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Comments on "Mortality Patterns Among Hard Rock Gold Miners Exposed To An Asbestiform Mineral" and "Asbestos Fiber Exposures in a Hard Rock Gold Mine"

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INCLUDED IN JOHNS-MANVILLE'S COMMENTS TO OSHA
APRIL 1976 REGARDING PROPOSED 0.5 FIBER STD/ASBESTOS

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On 9 October the Department of Labor, Occupational Safety and Health Administration (OSHA), issued a "Notice of Proposed Rulemaking for the Occupational Exposure to Asbestos" in the Federal Register. A reduction in the time-weighted average (TWA) from 2 fibers per cubic centimeter (cc) as promulgated on 7 June 1972 to a TWA of 0.5 fiber/cc is proposed, on the rationale that:

> Since the promulgation of the U.S. permanent asbestos standard, considerable new information has been forthcoming on the toxic effects of asbestos. This has been in two areas: In the widening spectrum of cancers associated with asbestos exposure, and in various manifestations of asbestos disease in individuals exposed to relatively low concentrations of dust.

One of the chief references used by OSHA as a basis for its reduction of the asbestos standard is:

> Gillam, J.D., Lemen, R.A., Archer, V.E., Wagoner, J.K., and Dement, J.: Morbidity and Mortality Among Hard Rock Miners Exposed to an Asbestiform Mineral. An. N.Y. Acad. Sci. (In press).

We reviewed this manuscript, and while its text was clearly an incomplete working draft, we assumed that it contained all the information available to OSHA from this particular study for use in preparing the proposed rule change. It was clear that the conclusions of this manuscript were a major component of the "new information...forthcoming" cited by OSHA. As scientists working in the field, and as parties vitally concerned with the proposed revision of the asbestos standard, we analyzed this manuscript for the dual purpose of identifying its "new information" and evaluating the quality and merit of the study. More eloquent than our comments,

criticisms, and questions is the fact that the report has undergone two major revisions since our initial critique. Our present comments are directed to the latest revision, which has resulted not only in a change in title, but a separation of the report into two distinct manuscripts, despite a reduction in the total body of information being presented.

CRITIQUE

In their introduction the authors refer to a series of other studies on asbestos-related mortality, neglecting, however, to recognize the limitations placed on them by the distinction of heavy exposure common to the other studies. The principal null hypothesis was that the study population has adjusted cause-specific death rates equal to an identified standard population (often the general U.S. population). Alternative hypotheses of interest have been either "one"- or "two"-sided. Excessive disease categories in the study population were thus "identified". In essence they have used excess categories from other studies in their references in comparison with the standard mortality of the population of South Dakota. To arrive at the conclusion that low-level asbestos exposures are suspect, acceptable experimental design demands: (1) strong evidence of excessive disease in the asbestos-related disease categories and (2) that all other possible explanations for the excessive disease are explored and convincingly ruled out.

The deaths in the observed and expected categories are quite "small" in the present cohort mortality study. Care is required to ensure that underlying assumptions are reasonably satisfied. The hazard and therefore

reluctance in drawing conclusions from such small numbers is well recognized. The formal statistical analyses are based on assumptions and approximations that are not taken into account in the Poisson model as used by the authors. For example, the modified life table technique they use adjusts for age by considering five-year age groups. However, death rates for many causes including lung cancer double within five-year age periods. Thus, if the age distribution of the study population differs from that of the standard population, the expected as calculated often is inaccurate and yields a distorted picture. Occupational cohorts defined by minimum years' experience may be skewed to younger ages, whereas the general population, in this case the state of South Dakota, is skewed to the older ages. The standard population rates for South Dakota are also empirical values, subject to the usual variation. This factor is especially important when regional rates (a state) are used. This phenomenon has been extensively studied for the Chi-square distribution, but apparently not for small-sample Poisson tests. The use of five-year calendar rates improves the sample size, but dynamic population rates further compound the problem of the instability of rates within the quinquennium.

Smoking, in addition, is an extremely important consideration. The general statements in the manuscript comparing the present study pouplation with a uranium miner study group substantively fail to cover this point. For example, the upper bound for a possible adjustment from the uranium miner study was in relation to a different standard population, a four-state regional rate, South Dakota not

included, and thus cannot be related to the current standard population. Failure to consider smoking habits adequately is a serious shortcoming of the study, particularly in the absence of assurances that all available smoking data have been incorporated into the study.

The influence of all factors is magnified in studies where a "few" observations mean the difference between statistically significant or not. For example, it is easily verified that modest increases in the "expected" rates alter the statistically significant findings of the study. Disease categories associated with asbestos exposure have not been treated separately. Virtually all other reported studies distinguish malignant pulmonary neoplasm from others. For example, ICD 160-164 includes nose, middle ear, accessory nasal sinuses; larynx; primary bronchus, trachea, and lung; unspecified primary or secondary of lung; mediastinum and unspecified thoracic organs. It is unreasonable to believe that the middle ear, accessory nasal sinuses, or mediastinum should be considered particularly vulnerable to the influence of the airborne particles since there is no, or at most very slight, direct contact between these structures and the respiratory airstream carrying particles. Actually none of these organs have shown an excess of malignancy, even in large populations exposed far more heavily to respirable asbestos than in the small population of this study. Tumors of these organs remote from the airstream, plus those tumors of a secondary nature, should be excluded in calculating excess occurrence of respiratory

malignancy in the context of this study. To be certain that none of these are included is especially important in view of the very small total number of tumors (10) reported, since two or three tumors of these occluded organs could falsely raise the "malignant neoplasms of the respiratory system" to a statistically impressive level.

ICD 162-163 account for well over 90 percent of the deaths in the 160-164 categories for the general population and should be isolated for the present study; no ICD 161 deaths were observed. For example, Table II presents 7 observed and 2.18 expected deaths in the $\geq$ 20 yrs. column. With the two nonlung cancer deaths removed and adjustments made for ICD 162-163 only, expected deaths of 2.0 result with a P-value (as the authors have calculated) of >0.10 rather than <.01. If any upward adjustment is then made for smoking, age distribution, etc., the P-value becomes even larger. (The 49 percent adjustment mentioned in the paper results in a P-value of >0.35.)

A discussion of the deficit of malignant deaths in sites other than the respiratory system is missing. The authors have discussed only the findings they feel to be consistent with asbestos exposure, while omitting those that are inconsistent with the conclusions. THis is evident from their grouping of four cases of silicosis, three cases of influenza and pneumonia, and one case of chronic obstructive lung disease in order to show a significant excess of nonmalignant respiratory disease related to the occupational environment; this is an elementary violation of acceptable analytical design. Further, it is unclear how silica-related deaths can be used to "implicate" asbestos. Removal of the silica-related deaths

eliminates the excess in the nonmalignant respiratory category. Silicosis, because of its virtual absence in the general population, contributes "nothing" to the expected rate.

A discussion of the findings of the 1960 study on this mine would be helpful in considering the present study. Because of the small numbers in the present study, it would have been especially helpful to have had an appropriate occupational control group included in this study. This can provide a workable alternative particularly in this situation where it is impossible to reconstruct individual exposure histories. However, investigation of the work locations prior to 1960 for lung cancer deaths may have been possible. Work location at the rock surface has been associated with excess deaths in at least one other study of gold miners in Rhodesia. Reasonable documentation of the work environment has not been achieved. For example, it is most likely that the free crystalline silica levels thirty to forty years ago were well above the current standard. Radiation and arsenic exposures over the past forty years are also impossible to determine. While it is not possible to document historical exposure levels for any category, it is equally unreasonable to ignore them when considering causes of death between 1960 and 1973.

Major attention is directed to current asbestos exposure in the mines; however, even this concern ignores obvious caveats. For example, selection of the 25 highest fiber concentrations from 200 air samples cannot possibly be considered to characterize the working environment. Since 200 air samples were already available, at the very least a random sample of those 200 should have been analyzed. The airborne fiber concentrations given by the authors are very low. Based on the discussion of

the paper, it appears that the airborne concentration of asbestos fibers per se is only a fraction (50 to 60 percent) of the total fiber concentrations quoted. Exposure levels to radon daughters, arsenic, silica, irritant gases, and hydrocarbons are poorly documented. It is entirely reasonable to question, for example, what the findings would be if 200 samples documenting arsenic levels had been taken, particularly since one of the authors has reported a suspected excess of lung cancers following long-term low-level exposure to arsenic concentrations apparently present in the mine. The discussion of arsenic exposures contained in the published original abstract is absent from the final draft.

SUMMARY

The claim of excessive rates in the asbestos-related malignant and non-malignant disease categories is clearly based on poor and incomplete data analyses. Even if the claim were based on valid analyses, data incriminating asbestos in the causation are entirely lacking. Exposure to cigarettte smoke, arsenic, silica, irritant gases, and hydrocarbons is concurrent with exposure to asbestos fibers. None of these potential causes of disease has been eliminated, controlled, or even accurately measured in this study.

The original one manuscript is now contained in two reports entitled: (1) "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral" and (2) "Asbestos Fiber Exposures in a Hard Rock Gold Mine." The second of the two reports presents conclusions based on data contained in the Mortality study in the paper listed first. This unusual if not unprecedented statement of conclusions based on data from a separate manuscript is as out of context as it is misleading. The summary

and conclusion section of a scientific manuscript must surely be based on results reported from the study.

Federal regulatory agencies, because of their ultimate responsibility in setting standards for public health and safety, must be insistent that all data used in setting such standards meet the highest requirements of scientific competency and accuracy.

Comments on "Mortality Patterns Among Hard Rock Gold Miners Exposed to an Asbestiform Mineral" and "Asbesto… Published by AsbestosClaims.Law
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