

1-00227

cc: J. A. Zapp, Jr.

June 9, 1975

MEMORANDUM

TO: R. M. LUCKRING, PHYSICS DEPT.
FROM: C. F. REINHARDT AND E. P. LEE, HASSELL LAB.

*I told Luckring it
was OK to give this
info to Carlsberg. Fred
Edel (see ref. letter).
-JSCM
6/16/75*

CARCINOGENICITY OF ASBESTOS FIBERS

Ref.: Letter R. M. Luckring to J. A. Zapp, Jr. 5-13-75.

We will attempt to answer the questions you posed in the reference letter in the order presented. First, you asked for our comments on the Gross et al. publication on Ingested Mineral Fibers attached to your letter. It indicates that the ingestion by rats of a diet containing as much as 5% asbestos over most of their life did not produce cancer. This, in addition to other similar experiments reported in the same article, indicates that ingested mineral fibers, including asbestos, do not present the hazard that has been associated with the inhalation of some mineral fibers.

As far as we know, there is no single report indicating tumor development in the gastrointestinal tract resulting from feeding asbestos dust to experimental animals.

Replies to the four additional questions you raised are as follows:

1. In a nationwide study of 2066 non-cigarette smoking asbestos workers, 73 deaths occurred but only two were due to lung cancer (Hammond and Selikoff 1973). One of the two men was a cigar and pipe smoker, and the other had never smoked regularly. From this observation, one can state that lung cancer is uncommon among asbestos workers who have no history of cigarette smoking and that if there is a risk involved, it is not great.

2. An initial study of 263 asbestos insulation workers who had a history of regular cigarette smoking revealed that 24 had died of lung cancer (Selikoff et al. 1968). However, given their smoking habits, only 2.96 such deaths would have been expected in the control smoking population. Using these figures there is about an eightfold increase in lung cancer deaths in asbestos workers who smoke cigarettes regularly versus non-asbestos workers who are regular cigarette smokers.

1421

DUP 0903572

PLAINTIFF'S
EXHIBIT

DUP-158

June 9, 1975

3. Of deaths that occurred among 532 asbestos workers exposed to asbestos dust 20 years or longer, 29 were due to gastrointestinal cancer, while 9.4 would have been expected in the control population (Selikoff et al. 1968). Although the data suggest that asbestos dust exposure increased the incidence of gastrointestinal cancer, the number of such deaths is too small to draw any definite conclusions.

A recent epidemiological survey of deaths among 17,800 U.S. and Canadian asbestos insulation workers showed that 16 deaths were due to stomach cancer versus 6.62 expected deaths from the control population; 26 observed versus 17.51 expected deaths from cancer of the colon-rectum; and 13 observed versus 3.31 expected deaths from cancer of the esophagus (Hammond and Selikoff, 1973).

Because of the small number of expected and observed deaths from cancer at these sites among workers with no history of cigarette smoking, no conclusive statement can be made that indicates a possible interaction between cigarette smoking and asbestos exposure as they might relate to gastrointestinal cancers. However, other surveys indicate a high degree of association between cigarette smoking and the occurrence of cancer of the esophagus.

In spite of the apparent relationship between exposure to asbestos dust and gastrointestinal cancer, the uniformly negative animal feeding studies cast some doubt on such a relationship.

4. Of 94 deaths observed among 370 asbestos workers, ten were due to mesothelioma, three were pleural mesotheliomas and seven peritoneal (Selikoff et al. 1968). This tumor is such a rare type that if the 370 subjects had been selected randomly from the general population, one would not expect any of them to die of mesothelioma.

Pleural and peritoneal mesotheliomas are felt to behave differently in that the peritoneal type have limited or no metastases (Thomson, 1969). However, pleural mesotheliomas have a range of malignancy where spread by blood and lymph can occur. More significant is the direct spread to the lungs, liver, vertebral bodies, serous sacs, pericardium and other pleural sacs.

We hope this information will be helpful to you and if you wish to discuss any of the above points, please give us a call.

References:

1. Hammond, E. C. and I. J. Selikoff: Proceedings of a Working Conference held at the International Agency for Research on Cancer, Lyon, France. IARC Scientific Publication No. 8., pp-312 (1973).
2. Selikoff, I. J., E. C. Hammond and J. Churg: JAMA 204:106 (1968).
3. Selikoff, I. J., J. Churg and E. C. Hammond: JAMA 188:22 (1964).
4. Thomson, J. G.: Pneumococcosis. Proceedings of the International Conference, Johannesburg, 150 (1969).

CJR:KPL:lja

DUP 0903573