Cox model with strata defined by smoking status (never, past, and current) and birth cohort decade with adjustment for pack-years of smoking (0, 1–19, 20–39, or ≥40 pack-years), time since last work in the sheet metal trade at start of follow-up (<1, 1–4, 5–9, or ≥10 years), years of sheet metal work beyond 20 years (20–24, 25–29, 30–34, or ≥35 years), spirometry categories, presence of pleural abnormalities, and profusion category. A pleural abnormality was defined as bilateral pleural thickening or plaques, with or without calcification.
[3]Pack-years of smoking, and years of sheet metal work entered as grouped continuous variables.
[4]Test for trend across categories for percent predicted FVC ($P = 0.0255$), FEV_1 ($P = <0.0001$) and FEV_1/FVC ($P < 0.0001$).

(not shown) found both age and duration of sheet metal work to be strong predictors of pleural changes ($P < 0.001$) thus controlling for age and years of sheet metal work in our Cox lung cancer models diminished the effects of pleural changes. In a Cox model (not shown) not adjusting for age or years in the sheet metal trade, pleural changes were significantly associated with lung cancer risk after control for smoking (HR = 1.24, 95% CI = 1.00–1.50). Our inability to detect a pleural effect in the adjusted model may be due in part to the requirement that workers have 20 or more years in the sheet metal trade for entry into our cohort, thereby diminishing availability of a reference group with low cumulative asbestos exposure.

One limitation of this study is that the radiologic interpretation and the classification of smoking status were determined at the time of the clinical exam, which could have preceded the date of death by more than a decade. It is possible that some of the workers who were categorized as without asbestosis could have developed parenchymal disease in the interim period. Prior analysis within this same cohort found that only 5% had progression from a normal chest x-ray to one classified as 1/0 or higher over a mean of 9 years [Welch et al., 2007], so misclassification of asbestosis is unlikely to explain the results.

The current study provides additional evidence that workers who experienced largely intermittent and indirect

TABLE X. Cox Model V2 Using Chest X-ray Predictors of COPD Mortality[1] in Sheet Metal Workers

Risk predictor	Number in model	No. of COPD cases	Hazard ratio[2]	95% Confidence limits lower	upper
Profusion Categories					
<1/0	9224	187	1.00	Ref	Ref
≥1/0	1032	59	1.71	1.26	2.30
Pleural Abnormalities					
Negative	8931	193	1.00	Ref	Ref
Positive	1325	43	0.96	0.70	1.31
Years Since Last Sheet Metal Trade Work at Exam					
<1	6588	77	1.00	Ref	Ref
1–4	1723	61	1.42	0.99	2.04
5–9	1152	61	1.62	1.09	2.42
≥10	746	47	1.85	1.16	2.94
Smoking Pack-Years[3]	10256	246	1.039	1.03	1.05
Years in Sheet Metal Trade Beyond 20 Years[3]	10256	246	1.024	1	1.05

[1]Cox proportional hazard analyses based on 10256 White males with 20 or more years in sheet metal trade, spirometry, and having data on other model covariates. Only workers with spirometry meeting ATS reproducibility were included.
[2]Stratified Cox model with strata defined by smoking status (never, past, and current) and birth cohort decade with adjustment for pack-years of smoking (0, 1–19, 20–39, or ≥40 pack-years), time since last work in the sheet metal trade at start of follow-up (<1, 1–4, 5–9, or ≥10 years), years of sheet metal work beyond 20 years (20–24, 25–29, 30–34, or ≥35 years), profusion score, and presence of pleural abnormalities.
[3]Pack-years of smoking, and years of sheet metal work entered as grouped continuous variables.

exposure to asbestos are at increased risk of asbestos-related diseases and at risk for COPD. The risk of lung cancer mortality increased sharply with parenchymal profusion score; however, the study provided additional evidence for increased risk among workers without radiographic asbestosis. The risk of cancers of the pleura and mesothelioma also were significantly increased among workers without radiological evidence of asbestosis or pleural abnormalities. Our study has several strengths including a large population with chest x-ray data classified by ILO criteria, and smoking histories on each member of the cohort. Nonetheless, our study is limited by a strong healthy survivor effect and an inability to address risks for workers who worked fewer than 20 years. The lack of workers with less than 20 years in the trade reduces available exposure contrasts in the Cox models and lessens the utility of trade work duration as an exposure surrogate. While our analyses controlled for smoking, it is impossible to exclude a contribution by other unmeasured risk factors such as welding fumes or other occupational lung carcinogens. However, confounding by these unmeasured exposures is unlikely to explain the steep and consistent patterns observed by profusion score.

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REFERENCES

Allison PD. 1995. Survival analysis using the SAS system—A practical guide. Cary, NC: SAS Institute Inc.

American thoracic society. 1987. Standardization of spirometry-1987 update. Am Rev Respir Dis 136:1285–1298.

American thoracic society. 1995. Standardization of spirometry-1994 update. Am Rev Respir Dis 152:1107–1136.

American thoracic society. 2004. Diagnosis and initial management of nonmalignant diseases related to asbestos. Am J Respir Crit Care Med 170(6):691–715.

Ameille J, Brochard P, Letourneux M, Paris C, Pairon JC. 2011. Asbestos-related cancer risk in patients with asbestosis or pleural plaques. Rev Mal Respir 28(6):e11–e17.

Anttila S, Karjalainen A, Taikina-aho O, Kyyronen P, Vainio H. 1993. Lung cancer in the lower lobe is associated with pulmonary asbestos fiber count and fiber size. Environ Health Perspect 110(2):166–170.

Becklake MR. 1976. Asbestos-related diseases of the lung and other organs: Their epidemiology and implications for clinical practice. Am Rev Respir Dis 114(1):187–227.

Bergdahl IA, Torėn K, Eriksson K, Hedlund U, Nilssonz T, Flodin R. 2004. Increased mortality in COPD among construction workers exposed to inorganic dust. Järvholm B 23:402–406.

Baker EL, Dagg T, Greene RE. 1985. Respiratory illness in the construction trades: 1. The significance of asbestos-associated pleural disease among sheet metal workers. J Occup Med 27:483–489.

Berry G. 1981. Mortality of workers certified by pemumoconiosis medical panels as having asbestosis. British J Ind Med 38:130–137.

Bilgrad R. 1995. National death index plus: Coded causes of death supplement to the national death index user's manual. Hyattsville, MD: Centers for Disease Control and Prevention.

Boyle CA, Decouflė P. 1990. National sources of vital status information: Extent of coverage and possible selectivity in reporting. Am J Epidemiol 131:160–168.

Cookson WO, Musk AW, Glancy JJ, de Klerk NH, Yin R, Mele R, Carr NG, Armstrong BK, Hobbs MS. 1985. Compensation, radiographic changes, and survival in applicants for asbestosis compensation. Br J Ind Med 42(7):461–468.

Coutts II, Turner-Warwich JC. 1987. Mortality in cases of asbestosis diagnosed by pneumoconiosis medical panel. Thorax 42:111–116.

Cullen MR, Barnett MJ, Balmes JR, Cartmel B, Redlich CA, Brodkin C et al. 2005. Predictors of lung cancer among asbestos-exposed men in the beta-carotene and retinol efficacy trial. Am J Epidemiol 161(3):260–270.

Dement J, Welch L, Haile E, Myers D. 2009. Mortality among sheet metal workers participating in a medical screening program. Am J Ind Med 52(8):603–613.

Dement JM, Welch L, Ringen K, Bingham E, Quinn P. 2010. Airways obstruction among older construction and trade workers at department of energy nuclear sites. Am J Ind Med 53:224–240.

Eisen EA, Robins AM, Greaves IA, Wegman DH. 1984. Selection effects of repeatability criteria applied to lung spirometry. Am J Epid 120:734–742.

Finkelstein MM. 2010. Absence of radiographic asbestosis and the risk of lung cancer among asbestos-cement workers: Extended follow-up of a cohort. Am J Ind Med 53(11):1065–1069.

Finkelstein M, Kusiak R, Suranyl G. 1981. Mortality among workers receiving compensation for asbestosis in Ontario. CMA J 125:259–262.

Hankinson JL, Odencrantz JR, Fedan KB. 1999. Spirometric reference values from a sample of the General U.S. population. Am J Respir Crit Care Med 159:179–187.

Hillerdal G. 1994. Pleural plaques and risk for bronchial carcinoma and mesothelioma: A prospective study. Chest 105:144–150.

Horm J. 1996. Assignment of probabilistic scores to national death index record matches NDI user's manual addendum. Hyattsville, MD: Centers for Disease Control and Prevention.

Huuskonen MS. 1978. Clinical features, mortality and survival of patients with asbestosis. Scand Journ Work Enviro 4:265–274.

IARC 2009. Monographs on the evaluation of carcinogenic risks to humans, volume 100 C, arsenic, metals, fibres, and dusts volume 100 C, A review of human carcinogens. Lyon, France: International Agency for Research on Cancer.

ILO U/C. 1980. International classification of radiographs of pneumoconiosis. Geneva: ILO.

Karjalainen A, Pukkala E, Kauppinen T, Partanen T. 1999. Incidence of cancer among Finnish patients with asbestos-related pulmonary or pleural fibrosis. Cancer Causes Control 10(1):51–57.

Kipen HM, Lilis R, Suzuki Y, Valciukas JA, Selikoff IJ. 1987. Pulmonary fibrosis in asbestos insulation workers with lung cancer: A radiological and histopathological evaluation. Br J Ind Med 44(2):96–100.

Liddell FD, McDonald JC. 1980. Radiological findings as predictors of mortality in Quebec asbestos workers. Br J Ind Med 37(3):257–267.

Loomis DP, Collman GW, Rogan WJ. 1989. Relationship of mortality, occupation, and pulmonary diffusing capacity to pleural thickening in the First National Health and Nutrition Examination Survey. Am J Ind Med 16(5):477–484.

Markowitz SB, Levin SM, Miller A, Morabia A. 2013. Asbestos, asbestosis, smoking, and lung cancer. New findings from the North American insulator cohort. Am J Respir Crit Care Med 188(1):90–96.

Nicholson WJ, Perkel G, Selikoff IJ. 1982. Occupational exposure to asbestos: population at risk and projected mortality–1980–2030. Am J Ind Med 3(3):259–311.

NCHS. 2013. National death index user's guide. Hyattsville, MD: National center for health statistics.

NIOSH 2011. LTAS Manual, LTAS. Net Version 3.0.3. Downloaded November 8, 2011. http://www.cdc.gov/niosh/LTAS/news.html.

Oksa P, Pukkala E, Karjalainen A, Ojajarvi A Huuskonen MS. 1997. Cancer incidence and mortality among Finnish asbestos sprayers and in asbestosis and silicosis patients. Am J Ind Med 31(6):693–698.

Omland O, Würtz ET, Aasen TB, Blanc P, Brisman JB, Miller MR, Pedersen OF, Schlünssen V, Sigsgaard T, Ulrik CS, et al. 2014. Occupational chronic obstructive pulmonary disease: A systematic literature review. Scand J Work Environ Health 40(1):19–35.

Paik NW, Walcott RJ, Brogan PA. 1983. Worker exposure to asbestos during removal of sprayed material and renovation activity in buildings containing sprayed material. Am Ind Hyg Assoc J 44:428–432.

Pencina MJ, Larson MG, D'Agostino RB. 2007. Choice of time scale and itseffect on significance of predictors in longitudinal studies. Stat Med 26:1343–1359.

Reid A, de Klerk NH, Ambrosini GL, Berry G, Musk AW. 2006. The risk of lung cancer with increasing time since ceasing exposure to asbestos and quitting smoking. Occup Environ Med 63(8):509–512.

Reid A, de Klerk NH, Ambrosini GL, Olsen N, Pang SC, Berry G, Musk AW. 2005. The effect of asbestosis on lung cancer risk beyond the dose related effect of asbestos alone. Occup Environ Med 62:885–889.

Robinson CF, Schnorr TM, Cassinelli RT, Calvert GM, Steenland NK, Gersic CM, Schubauer-Berigan MK. 2006. Tenth revision U.S. mortality rates for use with the NIOSH life table analysis system. J Occup Environ Med 48:662–667.

Roggli VL. 1990. Human disease consequences of fiber exposures: A review of human lung pathology and fiber burden data. Environ Health Perspect 88:295–303.

Rothman KJ, Boice JD. 1979. Epidemiologic Analysis with a programmable calculator. Washington, DC: US Government Printing Office.

Rothman KJ, Greenland S. 1998. Modern epidemiology. 2nd edition. Philadelphia, PA: Lippincott-Raven.

SAS Institute Inc. 2011. SAS/STAT® 9.3 user's guide. Cary, NC: SAS Institute Inc.

Selikoff IJ, Hammond EC. 1978. Asbestos-associated disease in United States shipyards. CA Cancer J Clin 28(2):87–99.

Selikoff IJ, Lilis R. 1991. Radiological abnormalities among sheet-metal workers in the construction industry in the United States and Canada: Relationship to asbestos exposure. Arch Environ Health 46(1):30–36.

Stampfer MJ, Willett WC, Speizer FE, Dysert DC, Lipnick R, Rosner R, Hennekens CM. 1984. Test of the National Death Index. Am J Epidemiol 119:837–839.

Steenland K, Beaumont J, Spaeth S, Brown D, Okun A, Jurcenko L, Ryan B, Phillips S, Roscoe R, Stayner L, et al. 1990. New developments in the life table analysis system of the national institute for occupational safety and health. J Occup Med 32:1091–1098.

Torén K, Järvholm B. 2013. Occupational exposure, to vapors gases, dusts and fumes and mortality in relation to chronic obstructive pulmonary disease among Swedish construction workers—A longitudinal cohort study. Chest. DOI:10.1378/chest.13–1429. [Epub ahead of print].

Wilkinson P, Hansell DM, Jassens J, Rubens M, Rudd RM, Taylot AN, McDonald JC. 1995. Is lung cancer associated with asbestos exposure when there are no small opacities on the chest radiograph. Lancet 345:1074–1078.

Welch LS, Haile E, Dement J, Michaels D. 2007. Change in prevalence of asbestos-related disease among sheet metal workers 1988–2004. Chest 131:1988–2004.

Welch LS, Michaels D, Zoloth SR. 1991. Asbestos-related disease among sheet-metal workers: Preliminary results of the National Sheet Metal Worker Asbestos Disease Screening Program. Ann NY Acad Sci 643:287–295.

Welch LS, Michaels D, Zoloth SR. 1994. The national sheet metal worker asbestos disease screening program: Radiologic findings. national sheet metal examination group. Am J Ind Med 25(5):635–648.

Williams PRD, Phelka AD, Paustenbach DJ. 2007. A review of historical exposures to asbestos among skilled craftsmen (1940–2006). J Tox Env Hlth, Part B 10:1940–2006.

Zoloth S, Michaels D. 1985. Asbestos disease in sheet metal workers: The results of proportional mortality analysis. Am J Ind Med 7:315–321.

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