Museum a chance to bid on it, disposed of the whole to the American Museum in New York. If it had to go abroad, there was certainly no place so suitable, and recent publications show how valuable it has been to American students. In the collection were the type specimens of a number of subspecies of birds peculiar to the British Islands, their description resulting from the critical (some would say hair-splitting) studies of recent times. The authorities of the American Museum, to the gratification of all concerned, presented these British specimens to the British Museum.

In spite of these causes of discontent, Lord Rothschild, who came to greatly regret having parted with the birds, wished to associate himself with the British Museum, and when he died, willed the whole institution to that Museum, to be kept up as a research center for zoologists. There was some discussion as to the acceptance of this gift, with its necessary obligations, but most zoologists warmly supported the proposal and the trustees took on the Tring Museum as a branch of the British Museum of Natural History. The advantages are many. There are the buildings and the great collections, with excellent facilities for work. The situation, distant from London, avoids the dreadful fogs of the metropolis, and (as was not then thought of) the danger from enemy bombs. It is possible, at Tring, to work in peace, without the innumerable interruptions which are unavoidable at the British Museum. Thus the place is a veritable haven of refuge and will undoubtedly contribute more and more to zoological science.

In Rothschild's day, it was found difficult or impossible to publish the numerous papers resulting from the work of the museum in the ordinary scientific journals. There was accordingly established a periodical called Novitates Zoologicae, which has now reached the forty-second volume. I am glad to see that the British Museum is keeping this going, and a bulky part of 180 pages, dated October, 1940, is now before me. It consists entirely of a monographic revision of the Mexican water beetles of the family Elmidae, by Dr. H. E. Hinton, of the British Museum. It must be admitted that not many of us are vitally interested in Mexican water beetles, as such, but the work is of wider value than its title would suggest. For example, Hinton has specially investigated the internal anatomy of his beetles, something an entomologist hardly ever does. He finds that the internal organs throw considerable light on taxonomic problems. Thus the genus *Cylloepus* is found to consist of two very distinct groups, which are treated as different genera. The beetles, without dissection, are easily separated by the presence or absence of a certain patch of tomentum, but ordinarily this would not be thought of as a generic character. The larvae are also described at considerable length, and the larval characters contribute to the system of classification. There is an interesting discussion of the problem of description. It is pointed out that it is impracticable and undesirable to enumerate all the characters of an insect, but at the other extreme, too short descriptions, even if they suffice to separate the insects from other known species, may fail utterly when numerous new species are discovered. Thus there has to be a middle-of-the-road policy, and the successful describer is he who knows his group, and can judge of the characters likely to prove important. The Mexican Elmidae have been so little known that all the species seen by Hinton have been described by two men only, 14 by Dr. David Sharp and 23 by Hinton. Many more undoubtedly remain to be discovered.

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ALCAPTONURIA IN A NEGRO FAMILY

ALCAPTONURIA is an outward manifestation of a very rare anomaly of protein metabolism in which homogentisic acid is excreted in the urine. This defect in body chemistry is hereditary and appears to be a rare recessive character in the Mendelian sense. Its mode of incidence is remarkably similar to that of albinism. Its presence, however, is not as strikingly apparent as is that condition. The error in metabolism is present from birth and persists throughout life.[1]

Comparatively few instances of alcaptonuria have been reported in this country. Of approximately 122 cases in the literature in 1929, only 17 occurred in the United States, the majority having been reported from European countries.[2] At the present time only 21 of the total number of recorded cases were reported in this country. As the condition is most often recognized by accident, undoubtedly more alcaptonurics exist than the records show.

Alcaptonuria in the American Negro has never been reported. We know that albinism exists among Negroes. Nothing, however, is known of the existence in this race of the other, less obvious, inborn errors of metabolism.

It is the purpose of this article to report the occurrence of alcaptonuria in two children of a Negro family. Fig. 1 is a diagram of the members of the family investigated. None but the two children was affected. The girl (D), 8 years old, was recognized during a routine examination in the out-patient clinic as a probable alcaptonuric from the atypical reduction of Benedict's solution given by her urine. On

[1] A. E. Garrod, "Inborn Errors of Metabolism," 2nd Edition, London, 1923.

[2] E. S. Bagnall, *New Eng. Jour. Med.*, 201: 422, 1929.

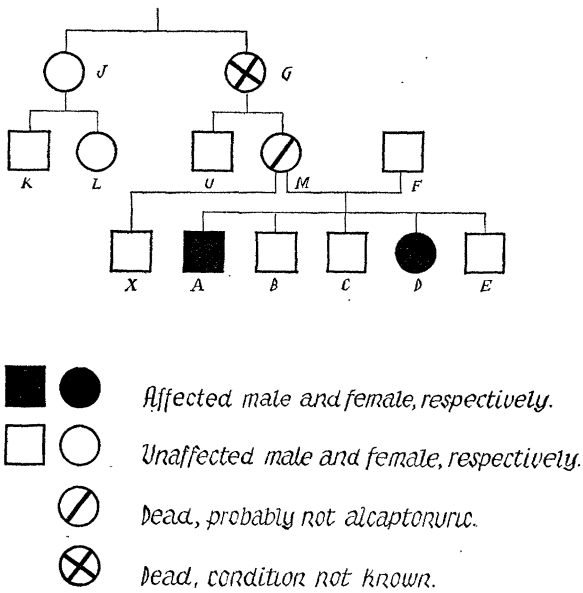

FIG. 1

examination, the urine was found to give the reactions characteristic of homogentisic acid: addition of 10 per cent. sodium hydroxide resulted in a brownish-black ring at the surface which gradually penetrated downward, ferric chloride caused a transitory blue color with each drop, and reduction of silver lactate and of ammoniacal silver nitrate proceeded rapidly at room temperature. The urine, as excreted, was amber, clear and acid. Quantitative analyses showed a concentration of homogentisic acid (Briggs's method[3]) in a random sample of 5 gm per liter with a homogentisic acid to nitrogen (H:N) ratio of 53.5:100. A larger sample, used for isolation of homogentisic acid, contained 4.4 gm per liter, and the H:N ratio was 45.1:100.

Investigation of the girl's four brothers revealed the fact that her eldest brother (A), 13 years old, also was excreting homogentisic acid. Qualitative tests were positive. The H:N ratio of a sample was 48.3:100. These ratios agree well with those given in the literature. The qualitative tests were confirmed by actual isolation of homogentisic acid from the lead salt obtained from the urine of both children.

The father (F) is not an alcaptonuric. Negative tests for urinary sugar on the mother (M), who died in the hospital in 1935, are considered evidence of the absence of any reducing substance in her urine. She probably was not alcaptonuric. The children's parents (F and M) were not related. The half-brother (X), 17 years old, the offspring of a previous marriage of the mother, is not an alcaptonuric. This would be expected if the error is a simple recessive character. It recalls the very interesting case of two alcaptonurics, brother and sister, whose parents later married others and had children, none of whom showed alcaptonuria.[4] The father's relatives are all in the Carolinas and thus unavailable for examination. The mother's brother (U) is not alcaptonuric. Unfortunately, the grandmother of the children (G) died one week before this study was undertaken. Whether or not she may have been alcaptonuric is not known. The grandmother's sister (J) and her children (K and L) are not alcaptonuric.

The two children (A) and (D) appear healthy and well nourished. They are typical American Negroes in every respect. That they have been alcaptonuric since infancy is attested by the fact that the father well recalls the staining of the bedding caused by the urine of these two children.

SUMMARY

A report of the occurrence of alcaptonuria in two children of a Negro family is presented. Other members of the family were investigated and found to be unaffected. This is the first evidence that this inborn error of metabolism exists in the American Negro.

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HUMAN GENE SYMBOLS

IN no field of science is there greater lack of uniformity with respect to the usage of symbols and greater lack of adherence to conventional genetic rules than in the field of human genetics. For instance one finds in recent human genetic literature the statement, "the three allelomorphic blood group genes A, B, and R," and the statement, "the blood group alleles M and N." Such usages lack logic and clarity and are not in accordance with generally accepted genetic rules.

For a long time it has been the custom in dealing with the genetics of lower forms to give all members of a given allelomorphic series the same basic symbol and to distinguish one member of a series from another by capitalization or by a superscript. For instance, the piebald alleles of the house mouse are designated as S and s, and the albino allelomorphic series of guinea pig coat color genes are designated as C, c^k, c^d, c^r, c^a. These same rules should be applied to all human alleles.

To indicate the genes responsible for the presence or absence of isoagglutinogens A and B and for the M and N agglutinogens the writer has used the following symbols: I^A—isoagglutinogen A; I^B—isoagglutinogen B; i—absence of A or B; A^m—agglutinogen M; A^n—agglutinogen N.

[3] A. P. Briggs, *Jour. Biol. Chem.*, 51: 453, 1922.

[4] A. E. Garrod, *Lancet*, 2: 1616, 1902.