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NIOSH REVIEW OF DCMA LEAD CHROMATE STUDY

To facilitate the identification of my references to this review, I have numbered certain paragraphs on the enclosed copy.

Paragraph 1.

The review states that Table 5 shows the latent period "to be 17 years at most". There is nothing in this table that indicates the length of the latent period. The data in the table show duration of employment, not the length of time that has elapsed from date of first exposure to date of death or to the closing date of the study (December 31, 1974). In plant 3, almost all the workers were hired prior to 1957, so for them the latent period would be at least 17 years. The reviewer may be under the mistaken impression that the workers in Plant 3 were hired in 1957, although the paper states on page 4 that the study population of Plant 3 was selected from "a list of employees on the wage roll as of June 30, 1957".

Paragraph 2.

Although latent periods are usually in the range of 20-35 years, as the author states, NIOSH personnel have indicated to me that an assumed latent period of 15 years is acceptable. In Plant 3, most of the subjects would probably satisfy the requirement of a 20-year latent period.

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Paragraph 3.

The small size of the sample is a valid point, but that is acknowledged by the author and it is the largest sample size that could be obtained in this country.

Paragraph 4.

I agree that the age distribution of the cohort should have been shown.

The author probably did not use crude death rates to compute expected numbers for the various causes of death analyzed in the report. The crude death rates for respiratory cancer shown in Tables 9 and 10 were presented to make points regarding trends in respiratory cancer, differences in rates among geographic regions, and differences between races. The author may have assumed it was understood that age-specific rates were used to compute all expected numbers, but he should have included such a statement in his paper to remove any doubts on this point.

There is no doubt, however, that age-sex-race specific rates were used to compute expected numbers for respiratory cancer and cancer of the stomach, as stated by the author on pages 13 and 14, and as cited by the reviewer in Paragraph 5. Since these two types of cancer were the ones with which the study was primarily concerned, it is difficult to understand the reviewer's criticism in Paragraph 4 that the SMR's were not adjusted for age and race.

Paragraph 5.

It is not clear which control group contains "considerable bias". The SMSA's that were used as control groups to compute SMR's for respiratory and stomach cancer had the least bias because they were in the same area as the plants and the rates took into account both age and race. Rates in these SMSA's were not used to compute SMR's for other causes of death because the numbers of deaths from other causes did not indicate an excess in the study populations.

Paragraph 6.

The "increasing gradient" of SMR's in Plant 1 is not significant since it is based on only 2 deaths. There was no

increasing gradient in Plant 2, as the author states, because there were no deaths with which to compute an SMR. The 2 deaths from respiratory cancer in Plant 2 were eliminated because these workers were hired after 1960 and died less than 2 years after they were hired.

In the last sentence of this paragraph, the reviewer states that records in Plant 3 were not available prior to 1957. As in Paragraph 1, the statement indicates the reviewer's mistaken impression that the study group in Plant 3 consisted of those hired in 1957.

Paragraph 7.

The statement that the SMR for digestive cancer in Plant 3 increased from 209.9 to 778.3 when the SMSA was used as a control population is not correct. The SMR, 209.9, is for cancer of all digestive organs combined, whereas the SMR, 778.3, is for cancer of the stomach only.

Paragraph 8.

The point is made here that a causal association between a disease and exposure to a given substance should not be ruled out because no such association has previously been reported in the scientific literature. This point is well taken.

Paragraph 9.

The gradient with increased exposure and latency for respiratory cancer in Plant 1 was based on only 2 deaths. In the Plant 3, the increasing gradient was very slight, the SMR increasing from 237.1 to 244.7, and resulting only from a drop in the expected number from 2.5 to 2.4.

Paragraph 10.

The reviewer suggests a re-analysis of the data, but does not specify what that analysis should be and how it would further clarify the excess respiratory and digestive cancer.

In general, I believe the author analyzed the data to the extent possible, given the acknowledged shortcomings in the data for the study group and in the comparison populations. The criticisms in this review were unduly severe, and all but two were not justified.

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