The data which lead to this speculation is mostly epidemiologic in character. There is unequivocal evidence that fibrosis of the lung (asbestosis) occurs in asbestos workers. Pleuritis is a frequent sequela of asbestos exposure and a rare malignancy of the pleura, mesothelioma, shows a remarkable increase in asbestos workers and in individuals living near asbestos-producing mines and factories. Neoplasms of the lung occur more frequently in individuals who have asbestosis or who are occupationally exposed to asbestos.

These findings, per se, would not lead to the sweeping speculations made by Thomson, Selikoff and others were it not for the additional finding that a large percentage of individuals over the entire world show "asbestos bodies" in their lungs at autopsy even though most have had no known occupational exposure to asbestos (Table I). Since "asbestos bodies" are specific for past exposure to asbestos or similar fibers, the speculation that all of us are under some increased threat of lung cancer due to the presence of ambient asbestos is valid.

Assessment of the magnitude of this threat is the crucial question. The dynamics of the situation will not permit us to wait until 1990 for a definitive answer. Forces such as the increased use of asbestos products and the general soiling of ambient air in our industrial technological society demand that we make some judgment now. If we cannot make this determination, we must direct our research into those channels which will be most likely to give us early and definitive proof.

Epidemiological studies are of prime importance and in fact present the most convincing evidence that asbestos may be dangerous to the working population, but the likelihood of proving that there is an increased risk to the public from asbestos in ambient air, by using epidemiologic methods, is very small. Even if all the factors which could influence its evaluation are known and a prospective study devised to demonstrate an effect by 1990, any conclusions from such a study would be subject to severe doubt because of the many variables which could not be controlled satisfactorily. For instance, it would be almost impossible to select a proper cohort which could be followed over the many years; the selection of matched controls would be almost insurmountable; the increasing use of asbestos and the differences in composition and possible exposure in various localities, and the complications introduced by cocarcinogens in our environment further becloud the issues and make it very unlikely that an equivocal answer could ever be forthcoming from this type of evidence alone.

TABLE I
Percentage of Asbestos Bodies in Lung Smears*

Thomson	(1963)	Cape Towne	26.4%**
Thomson	(1965)	Miami	27.2%**
Cauna et al.	(1965)	Pittsburgh	41.0%
Webster	(1965)	Johannesburg	39.2%
Meurman	(1966)	Finland**	57.6%
Anjilvel et al	(1966)	Montreal	48.0%
Cooper & Tabershaw	(1966)	San Francisco	45.0%

*in consecutive unselected autopsy series.
**thick sections

Adapted from Cooper, W. Clark. Asbestos as a Hazard to Health—Fact and Speculation. Arch Environ Health 15:283 (September) 1967.

The experimental approach can provide only tentative answers since it has been demonstrated that, while asbestos will produce irritation, plaques and malignancies in serous cavities, it is relatively inert as an over-all irritant to biologic materials. Furthermore, the translation of effect from any species of animals to man is difficult at best and is made even more questionable by marked variations in respiratory tract response by different animal species. The animal selected must most nearly resemble the human lung in order for the experiments to be meaningful. Difficulty is evidenced, for instance, in the fact that some animals, e.g. the cat and the guinea pig, will produce fibrosis and relatively few asbestos bodies, and others such as the mouse will produce relatively few asbestos bodies but no fibrosis.[4] This is probably related to the variations in the evolution of alveolar macrophages which Collet et al.[5] have demonstrated to differ among humans, cats, rabbits, and guinea pigs.

Exposure factors are not completely understood. Asbestos is said to be indestructible, but the term is a relative one; high heat, chemicals and body fluids produce changes in its physical and chemical properties in time. There is undoubtedly a difference in effect from a heavy, brief or continuous exposure as occurs in industry to a sustained minor exposure which may occur from ambient air pollution. Selikoff[6] has iterated many times that there is an increasing use of asbestos (Table II) and surmises that we are presently seeing lung malignancies "consequent upon inhalation of asbestos associated with the limited asbestos of some 30 years ago." The implication is clear that if this relationship (tons of asbestos produced to incidence of lung malignancy) is true, the multifold increase in its use has started an upward trend which will be reflected for the next 30 years and more. This extrapolation is unwarranted.

Increased production cannot be equated with increased hazard, especially in a substance like