environmental pollution with coal dust increased. Thus, this study presents presumptive data also that exposure to sulfur dioxide, at least at the levels found in air pollution, is not associated with specific pulmonary or cardiac symptoms or pathology.

In previous studies in Nashville, Zeidberg *et al.* (24) found that the average asthma attack rates on the 30 days with the highest SO_2 levels were significantly higher at the 5% level than the average attack rate on the 46 days with the lowest SO_2 levels. If the daily data on asthmatic attacks were deferred one day to take account of possible delayed SO_2 effects, the differences in rates were significant at the 1% level. However, even the authors acknowledged that asthma is not a very good disease to study in relation to single causes because "many factors have been listed as the cause of bronchial asthma," including allergens, meteorologic and aerometric factors, emotional factors, and infections. Any clinician treating asthmatics knows that trades involving the breathing of nonspecific dusts must be avoided by their patients. These Nashville studies found inverse relationships between attack rates and wind velocities, thus suggesting that still, quiet days with high probability of inversion prevailed when SO_2 was elevated. On such days, all pollutants including soiling, particulates, pollens, etc., as well as SO_2, would be increased.

CONCLUSION

As indicated in the opening paragraphs of this review, it is not the intent to imply that correlations between health effects and air pollution might not exist, but only to bring an awareness of frequently overlooked reports which suggest that the state of our knowledge at this moment is far from complete. Suggestions have been made which warrant further intensive investigation, but it is far to soon to draw any positive conclusions. It is to be hoped that those who are putting forth the "overwhelming, persuasive, and deceptive propaganda" will give pause to their efforts and take cognizance of these reports before they precipitate costly undertakings which may not be effective.

REFERENCES

1. PRINCI, F. Medical perspective in atmospheric hygiene. Chairman's Address to Section on Preventive Medicine, AMA Annual Meeting, Chicago, Ill. June 25, 1962.

2. GILLIAM, A. R., MILMORE, B. K., and LLOYD, J. W. Trends of mortality attributed to cancer of the lung: The declining rate of increase. *Cancer 14:* 622, 1961.

3. BROOKE, C. O. S. B. The incidence of cancer of the lung, 1932–1956. *Brit. J. Cancer 12:*481, 1958.

4. BEEBE, G. W. Lung cancer in World War I veterans: Possible relation to mustard-gas injury and 1918 influenza epidemic. *J. Nat. Cancer Inst. 25:*1231, 1960.

5. BARNES, B. O., and RATZENHOFER, M. One factor in increase of bronchial carcinoma. *J.A.M.A. 174:*2229, 1960.

6. HERDAN, G. The increase in the mortality due to cancer of the lung in the light of the distribution of the disease among the different social classes and occupations. *Brit. J. Cancer 12:*492, 1958.

7. MANOS, N. E., and FISHER, G. F. An index of air pollution and its relation to health. *J. Air Poll. Control Assn. 9:*5, 1961.

8. KREYBERG, L. 3:4 Benzpyrene in industrial air pollution: Some reflexions. *Brit. J. Cancer 13:*618, 1959.

9. HUEPER, W. C., *et al.* Carcinogenic bioassays on air pollutants. *Arch. Pathol. 74:*89, 1962.

10. WYNDER, E. L. Lung cancer in Venice: An epidemiological study. *Lancet 2:*1347, 1961.

11. MANOS, N. E. *Comparative Mortality Among Metropolitan Areas of the United States: 102 Causes of Death.* Pub. Health Service Pub. No. 562, 1957.

12. GILLIAM, A. G. Mortality attributed to lung cancer in the large cities of the United States in 1948 and 1949. *J. Nat. Cancer Inst. 15:*1307, 1955.

13. GILLIAM, A. G. Personal communication dated July 8, 1960.

14. MARI-CHANEZ, P. Development of spontaneous pulmonary tumors at high altitude in Strain A mice. *J. Nat. Cancer Inst. 28:*55, 1962.