OEM Online First, published on May 9, 2014 as 10.1136/oemed-2013-102000

ORIGINAL ARTICLE

Mortality experience among Minnesota taconite mining industry workers

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Received 21 November 2013
Revised 3 April 2014
Accepted 20 April 2014

ABSTRACT

Objective To evaluate the mortality experience of Minnesota taconite mining industry workers.

Methods Mortality was evaluated between 1960 and 2010 in a cohort of Minnesota taconite mining workers employed by any of the seven companies in operation in 1983. Standardised mortality ratios (SMR) were estimated by comparing observed deaths in the cohort with expected frequencies in the Minnesota population. Standardised rate ratios (SRR) were estimated using an internal analysis to compare mortality by employment duration.

Results The cohort included 31 067 workers with at least 1 year of documented employment. Among those, there were 9094 deaths, of which 949 were from lung cancer, and 30 from mesothelioma. Mortality from all causes was greater than expected in the Minnesota population (SMR=1.04, 95% CI 1.02 to 1.04). Mortality from lung cancer and mesothelioma was higher than expected with SMRs of 1.16 for lung cancer (95% CI 1.09 to 1.23) and 2.77 for mesothelioma (95% CI 1.87 to 3.96). Other elevated SMRs included those for cardiovascular disease (SMR=1.10, 95% CI 1.06 to 1.14), specifically for hypertensive heart disease (SMR=1.81, 95% CI 1.39 to 2.33) and ischemic heart disease (SMR=1.11, 95% CI 1.07 to 1.16). Results of the SRR analysis did not show variation in risk by duration of employment.

Conclusions This study provides evidence that taconite workers may be at increased risk for mortality from lung cancer, mesothelioma, and some cardiovascular disease. Occupational exposures during taconite mining operations may be associated with these increased risks, but non-occupational exposures may also be important contributors.

BACKGROUND AND SIGNIFICANCE

The iron mining industry in Minnesota began in the late 1800s with the discovery of hematite in northeastern Minnesota, within what is now known as the Mesabi Iron Range. Hematite, a high-grade ore, was excavated from the iron formation and shipped directly to steel mills. However, the high-grade ore became less abundant following heavy demand for its use in World War II. In the 1950s, with hematite reserves depleted, the mining and processing of low-grade taconite ore began.[1] Since then, the taconite mining industry in Minnesota has become the largest supplier of iron ore to the steel industry of the USA.[2]

Mining and processing of taconite iron ore results in potential exposure to non-asbestiform amphibole and non-amphibole elongate mineral particles (EMP), respirable silica, quartz and dust, and cleavage fragments.[3] The term 'EMP' refers to any mineral particle with a minimum aspect ratio of 3:1 that is of inhalable size. Cleavage fragments are fractured mineral EMPs created during the crushing and fracturing process.[4]

The Mesabi Iron Range is approximately 2.5 miles wide and 122 miles long and is divided into four mineralogical zones.[5] All zones have deposits of taconite along with quartz and iron silicates, but vary in the type of EMP.[6] The ore body in the eastern range, known as zone 4, contains iron-rich amphibole EMPs (primarily cummingtonite-grunerite), which is believed to be less than 1% fibrous.[7] The western end of the range, zone 1, contains almost exclusively non-asbestiform EMPs, primarily of quartz hematite, siderite, chamosite and greenalite.[6 8] Zone 2 is considered a transitional zone with some amphiboles appearing.[5] One mine operates in zone 4, one mine that is no longer in operation is located in zone 2, the remaining five mines are located in zone 1 which is roughly the western-most two-thirds of the entire Mesabi Iron Range. There is another mineralogical zone, zone 3, however, there are no mines located in this zone. The primary exposure in taconite operations is of non-asbestiform cleavage fragments however, due to the mineralogical differences in the eastern versus western zones, workers in the two zones may be exposed to different types of mineral particles.[3] There is an ongoing debate regarding these exposures and the health of miners which includes (1) whether the amphibole minerals mined in the eastern part of the iron range are a threat to human health and (2) whether exposure to non-asbestiform minerals, including cleavage fragments, poses any risk to human health.[4 9–12]

The history of public concern of the health of taconite miners and residents near the mining and processing facilities began in the early 1970s when EMPs, determined to be primarily grunerite, possibly including some asbestiform grunerite, were found in Duluth's drinking water supply as a result of taconite tailings that were disposed of into Lake Superior.[8 13] This prompted studies of the potential health effects from ingestion of Duluth water which did not show increased risk of malignant tumours in either laboratory animals or human populations.[14 15] The earliest studies of the health of taconite miners were carried out in the early 1980s. The first study[16] focused on a group of miners from Reserve Mining Company. The

To cite: Allen EM, Alexander BH, MacLehose RF, *et al*. *Occup Environ Med* Published Online First: [*please include* Day Month Year] doi:10.1136/oemed-2013-102000

authors reported no increased risk of respiratory cancers among the 5751 miners. Later, studies were conducted in 1988 with an update in 1992[17 18] and, similarly, did not report an excess mortality among the 3431 workers from Erie and Minntac mines. In 1997, the Minnesota Department of Health Cancer Surveillance System reported a 73% excess in cases of mesothelioma among men in the northeastern region of Minnesota between 1988 and 1996 as compared with the rest of the state.[19] This resurrected the concern over whether exposures from taconite mining and processing pose a threat to the health of the workers.

To address these lingering uncertainties regarding the health consequences of taconite mining, we conducted a mortality study of workers from multiple mines to characterise the overall health of the Minnesota taconite worker population.

METHODS

Study population

The occupational cohort for this analysis was enumerated in the early 1980s as part of the Mineral Resources Health Assessment Program (MRHAP). The program was developed by the University of Minnesota, School of Public Health, with the support of the Iron Range Resources and Rehabilitation Board. This was done as part of an effort to further research on health effects of mining and mineral processing. Investigators assembled a database of 68 737 individuals from employment records of the seven mines in operation in 1983, US Steel Corporation, Hanna Mining Company, Pickands-Mather and Company, Reserve Mining Company, Eveleth Taconite Company, Inland Steel Company, and Jones and Laughlin Corporation.

In 2008, the University of Minnesota launched the Taconite Workers Health Study (TWHS). The current mortality analysis was one component of the overall TWHS with an objective to update the health assessment of the cohort of 68 737 miners collected by MRHAP in 1983. The cohort included taconite workers and those who had worked in the earlier hematite mining operations. To focus the study on workers most likely to have been working after taconite mining began in the 1950s, the cohort used in this analysis was limited to those born in 1920 or later, leaving 46 170 individuals. Of these, 1927 were excluded, including 477 whose only record on file was an application with no further evidence of employment, 679 whose records were insufficient for vital status follow-up, and 539 for whom employment information was improbable, for example, began working at age 14 or younger. Those who died before reference mortality rates were available (before 1960, n=232), were also excluded, leaving 44 243 workers. To focus on workers with more stable employment in the taconite industry, this analysis was restricted to workers with at least 1 year of documented employment giving a study population of 31 067 workers. This exclusion removed workers who did not stay in the industry, and also summer workers, often students who only worked a few months.

Vital status ascertainment

The mortality analyses covered the period from 1960 (when complete reference mortality rates were available) through 2010. The vital status of cohort members as of 31 December 2010 was ascertained through several sources including the Social Security Administration (SSA), the National Death Index (NDI), Minnesota Department of Health, and other state health departments. Social security numbers and names of all cohort members were sent to the SSA and were returned with a vital status of *deceased*, *alive*, or *unknown*, with the state of death and date of death identified for decedents. Cohort members who died in Minnesota, or whose state of death was unknown, were sent to the Minnesota Department of Health to ascertain causes of death. NDI, established in 1979, is a national death registry designed to facilitate health investigations. For those who died outside of Minnesota in the year 1979 or later, causes of death were obtained from NDI Plus. For individuals who died before 1979, death certificates were obtained from the state health department from the state in which the individual died. Additional tracing was done on those whose vital status was unknown and, if found to be deceased, their death certificates were obtained. Underlying and contributing causes of death were coded to the International Classification of Disease (ICD) version current at the year of death. The ICD codes were obtained directly from the Minnesota Department of Health and the NDI. All other death certificates were reviewed and coded by a nosologist.

Individuals who were identified as deceased, but whose death certificates were not found, were classified as 'Presumed Dead'. The date of death provided by the SSA was recorded as the vital status date and the cause of death was classified as 'Unknown.' Individuals identified as 'Unknown' by the SSA were traced via a commercial tracing vendor that uses credit bureau address updates. For those who were found to have had recent address activity, their vital status was recorded as 'Presumed Alive' with a vital status date as the most recent date recorded from the web tracing tools. The vital status date for the remaining individuals with an unknown vital status was their last date of employment.

Given the size of the cohort, detailed abstraction of all work histories in the cohort was not feasible, and duration of employment was the primary exposure measure of interest. For this analysis, work records of cohort members were reviewed with the first and last dates of employment abstracted, as well as the last date of activity on the work record. In 4.5% of the data, the work records contained start dates but were missing end dates. In this case, the last date of activity was used as the end date to calculate duration of employment. For roughly 92% of the study population, we also had location (zone 1, 2, or 4) of employment.

Data analysis

The mortality rate of the cohort was compared with that of the Minnesota population to estimate standardised mortality ratios (SMR), and 95% CIs adjusted for sex, and 5-year age and calendar period. Person-time at risk was accrued from the first date of employment until the date of death or the end of the follow-up period (31 December 2010). The expected number of deaths was calculated by applying age, calendar time, and cause-specific mortality rates of the Minnesota population to the person-year observations of the study population. SMRs were obtained by computing the ratio of the observed-to-expected number of deaths for the overall mortality and specific causes of death. In addition to overall SMRs, workers with any evidence of employment in zones 1, 2, and 4 were grouped and SMRs for mesothelioma and lung cancer were estimated for each zone.

To further explore summary results for selected causes of death from the SMR analysis, an internal analysis of mesothelioma, lung cancer, hypertensive heart disease, and ischemic heart disease by duration of employment was undertaken. Mesothelioma was captured under ICD-10 code C45, lung cancer was captured under ICD-7 code 162, ICD-8 code 162, ICD-9 code 162, and ICD-10 codes C33 and C34, hypertensive heart disease was captured under ICD-7 codes 440–443, ICD-8

codes 400.1, 400.9, 402, and 404, ICD-9 codes 402 and 404, and ICD-10 codes I11 and I13, and ischemic heart disease was captured under ICD-7 code 420, ICD-8 codes 410–414, ICD-9 codes 410–414 and 429.2, and ICD-10 codes I20, I21, I22, I24, I25, I51.3, and I51.6. Exposure categories were grouped by duration of employment into four exposure categories (1 year, 2–5 years, 6–14 years, and 15+ years). Those who worked 2–5 years were considered most representative of taconite workers with low but stable employment; those who worked less than 2 years were thought to be either transient workers or individuals whose work records were incomplete. Therefore, the 2–5 year exposure group, representing 35% of the study cohort, was used as the reference. Standardised Rate Ratios (SRRs) were computed by standardising to the age and sex distribution of the total study population. Taylor-series-based 95% CIs were calculated for each specific SRR. All SMRs and SRRs were calculated using the Life Table Analysis System (LTAS) V.3.0 software.[20]

RESULTS

This cohort of 31 067 taconite workers with at least 1 year of documented employment was predominantly male (93%), contributed 1 152 966 person-years of observation, and experienced 9094 deaths. Their mean and median durations of employment were 9.4 and 6 years, respectively. Table 1 shows demographic information of the entire cohort and for those with selected causes of death.

The mortality rates from all causes (SMR=1.04, 95% CI 1.02 to 1.06) and all cancers (SMR=1.04, 95% CI 1.00 to 1.08) were higher than the Minnesota population. Among specific cancers, mortality rates for lung cancer (SMR=1.16, 95% CI 1.09 to 1.24) and mesothelioma (SMR=2.77, 95% CI 1.87 to 3.96) were significantly higher than expected. The mortality rate for cardiovascular disease was also elevated (SMR=1.10, 95% CI 1.06 to 1.14), specifically for hypertensive heart disease (SMR=1.81, 95% CI 1.39 to 2.33) and ischemic heart disease (SMR=1.11, 95% CI 1.07 to 1.16). Table 2 shows selected SMRs for the taconite workers cohort. Only one death each for asbestosis and silicosis was observed.

The mortality rates were elevated for mesothelioma and lung cancer in all three zones of the iron range. Among the 20 282 workers who ever worked in zone 1, the SMRs for mesothelioma and lung cancer were 1.85 (95% CI 0.98 to 3.16) and 1.18 (95% CI 1.09 to 1.27) respectively. Among the 5580 workers who ever worked in zone 2, the SMRs for mesothelioma and lung cancer were 7.38 (95% CI 4.30 to 11.82) and 1.43 (95% CI 1.26 to 1.63) respectively. Among the 6501 workers who ever worked in zone 4, the SMRs for mesothelioma and lung cancer were 3.17 (95% CI 1.37 to 6.25) and 1.23 (95% CI 1.07 to 1.40), respectively.

The internal analysis of mesothelioma, lung cancer, hypertensive heart disease, and ischemic heart disease by duration of employment showed elevated but imprecise SRRs when comparing those with 6–14 years, and 15+ years, to those with 2–5 documented work years for hypertensive heart disease. There was no significant elevation in SRRs for mesothelioma, ischemic heart disease and lung cancer (table 3).

DISCUSSION

In this study of Minnesota taconite iron ore miners, an overall higher than expected mortality rate from all causes was observed among taconite workers. Specifically, elevated causes of death from respiratory cancers (including lung cancer and mesothelioma) and cardiovascular disease (including hypertensive heart disease and ischemic heart disease) were identified. These rates were elevated in all three zones of the iron range for mesothelioma and lung cancer. An internal analysis comparing the association between duration of employment and these causes of death did not show a statistically significant elevation in risk for any duration of employment category for mesothelioma, lung cancer, hypertensive heart disease and ischemic heart disease mortality.

Studies of the morbidity and mortality of miners were first carried out in the early 1980s. Higgins *et al*[16] followed a cohort of 5751 men employed at Reserve Mining Company from 1952 to 1976. The study showed no increases in observed respiratory cancers compared to the USA and Minnesota. Cooper *et al*[17 18] studied mortality through 1988 in a cohort of 3431 male workers from Erie and Minntac mines between 1959 and 1977. Total observed deaths were fewer than expected when compared to US and Minnesota death rates. The investigators reported no significantly elevated SMRs for any cause of death. Though these first studies of the health of taconite miners did not show increased risk of mortality, it is important to note that mesothelioma was not captured systematically in mortality registries until 1999 when the ICD V.10 was introduced giving mesothelioma a unique ICD code. Additionally, the follow-up times were not long enough to capture many of the potential cases given the relatively long latency period which, for mesothelioma, is estimated to have a median duration of 32 years.[21] Aside from these two studies that followed a small number of workers over a relatively short amount of time, there has been no comprehensive look at the health of taconite miners across the entire Mesabi iron range.

Several occupational studies have been conducted that evaluate the health risk to workers exposed to non-asbestiform EMPs in other occupational settings. These include studies of talc miners in upstate New York and Homestake gold miners in South Dakota. In a 2002 mortality study of talc miners, Honda *et al*[22] reported an excess in mortality from all cancers, lung cancer, ischemic heart disease, and non-malignant respiratory disease. A 2012 follow-up commentary argued that talc ore exposure also increases the risk of mesothelioma,[11] though that conclusion has been debated.[12] Though the authors argue, the lack of an exposure-response relationship indicates the lung cancer excess may not be related to talc ore dust; rather it might be explained by a relatively high smoking rate in the population,[22] it is unlikely that confounding by smoking accounts fully for the lung cancer excess observed in the study.[4] The results of these studies have been argued further, as the composition of industrial-grade talc has been redefined. Industrial-grade talc deposits are a complex mixture of mineral particles that vary substantially and may rarely include asbestos fibers.[23] Price[23] argues that elevated rates of mesothelioma found in New York talc miners are a result of previous occupational exposure to commercial asbestos. Several studies of miners at the Homestake gold mine in South Dakota were done in the 1970s and 1980s.[24–26] An excess of respiratory cancer was reported in the earliest study,[24] and a small excess of lung cancer was reported in the studies by McDonald *et al*[25] and Steenland and Brown. [26] The results of these studies suggest a weak association between dust exposure and lung cancer and like the studies of talc miners, no dose-response relationship was observed.[4] The studies of New York talc miners and Homestake gold miners cannot definitively conclude whether exposure to non-asbestiform minerals poses any risk to human health.

The elevated risk of lung cancer and mesothelioma as a result of exposure to asbestiform EMPs is well documented in the

Table 1 Characteristic of taconite workers with selected causes of death

	Selected cause of death									
	Mesothelioma		Lung cancer		Hypertensive heart disease		Ischemic heart disease		Total cohort	
	N	Per cent	N	Per cent	N	Per cent	N	Per cent	N	Per cent
Duration of employment (years)										
1	4	13.33	123	12.96	6	9.68	241	11.03	4353	14.01
2–5	8	26.67	250	26.34	14	22.58	576	26.36	10 839	34.89
6–14	6	20.00	239	25.18	18	29.03	545	24.94	9072	29.20
15+	12	40.00	337	35.51	24	38.71	823	37.67	6803	21.90
Sex										
Male	30	100.0	915	96.42	58	93.55	2143	98.08	28 860	92.90
Female	0	0	34	3.58	4	6.45	42	1.92	2202	7.09
Unknown	0	0	0	0	0	0	0	0	5	0.02
Age at hire (yrs)										
<20	12	40.00	247	26.03	15	24.19	628	28.74	11 635	37.45
20–29	14	46.67	494	52.05	34	54.84	1163	53.23	15 962	51.38
30–39	4	13.33	165	17.39	10	16.13	312	14.28	2851	9.18
40+	0		43	4.53	3	4.84	82	3.75	619	1.99
Decade of hire										
<1950	6	20.00	289	30.45	20	32.26	799	36.57	4557	14.67
1950–1959	17	56.67	442	46.58	21	33.87	954	43.66	9072	29.20
1960–1969	6	20.00	143	15.07	14	22.58	272	12.45	6897	22.20
1970–1979	1	3.33	72	7.59	6	9.68	157	7.19	10 332	33.26
>1980	0		3	0.32	1	1.61	3	0.14	209	0.67
Vital status										
Alive									20 814	67.00
Dead	30	100.0	949	100.0	62	100.0	2185	100.0	8952	28.82
Presumed alive									1157	3.72
Presumed dead									144	0.46
Total	30	100.0	949	100.0	62	100.0	2185	100.0	31 067	100.0

literature.[4 27–29] However, risk of exposure to non-asbestiform ampbibole and non-amphibole EMPs as found in taconite mining operations, is not understood, and evidence of their toxicity is inconclusive.[4] Our results indicate an increased risk for mesothelioma and lung cancer among taconite workers with at least 1 year of employment, but no exposure-response association for duration of employment was detected. Mortality from cardiovascular disease, specifically hypertensive heart disease and ischemic heart disease, were also increased. Major risk factors for the development of heart disease include hypertension, diabetes and cholesterol. Lifestyle factors, such as smoking, physical activity, and diet also play a role in disease risk. This study result suggests that lifestyle factors likely contribute to disease burden in this working population. However, occupational risk cannot be ruled out entirely. Other workplace factors, such as stress, noise, vibration, extreme temperature and shift work, may also affect cardiovascular disease risk.[30 31] Additionally, environmental factors, such as particulate air pollution, have also been shown to increase the risk of cardiovascular events from short and long-term exposure,[32–36] and elevated cardiovascular mortality has been identified in other working cohorts.[37] Thus, a combination of workplace and lifestyle factors may be contributing to the excess in cardiovascular disease in this taconite workers cohort.

The following limitations should be considered when interpreting the results of this analysis. Instead of specific exposure measurements for this analysis, duration of employment in the taconite mining industry was meant as a proxy for exposure averaged across all jobs and locations on the range. Our estimate of employment duration was measured as the last date of employment minus the start date. This crude measure of employment duration does not take into account any gaps in work history which could result in employment duration misclassification. Individuals who appear to have worked more than 15 years may have a much shorter cumulative work history when considering gaps in employment. We did not have access to information on some confounding variables, most notably smoking status which is a major risk factor for lung cancer and cardiovascular disease. Though we could not adjust for smoking in this analysis, it is possible that smoking explains at least some of this excess risk in lung cancer mortality especially given that working cohorts typically have higher smoking rates than the general population, and because of the high attributable risk for smoking.[38] Smoking however, is not a risk factor for mesothelioma, thus, the high mortality ratio of mesothelioma suggests that there may be occupational exposures to account for some of the increased risk of these diseases.

The risk of mesothelioma may also be underestimated, as the specific ICD code for this disease was not available until 1999, thus, earlier cases were misclassified as another disease. The lower percentage of mesothelioma cases, as compared to other causes of death (table 1) of those who were hired prior to 1950, the earliest exposed, may represent this misclassification. These undercounted mesotheliomas may have had more hematite exposure or exposure to the taconite processes in their earlier work. However, identifying other potential mesothelioma cases

Table 2 Selected SMRs for Minnesota Taconite Workers with ≥1 year employment*

Underlying cause of death	Observed	Expected	SMR	95% CI
All causes	9094	8764.69	1.04	1.02 to 1.06
All cancers	2710	2609.86	1.04	1.00 to 1.08
Respiratory	981	846.74	1.16	1.09 to 1.23
Larynx	26	23.84	1.09	0.71 to 1.60
Trachea, bronchus, lung	949	815.67	1.16	1.09 to 1.24
Pleura	1	1.81	0.55	0.01 to 3.08
Mesothelioma	30	10.82	2.77	1.87 to 3.96
Heart diseases	2676	2435.81	1.10	1.06 to 1.14
Hypertensive heart disease	62	34.17	1.81	1.39 to 2.33
Ischemic heart disease	2185	1964.93	1.11	1.07 to 1.16
Cerebrovascular disease	391	384.30	1.02	0.92 to 1.12
Hypertension w/o heart disease	35	52.80	0.66	0.46 to 0.92
Respiratory Diseases	582	621.19	0.94	0.86 to 1.02
COPD	363	369.89	0.98	0.88 to 1.09
Asbestosis	1	2.90	0.35	0.01 to 1.92
Silicosis	1	1.09	0.91	0.02 to 5.09
Transportation injuries	339	329.15	1.03	0.92 to 1.15
Other injury	239	221.75	1.08	0.95 to 1.22
Violence	289	258.41	1.12	0.99 to 1.26

*Adjusted for age, calendar period, and sex.
SMR, Standardised mortality ratios.

using previously used rubrics[39] would not change the interpretation that taconite workers have elevated rates of mesothelioma. It is also important to note that the cases were identified as primary causes of death, and do not capture incident cases or contributing causes of death and, therefore, do not accurately reflect the total disease burden in the cohort.

Although the SMR for mesothelioma was elevated, the internal analysis did not identify an association by duration of employment. One possible explanation of this is if the elevated risk of mesothelioma is related to work in the taconite industry, that risk may not be a function of time, rather a function of specific exposures while performing certain job tasks. Likewise, the internal analysis did not show an increased risk of lung cancer, hypertensive heart disease, and ischemic heart disease by duration of employment, suggesting that other lifestyle factors are potentially contributing to the elevated SMRs. These results could also have been affected by the crude employment duration measure resulting in misclassification of time worked.

The analysis by zone was a cursory examination of the risk across the iron range, since it evaluated any work in a zone. It does not allow for comparison of risk between zones, but only suggests the risk of mesothelioma and lung cancer is elevated with employment in each zone of the iron range. We limited the analysis to iron mining workers who were born in 1920 or later and who had at least 1 year of documented employment. Restricting the cohort further to those born in 1930 or later (excluding an additional 8504 workers) in order to potentially better focus on taconite mining did not substantially change the results and interpretation of this study (lung cancer SMR=1.15, 95% CI 1.04 to 1.27, mesothelioma SMR=3.59, 95% CI 2.16 to 5.60). Examination of the entire cohort of 44 243 individuals, including those with less than 1 year of documented employment, likewise did not substantially change the results and interpretation of this study (lung cancer SMR=1.20, 95% CI 1.14 to 1.27, mesothelioma SMR=2.89, 95% CI 2.11 to 3.87).

This study has some notable strengths, including the large size and long follow-up of the cohort and the high proportion of workers whose vital status was ascertained. Vital status was found on 98% of the eligible cohort, and few workers (4%) were excluded from the analysis due to data quality problems. Additionally, this study captured mortality from mesothelioma; early mortality studies of taconite workers were unable to evaluate mesothelioma until 1999 when ICD-10 became available.

Table 3 Standardised rate ratios by duration of employment, adjusted for age, calendar period, and sex

	Cause of death							
	Mesothelioma		Lung cancer		Hypertensive heart disease		Ischemic heart disease	
Employment duration (years)	Obs	SRR (95% CI)	Obs	SRR (95% CI)	Obs	SRR (95% CI)	Obs	SRR (95% CI)
1	4	1.14 (0.34 to 3.81)	123	1.01 (0.81 to 1.26)	6	0.90 (0.34 to 2.41)	241	0.88 (0.76 to 1.03)
2–5 (ref)	8	1.0	250	1.0	14	1.0	576	1.0
6–14	6	0.77 (0.26 to 2.25)	239	1.01 (0.85 to 1.21)	18	1.29 (0.63 to 2.63)	545	0.99 (0.88 to 1.11)
15+	12	1.08 (0.44 to 2.67)	337	0.94 (0.79 to 1.13)	24	1.84 (0.82 to 4.11)	823	0.98 (0.88 to 1.10)

SRR, Standardised rate ratios.

This study allowed us to characterise the mortality of the entire Minnesota mining population as compared to the rest of Minnesota, as well as capture information specific to where miners worked by zone which has not been done before. The analysis identifies a need for future studies with more refined exposure estimates to evaluate the extent to which mining-related exposures specifically contribute to disease burden and will be the next step in our evaluation of the health of taconite mining workers.

CONCLUSION

In summary, this analysis suggests taconite workers may be at increased risk for mortality from some cancers and cardiovascular diseases. Duration of employment did not appear to be associated with the mortality risk. However, based on the limited way exposure potential was evaluated, we cannot say for sure what the role of actual workplace exposures play in the disease excess. Additional investigation is warranted.

What this paper adds

- Mining and processing of taconite results predominantly in exposure to non-asbestiform amphibole and non-amphibole minerals.
- The health risks of these exposures are uncertain.
- Increased mortality rates from mesothelioma, lung cancer and some cardiovascular disease among taconite workers were observed.

Acknowledgements The authors thank Alan Bender and Allan Williams of the Minnesota Department of Health for thoughtful input. The authors would also like to acknowledge the efforts made by study staff members Richard Hoffbeck, Diane Kampa, Nancy Pengra, and Leslie Studenski.

Contributors All authors participated in the study design, analysis, and interpretation of the data. All authors assisted in revision and approved the final manuscript.

Funding This study was funded by the State of Minnesota. The views expressed are the authors' and do not reflect the State of Minnesota. EM Allen was supported in part by the Midwest Center for Occupational Safety and Health training grant CDC/NIOSH 2T42 OH008434.

Competing interests None.

Ethics approval Ethics approval for this study was provided by the University of Minnesota Institutional Review Board.

Provenance and peer review Not commissioned; externally peer reviewed.

REFERENCES

1 Berndt ME, Brice WC. The origins of public concern with taconite and human health: reserve mining and the asbestos case. *Regul Toxicol Pharmacol* 2008;52 (S1):S31–9.
2 Minnesota Department of Natural Resources. Digging in MN minerals: Taconite 2011 (Retrieved 22 August 2011). http://www.dnr.state.mn.us/education/geology/digging/mining.html
3 Hwang J, Gurumurthy R, Raynor P, *et al*. Comprehensive assessment of exposures to elongate mineral particles in the taconite mining industry. *Ann Occup Hyg* 2013;57:966–78.
4 Department of Health and Human Services, National Institute for Occupational Safety and Health. Asbestos fibers and other elongate mineral particles: State of the science and roadmap for research, 2011: Publication No. 2011-159.
5 McSwiggin PL, Morey GB. Overview of the mineralogy of the Biwabik Iron Formation, Mesabi Iron Range, northern Minnesota. *Regul Toxicol Pharmacol* 2008;52(S1):S11–25.
6 Jirsa MA, Miller JD, Morey GB. Geology of the Biwabik Iron Formation and Duluth Complex. *Regul Toxicol Pharmacol* 2008;52(S1):S5–10.
7 Wilson R, McConnell EE, Ross M, *et al*. Risk assessment due to environmental exposures to fibrous particulates associated with taconite ore. *Regul Toxicol Pharmacol* 2008;52(S1):S232–45.
8 Langer AM, Maggiore CM, Nicholson WJ, *et al*. The contamination of Lake Superior with amphibole gangue minerals. *Ann NY Acad Sci* 1979;330:549–72.
9 Williams C, Dell L, Adams R, *et al*. State-of-the-science assessment of non-asbestos amphibole exposure: is there a cancer risk? *Environ Geochem Health* 2013;35:357–77.
10 Yang H-Y, Shie R-H, Chen P-C. Carving of non-asbestiform tremolite and the risk of lung cancer: a follow-up mortality study in a historical nephrite processing cohort. *Occup Environ Med* 2013;70:852–7.
11 Finkelstein MM. Malignant Mesothelioma Incidence Among Talc Miners and Millers in New York State. *Am J Ind Med* 2012;868(April):863–8.
12 Nolan RP, Gamble JF, Gibbs GW. Letter to the editor on commentary: malignant mesothelioma incidence among talc miners and millers in New York state by M M Finkelstein. *Am J Ind Med* 2013;56:1116–18.
13 Gamble JF, Gibbs GW. An evaluation of the risks of lung cancer and mesothelioma from exposure to amphibole cleavage fragments. *Regul Toxicol Pharmacol* 2008;52 (S1):S154–86.
14 Hilding AC, Hilding DA, Larson DM, *et al*. Biological effects of ingested amosite asbestos, taconite tailings, diatomaceous earth and Lake Superior water in rats. *Arch Environ Health* 1981;36:298–303.
15 Levy BS, Sigurdson E, Mandel J, *et al*. Investigating possible effects of asbestos in city water: surveillance of gastrointestinal cancer incidence in Duluth, Minnesota. *Am J Epidemiol* 1976;103:362–8.
16 Higgins IT, Glassman JH, Oh MS, *et al*. Mortality of Reserve Mining Company employees in relation to taconite dust exposure. *Am J Epidemiol* 1983;118:710–19.
17 Cooper WC, Wong O, Graebner R. Mortality of workers in two Minnesota taconite mining and milling operations. *J Occup Med* 1988;30:506–11.
18 Cooper WC, Wong O, Trent LS, *et al*. An updated study of taconite miners and millers exposed to silica and non-asbestiform amphiboles. *J Occup Med* 1992;34:1173–80.
19 Minnesota Cancer Surveillance System Epidemiology Report. *Cancer incidence rates in Northeastern Minnesota*. Minnesota Department of Health, 1999.
20 Centers for Disease Control and Prevention. Life Table Analysis System. 2013. http://www.cdc.gov/niosh/ltas
21 Lanphear BP, Buncher CR. Laten period for malignant mesothelioma of occupational origin. *J Occup Med* 1992;34:718–21.
22 Honda Y, Beall C, Delzell E, *et al*. Mortality among workers at a Talc mining and milling facility. *Ann Occup Hyg* 2002;46:575–85.
23 Price B. Industrial-grade talc exposure and the risk of mesothelioma. *Crit Rev Toxicol* 2010;40:513–30.
24 Gillam JD, Dement JM, Lemen RA, *et al*. Mortality patterns among hard rock gold miners exposed to an Asbestiform mineral. *Ann N Y Acad Sci* 1976;336–44.
25 McDonald JC, Gibbs GW, Liddel FDK, *et al*. Mortality after long exposure to cummingtonite-grunerite. *Am Rev Respir Dis* 1978;118:271–7.
26 Steenland K, Brown D. Mortality study of gold miners exposed to silica and nonasbestiform amphibole minerals: an update with 14 more years of followup. *Am J Ind Med* 1995;27:217–29.
27 McDonald JC, McDonald AD. The epidemiology of mesothelioma in historical context. *Eur Respir J* 1996;9:1932–42.
28 Robinson BWS, Musk AW, Lake RA. Malignant mesothelioma. *Lancet* 2005;366:397–408.
29 Robinson BM. Malignant pleural mesothelioma: an epidemiological perspective. *Ann Cardiothorac Surg* 2012;1:491–6.
30 Kawachi I, Colditz GA, Stampfer MJ, *et al*. Prospective study of shift work and risk of coronary heart disease in women. *Circulation* 1995;92:3178–82.
31 Price AE. Heart disease and work. *Heart* 2004;90:1077–84.
32 Brook RD, Rajagopalan S, Pope CA, *et al*. Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 2010;121:2331–78.
33 Dockery DW, Pope CA, Xu X, *et al*. An Association between Air Pollution and Mortality in Six U.S. Cities. *N Engl J Med* 1993;329:1753–9.
34 Miller KA, Siscovick DS, Sheppard L, *et al*. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 2007;356:447–58.
35 Pope CA III, Burnett RT, Thurston GD, *et al*. Cardiovascular mortality and long-term exposure to particulate air pollution: epidemiological evidence of general pathophysiological pathways of disease. *Circulation* 2004;109:71–7.
36 Bhatnagar A. Environmental cardiology: studying mechanistic links between pollution and heart disease. *Cir Res* 2006;99:692–705.
37 Gallagher LG, Ray RM, Li W, *et al*. Occupational exposures and mortality from cardiovascular disease among women textile workers in Shanghai, China. *Am J Ind Med* 2012;55:991–9.
38 Centers for Disease Control and Prevention. Current cigarette smoking prevalence among working adults-United States 2004–2009. *MMWR Morb Mortal Wkly Rep* 2011;60:1305–9.
39 Sullivan PA. Vermiculite, respiratory disease, and asbestos exposure in Libby, Montana: update of a Cohort Mortality Study. *Environ Health Perspect* 2007;115:579–85.

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Occup Environ Med published online May 9, 2014
doi: 10.1136/oemed-2013-102000