cluded these other works (*2–5*) in the list of references. Perhaps it was an oversight on their part or possibly they are unfamiliar with these previous reports. If the latter is the case, I am happy to call these articles to their attention.

MICHAEL A. DAVIS
Harvard Medical School
Boston, Massachusetts

REFERENCES

1. BUSSE W, REED C, TYSON I, et al: Prolonged retention of radioactivity following perfusion lung scan in asthmatic patients. *J Nucl Med* 14: 837–839, 1973

2. DAVIS MA: Long-term iron retention in lungs perfused with iron hydroxide aggregates. *J Nucl Med* 12: 349–350, 1971

3. DAVIS MA, HOLMAN BL: Radiopharmaceuticals for perfusion scanning. In *Progress in Nuclear Medicine*, vol 3: *Regional Lung Function in Health and Disease*, Holman BL, Lindeman J, eds, Basel, S. Karger Press, 1973, pp 10–36

4. DAVIS MA: Long term retention and biologic fate of ^{99m}Tc-iron hydroxide macroaggregate. In *Radiopharmaceuticals and Labelled Compounds*, vol 2, Vienna, IAEA, 1973, pp 43–63

5. SPENCER RP: Variation in pulmonary retention of ^{131}I-macroaggregated albumin. *Thorax* 27: 332–333, 1972

THE AUTHOR'S REPLY

Our interest in this phenomenon has been primarily in patients with asthma. In a subsequent publication (*1*) we reported additional studies that further elucidated this abnormal retention of the radioactivity by the lungs of asthmatic patients. Briefly, we found that the Bordetella pertussis-treated mouse, which in many respects resembles the atopic state (*2,3*), retained a higher percentage of the injected radioactive ^{131}I-MAA at 24 hr than did the untreated mouse (24% versus 12%, $p = 0.001$). In addition, in vitro studies measuring degradation of the ^{131}I-MAA particle by plasma and leukocytes demonstrated a decreased ability to degrade the aggregated albumin particle by plasma and leukocytes from patients with asthma. Patients with chronic obstructive lung disease had normal in vitro degradation of the albumin particles. Other disease states, however, were not investigated.

The significance of this observation remains unclear. In asthma, however, the sputum is characteristically thick and viscous. There is evidence that sputum from patients with asthma interferes with tryptic digestion (*4*). We have speculated that the same process that is active in degrading the MAA particle is also active in reducing sputum viscosity.

Dr. Davis' comments are appreciated and they bring to our attention his and Dr. Spencer's work.

WILLIAM W. BUSSE
University Hospitals Center
for Health Sciences
Madison, Wisconsin

REFERENCES

1. BUSSE WW, REED CE: Abnormal degradation of macroaggregated albumin particles in patients with asthma. *J Allergy Clin Immunol* 53: 271–277, 1974

2. REED CE: Pertussis sensitization as an animal model for the abnormal bronchial sensitivity of asthma. *Yale J Biol Med* 40: 507–515, 1968

3. SZENTIVANYI A: Effect of bacterial products and adrenergic blocking agents on allergic reactions. In *Immunological Diseases*, Samter M, ed, Boston, Little, Brown and Co, 1971

4. SMITH JM: Interference with tryptic digestion by sputum from asthmatic patients. *Am Rev Respir Dis* 88: 858–860, 1963