Resolved: (1) That papers presented on the programs of the American Association for the Advancement of Science should not refer to commercial trade names of products where this can be avoided, in order to eliminate the criticism which might be made that such reference in scientific papers is in any way associated with advertising.

(2) That papers for the programs be accepted with the understanding that full scientific information is available for presentation and discussion if pertinent to the specific phase of the research being reported or described.

It was voted that nominations for emeritus life memberships under the Jane M. Smith fund be referred to the Committee on Emeritus Life Memberships, with power.

The report of the Committee on Source Books in the History of Science was read and accepted. The general secretary presented a progress report (1) of the Committee on Radio Program at Saint Louis and (2) on the activities of the Committee on the Place of Science in Education. After considerable discussion on the field and the work of the Committee on Adult Education, the report from the committee was accepted.

The Limnological Society of America was accepted as an *affiliated* society. (The society has a total membership of 238 members. Of this number 170 are members of the association, 82 of these being fellows. The society is entitled to one representative in the council of the association.)

The Alpha Chi Sigma Fraternity was accepted as an *associated* society. (This society has a total membership of 10,583. Of this number 1,232 are members of the association, 568 of these being fellows.)

The Chi Beta Phi Scientific Association was accepted as an *associated* society. (This society has a total membership of 1,449. Of this number 36 are members of the association, 31 of these being fellows.)

The permanent secretary reported for record the appointment of Dr. H. S. Jennings as representative of the association to the meeting of the British Association for the Advancement of Science, held in Norwich, September 4–11, 1935.

The permanent secretary reported for record the appointment of Dr. J. McKeen Cattell and Professor M. S. Vallarta, Massachusetts Institute of Technology, as representatives of the association to the seventh American Scientific Congress held in Mexico City, September 8–17, 1935.

The chairman of the executive committee and the general secretary were appointed a committee to consider the possible relations of the association with scientific organizations and workers in science in Mexico, and to present at a later meeting such recommendations as they feel should be made.

Dr. H. G. Moulton was elected a fellow of the association.

It was voted that the spring meeting of the executive committee in 1936 be held in Lancaster, Pa.

The permanent secretary presented a progress report on the organization of a Southeastern Division.

The permanent secretary reported for record the receipt of the annual report of the Joseph A. Holmes Safety Association.

The following resolutions from the American Pharmaceutical Association were read and placed on record:

Resolved, That the American Pharmaceutical Association desires to express its gratification to the American Association for the Advancement of Science for the creation of a Section on Pharmacy and for the opportunity to present a separate program illustrating the contributions of pharmacy to the advancement of science.

Resolved, That the American Pharmaceutical Association appoint two delegates to the Seventh American Scientific Congress to be held in Mexico City from the 8th to the 17th of September of this year and that those delegates be instructed to participate as fully as possible in all the efforts to make the Congress a success.

Two resolutions were presented from the Ecological Society of America on (1) preservation of natural areas and (2) program of the Quetico-Superior Council. The committee expressed its general approval and indicated that the substance of these resolutions was contained in resolutions previously adopted by the council.

The meeting adjourned, to meet at the Jefferson Hotel in Saint Louis.

HENRY B. WARD,
Permanent Secretary

SPECIAL ARTICLES

FURTHER EVIDENCE FOR THE PRESENCE OF A TOXIC FACTOR IN PERNICIOUS ANEMIA[1]

VERY recently, Mermod and Dock[2] reported their confirmation of Massa and Zolezzi's[3] finding that the intravenous injection of repeated doses of the dye, Congo Red, produces effects similar to those of liver

[1] From the department of physiology and pharmacology, University of Louisville School of Medicine. This investigation has been made with the assistance of a grant from the Committee on Therapeutic Research, Council on Pharmacy and Chemistry, American Medical Association.

[2] C. Mermod and W. Dock, SCIENCE, 82: 155, 1935.

[3] M. Massa and G. Zolezzi, *Klin. Wochnschr.*, 14: 235, 1935.

extract in patients with pernicious anemia. Mermod and Dock point out that this observation, together with the known effectiveness of Congo Red in neutralizing certain toxic substances, calls for a further exploration of the old theory that pernicious anemia is due to the presence of some toxic agent. During the past two years we have accumulated a certain amount of evidence for the presence of a toxic factor in pernicious anemia.

Sterile morning urine specimens obtained from eight untreated pernicious anemia patients (two males and six females) were injected intramuscularly into pigeons. Eight pigeons were used for each urine. Four of the birds received 0.1, 0.5, 1.0 and 1.5 cc of urine per 100 gm of pigeon respectively for five successive days. The other four pigeons were given corresponding amounts of urine previously heated to 100° C. for two hours. Daily reticulocyte counts were made for two weeks prior and for six weeks subsequent to the first injection. Each of the eight unheated urines produced a decrease in the reticulocytes of the pigeon below the significant level of 5 per cent. (Repeated daily counts on more than one hundred fifty control pigeons by a method already reported[4,5] have never shown a minimum reticulocyte percentage of less than 5.) The significant minimal counts varied from 0 to 4 per cent. with an average of 2 per cent. The peaks of the decreases were obtained on the 5th to the 8th days. The reticulocyte decreasing substance is thermolabile, inasmuch as none of the pigeons injected with the heat-treated urines showed a reduction in reticulocytes. Likewise, pigeons receiving corresponding doses of six normal human urines, unheated and heated, failed to show a reticulocyte decrease. The four urines containing the greater concentrations of the reticulocyte decreasing principle were definitely toxic, since 15 of the 21 birds injected with these urines died in from one to eight days following the first injection. Indeed one urine was so toxic that smaller doses (.025 and .05 cc) were necessary in order to demonstrate the reticulocyte decreasing effect. On the other hand, of the 32 pigeons injected with the heat-treated pernicious anemia urines which were without reticulocyte decreasing effect, all but one survived. Likewise the 48 pigeons receiving the normal unheated and heated human urines survived. The toxicity, therefore, is associated with the reticulocyte decreasing factor and is very probably due to it.

Following the primary decrease in reticulocytes, most of the surviving pigeons showed a subsequent reticulocytosis. This reticulocyte stimulating effect was partially retained by the heat-treated pernicious anemia urines. A detailed consideration of the significance of this reticulocyte stimulating substance previously reported to be present in normal human urine[4,5] is outside the scope of this progress note. However, the possibility of the identity or similarity of this second urinary substance and the anti-pernicious anemia principle is suggested by the reports of Decastello[6] and one of us.[7] Obviously more work is necessary, although these findings also throw doubt on the current deficiency theory as a complete explanation of the pathogenesis of pernicious anemia.

Thus far, we have examined the urines of two treated pernicious anemia patients and have found the toxic, reticulocyte decreasing substance absent. Interestingly enough, the concentration of the reticulocyte stimulating principle in these two urines was definitely decreased.

It is apparent, therefore, that urine from untreated patients with pernicious anemia contains both a thermolabile, comparatively toxic, reticulocyte decreasing factor and a partially thermostable, relatively nontoxic, reticulocyte stimulating principle for the pigeon. Normal human urine contains the latter but not the former, or at least not in the quantities of urine used. What relation the urinary reticulocyte decreasing principle bears to the toxic substances reported by Macht,[8] Mermod and Dock,[9] and Kingisepp[10] as present in the plasma of untreated pernicious anemia patients is largely speculative at present. If our impression that the principle acts through depressing erythrogenesis in the bone-marrow should prove to be correct, the production of experimental pernicious anemia in mammals by the separation and administration of sufficient quantities of the reticulocyte decreasing factor is not beyond the realm of possibility.

G. E. WAKERLIN
H. D. BRUNER

SOME OBSERVATIONS ON ULTRA-VIOLET IRRADIATED AMEBAS

PRELIMINARY to a study of the manner in which various saline solutions modify the action of ultra-violet light on protoplasm a number of exploratory experiments were done upon *Amoeba proteus*. In the course of these experiments certain results of more general interest were obtained.

(1) Changes in the prominent food vacuoles of approximately 15 amebas were observed during and

[4] G. E. Wakerlin, H. D. Bruner and J. M. Kinsman, *Proc. Am. Physiol. Soc.*, p. 136, 1935.
[5] G. E. Wakerlin and H. D. Bruner, *Arch. Int. Med.*, in press.
[6] A. Decastello, *Med. Klin.*, 31: 377, 1935.
[7] G. E. Wakerlin, *Proc. Soc. Exp. Biol. and Med.*, 32: 1607, 1935.
[8] D. I. Macht, *Jour. American Medical Association*, 89: 753, 1927.
[9] C. Mermod and W. Dock, *Proc. Soc. Exp. Biol. and Med.*, 32: 373, 1934.
[10] G. Kingisepp, *Klin. Wochnschr.*, 13: 1820, 1934.