and co-workers predict that the rate will go down even if we do not reduce air pollution, so that a simple decrease in rate is no proof of causal relationships. Another possible interpretation of this data is that if air pollution and lung cancer have a causal relationship, whatever it is in the air that was responsible for this has already begun to decrease. One could speculate that substitution of oil for coal, beginning about 1930 in significant amounts, has already begun to remove the lung carcinogens from the air.

In 1958, C. O. S. B. Brooke of the Finsbury Health Center, London, examined carefully the statistics on lung cancer deaths for England and Wales for the 1932–1956 period (3). He interpreted the data as suggesting that cancer of the lung seen now may have been at least partly determined during the patient's teens. Further, an almost explosive increase in bronchocarcinogenic factors appears to have occurred early in the century, although some regression may have occurred since 1915. In brief, his data support generally, if not in every detail, the data of Gilliam *et al.* quoted above. Beebe (4), in 1960, analyzed lung cancer deaths among World War I veterans, and concluded that lung cancer was slightly increased in those who had been subjected to mustard gas poisoning in 1918, although no such increase was observed in those who had pneumonia or wounds of the extremities.

Barnes and Ratzenhofer (5) reviewed 26,546 autopsies at the University of Graz, Austria, finding 868 cases of lung cancer. Apparently lung cancer is far more prevalent than other malignancies as a cause of death in persons having tuberculosis. Since the advent of modern chemotherapy for tuberculosis, more people survive tuberculosis than previously, living on to develop lung cancer at a later date. Confirming this, Herdan (6), of the University of Bristol, pointed out that certain apparently unrelated occupational groups showed a significantly high or low standardized mortality ratio for respiratory tuberculosis in 1930, and these same groups now have a significantly high or low ratio for lung cancer, with their tuberculosis mortality greatly reduced. It was suggested that sulfa drugs and antibiotics have reduced mortality due to epidemic and inflammatory lung disease, thus making way for the action of a disease of genetic origin. This would affect males more than females because males are more subject to recessive genes that transmit lethal conditions.

In another statistical paper, Manos and Fisher of the U.S. Public Health Service found (7) high positive correlations of various indices of air pollution that they developed with the following diseases: cancer of the esophagus, stomach, trachea, bronchus, and lung; arteriosclerotic heart disease, including coronary disease; and chronic endocarditis not specified as rheumatic and other myocardial degeneration. Although there may be some good reason for such correlations in some of these conditions, it is difficult to postulate the rationale for others, such as chronic endocarditis. This suggests the possibility of some third factor common to all the diseases and also to an air pollution index.

Kreyberg (8), of the University of Oslo, found that the air in one gas works contained amounts of benzpyrene corresponding to some 5,000 cigarettes daily for the 40-hour week, but only a moderate excess, if any, of lung cancer in the gas workers. It is possible that there may be no relationship at all between lung cancer and benzpyrene. This possibility was also suggested by Hueper *et al.* (9), who could find no correlation between reported lung cancer mortality and the amount of 3:4-benzpyrene or the carcinogenic potency (determined by injection under the skin of mice) of particulates found in the air of eight specially selected U.S. metropolitan areas. There has been a tendency on the part of many air pollution investigators to use benzpyrene content of the air as a measure of pollution, and hence of the hazard of lung cancer. These data suggest that this cannot be done.

Wynder (10), of the Sloan-Kettering Institute for Cancer Research, found that the lung cancer incidence in Venice, presumably with a very low air pollution level, at least from auto exhausts (because of its canals), was no different from other Italian cities. This suggests that the elimination of auto exhaust might not influence lung cancer rates.

Perhaps one very distressing situation in regard to lung cancer is the lack of reliable statistical data on the incidence of lung cancer in various cities. Thus Manos (11), of the U.S. Public Health Service, published a large volume providing mortality indices for various causes of death, including lung cancer, in a series of cities in the United States. These indices presumably provide a measure of the amount by which a given cancer rate in a given city exceeds or