

**HEALTH STUDY
OF
ITALIAN TALCS**

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PREFACE

For several years the scientific community has been gathering data and preparing numerous studies in an effort to bring facts to the public in lieu of incomplete and, in far too many cases, inaccurate allegations of supposed hazards to which the public may be exposed.

The report which follows was undertaken by the Department of Occupational Medicine of the University of Turin under the direction of Dr. G.F. Rubino, an internationally recognized epidemiologist. It was presented to the American Industrial Health Conference in San Francisco on April 15, 1975.

The study was conducted on 1794 miners and millers who worked in the talc mines and mills of Pinerola in Northern Italy. (These mines are the source of the Italian Talc which is used extensively throughout the world and has become the world standard of quality). The study is unique in that the Pinerola region is the only talc producing area in the world where its miners and millers have remained stationary and, for the most part, been involved in talc operations only.

This tradition provides a valuable opportunity to study a large group of people exposed over many decades to levels of talc far in excess of that to which any consumer could conceivably be exposed. This experimental design is thoroughly comprehensive and statistically conclusive.

It should be noted that the Italian Talc is of cosmetic quality due to its purity. The report reassures the cosmetic manufacturer that Italian talc which has been tested for purity and offered by us as cosmetic talc is entirely safe for the end intended.

WHITTAKER, CLARK & DANIELS, INC.

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**MORTALITY STUDY
OF
TALC MINERS AND MILLERS**

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Lo Giudice/MC/D/0325

INTRODUCTION

The purpose of this research is to ascertain the causes of death in talc miners and millers.

Our study was conducted on a talc mineralogically very pure. All the subjects of our study have worked only in the mines or mills of the talc operations in the Pinerolo Valley.

This particular talc is of the highest standard of purity.

When analyzed by X-Ray Diffraction and by Electron Microscopy it has never been found to contain any detectable amount of non talc minerals, such as tremolite or chrysotile.

Kleinfeld et al. made a similar study in 1967 in N.Y. State on a group of 260 talc miners and millers exposed to an industrial talc. As pointed out by these authors this talc contained other silicates such as serpentine, tremolite, anthophyllite and other ingredients predominantly carbonates. Free silica was also present in the dust, usually averaging not more than 3 percent. Therefore our present epidemiological study provides a basis for comparing subjects exposed to an impure industrial type of talc to subjects exposed to a very pure cosmetic type of talc.

TYPE OF STUDY AND DATA COLLECTION

We have employed a prospective historic study consisting in the follow-up on all male subjects who began to work in the years between 1921 and 1950 and who have been exposed for at least one year to talc in the mines or in the mills.

We have gathered a total of 1514 miners and 478 millers qualified by our definition of the sample.

In choosing the control sample we thought it advisable to use the method of matching rather than a comparison with national death rates which would potentially involve the influence of nuisance variables (feeding habits, ethnic characteristic differences, etc...).

Control subjects were taken from the population of the town of Alba, because it presents the same ethnic characteristics and similar social and economic conditions.

Alba is a center, about 38 miles from the Pinerolo Valley with a dominant traditional agricultural activity and very little industrial development involving any obvious hazard of occupational disease or carcinogenesis to its workers.

For every subject of the exposed group a control subject was taken from the City Hall records with the same sex and birth date and still alive at the time the exposed subject was beginning his employment. The first control subject meeting these requirements was selected.

With the information we had gathered a follow-up procedure was instituted for the exposed subjects who had left employment and whose vital status could not be determined from the company records, as well as for the control subjects.

The City Hall registries of the birth place or death place provided information on vital status and possibly on cause of death.



When the cause of death was not found in the City Hall records or if there was some doubt about its reliability, a further inquiry was started with relatives of the deceased, attending physicians (whenever they could be found) or examining the hospital records for hospital deaths.

Follow-up is 88.9% complete on miners and 91.6% complete on millers. Slide 1 show the follow-up and slide 2 the vital status of the exposed population and their controls, as of June the 30th, 1974.

The subsequent statistical analysis was conducted on 1346 miners and 438 millers on whom follow-up was complete.

As we were already in possession of air dust concentration in the mines and in the mills (this is shown in slide 3) from 1948 till today, we were able to determine for each worker the amount of exposure to which he had been subject.

As no change modifying the environment occurred to 1949 the level of exposure was considered to be uniform from 1921 to 1949.

As you can see from the slide, the level of exposure is quite different between miners and millers.

Slide 4 shows the distribution of all workers, on whom follow-up was complete, according to the Exposure Level.

The Exposure Level was figured out by the addition of the number of particles per cubic centimeter for every year at work.

RESULTS

Slide 5, Table 4 shows that the observed overall mortality of talc miners is lower than expected. There are 704 observed deaths compared with 791.2 expected deaths.

No relationship exists between the Ratio of observed to expected deaths and level of exposure for the overall mortality, all malignant neoplasms, lung cancer and gastro-intestinal cancer. On the contrary respiratory diseases show an increase in the Ratio with increasing exposure. This is also graphically represented in slide 6.

Slide 7 shows a similar pattern on overall mortality for millers (227 observed deaths compared to 258.4 expected) and in contrast with miners also exposure factor does not show any influence in increasing mortality from respiratory diseases.

In miners, as shown in slide 8 the Ratio of observed to expected deaths from respiratory diseases increases with increasing interval between first exposure and death (latency).

All malignant neoplasms decrease in miners with increasing latency following the trend of the overall mortality. The trend of the mortality from all causes, considering the latency time, is reported respectively for miners and millers in slide 9. For millers, as shown in slide 10, no relationship exists between Ratio and latency for any cause of death.



Lo Giudice/MCD/0327

DISCUSSION

The above tables show that the mortality (overall mortality) is higher in the control group than in the miners and the millers. This could be due:

- a. to a *biased selection* of the exposed group.
- b. to a *difference in recording* mortality in the exposed and in the control group.
- c. to the well known effect whereby occupational groups experience a lower mortality than controls taken from general population because of a *selection upon starting work*.
- d. to a *beneficial effect of talc* exposure.
- e. to a *lack of comparability* of the two groups.

Cause (a), biased selection, can be excluded because all the workers who have been enrolled between 1921 and 1950 and who have worked at least one year (therefore, including a substantial number with a very long period of observation) were included in the study.

Cause (b), difference in recording mortality, seems very unlikely. Methods of data collection on mortality were the same for the two groups (exposed and controls respectively) and in both groups cases were lost to follow up for reasons not related to mortality (accidental destruction of record files at the local City Hall registries, carelessness of clerical workers, etc...).

Cause (c), selection upon starting work, is less easy to discard, although a definite indication that it cannot provide the entire explanation for the higher mortality in the control group comes from the uniformity of the mortality Ratio during the observation period.

Assuming that the lower mortality of the exposed group is due to selection of more healthy individuals, the effect would have vanished after one or two decades: on the contrary it remains practically unchanged for the whole period of observation (see latency tables).

Cause (d), a beneficial effect of talc, can be discarded on general grounds and because the lower mortality is present in both miners and millers and uniformly for most causes of death, except for respiratory diseases in miners. But it may be taken into consideration in evaluating the extremely low mortality rate from lung cancer in both miners and millers. This hypothesis can be supported by some studies in accordance with our findings: Kennaway and Kennaway and more recently Goldman, Ashley, Enterline and Liddell have found a strikingly low rate of lung cancer in coal miners. Similar results were obtained by Gooding who found an incidence of 0.5% of lung cancer in the autopsied cases among 592 coal miners in South Wales.

By contrast Doll, in reviewing all the autopsies of male subjects in general hospitals in England has found an autopsy lung cancer incidence of 6.2%.

The hypothesis of a protection against lung cancer induced by organic or inorganic dust was first suggested in 1952 by Kennaway and recently supported by Speizer who states that exposure to some mineral or organic dust induces some varying degree of hypersensitivity reactions which develop an immunological protective status and enable the host to recognize and to destroy the cells which altered immunological patterns (such as carcinogen ones).

Moreover, it is well known that inert particles (such as coal, bentonite, aluminum salts) act as adjuvants when introduced in the organism, by increasing the antibody level and macrophage response. Some studies have also shown that in absence of radioactive or specific cancerogenic materials (haematite or asbestos) in the ground, no risk of lung cancer excess is present for miners.



Cause (e), lack of comparability, could be the main contributor to the observed differences. As already mentioned male controls were matched by age to exposed individuals and drawn from a population similar in economic and social structure, general habits and degree of urbanization. However, it could be possible that the matched population exhibits (for not easily understandable reasons) a constantly higher death rate in respect to the whole population of the Piedmont region. But this is not easy to verify because of the lack of regional statistical data (so far back in the past) fit for this comparison. It may be concluded that the comparison between the controls and the exposed has to be considered with caution.

The lower mortality among the exposed workers cannot lead to a final conclusion and be accepted without reserve even if no doubt would exist on the adequacy of the comparison with the control group.

However, further insight may be gained by the internal comparison of groups of workers exposed to difference total dose of particles and by splitting total mortality by cause.

For millers no clear dose effect relationship shows up either for total mortality, considered over time of latency, or for causes of death.

For miners the only clear pattern emerges for respiratory causes of death: here, in spite of the lower overall mortality in the exposed group, an excess of mortality is present both in the exposed versus controls comparison and as an increasing mortality with increasing dose and latency period.

Considering the trend of mortality from respiratory diseases in relation with dose and latency, respectively in miners and in millers, it is possible to assume that the inducing factor is the silica rather than the talc.

The deficit of tumours in the exposed in respect of controls is supported by the absence of any dose-effect pattern and is more easily interpreted as just one aspect of the lower mortality of the exposed group, although a low rate of lung cancer as reported above, may have an alternative explanation.

In conclusion exposure to silica in talc mines increases the risk of death from respiratory causes.

No increase of risk is detectable, under the slightly less than optimal conditions of the study, for other causes of death in miners, nor for any causes of death in talc exposed millers.

While Kleinfeld's findings provide evidence of cancerogenicity risk when talc mining or milling is a source of asbestos exposure caused by fibrous contamination, our conclusions support the thesis of no cancerogenic effect attributable to pure talc.

Table 1

SLIDE 1

Follow-up Status of talc Miners and Millers (1921-1974)			
MINERS	Total	Found	Unknown
Number	1514	1346	168
Percent	100	88.9	11.1
MILLERS			
Number	478	438	40
Percent	100	91.6	8.4

Table 2

SLIDE 2

VITAL STATUS OF 1346 MINERS AND 438 MILLERS WITH COMPLETED FOLLOW UP AND OF RESPECTIVE CONTROLS (ON JUNE 1974).			
	DEAD	ALIVE	DEATH CERTIFICATES OBT.
Miners - Number	704	642	667
Percent	52.3	47.7	94.7
CONTROLS - Number	851	495	813
Percent	63.2	36.8	95.5
MILLERS - Number	227	211	218
Percent	51.8	48.2	96
CONTROLS - Number	275	163	254
Percent	62.8	37.2	92.4

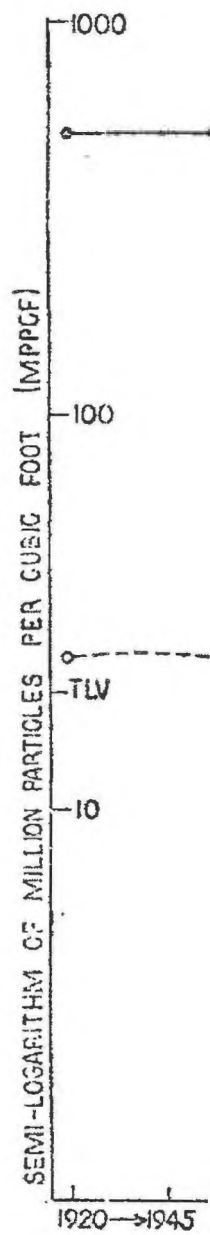


FIG.1
DUST CONCENTRATION IN AIR

SLIDE 3

ALONG OBSERVATION PERIOD.

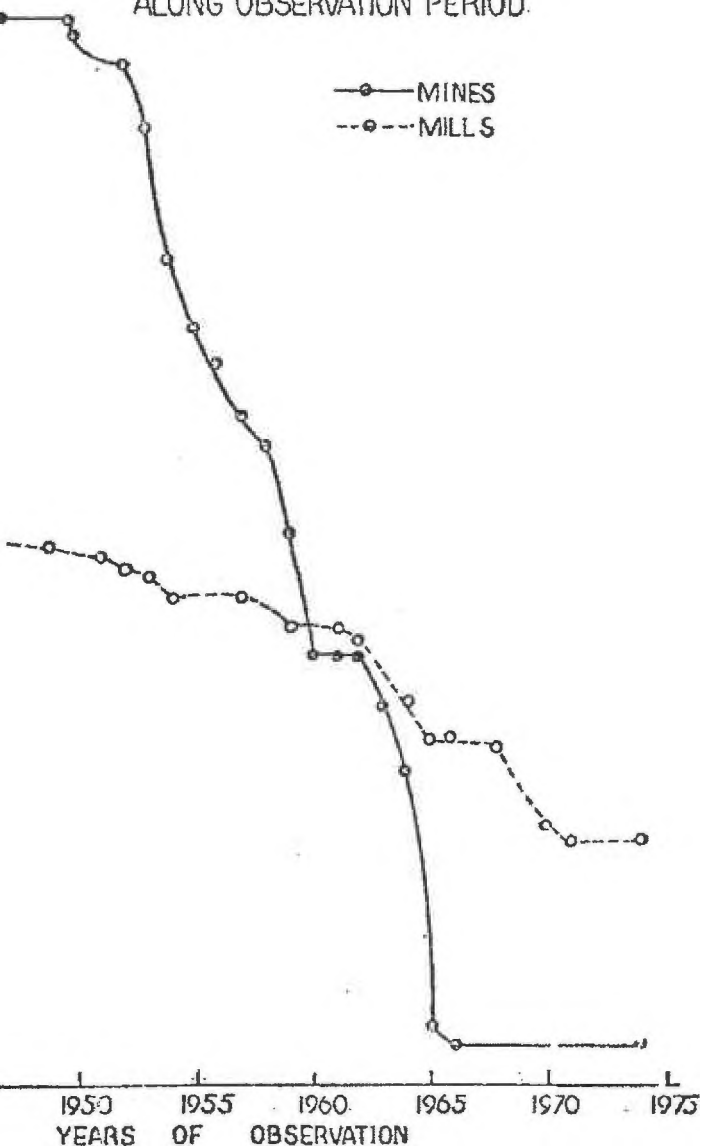


Table 3

SLIDE 4

DISTRIBUTION OF TOTAL NUMBER OF INHALED PARTICLES IN MINERS AND MILLERS WITH COMPLETED FOLLOW-UP				
	EXP.LEVEL I	EXP.LEVEL II	EXP.LEVEL III	
MINERS Σ pp/ml/yr	< 60000	60000 - 200000	> 200000	TOTAL
number	405	423	518	1346
percent	30.1	31.4	38.5	100
MILLERS Σ pp/ml/yr	< 5000	5000-15000	> 15000	TOTAL
number	163	144	131	438
percent	37.2	32.9	29.9	100

Table 4

SLIDE 5

MINERS: OVERALL MORTALITY

Observed deaths and Expected Deaths by Interval between first Exposure and Death (Latency).

LATENCY	Exposed v. Controls Comparison					Internal Comparison					
	Total Exposed		Controls			Exp Level I		Exp Level II		Exp Level III	
	Obs/Exp	Ratio	Obs/Exp	Ratio		Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio
0-1	68/84.2	0.81**	100/89.8	1.19**		38/24.5	1.47**	32/23.4	1.37**	0/18.75	0 **
11-20	110/119.6	0.92	127/117.8	1.08		40/33.7	1.19	42/35.4	1.19	28/30.7	0.71*
21-30	203/208.8	0.97	206/200.2	1.03		62/59.5	1.04	60/55.1	1.09	81/87.2	0.93
31-40	188/207	0.91	220/200.9	1.10		50/53.7	0.93	41/41.4	0.99	87/89.1	1.04
41-50	125/157.5	0.80**	184/152.4	1.21**		41/40.6	1.01	32/28.8	1.07	53/55.2	0.96
50 +	8/14.1	0.64*	14/8.9	1.57*		2/2.6	0.77	2/2.04	0.98	5/4.4	1.14
Total	704/791.2	0.89**	851/763.3	1.11**		231/214.7	1.08	209/187.2	1.12*	284/288.4	0.98*

* Statistically significant at 5% level

** Statistically significant at 1% level

Table 5

SLIDE 6

MILLERS: OVERALL MORTALITY

Observed deaths and Expected Deaths by Interval between first Exposure and Death

LATENCY	Exposed v. Controls Comparison					Internal Comparison					
	Total Exposed		Controls			Exp Level I		Exp Level II		Exp Level III	
	Obs/Exp	Ratio	Obs/Exp	Ratio		Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio
0-10	16/29.6	0.54**	43/29.4	1.46**		14/7.8	1.84**	2/5.2	0.38	0/2.9	0
11-20	46/41.7	1.10	38/40.2	0.90		20/18.7	1.20	23/18.8	1.24	3/11.0	0.27**
21-30	68/67.8	0.97	89/87.1	1.03		32/27.8	1.17	20/14.5	1.38	14/23.7	0.62*
31-40	57/72.5	0.79**	87/71.5	1.22**		23/21.2	1.08	15/10.3	1.46	19/24.4	0.78
41-50	38/42.2	0.90	37/32.8	1.13		10/12.9	0.78	4/3.7	1.08	24/21.8	1.11
50 +	4/4.8	0.83	3/2.5	1.20		3/2.8	1.15	0/0.15	0	1/1.4	0.71
Total	227/258.4	0.88**	275/243.6	1.13**		102/88.3	1.15	64/52.4	1.22*	81/84.1	0.73**

* Statistically significant at 5% level

** Statistically significant at 1% level

Table 6

SLIDE 7

MINERS: OBSERVED/EXPECTED DEATHS AND RATIO BY CAUSE

CAUSE OF DEATH	Exposed v. Controls Comparison				Internal Comparison					
	Total Exposed		Controls		Exp Level I		Exp Level II		Exp Level III	
	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio
Infectious diseases	56/66	0.85	75/64.5	1.16	21/17.5	1.20	15/14.8	1.01	20/23.4	0.5
All tumors	100/129.5	0.77**	155/125.1	1.2**	38/31.8	1.19	28/25.5	1.10	34/41.7	0.82
Lung cancer	9/20.2	0.45**	31/19.5	1.58**	3/2.7	1.11	1/1.9	0.53	5/4.4	1.14
Gastrointest. cancer	46/49.9	0.92	51/49	1.10	19/14.9	1.28	16/11.1	0.90	16/10.5	0.92
Various s. diseases	9/9.9	0.91	10/9.8	1.03	3/2.8	1.07	2/2.3	0.87	4/3.9	1.03
Cardiovasc. diseases	208/270.1	0.73**	334/265.5	1.23**	66/63	1.05	71/55.7	1.27**	71/86.2	0.82*
Respiratory diseases	142/102	1.39**	58/97.5	0.59**	28/36.9	0.72*	38/37	1.03	76/67.4	1.13
Gastrointest. diseases	49/59.1	0.83*	69/57.2	1.19*	16/14.8	1.08	12/12.7	0.94	21/21.5	0.98
Sentivity	12/11	1.09	10/10.8	0.93	5/4	1.25	3/2.8	1.07	4/4.9	0.81
Accidents	68/64.7	1.05	80/62.6	0.96	23/20.8	1.11	27/21	1.29	18/25.4	0.70
All other causes	23/33.5	0.68**	43/32	1.34**	11/7.7	1.43	4/5.2	0.77	8/9.8	0.82

* Statistically significant at 5% level

** Statistically significant at 1% level

Table 7

MILLER: OBSERVED/EXPECTED DEATHS AND RATIOS BY CAUSE

SLIDE 8

USE OF HEALTH	Exposed v. Controls Comparison			Internal Comparison						
	Total Exposed			Exp Level I		Exp Level II		Exp Level III		
	Obs/Exp	Ratio	Controls	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio	
Infectious diseases	14/27.6	0.50	21/17.2	1.22	7/5.4	1.30	6/4.9	1.22	1/3.7	0.27
All tumors	42/46.2	0.91	48/43.5	1.10	18/16.2	1.11	13/9.7	1.34	11/15.4	0.71
Lung cancer	4/7.6	0.53	11/7.3	1.51	3/1.61	1.86	1/0.79	1.27	0/1.26	0
Gastrointestinal cancer	16/19	0.84	21/17.9	1.17	8/6.7	1.19	4/3.8	1.11	4/5.7	0.70
Nervous system diseases	4/2.5	1.60	1/2.5	0.40	2/1.5	1.33	0/0.7	0	2/1.8	1.11
Circulatory diseases	72/92	0.78**	106/85.8	1.23**	28/26.6	1.05	20/14.2	1.41	24/20.2	0.79
Respiratory diseases	25/31.5	1.16	17/20.3	0.84	11/9.9	1.11	9/8	1.50	5/8.2	0.60
Gastrointestinal diseases	20/21.8	0.92	22/20.1	1.09	7/7.3	0.96	4/4.3	0.93	9/8.7	1.03
Serility	4/4.6	0.87	5/4.4	1.14	2/1.9	1.05	2/0.6	3.33	0/0.6	0
Accidents	28/26.7	1.05	24/25.2	0.95	15/11	1.36	7/8.3	0.84	6/8.8	0.68
All other causes	9/9.8	0.92	10/9.2	1.09	3/3.5	0.86	3/1.7	1.76	3/3.7	0.81

* Statistically significant at 5% level

** Statistically significant at 1% level

Table 8

SLIDE 9

MINERS: OBSERVED AND EXPECTED DEATH BY INTERVAL BETWEEN FIRST EXPOSURE AND DEATH
(Exposed v. Controls Comparison)

CAUSE OF DEATH	LATENCY					
	< 20		20 - 40		> 40	
	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio
All tumors	18/20.1	0.95	55/73.8	0.75**	28/35.6	0.73*
Lung cancer	1/1.98	0.51	6/14.2	0.42**	2/4.1	0.48
Gastrointest. cancer	5/6.9	0.72	18/19	0.95	13/14.6	0.89
Respiratory diseases	26/22.5	1.18	77/54.4	1.42**	38/25.2	1.43**

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

Table 9

SLIDE 10

MILLERS: OBSERVED AND EXPECTED DEATHS BY INTERVAL BETWEEN FIRST EXPOSURE AND DEATH
(Exposed v. Controls Comparison)

CAUSE OF DEATH	LATENCY					
	< 20		20 - 40		> 40	
	Obs/Exp	Ratio	Obs/Exp	Ratio	Obs/Exp	Ratio
All tumors	8/10.6	0.75	24/28.7	0.84	7/7.5	0.93
Lung cancer	0/1.5	0	1/1.5	0.67	3/4.5	0.67
Gastrointest. cancer	5/5.1	0.98	8/11.1	0.72	3/2.9	1.03
Respiratory diseases	7/8.2	0.85	17/11.6	1.47*	1/1.7	0.59

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