TABLE IV

Epidemiologic Studies of Asbestos Exposures and Lung Cancer*

PLACE	AUTHOR	YEARS FOLLOWED	NO WITH LUNG CANCER	COMPARISON Ob/Expected
United Kingdom	Doll	31	11/39 deaths	+0.8 expected
Quebec	Braun & Truan	5	9/87 deaths	+6.0 expected
Pennsylvania	Mancuso & Coulter	20	19/186 deaths	+5.61 expected
New York & N. Jersey	Selikoff et al.	30	45/255 deaths	+6.6 expected
California	Dunn & Weir	7.1	10/41 deaths	+2.8 expected
Dresden	Jacob & Anspach	39	34/150 deaths	+11.9 expected
U.S.A.	Enterline deaths	24/286	12	+11.9 expected

*Adapted from W. Clark Cooper—Asbestos as a Hazard to Health—Fact and Speculation, Arch. Environ. Health, 15:285 (September) 1967.

malignancy of smokers who are not exposed to asbestos. The metal complexes in the asbestos fibers (nickel, chromium, etc.), benzpyrene and others carcinogenic agents absorbed on the asbestos, allergic response which produces altered immunity, and the increased burden from urban populations or smoking are now postulated to be the major co-factors which make asbestos carcinogenic.[12]

A dose too small to produce fibrosis, i.e. asbestosis, may, nevertheless lead to lung cancer. A dose large enough to produce asbestosis may cause malignancy if the worker does not die first from his pneumoconiosis. However, many men, after a lifetime of work with asbestos products—specifically insulators—show no evidence of any untoward pulmonary pathology. Again, in all of these, the amount of asbestos inhaled is unknown. When the dose is known to be related to the incidence and severity of a disease (and it grossly appears to be the case in asbestosis), it is safer to postulate that a non-threshold relationship exists. From a practical standpoint, however, asbestos would be an unusual disease agent if it did not have such a threshold. "Proof," however, will always be a speculation, as it is for example in determining the carcinogenic level of radiation. There is no way of estimating this threshold level, which at present needs more industrial hygiene studies.

Our findings support those of other investigators who state that pleuritis occurs in about 25% of insulation asbestos workers. The incidence of mesothelioma in this group is extremely large, considering the rarity of the tumor. Selikoff reporting on a series of 124 deaths from neoplasia in insulators found 10 with mesothelioma.[6] This malignancy has also occurred in increased incidence in some populations which did not work directly with asbestos. In a very large proportion in South Africa and in a considerable number of the the victims of this malignancy in the United States, environmental exposure is postulated as the cause since they lived close to asbestos plants using especially amosite or crocidolite.[13] The evidence is strong that the source of the agent was indeed airborne from the asbestos factories or mines, even though many of the individuals did not show an underlying asbestotic fibrosis. However, the ambient concentration or indeed the specific type of asbestos to which the person was exposed and which could be responsible for the disease has never been determined, either in the neighborhood or in the working environment.

An increased incidence of gastrointestinal malignancy is still being investigated, but the statistical inference is strong that there is an increased susceptibility in those exposed to asbestos. This will probably be clarified in time. Emphysema or chronic bronchitis, if it does occur as a result of inhalation of asbestos dust, would always be obscured by the underlying pneumoconiosis; and the diagnosis of these conditions would always be considered a secondary rather than a primary disease.

A link and perhaps the key to the problems lie in understanding the role of the "asbestos body" in the pathogenesis of the different tissue responses. Our experience supports the statement of Gough:[14] "Asbestos is diagnosed histologically by the association of asbestos bodies and asbestos fibers with fibrosis." However, what the "asbestos body" means in individuals who apparently have never been exposed to asbestos is not clear. If this can be explained, inferences then could be made with some certainty regarding the potential environmental hazard.

The presence of "asbestos bodies" in the lung is a biological indicator of *exposure* to asbestos. Whether or not other mineral fibers can produce an "asbestos body" is not pertinent in an environmental sense. The highest concentration of mineral fiber in ambient air is overwhelmingly asbestos and hence one can assume that "asbestos bodies are almost entirely derived from asbestos fibers." "Asbestos bodies" probably can be found in every lung at autopsy if searched for long and hard enough and an occasional one is adventitious and simply indicates that mineral fibers make up a part of the dust in our environment.

The fibrosis of asbestosis is nonspecific and has no special structural characteristics except for the presence of "asbestos bodies." Hence, interstitial