ogists (and other scientists) can receive the communication. With Esperanto the same is at present true, with the additional limitation that no communications are being offered. But I should be willing to join my colleagues in any generally attempted solution, even if it means sitting up nights with an Urdu grammar or a Quechuan wordbook.

ANCEL KEYS

Laboratory of Physiological Hygiene
University of Minnesota

The Hemolytic Substance Present in Animal Tissues

TYLER (*1*) has recently reported hemolytic activity in saline extracts of fetal and adult guinea pig liver. The lytic activity varied in different fractions of the extracts, which were obtained by centrifugation at 2,500 rpm and at 25,000 and 100,000 × *g*; there were also relative differences between adult and fetal extracts. This was taken as "substantial evidence" of differences in the rate of enzymatic (hemolytic) activity between fetus and adult, which were considered not to be due to differences in enzyme concentrations but in inhibitor.

Tyler makes the following statements and suggests alternative theories concerning the nature and mode of action of the hemolytic substance present in animal tissues:

1. An unidentified hemolytic agent occurs in normal and pathological tissues.

2. Plasma or serum and possibly brain contain inhibitory substances of the hemolytic agent, which are thus far undefined.

3. Practically no attention has hitherto been paid to intracellular inhibitors of hemolysis.

4. The lytic agent is an enzyme acting directly on the cell membrane, which is its substrate, with inactivation resulting from the ability of inhibitors to act as competitive substrates; or

5. The lytic agent is enzymatically produced, with inhibition taking place through the formation of an inhibitor-lysin complex.

The following facts (*2–7*) refer to each of the above-mentioned points:

1. The hemolytic substance present in plasma and animal tissues has been isolated in crystalline form (*2*) and identified as *cis*-vaccenic acid [$CH_3 \cdot (CH_2)_5 \cdot CH{=}CH \cdot (CH_2)_9 \cdot COOH$] (*8*).

2. The inhibitors of hemolysis present in plasma are albumin globulin, calcium, cholesterol, and lecithin. Their quantitative interrelationship with the hemolytic acid has been examined. Phosphate and, in pathological conditions, hematin and porphyrins act as accelerators. Extraneous circulating hemolytic acid has no effect on erythrocytes under normal conditions because of a large excess of inhibitors in the plasma. It may, however, be a contributory factor to hemolysis in certain pathological conditions, as, e.g., in blackwater fever (*4*).

3. Normal erythrocytes contain the hemolytic acid, which is most likely related to their normal life span. Hemoglobin and stromatin act as powerful *intracellular* (intracorpuscular) inhibitors of lysis during most of the life of the normal erythrocyte. Only in aging erythrocytes does the hemolytic acid seem to become dissociated from its complex with stromatin.

4. No experimental evidence exists for the contention that the body contains a hemolytic enzyme, whose substrate is the cell membrane. As this is a lipoprotein, it can only be acted upon through one of its constituents. The supposition that the enzyme is proteolytic can be excluded. It could therefore be solely concerned with the degradation of the lipid component, as was first suggested by Bergenehm and Fahraeus (*9*). However, the claim advanced by these authors in favor of the existence of (hemolytic) lysolecithin produced from lecithin by an enzyme (i.e., lecithinase) was not supported by direct experimental proof—as, for instance, isolation of either the enzyme or the lysolecithin—but was based on indirect evidence and analogies. These have not stood the test of critical reexamination. Their claim must therefore be abandoned.

5. The claim that the hemolytic agent, though not an enzyme, is enzymatically produced, may be made for innumerable substances that are not ingested but changed or synthesized by the body.

As the structure of the hemolytic acid present in animal tissues is now established, there seems to be no need of further speculation regarding its nature.

H. LASER

Molteno Institute
University of Cambridge

References

1. TYLER, D. B. *Science*, **112**, 291 (1950).
2. LASER, H. *J. Physiol.*, **110**, 338 (1949).
3. ———. XVII Intern. Physiol. Congress, Abstr. of Communications, 160 (1947).
4. ———. *Nature*, **161**, 560 (1948).
5. LASER, H., and FRIEDMANN, E. *Ibid.*, **156**, 507 (1946).
6. LASER, H. I Intern. Congress of Biochemistry, Abstr. of Communications, 454 (1949).
7. MORTON, I. D., TODD, A. R., and LASER, H. *Ibid.*, 239 (1949).
8. MORTON, I. D., and TODD, A. R. *Biochem. J.*, **47**, 327 (1950).
9. BERGENEHM, B., and FAHRAEUS, R. *Z. ges. exptl. Med.*, **97**, 555 (1936).

Professorships in Foreign Countries

OPPORTUNITIES for foreign exchange professorships and visiting professorships are plentiful. The Fulbright Exchange Program is expanding; the Smith-Mundt Act provides for visiting professorships; teaching opportunities exist in the American Military Governments of Japan and Germany; and many positions are available through the Unesco fellowships, scholarships, and educational exchange programs. My experience in this type of work began in 1946–47 when, as consultant in biology for AMG in Korea, I had the opportunity to teach in several Korean colleges and universities (*Science*, **107**, 31, [1948]). In June 1950 I was visiting professor of parasitology at Seoul National University Medical School. In Japan, after the evacuation, I discussed at length the experiences, impressions, and conclusions of several others who taught in Korea. Combining their ideas and mine, I here present some general reactions and recommendations with the hope they will help anyone who plans to accept a foreign professorship.