FILE NAME: Talc (TALC)

DATE: 1982 June

DOC#: TALC099

DOCUMENT DESCRIPTION: Medical Journal Article - The Mortality Experience of Upstate NY Talc Workers

The Mortality Experience of Upstate New York Talc Workers

W. T. Stille, D.P.H., and Irving R. Tabershaw, M.D.

Deaths for a 31-year period (1948 to 1978) were analyzed in a historical prospective cohort study of 655 white male talc workers. Death rates from all causes, from cancer of the respiratory system, and from nonmalignant respiratory disease were not significantly different from those of the U.S. white male population. However, significant differences for these causes of death were found among workers who had previous occupational histories. An analysis of the latency periods of the observed lung cancer suggests that exposure to an etiologic agent during previous work experience may play a role in the development of lung cancer.

Talc pneumoconiosis was first associated with talc mining and milling by Thorel in 1896[1] and since that time this association has been repeatedly documented in studies from various geographic sites. However, there are a number of contradictory reports regarding disability from talc pneumoconiosis and its effect on working capacity and respiratory function. The contradiction in these reports has been ascribed to the differences in the mineral composition of the various talcs. Kleinfeld et al,[2,3] in a series of reports, confirmed the increased incidence of talc pneumoconiosis and noted that among the talc workers who were 60 to 79 years of age and had 15 or more years of exposure the incidence of lung cancer was four times that among the general population. Their follow-up study repeated these findings but noted that the observed deaths from 1945 to 1959 dropped to approach the expected mortality between 1960 and 1969. The authors attributed this improved mortality experience to better environmental dust control.[3]

Brown and Wagoner,[4] using a similar cohort from the same population base; i.e., talc miners and millers in upstate New York, also found a statistically significant increase in the occurrence of bronchogenic cancer. The average latency for these cancer deaths was 20 years. They noted that data on several variables, particularly cigarette smoking and previous work history, were not available. Thus, from the work of Kleinfeld et al[2,3] and Brown and Wagoner, it appeared that talc mining in upstate New York is associated with an increase in lung cancer. Selevan et al,[5] in studying workers in Vermont talc mines and mills, showed an excess of deaths due to nonmalignant respiratory disease among millers and an excess of lung cancer mortality among miners. The lung cancer rate was similar to that found by Brown and Wagoner in their cohort of New York State workers. Selevan et al concluded, however, that agents other than talc, "either alone or in combination with talc dust, affect mine workers. The possible role of radon daughter exposures for this cancer mortality risk cannot be eliminated."[5]

The present study includes all workers in one talc mine and mill (TMX) in upper New York State who were employed between 1948, when operations began, and Dec 31, 1977.

Materials and Methods

The work force consisted of 744 persons employed between Jan 1, 1948 and Dec 31, 1977. Thirty-five women, primarily employed in office and administrative jobs, were not included because of the small numbers and a lack of exposure to talc. Nearly all employees were white and records indicate that to date all decedents have been white. The vital status of 36 (5.1%) men remained unresolved after Social Security and New York State Motor Vehicle Bureau records were searched. The maximum cohort available for analysis consisted of 708 white men, 113 of whom were known to be dead by Dec. 12, 1978, which was established as the vital status cutoff date.

Demographic data (microfilmed from employment records) consisted of the following: worker identifiers; sex; date of birth, employment and death; cause of death and information on prior employers. Data on cigarette smoking were not available. The occupational history included dates of work, job codes, and work location codes. Records were updated for the dates and causes of death as such information became available. Death certificates were coded according to the eighth revision of the International Classification of Diseases by a trained nosologist. Comparisons of observed deaths by age and year of death with those expected in the U.S. white male population were done by means of the modified life-table methods described by Monson.[6] All standard mortality ratios (SMRs) were computed by this program. Fifty-three workers whose records lacked dates of birth or other significant data had to be

From Tabershaw Occupational Medicine Associates, 6110 Executive Blvd., Rockville, MD 20852. Dr. Stille was formerly Senior Epidemiologist. Dr. Tabershaw is Senior Occupational Physician.

WES001126

ork is associated
al,[5] in studying
ills, showed an
spiratory disease
ancer mortality
. similar to that
ort of New York
, however, that
ombination with
le role of radon
ty risk cannot be

in one talc mine
e who were em-
began, and Dec

rsons employed
irty-five women,
rative jobs, were
rs and a lack of
were white and
have been white.
ined unresolved
g Motor Vehicle
aximum cohort
ite men, 113 of
1978, which was

m employment
rker identifiers;
; cause of death
ata on cigarette
tional history in-
: location codes.
uses of death as
certificates were
the International
logist. Compari-
death with those
in were done by
ds described by
MRs) were com-
s whose records
data had to be

ille & Tabershaw

Table 1 -- Observed and Expected Numbers of Deaths and SMRs Among TMX Workers (655 White Males With 11,350 Years at Risk).

Cause of Death ICD,* 8th Revision	Deaths Observed	Expected	SMR
All deaths	113	107	106
Tuberculosis (010-019)†	3	.7	414
All cancer (140-209)	25	20.5	122
Digestive system (150-159)	6	5.7	105
Esophagus (150)	1	.5	216
Stomach (151)	1	1.1	92
Liver (155-156)	2	.4	511
Pancreas (157)	1	1.1	89
Respiratory system (160-163)	11	6.8	163
Lung (162)	10	6.4	157
Prostate (185)	1	1.3	79
Kidney (189)	1	.5	195
Brain (191-192)	1	.7	148
Lymphosarcoma (200)	1	.5	208
Hodgkin's disease (201)	2	.3	631
Leukemia (204-207)	2	.9	229
All lymphopoietic cancer (200-209)	5	2.1	230
All circulatory system (390-468)	48	53.7	89
Myocardial infarction (410-413)	33	38.3	86
Cerebrovascular disease (430-438)	5	7.1	71
All respiratory disease (460-519)	10	6.1	164
Pneumonia (480-486)	4	2.2	182
Emphysema (492)	2	1.7	121
All digestive system (520-577)	3	5.3	57
External causes of death (800-998)	11	12.6	87
Motor vehicle accidents (810-827)	4	4.3	93
Suicide (950-959)	2	2.8	70

* ICD indicates International Classification of Diseases
† Numbers refer to ICD code

Table 2 -- Observed and Expected Numbers of Deaths and SMRs Among Talc Workers With Known Prior Employment Before Work at the TMX (540 White Males With 9,168 Years at Risk).

Cause of Death ICD,* 8th Revision	Deaths Observed	Expected	SMR
All deaths	90	60.6	148*
Tuberculosis (010-019)†	3	.4	680*
All cancer (140-209)	22	11.6	182*
Digestive system (150-159)	6	3.0	201
Esophagus (150)	1	.3	382
Stomach (151)	1	.6	180
Liver (155-156)	2	.2	1,013*
Pancreas (157)	1	.6	164
Respiratory system (160-163)	8	4.0	228‡
Lung (162)	8	3.7	214
Kidney (189)	1	.3	326
Brain (191-192)	1	.5	206
Lymphosarcoma (200)	1	.3	326
Hodgkin's disease (201)	2	.2	849
Leukemia (204-207)	2	.5	391
All lymphopoietic cancer (200-209)	5	1.3	374*
All circulatory system (390-468)	36	28.0	129
Myocardial infarction (410-413)	27	20.3	133
Cerebrovascular disease (430-438)	3	3.1	97
All respiratory disease (460-519)	9	2.9	307*
Pneumonia (480-486)	3	1.0	274
Emphysema (492)	2	.7	278
All digestive system (520-577)	1	3.5	28
External causes of death (800-998)	11	9.8	112
Motor vehicle accidents (810-827)	4	3.4	119
Suicide (950-959)	2	2.2	92

* Indicates statistical significance at the 1% level
† Numbers in parentheses refer to ICD codes
‡ Indicates statistical significance at the 5% level

dropped from the life-table analyses. Thus, the study cohort consisted of 655 white males, a relatively small sample size and therefore a limitation on the study.

Results

Table 1 shows the numbers of observed and expected deaths in the study cohort of 655 white male talc workers. Although the SMRs are elevated, the numbers of deaths from all causes, from cancer of the respiratory tract and lung from nonmalignant respiratory disease, or from other causes of death are not significantly different from those occurring in the U.S. white male population. Numbers of deaths from circulatory diseases, digestive tract diseases, and accidental and external causes are lower, but not significantly so. The SMR of 106 for all causes of death has 95% confidence limits from 87 to 128, thus the total talc worker population has an SMR within the usual range of variation from the national data. Since people who are chronically ill or disabled are usually unemployable and have higher death rates, healthy working populations typically have SMRs below 100. The SMRs in an occupational group with values over 100 indicate either possible work place hazards or possible confounding with non-occupational exposures.

One such non-occupational exposure is smoking, and it is noted that the SMR of 163 for respiratory cancer in this population is consistent with a smoking effect. While the actual smoking experience of this population is not known, insight into it is gained from knowing the smoking patterns of current TMX workers, many of whom are included in the study cohort. Among 151 workers given a medical examination at TMX in 1979, 69 (46%) were current smokers, 38 (25%) were past smokers and 44 (29%) were nonsmokers. The percentage of current smokers is higher than in the general population, but is consistent with patterns seen in the mining industry.

The SMR for all causes of death, 106, may also reflect in part inclusion in the cohort of everyone ever employed during the study period, regardless of their length of employment. Universal cohort membership generally tends to increase SMRs, since short-term workers often have a greater mortality experience than long-term employees. Most study designs exclude workers with less than one year of working experience. The practice was not followed i-

WES001127

Table 3 – Observed and Expected Numbers of Deaths and SMRs Among Talc Workers With No Known Work Prior to TMX Employment (115 White Males With 2,184 Years of Risk).

Cause of Death ICD, 8th Revision	Deaths Observed	Expected	SMR
All deaths	23	45.9	50*
All cancer (140-209)†	3	9.0	33
Lung (162)	2	2.6	76
Prostate (185)	1	.8	120
All circulatory system (390-458)	12	25.9	46
Myocardial infarction (410-413)	6	18.0	33
Cerebrovascular disease (430-438)	2	4.0	50
All respiratory diseases (460-519)	1	3.2	31
Pneumonia (480-486)	1	1.1	90
All digestive system (520-577)	2	1.8	112

* Indicates statistical significance at the 1% level; note that this SMR is lower than expected with respect to the U.S. national data

† Numbers in parentheses refer to ICD codes

this study in order to maximize cohort size.

The use of the U.S. white male population as the external comparative group does not necessarily describe the TMX mortality experience, since the mortality experience of the U.S. white male population is likely to vary substantially from that of this cohort. For example, the New York State cancer death rate is 199.24 compared to the national average of 174.04, approximately a 13% difference.[7]

Since exposures prior to employment at the TMX could have included carcinogens and other substances hazardous to the lungs, the results have been analyzed to investigate this possibility. Table 2 shows the observed and expected numbers of deaths and the SMRs for all workers whose job histories show prior work before employment at the TMX. Deaths for all causes are significantly increased, as are deaths for a number of cancers and for nonmalignant respiratory diseases. Since the cancers and lung diseases typically have long latencies, the possibility exists that exposures prior to work at the TMX were responsible for at least some of these diseases.

Table 3 shows the observed and expected numbers of deaths for workers with no known employment prior to the TMX. Mortalities for all disease categories do not differ significantly from those for the comparable U.S. population. In fact, the SMRs from all cancers and all respiratory tract diseases (33 and 31, respectively) were among the lowest of all the mortalities. It should be noted that workers whose employment records were devoid of work history data were judged as having had no known prior employment and thus were included in Table 3. If some of those workers did indeed have work experience before employment at the TMX, the effect would be to increase the SMRs. However, even with this possible compromising effect, Table 3 shows a remarkably healthy work force, with most mortalities at 50% or less of those of the comparable U.S. population. As noted earlier, however, the small sample size limits the conclusions that can be drawn from this study.

Five of the 12 lung cancer cases, which are included in Table 5, were substantially older than the median age of

Table 4 – Age at Death and Segments of Work History of Workers at the TMX With Bronchogenic Carcinoma.

Case No./Death, yr	Age at	Pre-TMX Employment	Years or Days Employed at the TMX	Post-TMX Employment
1/39		10	4	7
2/49		8	17	6
3/53		8	47 days	27
4/53		13	56 days	23
5/54		12	3	21
6/55		21	3	13
7/59		16	345 days	24
8/62		26	17	1
9/64		25	17 days	21
10/79		39	8 days	22

the entire cohort at age of hire. For those workers with TMX-only employment, 63% were under 25 when hired, contrasted with 35% for those with previous experience. The difference in age at hire between the two cohorts may partially explain the deficit in disease mortality among TMX-only workers and the lung cancer experience of those with other work history.

Discussion

Workers with previous jobs before employment at the TMX were found to have high mortalities, as shown in Table 2. Workers with no known work history before employment at the TMX were found to have much lower mortalities (Table 3). Thus, it would seem that exposures to health hazards occurred in these earlier jobs and that these exposures are causally related to the higher rates of death. Obviously, mines and mining operations are not uniform with respect to the occurrence of health hazards and, as previously stated, Selevan et al[5] have described increased rates of lung cancer in some talc miners exposed to radon daughters. No significant radon daughter exposures occurred at the TMX.[8,9] The low mortalities given in Table 3 are in agreement with the absence of radon daughter and other related hazardous exposures at the TMX. However, mortality studies such as ours lend themselves to various conclusions. Lacking data on smoking and other personal risk factors, we sought to obtain confirmation for our interpretations by an independent analysis of lung cancer latencies.

Data for an analysis of the lung cancer latencies are given in Table 4. For each of the ten cases of lung cancer, the employee's years of occupational history are given as the following: pre-TMX employment (employment was deter-

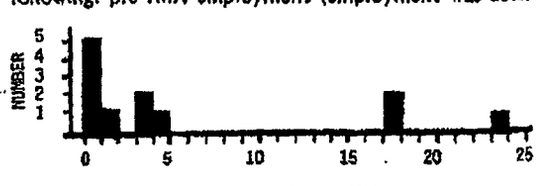


Fig. 1 – Number of lung cancer cases by years of employment at TMX.

WES001128

Table 5 -- Latencies of Bronchogenic Carcinomas in TMX Workers

Latency, *yr	Bronchogenic Carcinomas in TMX Workers Whose Exposure is Hypothesized to Begin: On Employment at the TMX	Bronchogenic Carcinomas in TMX Workers Whose Exposure is Hypothesized to Begin: At Age 18, Prior to TMX Employment
<10	1	–
10-14	1	–
15-19	2	–
20-24	5	1
25-29	3	1
30-34	–	1
35-39	–	4
40-44	–	3
45-49	–	1
50-54	–	–
55-59	–	–
>60	–	1
Total	12	12
Latency	19.9	38.2

* From onset of exposure to diagnosis or death

mined arbitrarily to have begun at age 18); tenure at the TMX; and the period from TMX employment until the diagnosis of lung cancer or death. Although not included in Table 4, two additional persons with lung cancer should be noted. The first was surgically treated for lung cancer at age 47 after 137 days of employment at the TMX. At age 57 he died of pneumonia. His significant occupational periods were 29 years pre-TMX, 137 days of TMX tenure, and zero years post-TMX to the time of diagnosis of lung cancer. The second individual died of lung cancer after the cutoff date. His significant occupational periods were 15 years pre-TMX, 23 years of TMX tenure, and six years post-TMX.

To explore the nature of the dose-response relationship, column 3 in Table 4, which lists numbers of years or days employed at the TMX, is graphically represented in the Figure. The two additional cases of lung cancer just described are included. The clustering of nine of the 12 cases within the first five years of employment at the TMX shows an inverse dose response, i.e., higher risks of lung cancer with less occupational exposure to talc. The lack of a dose response, in terms of years of exposure at the TMX, is in agreement with our findings that workers with "exclusive" TMX employment seem to be at no considerable risk of having lung cancer develop.

Knowledge of the latency period of a disease permits determination of the probable time at which pathological processes began. Thus, the analysis of disease latencies can be useful in attributing or refuting evidence as to when causal exposures occurred. Armenian and Lilienfeld[10] have reviewed the latency periods of a number of cancers. They present evidence that leukemias in different populations and under different conditions have median latency periods and latency variabilities that are quite similar. They also reviewed data that indicate that the intensity of exposure has very little effect on latencies of other neoplasms. Hence, we hypothesize that if occupational talc exposure results in pulmonary exposure to a carcinogen, then the distribution of TMX workers lung cancer latencies should be similar to those reported for lung cancer in other studies. However, if such exposure is not carcinogenic to the lung, then the distribution of TMX lung cancer latencies would be dissimilar to lung cancer latencies reported elsewhere.

To test the hypothesis that carcinogenic exposure began at TMX, the median lung cancer latency of 36.5 years, as calculated by Armenian and Lilienfeld,[10] was compared to the latency period from the onset of TMX employment to the diagnosis of each lung cancer. As can be seen from Column 2 in Table 5, the average latency period of 19.9 years for lung cancer among the TMX workers is almost half the 36.5 years noted above. The Chi Square goodness of fit test provides a quantitative basis for rejecting the hypothesis at the 5% level of statistical significance.

If the occupational talc exposures to the TMX are not carcinogenic, then the question shifts to what other exposures occurred that may have caused the lung cancers. Since each of the subjects with lung cancer included in Table 4 had between eight and 39 years of possible occupational exposure from the time of his 18th birthday to employment at the TMX, this prior period of exposure may be related to subsequent cancers. To test this possibility, we hypothesized that the causal exposure for the lung cancer occurred before employment at the TMX. The data in the last column of Table 5 reveal a much more reasonable relationship to the lung cancers reported by Armenian and Lilienfeld. This second hypothesis, that the causal exposure began before TMX employment, yields an average latency period of 38.2 years, which is only slightly longer than the Armenian and Lilienfeld median of 36.5 years. The Chi Square goodness of fit is almost zero, indicating a very close agreement between the lung cancer worker latency periods and those of talc workers when exposures prior to TMX employment are hypothesized as carcinogenic. Hence, the initiation of cigarette smoking in the late teenage years, World War II exposures, excessive exposures to mineral dust in the past, diet and nutritional status, absence of complete work history, preexisting diseases, and other unknown etiologic agents could contribute to the etiology of the observed lung cancers, while exposures at TMX seem to be noncarcinogenic. It should be noted that nine of the 12 employees with lung cancer worked underground in TMX, which suggests that their other jobs also were likely to have been underground. Review of the death certificates tends to confirm the observation that these men worked in occupations known for their dusty environment. The employment policy of TMX to preferentially hire experienced workers appears to have resulted in the confounding of prior hazardous exposures with employment at TMX.

Summary and Conclusions

A historical prospective mortality study of workers at an upstate New York talc mine demonstrated elevated mortali-

WES001129

ties but no significant increases in the numbers of deaths from lung cancer, from nonmalignant respiratory disease, and from all causes. However, workers with exposures in other jobs prior to work at the TMX were found to have excessive mortality from lung cancer and from nonmalignant respiratory tract disease. Exposures in jobs held prior to TMX employment also were implicated in an independent analysis of lung cancer latencies.

Acknowledgment

Technical and editorial contributions were made to this communication by Maureen A. Vogt and M. James Sharpe. Juliet A. Foster, Pamela J. Trotter, and Deborah Rogers aided in data collection and processing.

References

1. Thorel C: Die speicksteinlunge (ein beitragzur pathologischen anatomic der straublungen). *Beitr Path Ant* 28:85-101, 1896.

2. Kleinfeld M, Mossite J, Kooyman O, et al: Mortality among talc miners and millers in New York State. *Arch Environ Health* 14:663-667, 1967.

3. Kleinfeld M, Messite J, Zaki MH: Mortality experiences among talc workers: A follow-up study. *JOM* 16:345-349, 1974.

4. Brown DP, Wagoner JK: Occupational exposure to talc containing asbestos: III. Retrospective cohort study of mortality. U.S. Dept. of HEW, (NIOSH) Pub. 80-115:31-33, 1980.

5. Selevan SG, Dement JM, Wagoner JK, et al: Mortality patterns among miners and millers of non-asbestiform talc: Preliminary report. *J Environ Pathol Toxicol* 2:273-284, 1979.

6. Monson RR: Analysis of relative survival and proportional mortality. *Comp Biol Res* 7:325-332, 1974.

7. Mason TJ, McKay FW: U.S. Cancer Mortality by County: 1950-1969. HEW (NIH) Pub. 74-615:685, 1974.

8. Health and Safety Report, Health and Safety Spot Inspection (Radiation), American Mine, Gouverneur Talc Co., Inc., Balmat, St. Lawrence County, N.Y., Feb 28, 1973. U.S. Department of Interior, Bureau of Mines Health and Safety Activity.

9. Health and Radiation Inspection Report, Gouverneur Talc Co., Mine No. 1 and Mill, Balmat, N.Y., March 24, 1976. U.S. Department of Interior, Mining Enforcement and Safety Administration.

10. Armenian HK, Lilienfeld AM: The distribution of incubation periods of neoplastic diseases. *Am J Epidemol* 99:92-100, 1974.

Shaping Moral Neuters

Common to the various "moral education" approaches offered today is the idea that there is nothing right or wrong and that to teach any substantive ethical percept or idea is to "indoctrinate" students. In one approach, students are invited to "clarify" their own values; the focus is on "rationality," "creativity," "autonomy" and "process." In another approach, students are asked to dwell on ethical dilemmas such as when lying can be justified, or on public policy questions such as the morality of racial quotas.

The problems with these approaches are obvious to anyone raised in the basic morality. In the values approach, no one need fear a teacher's judgment if his values are wrong, because there are no wrong values (other than, of course, the value of opposing "values" education). Upon learning that all choices are equally valid, a student will be educated into skepticism about morality, fertile soil for wrong action. . . .

Today's "moral education" fails in the most critical way because it addresses the intellect but not the emotions. No one is taught to dislike and certainly not to hate anything. If young Americans are shaped, they are unfortunately shaped to be moral neuters. The inculcation of the basic morality in the minds *and* hearts of young people, as given in American education since the 17th Century, has for the most part ceased to be in the latter part of the 20th.

— From "Teaching Morality in Public Schools" by Terry Eastland in *The Wall Street Journal*, Feb. 22, 1982.

WES001130