exposure-response curve. He arrived at a value of 10 f/cc-years, a figure considerably lower than that derived by Berry et al. (Ex. 84–20). One significant factor that may account for the difference in estimates between these two studies in a difference in the length of their followup period (i.e., it is considerably longer in the Finkelstein study). In addition, the workers studied by Finkelstein may have also been exposed to silica which was used in the production process. If there was an excess risk of silicosis from such exposure which was mistaken for asbestosis then the exposure level resulting in a 1 percent probability of developing asbestosis would have been overestimated. There were no data published on the silica exposure levels to determine if this was a possibility.

Dement et al. (Ex. 84–37) studied the risk of asbestosis in 1,261 males employed for one or more months in a chrysotile asbestos textile operation between January 1, 1940 and December 31, 1965. Mortality followup using data from death certificates was from January 1, 1940 to December 31, 1975 and was 98 percent complete. The method used in this study to assign exposures to individuals was described previously (48 FR 51102). There was a total of 33,141 person-years of observation, and 24 deaths were ascribed to "other respiratory diseases" (ICDA 751–527), the category that includes asbestosis. Of the 24 deaths in this category, asbestosis or pulmonary fibrosis was the underlying cause of 17 deaths. Nineteen of these 24 deaths occurred 20 or more years after initial employment. The overall Standardized Mortality Ratio (SMR) for this category was 552. There was also an increased SMR for deaths due to cardiovascular disease, which is consistent with other observations among asbestos workers. The authors note that "a review of death certificates for the 105 deaths found [that] 6 [certificates] mention asbestosis or pulmonary fibrosis as a contributing condition."

In the study by Dement et al. (Ex. 84–37), there was little difference in SMRs for the "other nonmalignant respiratory diseases" by years since initial employment. For the group observed 10–19 years after first employment, the SMR was 521; for 20–29 years it was 565; and for greater than 30 years, the SMR was 570. It is noteworthy that even in the group observed 30 or more years after first employment, the SMR remains at an elevated level and showed no decline. The authors also derived SMRs for white males with 15 or more years since first exposure. These were 362, 84, and 879 for the exposure categories less than 1,000 f/cc-days (2.7 f/cc-years), 1,000–10,000 f/cc-days (2.7–27.4 f/cc-years), and 10,000–40,000 f/cc-days (27.4–109.6 f/cc-years), respectively. The excesses were statistically significant for both the first and the third exposure categories, and both of these cumulative exposures are below the cumulative exposure that would be caused by 50 years of exposure at the existing PEL of 2 f/cc. Finally, since other investigators (Selikoff, Ex. 84–90; Elmes and Simpson, (Ex. 84–42) who have studied the mortality of asbestos workers have shown that relying only on death certificates for ascertainment of cases causes a significant number of asbestosis deaths to be missed, the risk estimates reporterd by Dement et al. (Ex. 84–37) may be understated.

4. Summary of the Evidence of Asbestosis

OSHA believes that the studies of Berry et al. (Ex. 84–20) and Finkelstein (Ex. 84–44) show a clear dose-response relationship between asbestos exposure and asbestosis, and substantial excess risks due to asbestosis close to or below 100 f/cc-years, the cumulative lifetime exposure permitted by the 2 f/cc standard. The risk of mortality due to asbestosis in the work by Dement et al. (Ex. 84–37) was also in excess in workers exposed to less than 100 f/cc-years, despite the problems of underascertainment of cases. Because asbestosis morbidity is a better indication of risk than is mortality, OSHA has included the studies of Berry et al. (Ex., 84–20) and Finkelstein (Ex. 84–44) in a quantitative risk assessment for asbestosis (see Section V).

In his testimony Dr. Weill concluded that asbestosis deaths would be rare to non-existent under a two fiber standard and a disease of the past at a revised standard of 0.5 f/cc. He stated that:

> . . . We are able to detect asbestosis with greater sensitivity. It means we are going to be seeing less severe disease. . . .
>
> The asbestosis that is being seen generally now around the country again, I think by wide agreement . . . is at a low level, even now. This is associated with the exposures of the last several decades, when we know certainly in most instances and particularly in end-product use the exposures would still have been relatively uncontrolled.

In response to questions on the same issue Dr. Lewinsohn noted:

> I think asbestosis as it was originally described is a vanishing disease, yes. I think that asbestosis is a different disease than we see now, if it still exists. It's much milder. It's less likely to be fatal. It's less likely to produce significant impairment. . . .
>
> I think the levels of exposure have diminished and the changes, the disease itself is different. You don't see the full blown picture of asbestosis with people who die from asbestosis after less than 10 years' exposure with severely damaged lungs, with heart failure. . . .
>
> What you see today is somebody who has pleural changes or somebody who has very minimal radiological features of asbestosis and who probably goes on to live a reasonably normal life span. . . .

The view that asbestosis mortality and severe asbestosis morbidity is on the decline is corroborated by the work of Berry et al. (84–20) who show that as the cumulative dose of asbestos decreases, more cases are diagnosed as having crepitations only or as being possible asbestosis, in contrast to certified asbestosis. Nonetheless, severe cases (Barry et al. Ex. 84–20), disabled cases (Finkelstein, Ex. 84–44), and deaths due to asbestosis (Ex. 84–37) have been found to occur in workers with estimated cumulative exposures well below 100 f/cc-years. Although the clinical impressions of Drs. Weill and Lewisohn regarding a shift in the severity of asbestosis as exposures have declined in the past few decades may be correct, their conclusions regarding the eventual absence of disabling asbestosis and death due to asbestosis at the current standard of 2 f/cc are contradicted by the evidence from epidemiological studies mentioned above. In addition, Dr. Selikoff, under cross examination by Ms. Nash, tesified that he is still seeing cases of severe asbestosis more than 10 years since the 2-f/cc standard became effective:

> Nash: . . . In your clinical observations, are you continuing to see cases of asbestosis?
>
> Selikoff: Oh, yes.
>
> Nash: Are you continuing to see advanced cases of asbestosis—?
>
> Selikoff: . . . We certainly do. We see deaths. But I have not had the experience of seeing what would happen at 0.1 [f/cc]. But I'm also not willing to expose a large number of people to 0.1 as guinea pigs, so that I can come along twenty years later and give you the answer.
>
> Nash: So you would then obviously believe there's a risk of exposing people at higher levels and developing asbestosis . . .?
>
> Selikoff: Oh, no question—there is no question about that. That we already know from our extrapolation. (Tr. 7/2, p. 173)

Based on this testimony and the epidemiological data discussed above, as well as the results from OSHA's risk assessment (see Section V), OSHA finds that a reduction of the current 2-f/cc PEL will result in a continued decline in asbestosis incidence.

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