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New Data on the Extraction of B_1 From Natural Material (Yeast)

Investigation was made to determine the most suitable conditions for the extraction of B_1 from yeast.

In a series of assays, using yeast from the same container and using the same enzyme (papain) but a different pH with each assay, it was found that optimum results were obtained at pH 1.0–1.5.

At the same time it was confirmed that synthetic B_1 is best conserved at pH 4.0–4.5.

For the checking of the B_1 the colorimetric method of Melnick and Field was used.

Further work is needed to determine whether the findings apply to the extraction of B_1 from other natural materials; also, on the use of different enzymes or a combination of enzymes.

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The Rh System in the Chimpanzee

The present writer's theory of multiple allelic genes, to account for the hereditary transmission of the Rh-Hr blood types, has received adequate confirmation from family and statistical studies (A. S. Wiener, E. B. Sonn, and H. R. Polivka. *Proc. Soc. exp. Biol. Med.*, 1946, **61**, 382). On the other hand, no substantial evidence has been adduced to support Fisher's theory of gene triplets, which only leads to contradictions and paradoxes (A. S. Wiener. *Brit. med. J.*, 1946, **1**, 982; J. Murray. *Brit. J. exp. Path.*, 1946, **27**, 102). A new argument for Fisher's theory has now been advanced in your columns by Mourant and Race (*Science*, 1946, **104**, 277).

M. Wade and I (*Science*, 1945, **102**, 177) reported that the bloods of every one of 15 chimpanzees tested did not absorb anti-Rh', anti-Rh'', or anti-Rh, agglutinins from human antisera, but did absorb anti-Hr'. This is confirmed by tests on a single additional chimpanzee by Mourant and Race, who also report that the blood of their chimpanzee did not absorb the anti-Hr'' agglutinin. Based on this finding, Mourant and Race conclude that the factors Rh'' and Hr'' are absent from chimpanzee blood and suggest that the hypothetical locus E-e of Fisher is lacking in this species. They consider this apparent separation of one gene pair from Fisher's three

sets of hypothetical genes an argument favoring Fisher's theory of closely linked genes, as against my multiple allele theory.

The reasoning used by Mourant and Race has a number of fallacies which can best be demonstrated by citing analogous observations involving other blood agglutinogens. Rhesus red cells are not clumped by, nor do they absorb, human anti-Rh, agglutinins, which, according to Mourant and Race, would indicate that the Rh, factor is entirely lacking in this species. However, the original antisera for detecting the Rh, factor were prepared by injecting Rhesus blood into rabbits and guinea pigs; in fact, that is how the Rh factor got its name. The correct conclusion is that Rhesus blood does not contain a factor identical with human Rh,—only a related factor, that is, an Rh₀-like factor. Similarly, it seems highly likely that chimpanzee blood actually does contain Rh''-like or Hr''-like factors, or both.

Another obvious fallacy is to assume that every separate agglutination reaction given by an antigen proves the presence of comparable separable components within the antigen. The agglutination test is merely a diagnostic test, and one might just as unreasonably conclude that every time a new qualitative test is devised for a chemical substance this proves the presence of another structure within its molecule. K. Landsteiner (*Specificity of serological reactions*. (Rev. ed.) Cambridge, Mass.: Harvard Univ. Press, 1945. Pp. 114–116) has repeatedly demonstrated how simple chemical compounds can give rise to several distinct but specific immune antibodies, and he has also demonstrated that the number of qualitatively different antibodies is not necessarily correlated with the existence of distinct structures within the antigen molecule. If we were to apply Mourant and Race's arguments in the case of the A-B-O blood groups and the M-N types, we would be faced with a number of queer paradoxes. Studies on the evolution of the M agglutinogen reveal the existence of at least four distinct partial antigens in the human M agglutinin and two partial antigens in the N agglutinin (A. S. Wiener. *Amer. Nat.*, 1943, **77**, 199). According to Fisher, it would therefore be necessary to postulate that agglutinin M of human blood is determined by a gene complex, $M_iM_{ii}M_{iii}M_{iv}$, while agglutinin N is determined by a linked gene complex, N_iN_{ii} . This leads to a situation where corresponding portions of a pair of homologous chromosomes are not homologous, and, if this conclusion were correct, it would be very strange that in millions of tests no evidence of crossing-over, such as a blood $M_iM_{ii}N_{ii}$, has ever been obtained. It seems much more reasonable to conclude that the complicated M and N agglutinogens of human blood are each determined by corresponding genes forming an allelic pair, in accordance with the generally accepted theory of Landsteiner and Levine. The reactions of the bloods of chimpanzees and monkeys with anti-M and anti-N sera can be explained most reasonably and simply by postulating the presence in these species of M-like and N-like agglutinogens rather than portions of a complicated gene complex; that is, the phenomena described are undoubtedly examples of the evolution of complicated chemical