"pseudo-asbestos bodies." Recently Davis[5] and Collet[6] have demonstrated the intracellular development of "asbestos bodies" secondary to amorphous material.

Asbestos occupies an anomalous position in its ability to produce lung damage, causing diffuse fibrosis and possibly also lung cancer. It is anomalous inasmuch as no other silicate has been shown to possess this ability. The fibrosis encountered in talcosis is limited to the tremolite variety, which is asbestos-like. On the other hand, the pneumoconiosis associated with the mining of mica (another silicate) has not been investigated experimentally, and the fibrogenic activity of the dusts coexisting with the mica has not been evaluated. Other silicates may evoke either minimal or relatively slight inflammatory changes, and are incapable of producing lung cancer.

Inasmuch as certain polycyclic hydrocarbons and trace metals known to be potent cancer-producing agents have been demonstrated in asbestos, particularly nickel, chromium and benzopyrene, the possibility exists that the trace metals and/or the polycyclic hydrocarbons may be solely responsible for the observed pathogenicity.

This possibility is supported by two findings:

1. The report of Braun and Truan[7] which indicated that the prevalence of lung cancer among Canadian asbestos miners was no greater than that of the general population. This was in contrast to the report of Doll[8]

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(5) Davis, J.M.G. Electron-Microscope Studies of Asbestosis in Man and Animals. Ann. N.Y. Acad. Scie. 132:98-111, 1965.

(6) Collet, A. Personal Communication, 1965.

(7) Braun, Daniel C. and Truan, T. David. An Epidemiological Study of Lung Cancer in Asbestos Miners. AMA Arch. Ind. Health 17:634-653, June 1958.

(8) Doll, R. Mortality from Lung Cancer in Asbestos Workers. Brit. J. Ind. Health 12:81, 1955.