#18

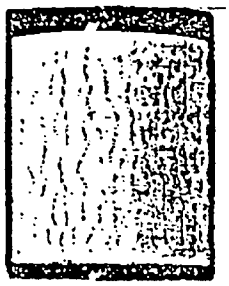
*Reprinted from*

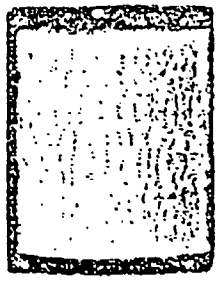
INHALED PARTICLES IV

Edited by

W. H. WALTON

PERGAMON PRESS OXFORD AND NEW YORK 1977





J&J-0065613

JNJ 000051763

# TALC—RECENT EPIDEMIOLOGICAL STUDIES

GAVIN HILDICK-SMITH

*Rutgers Medical School and Johnson & Johnson, New Brunswick, N.J., U.S.A.*

*Abstract*—Talc dusts are categorized into cosmetic grades consisting predominantly of talc mineral free of asbestos, and industrial grades containing varying amounts of mineral talc combined with other mineral dusts.

Concern has been expressed on the hazard to health of talc dusts encountered in the environments of talc-mine employees and of consumers. Data obtained from recent epidemiological studies of mine employees exposed to cosmetic-grade talc dusts are reviewed to assess the health hazard of talc mineral dust.

Study of the respiratory system of talc-mine employees exposed to cosmetic grade talc dusts shows that the pulmonary function and chest X-ray findings were similar to those observed in control subjects, but an increase in symptoms was noted in smoking talc miners.

Review of the cause of death of talc-mine millers exposed to 20 mppcf cosmetic talc dust for as long as 40 years showed no difference in the incidence of the causes of death from data obtained from a control cohort; miners exposed to 600 mppcf of talc mineral dust (containing 5% quartz) showed a higher incidence of respiratory deaths than the controls.

The incidence of deaths from pulmonary tumours and all tumours was numerically lower for the talc millers and statistically lower for talc miners than recorded for the control cohort, indicating that cosmetic-grade talc is not a carcinogen.

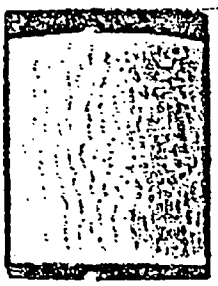
The short exposure to small amounts of cosmetic-grade talc in the normal use of talc-containing cosmetic products indicates that cosmetic-grade talc as used in such products is not a hazard to health.

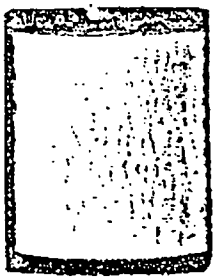
RECENTLY attention has been focused on the health hazard of industrial and cosmetic-grade talc dusts used by industry and in consumer products, on which there are limited epidemiological data by which to assess their precise effect on health.

Talc, which is hydrated magnesium silicate $Mg_3Si_4O_{10}(OH)_2$ (PAULING, 1930), was known to the ancient Egyptians, Assyrians and Chinese and has been used ever since. The current largest use of talc is in such industrial products as ceramics, paint and paper and industrial processes like the manufacture of rubber goods. The talc used in such products and industries is referred to as industrial talc (THOMPSON, 1974) as it is usually composed of talc combined with a variety of mineral dusts to provide the desired physical properties. Industrial talcs can have a low mineral talc content and may contain free silica and other minerals and can contain asbestos.

Due to the possible health hazard of industrial talc dusts, government regulations control the amount and type of dust to which workers are exposed. At this time the maximum talc dust exposure is defined as a Threshold Limit Value (TLV) of 20 mppcf







J&J-0065614

JNJ 000051764

nonfibrous particles and five fibres per ml in a working environment based on an 8-h day for a 5-day working week (A.C.G.I.H., 1973). The grade of talc that is used in cosmetic and toiletry products is called cosmetic talc which is mined from selected talc deposits and can be defined as containing at least 90% mineral talc that is free of detectable amounts of asbestos, but associated with limited amounts of other minerals.

Talc was mined and used industrially for many decades until THOREL (1896) published the first report of talc pneumoconiosis. Talc pneumoconiosis was subsequently reported in workers exposed to industrial-grade talcs, usually before industrial environments were improved (DEVILLIERS, 1961; DREESSEN, 1933; GREENBERG, 1947; MILLMAN, 1947; PORRO *et al.*, 1942). Unfortunately most published medical reports on talc dusts relate to the use of industrial-grade talc and provide no precise data on the composition or the amount of talc dust to which workers were exposed. The exact factors responsible for any ill effects reported cannot, therefore, be accurately assessed. In order to determine the effect of talc dusts on health, it is important to identify the amount and composition of talc dusts in the working environment.

Epidemiological studies have been initiated with cosmetic-grade talc dusts in an attempt to assess the effect of talc mineral dust on the respiratory system and the result of prolonged exposure to such dust.

A review of recent epidemiological studies is submitted to assist in evaluating the effect of exposure to cosmetic-grade talc dusts on mine employees and assess whether the normal use of cosmetic-talc products is a hazard to the health of the consumer.

## PULMONARY MORBIDITY STUDIES

KLEINFELD *et al.* (1965) reported that talc millers exposed for an average of 19 years to 62.3 mppcf of an industrial talc containing tremolite, anthophyllite and free silica had a significantly higher incidence of respiratory symptoms, abnormal pulmonary function tests and chest X-ray findings than recorded for a control group.

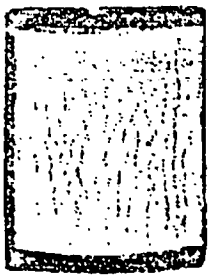
GREEN and SYLWESTER (unpublished) initiated a prospective study of talc-mine employees designed to monitor respiratory system function in relation to a cosmetic talc dust environment. Data obtained on seventy mine employees were compared with similar data obtained from a control group of subjects employed in non-dusty industries.

The British Medical Research Council questionnaire was used to obtain health data, and pulmonary function information was gathered by use of a Stead–Wells spirometer. AP and lateral chest X-rays were taken under controlled conditions and read by trained personnel.

The main employees had worked for an average duration of 4.6 years and were exposed to an average dust count of 7.6 mppcf. The cosmetic grade talc produced by the mine undergoes a purification procedure in the mill so that the final product consists of more than 90% platy talc, free of asbestos and free silica.

The seventy talc-mine employee population contained eight non-smoking subjects and sixty-two who smoked or had smoked. The eight non-smokers were symptom-free and showed no difference in pulmonary function tests or chest X-ray findings from the control subjects. The data on miners who smoked were compared with data from





J&J-0065615

JNJ 000051765

a control group matched for age and duration of employment drawn from non-dusty industries and consisted of 247 subjects who smoked.

Review of the pulmonary function data obtained on the smoking miners and smoking and non-smoking control subjects showed no statistically significant difference in the measurements of FVC or $FEV_1$ (when assessed as per cent predicted values using predictions derived from Vermont non-mineral industries, taking into account the person's age, sex, height, and smoking habits) or in the $FEV_1$/FVC ratios (Table 1a).

TABLE 1. COMPARISON OF PULMONARY MORBIDITY IN TALC MINERS (SMOKERS) AND CONTROLS (after GREEN and SYLVESTER, unpublished)

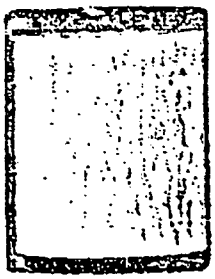
| | Talc miner smokers | Smoking controls | Non-smoking controls |
|---|---|---|---|
| Sample size | 62 | 247 | 122 |
| (a) *Lung Function Variables* | | | |
| No. cigarettes/day | 24.8 | 20.5 | 0 |
| $FEV_1$/FVC (%) | 81.4 | 81.0 | 82.5 |
| FVC (% of predicted) | 103.5 | 100.2 | 102.1 |
| $FEV_1$ (% of predicted) | 101.9 | 100.0 | 103.1 |
| Lung X-ray localized opacities (no. of cases) | 2 | 9 | 8 |
| Lung X-ray diffuse opacities (no. of cases) | 4 | 15 | 10 |
| (b) *Other Variables* | | | |
| Cough (no. of cases) | 14 (23%) | 49 (20%) | 5 (4%)† |
| Phlegm (no. of cases) | 14 (23%) | 23 (9%)† | 5 (4%)† |
| Wheeze (no. of cases) | 20 (32%) | 51 (21%)* | 15 (12%)† |
| Wheeze + shortness of breath (no. of cases) | 7 (11%) | 30 (12%) | 8 (7%) |
| No symptoms (no. of cases) | 7 (11%) | 94 (38%) | 89 (73%) |
| Cough for 3 months of year/subjects with cough | 9/14 = 64% | 41/49 = 84% | 2/5 = 40% |
| Phlegm for 3 months of year/subjects with phlegm | 10/14 = 71% | 19/23 = 83% | 3/5 = 60% |

* = $P < 0.10$. † = $P < 0.01$.

Review of the chest X-ray findings shows fewer localized and diffuse opacities were seen in the smoking talc workers than the smoking or non-smoking control subjects (Table 1a).

The data obtained on pulmonary symptoms show that smoking workers reported an incidence of cough and wheezing similar to that of smoking non-talc workers but higher than non-talc working non-smokers. A higher incidence of phlegm was reported for the smoking miners than smoking and non-smoking control. There was no difference in incidence in the three groups studied when assessed for cough or phlegm production over a period of 3 months for each year or wheeze combined with shortness of breath for a similar period (Table 1b).





## CAUSE OF DEATH OF TALC-MINE EMPLOYEES

The long-term follow-up of workers exposed to mineral dusts has revealed that the critical environmental factors which determine the nature and rate of onset of disease, are dust count, length of exposure and specific type of dusts. ENTERLINE *et al.* (1973) showed that there was a critical level (125 mppcf-years) of exposure to chrysotile asbestos dust and duration of employment of chrysotile mine workers below which pulmonary cancer did not occur.

Epidemiological studies on the cause of death of talc-mine employees can be valuable in assessing the rate of onset and type of disease produced by specific talc dusts encountered in an industrial environment. Epidemiological studies of the cause of death of talc miners were initially conducted on workers exposed to industrial grade talc containing asbestos minerals and later on those exposed to cosmetic-grade talc dust free of asbestos.

KLEINFELD *et al.*, (1967) published data on the cause of death of miners exposed for more than 15 years to industrial talc dust containing tremolite and anthophyllite and reported an incidence of malignancy that was four times greater in the older miners (60–79 years) than the general public in contrast to miners in the age group 45 to 59 years.

In a subsequent follow-up publication KLEINFELD *et al.*, (1974) reported on the cause of death of workers from the same mine and showed that the incidence of deaths due to malignancy in the miners was not statistically different from the expected incidence in the general population.

Kleinfeld attributed the drop in incidence of malignancy to the reduction in dust counts, a median dust range of 413 to 30 mppcf before 1945, to 63 to 3 mppcf in 1945 to 1965.

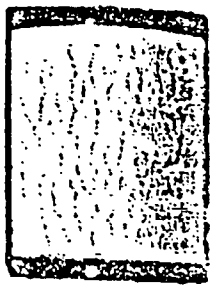
RUBINO *et al.* (unpublished) conducted a study on the cause of death of Italian talc-mine employees to establish whether the mining of cosmetic-grade talc free of asbestos was associated with causes of death different from those observed in a control population. The study consisted of reviewing the cause of death obtained from death certificates of employees who began mine work between 1921 and 1950, and who had worked for a minimum of 1 year. The control data were obtained from the records of subjects who lived in a non-industrial rural community 38 miles from the mine by randomly selecting data on male subjects who matched the birth date of individuals in the mine employee cohort and recording the cause of death of those who had died after 1921.

The data on a total population of 1784 mine employees consisting of 1346 miners (88.9% of the total miner census) and 438 millers (91.6% of total miller census) were compared with the data obtained from the control cohort (Table 2A).

Table 2B presents an analysis of the years of exposure of 1325 miners (mean 14.9 years) and 428 millers (mean 15.8 years). Four hundred and fifty-seven (34%) miners and 170 (39%) of the millers had 20 years' exposure and 206 (15%) miners and 57 (13%) millers had more than 30 years' exposure to talc dust.

The air dust content was determined by reviewing records obtained from 1948 until today, with the air dust concentration being constant from 1921 to 1949.

Records of dust counts show that miners were exposed to 600 mppcf of talc dust





J&J-0065617

JNJ 000051767

TABLE 2. DEATH STATISTICS OF TALC-MINE EMPLOYEES COMPARED WITH CONTROLS (after RUDINO *et al.*, unpublished)

A. *Vital status of 1346 miners and 438 millers with completed follow up compared with respective controls*

| | | Dead | Alive | Death certificates obtained |
|---|---|---|---|---|
| Miners | Number | 704 | 642 | 667 |
| | Per cent | 52.3 | 47.7 | 94.7 |
| Controls | Number | 851 | 495 | 813 |
| | Per cent | 63.2 | 36.8 | 95.5 |
| Millers | Number | 227 | 211 | 218 |
| | Per cent | 51.8 | 48.2 | 96 |
| Controls | Number | 275 | 163 | 254 |
| | Per cent | 62.8 | 37.2 | 92.4 |

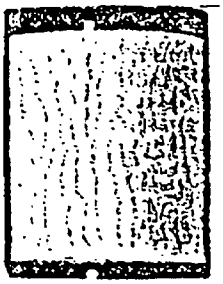
B. *Length of exposure to talc of 1325 miners and 428 millers*

| Years of exposure | Miners | | Millers | |
|---|---|---|---|---|
| | Number | Per cent | Number | Per cent |
| 1–9 | 652 | 49.2 | 190 | 44.4 |
| 10–19 | 216 | 16.3 | 68 | 15.9 |
| 20–29 | 251 | 18.9 | 113 | 26.4 |
| 30–39 | 200 | 15.1 | 50 | 11.7 |
| 40–49 | 6 | 0.5 | 7 | 1.6 |
| Total | 1325 | 100 | 428 | 100 |

C. *Miners—observed* vs. *expected deaths and ratios*

| | Exposed *vs.* controls comparison | | | |
|---|---|---|---|---|
| | Total exposed | | Controls | |
| Latency | Obs./Exp. | Ratio | Obs./Exp. | Ratio |
| 0–1 | 68/84.2 | 0.81† | 100/83.8 | 1.19† |
| 11–20 | 110/119.6 | 0.92 | 127/117.2 | 1.08 |
| 21–30 | 203/208.8 | 0.97 | 206/200.2 | 1.03 |
| 31–40 | 188/207 | 0.91 | 220/200.9 | 1.10 |
| 41–50 | 126/157.5 | 0.80† | 184/152.4 | 1.21† |
| 50+ | 9/14.1 | 0.64* | 14/8.9 | 1.57* |
| Total | 704/791.2 | 0.89† | 851/763.3 | 1.11† |

* Statistically significant at 5% level.
† Statistically significant at 1% level.



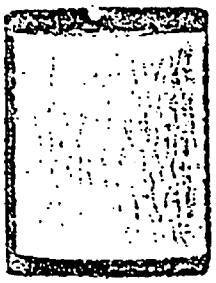


TABLE 2—*continued.*

D. *Millers—observed* vs. *expected deaths and ratios*

| | Exposed vs. controls comparison | | | |
|---|---|---|---|---|
| | Total exposed | | Controls | |
| Latency | Obs./Exp. | Ratio | Obs./Exp. | Ratio |
| 0–10 | 16/29.6 | 0.54† | 43/29.4 | 1.46† |
| 11–20 | 46/41.7 | 1.10 | 36/40.2 | 0.90 |
| 21–30 | 66/67.9 | 0.97 | 69/67.1 | 1.03 |
| 31–40 | 57/72.5 | 0.79† | 87/71.5 | 1.22† |
| 41–50 | 38/42.2 | 0.90 | 37/32.8 | 1.13 |
| 50+ | 4/4.5 | 0.89 | 3/2.5 | 1.20 |
| Total | 227/258.4 | 0.88† | 275/243.5 | 1.13† |

† Statistically significant at 1% level

E. *Miners—observed/expected deaths and ratio by cause*

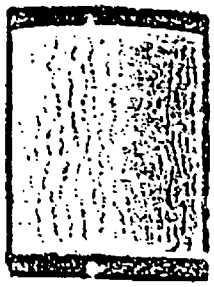
| | Total exposed | | Controls | |
|---|---|---|---|---|
| Cause of death | Obs./Exp. | Ratio | Obs./Exp. | Ratio |
| Infectious diseases | 56/66 | 0.85 | 75/64.5 | 1.16 |
| All tumours | 100/129.5 | 0.77† | 155/125.1 | 1.24† |
| Lung cancer | 9/20.2 | 0.45† | 31/19.5 | 1.59† |
| Gastrointestinal cancer | 46/49.9 | 0.92 | 54/49 | 1.10 |
| Nervous system diseases | 9/9.9 | 0.91 | 10/9.8 | 1.02 |
| Cardiovascular diseases | 208/276.1 | 0.75† | 334/265.5 | 1.26† |
| Respiratory diseases | 142/102 | 1.39† | 58/97.5 | 0.59† |
| Gastrointestinal diseases | 49/59.1 | 0.83* | 68/57.2 | 1.19* |
| Senility | 12/11 | 1.09 | 10/10.8 | 0.93 |
| Accidents | 68/64.7 | 1.05 | 60/62.6 | 0.96 |
| All other causes | 23/33.5 | 0.69† | 43/32 | 1.34† |

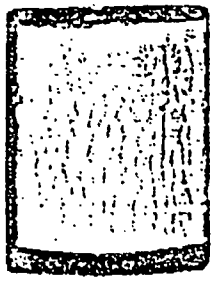
* Statistically significant at 5% level.
† Statistically significant at 1% level.

F. *Millers—observed/expected deaths and ratio by cause*

| | Total exposed | | Controls | |
|---|---|---|---|---|
| Cause of death | Obs./Exp. | Ratio | Obs./Exp. | Ratio |
| Infectious diseases | 14/17.6 | 0.80 | 21/17.2 | 1.22 |
| All tumours | 42/46.2 | 0.91 | 48/43.5 | 1.10 |
| Lung cancer | 4/7.6 | 0.53 | 11/7.3 | 1.51 |
| Gastrointestinal cancer | 16/19 | 0.84 | 21/17.9 | 1.17 |
| Nervous system diseases | 4/2.5 | 1.60 | 1/2.5 | 0.40 |
| Cardiovascular diseases | 72/92 | 0.78† | 106/85.9 | 1.23† |
| Respiratory diseases | 25/21.5 | 1.16 | 17/20.3 | 0.84 |
| Gastrointestinal diseases | 20/21.8 | 0.92 | 22/20.1 | 1.09 |
| Senility | 4/4.6 | 0.87 | 5/4.4 | 1.14 |
| Accidents | 28/26.7 | 1.05 | 24/25.2 | 0.95 |
| All other causes | 9/9.8 | 0.92 | 10/9.2 | 1.09 |

* Statistically significant at 5% level.
† Statistically significant at 1% level.





J&J-0065619

JNJ 000051769

from 1920 to 1950, at which time the dust level was progressively reduced to a level of 2 mppcf by 1965, where it has remained. The millers were exposed to a dust level of 30 mppcf in 1920 when this level was gradually lowered to 10 mppcf by 1975.

The cosmetic-grade talc to which the miners were exposed consists of platy talc free of asbestos minerals but containing 5% of silica dust which arises from the footwall rock in the mine. The talc dust to which the millers were exposed was not less than 90% pure talc, free of asbestos but containing 0.5% by weight of free silica and 3–4% by weight of chlorite and 1–2% by weight of carbonate (WAGNER *et al.*, this Symposium; pp. 647–652).

A review of the overall data on total deaths in the control groups and those of the miners and millers shows that for the miners there were 704 deaths as opposed to 851 deaths in the control group, with 227 deaths in the miller group as opposed to 275 deaths in the control group. The data for the miners (Table 2C) show no relationship exists between the ratio of observed to expected deaths among miners except in those with 41–50 years' exposure in which there is a significantly lower number of deaths than the expected values.

The total number of deaths observed in the miners is significantly less than those for the control cohort. Review of the observed and expected deaths in the miller cohort shows a significantly lower number of deaths in the millers of the age group with 31–40 years of exposure and a significantly lower overall incidence of death for the millers (Table 2D).

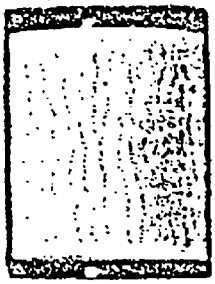
Review of death in miners in the major categories analysed (Table 2E) shows a significantly higher incidence of deaths from respiratory disease. In contrast, in the case of deaths due to all tumours as well as lung cancer and cardiovascular disease, the observed deaths are statistically lower than those noted in the control group.

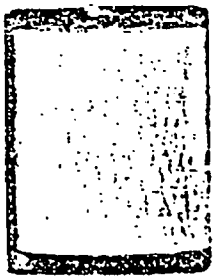
Review of the cause of death in the miller cohort shows no difference between observed and expected deaths except in cardiovascular deaths which showed an observed number of deaths below expected values (Table 2F).

The data obtained from this study show the miners exposed to 600 mppcf cosmetic talc mineral dusts containing 5% silica have a higher incidence of deaths from respiratory disease than the control group or the millers exposed to 20 mppcf cosmetic talc dust containing less than 0.5% free silica. In addition, the lower incidence of overall tumours and lung cancer reported in the mine employees in contrast to the control group indicates that dust encountered in the mine or cosmetic-grade talc dust encountered in the mill is not a carcinogen in the population studied.

## DISCUSSION

In recent years interest has been focused on the potential hazard of talc dusts present in industrial and consumer environments. As industrial and consumer talcs can be significantly different in their mineral composition, a review of recent epidemiological studies has been undertaken to identify the potential hazard to health of mine workers and consumers exposed to cosmetic-grade talc dusts. The effect of industrial grade talcs on the health of those exposed to them will be determined by appropriate epidemiological studies.





J&J-0065620

JNJ 000051770

Initial data obtained from an ongoing epidemiological study of pulmonary function in talc-mine workers exposed to cosmetic-grade talc have been submitted.

The data show that non-smoking talc miners showed no difference in incidence of pulmonary symptoms, chest X-ray and pulmonary function findings from the control population studied. The workers who smoked also showed no difference in pulmonary function tests and chest X-ray findings from the control subjects. The smoking talc workers had a similar incidence of cough and wheeze but a higher incidence of phlegm as compared to the smoking and non-smoking control subjects.

These data indicate that workers exposed to the cosmetic-grade talc dust environment described did not show evidence of pulmonary disease. The significance of increased incidence of phlegm in miners who smoke, in the absence of abnormal pulmonary function and chest X-ray findings, has yet to be determined.

It is of interest to note that talc dust is not an allergen and that available data show that talc dust is readily ingested by pulmonary macrophages, which does not alter the viability of the cells or their phagocytosis of colloidal particles (DI LUZIO, unpublished).

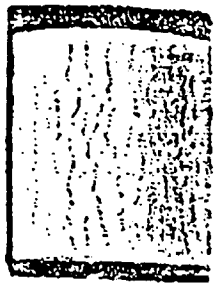
In college-age subjects, smoking has been shown to induce a significant increase in incidence of cough, phlegm and wheezing as opposed to the incidence noted in a non-smoking control group (PETERS and FERRIS, 1967). In addition, in a survey of respiratory symptoms reported among employees working in a chrysotile asbestos mine (MCDONALD *et al.*, 1972), it was shown that smoking was a determinant in producing bronchitic symptoms. ROSSITER and WEILL (1974) have indicated that smoking in a dust environment produces an adverse synergistic effect which is demonstrated by pulmonary lung function tests. These data suggest that workers exposed to a dust environment who smoke may run a higher risk of developing pulmonary disease than non-smokers. Prospective study of workers who smoke in dusty environments will, however, determine whether or not pulmonary disease develops.

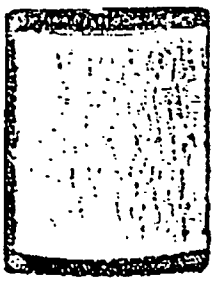
As long-term exposure to dusts may produce pulmonary fibrosis or pulmonary cancer, the evaluation of the cause of death of workers who have had prolonged exposure to cosmetic-grade talc dust is important. RUBINO *et al.* (unpublished) showed that miners exposed to 600 mppcf talc dust containing *circa* 5% of free silica arising from the rock wall in the mine had a higher incidence of death from respiratory disease as compared to the controls. Millers exposed to cosmetic grade talc dusts at an average level of 20 mppcf for periods up to 40 years showed an incidence of respiratory disease similar to that for the control cohort, which suggests that this level of cosmetic talc dust exposure does not induce a degree of pulmonary disease that is responsible for death.

Kleinfeld reported that miners exposed to levels as high as 413 mppcf of industrial-grade talc containing tremolite had a higher incidence of pulmonary cancer in the older age group than control subjects and later reported that the difference was not noted when the dust level was lowered.

MERLISS (1971), BLEJER and ARLON (1973), HENDERSON *et al.* (1971) and PELFRENE and SHUBIK (1975) suggested that talc containing chrysotile or talc alone might be carcinogenic.

The study conducted by RUBINO *et al.* (unpublished) showed that total tumours and pulmonary tumours reported were numerically lower for millers and statistically





lower for miners compared with the incidence in the control cohort. These data would indicate that talc is not a carcinogen. This observation is supported by a study conducted in hamsters by WEHNER (unpublished) and those reported by SMITH (1965, 1973), POTT and FRIEDRICHS (1972) and GROSS *et al.* (1970) where animals which had talc given intrathoracically or inhaled it for prolonged periods and followed to natural death did not develop cancer.

Review of exposure time and dust concentration encountered by the consumer, in contrast to that of the talc-mine employees exposed to cosmetic talc dust, gives an indication of the difference in exposure to talc dust. A consumer exposed to cosmetic talc dust for one minute a day would be exposed to talc dust for one 480th the daily 8-h exposure time of a talc miner or miller.

The normal usage of a commercially available cosmetic talc dusting product exposes the consumer to a total weekly exposure of talc of 0.102 mppcf-hours (SIVERTSON, unpublished). The talc millers in the study reported by RUBINO *et al.* (unpublished) were exposed to an average dust count of 20 mppcf (the TLV for talc) which provides a weekly time weighted average of 800 mppcf-hours per week of talc dust which is 7800 times greater than the weekly time weighted dust count exposure to which a consumer is exposed.

The data submitted indicate that the normal use of cosmetic products containing cosmetic-grade talc is not a hazard to health.

## REFERENCES


AMERICAN CONFERENCE OF GOVERNMENTAL INDUSTRIAL HYGIENISTS (1973). *Threshold Limit Values for Chemical Substances and Physical Agents in Workroom Environments.* A.C.G.I.H., Cincinnati, OH.

BLEJER, H. P. and ARLON, R. (1973) *J. occup. Med.* **15**, 92–97.

DEVILLIERS, A. J. (1961) *Occup. Hlth Rev.* **13**, 3–14.

DI LUZIO, N. (unpublished) Personal communication, 1975.

DREESSEN, W. C. (1933) *J. ind. Hyg. Toxicol.* **15**, 66–78.

ENTERLINE, P., DE COUFLE, P. and HENDERSON, V. (1973) *Br. J. ind. Med.* **30**, 162–166.

GREEN, G. and SYLVESTER, D. (unpublished) Personal communication, 1975.

GREENBERG, L. (1947) *Yale J. biol. Med.* **19**, 431–501.

GROSS, P., DE TREVILLE, R. T. P. and CRALLEY, L. J. (1970) *Pneumoconiosis, Proceedings International Conference, Johannesburg, 1969* (edited by SHAPIRO, H. A.) pp. 220–224. Oxford Univ. Press, Cape Town.

HENDERSON, W. J., JOSLIN, C. A. F., TURNBULL, A. C. and GRIFFITHS, K. (1971) *J. Obstet. Gynaec. Br. Commonw.* **78**, 266–272.

KLEINFELD, M., MESSITE, J., SHAPIRO, J. and SWENCICKI, R. (1965) *Archs envir. Hlth* **10**, 431–43.

KLEINFELD, M., MESSITE, J., KOOYMAN, O. and ZAKI, M. (1967) *Archs envir. Hlth* **14**, 663–667.

KLEINFELD, M., MESSITE, J. and ZAKI, M. H. (1974) *J. occup. Med.* **16**, 345–349.

MCDONALD, J. C., BECKLAKE, M. R., FOURNIER-MASSEY, G. and ROSSITER, C. E. (1972) *Archs envir. Hlth* **24**, 358–363.

MERLISS, R. R. (1971) *Science, N.Y.* **173**, 1141–1142.

MILLMAN, N. (1947) *Occup. Med.* **4**, 391–394.

PAULING, L. (1930) *Proc. Nat. Acad. Sci. U.S.A.* **16**, 123–129.

PELFRENE, A. and SHUBIK, P. (1975). *Nouv. Presse méd.* **4**, 801–803.

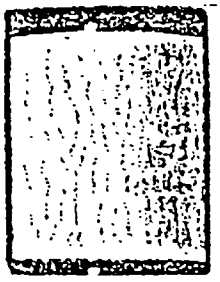
PETERS, J. M. and FERRIS, B. G. (1967) *Am. Rev. resp. Dis.* **95**, 774–782.

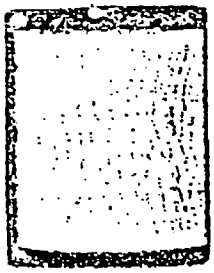
PORRO, F. W., PATTON, J. R. and HOBBS, A. A. JR. (1942) *Am. J. Roentg.* **47**, 507–524.

POTT, F. and FRIEDRICHS, K. H. (1972) *Naturwissenschaften* **59**, 318.

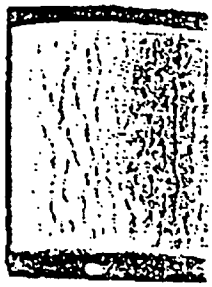
ROSSITER, C. E. and WEILL, H. (1974) *Bull. Physiopath. resp.* **10**, 717–725.

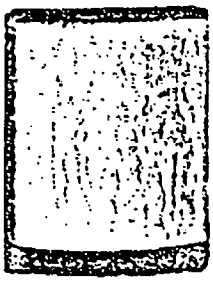
RUBINO, G. F., SCANSETTI, G., PIOLATTO, G. and ROMANO, C. (unpublished) Meeting of American Occupational Medical Association, San Francisco, April 1975.



SIVERTSON, J. N. (unpublished) Personal Communication, 1974.
SMITH, W. E., MILLER, L., ELSASSER, R. E. and HUBERT, D. D. (1965) *Ann. N.Y. Acad. Sci.* 132, 456–488.
SMITH, W. E. (1973). *Am. ind. Hyg. Ass. J.* 34, 227–228.
THOMPSON, C. S. (1974) International Circular, IC-863, pp. 22–44. U.S. Bureau of Mines, Washington.
THOREL, C. (1896) *Beitr. path. Anat.* 20, 85–101.
WEHNER, A. P. (unpublished) XVIII International Congress on Occupational Health, Brighton, England, September, 1975.





## DISCUSSION

D. MALCOLM: Was the method of estimating talc dust the same from 1921 to 1975?

Dr HILDICK-SMITH: No.

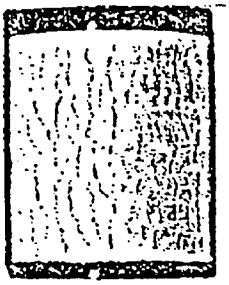
D. MALCOLM: In 1948, when talc was used as a mould lubricant for casting battery grids, some fifty casters were X-rayed, and some showed early nodulation. Six have since died of pneumoconiosis. The type of talc and concentration were not known. Some men may have been ex-miners.

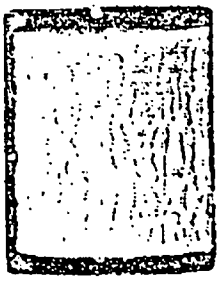
G. W. WRIGHT: Did you find any X-ray abnormalities in the men about whom you are reporting?

Dr HILDICK-SMITH: No.

J. C. MCDONALD: I am unclear and concerned about the choice of controls in the mortality study and their comparability with the study population. It is stated only that they were a random sample of persons of the same birth date from a neighbouring community. Miners are a self-selected group who survive long enough to be employed in heavy work. Were the controls similarly selected from survivors to the same point in time?

Dr HILDICK-SMITH: The controls were selected from agricultural workers living 40 miles from the mine. Each control had a birth date of a mine employee and had to be alive in 1921.





J&J-0065624

JNJ 000051774