despite the smaller number of observations, the continuity of the light curves has been well maintained.

Cyrus F. Fernald, of Wilton, Maine, again heads the list, with a total of 4,206 observations. He observed on 143 nights, and reports having spent 207 actual hours at observing, an average of twenty stars per hour. This is a remarkable record, and well attests the value of having a finely mounted and properly adjusted telescope—an 8-inch Springfield reflector—combined with considerable experience and plenty of enthusiasm. How much time Mr. Fernald spends in listing his reports would be of interest.

In the 2,100 to 2,300 class are Holt, of Tucson, Ariz.; deKock, of South Africa; Cilley, of Lewisburg, W. Va.; and Peltier, of Delphos, Ohio. In the 1,100 to 1,800 class are Mrs. Kearons, and Messrs. Hartmann, Jones and Chandra.

Nine observers made between 500 and 1,000 estimates each, and eleven, between 200 and 500. These twenty-nine observers made 87 per cent. of all the observations, but the other 61 contributors, 13 per cent., have added materially to the cause.

Special mention should be made of the interest shown by our Canadian observers. We now have five active contributors from that country, with a total of 914 observations for the year. Our South African observers contributed 4,179 observations; from India came 1,602, and from Mexico, 952. Australia, Argentina and Japan contributed 372, 96 and 87, respectively. Our 74 American observers accumulated a total of 24,888 observations.

The eleven observers of the Milwaukee Astronomical Society contributed 1,518 observations; the three from the Fall River, Mass., group, 2,630; and the three in Portland, Maine, 431.

Personnel: Mrs. Helen S. Federer has acted as Pickering Memorial assistant throughout the year. She has acted as custodian of the records, plotting the observations and picking up discrepancies when they occurred. She has also looked after the correspondence and mailing out of *Bulletins, Annals* and so forth, thus allowing the recorder to spend much of his time on the discussion of the variables.

When the million mark will be attained is still a question, but to date the American Association of Variable Star Observers has reached a grand total of 880,000 observations in the 31 years since it began its work. We must not permit the variables to go unobserved, even in these war-torn times; an ever-continuing history of the activities of our variables must be maintained, in so far as it is possible. But first and foremost must come the winning of this war, and the sooner the better for civilization and for science.

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SPECIAL ARTICLES

PIMELIC ACID, BIOTIN AND CERTAIN FUNGI

EVIDENCE that pimelic acid is utilized by the diphtheria bacillus for the synthesis of biotin has been presented by du Vigneaud, Dittmer, Hague and Long.[1] Eakin and Eakin[2] report that the synthesis of biotin by *Aspergillus niger* was increased by the addition to the medium of pimelic acid, and the effect was enhanced by cysteine or cystine. However, du Vigneaud and associates found that pimelic acid did not replace biotin in its growth-stimulating effect on yeast. We have attempted without success to replace biotin with pimelic acid for thirteen fungi which suffer from a biotin deficiency.

The following organisms were used in one series of experiments: *Ceratostomella ips* #255, *C. ips* #438, *C. microspora, C. montium, C. obscura, C. penicillata, C. pini, C. radicicola, Grosmannia serpens, Fusarium avenaceum, Neurospora sitophila* 56.2 and *N. tetraspora* S_1. None of these fungi makes more than slight growth on a mineral-dextrose medium containing asparagine and purified agar unless biotin is present. The addition of 0.05 μ g of biotin to a tube containing 8 ml of the basal medium permits luxuriant growth.[3]

Negative results were obtained when the 0.05 μ g of biotin was replaced with 0.05 μ g of pimelic acid. No benefit was observed when the quantity of pimelic acid was increased to 0.1 μ g per tube containing 8 ml of medium.

Sulfur is furnished in our basal medium as $MgSO_4$. The medium used for the cultivation of the diphtheria bacillus contained *l*-cystine. Eakin and Eakin found that cysteine or cystine markedly increased the formation of biotin by *Aspergillus niger* in the presence of pimelic acid. However, none of the twelve fungi listed above grew when 0.1 μ g of pimelic acid and 1 mg of *l*-cystine, 0.1 μ g of pimelic acid and 1 mg of glutathione or 0.1 μ g of pimelic acid and 1 mg of methionine were added to the basal medium instead of biotin. Excellent growth was obtained when the pimelic acid in the above media was replaced by

[1] Vincent du Vigneaud, Karl Dittmer, Eleanor Hague and Barbara Long, SCIENCE, 96: 186, 187, 1942.

[2] Robert E. Eakin and Esther A. Eakin, SCIENCE, 96: 187, 188, 1942.

[3] Some of these fungi must be supplied also with thiamine or pyridoxine or with both vitamins in addition to biotin.

0.1 μ g of biotin methyl ester, which shows that the failure to grow in the tubes containing the pimelic acid was because the pimelic acid did not replace biotin, not because the medium was injurious; du Vigneaud and associates obtained maximum growth of the diphtheria bacillus under their experimental conditions with 1.5 μ g of pimelic acid, and a marked effect with 0.05 or 0.1 μ g. Eakin and Eakin, however, used as much as 1 mg of pimelic acid per culture, and report that 20 μ g per 12 ml culture gave maximum results. We obtained no growth from the addition per tube of the basal medium 0.1 mg of pimelic acid and 1 mg of *l*-cystine, or 1 mg of pimelic acid and 1 mg of cystine. Growth was obtained when 0.1 μ g of biotin was added to these media, demonstrating that lack of growth in the media containing pimelic acid was because of insufficient biotin, or physiologically equivalent substances, and not because of too much pimelic acid.

Ashbya (*Nematospora*) *gossypii*, the organism used by Kögl as a means of bioassay in the original isolation of biotin, was also tested.[4] Negative results were obtained when 1 μ g or 100 μ g of pimelic acid were added in place of biotin to 8 ml of a basal medium and when biotin was replaced with 1 μ g or 100 μ g of pimelic acid together with 1 mg of *l*-cystine. The results were negative also when the medium containing pimelic acid or pimelic acid and *l*-cystine was further supplemented with 1.5 mg of casein hydrolysate per tube.

It appears that the thirteen fungi we used are not able to synthesize biotin from pimelic acid, or from pimelic acid and *l*-cystine, under our experimental conditions. This should not be interpreted to mean that other organisms can not construct biotin from pimelic acid and *l*-cystine, nor that pimelic acid is not a precursor of biotin. The relation of microorganisms to thiamine and its thiazole and pyrimidine intermediates have demonstrated that some organisms have no synthetic power for thiamine and require it in molecular form; others have incomplete synthetic power and can construct the vitamin if furnished the proper intermediates, but not otherwise; while still others are able to make thiamine from the minerals and sugar in a basal medium. A somewhat similar situation may exist with regard to biotin. If it does, the fungi we have used appear to require biotin as such.

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[4] *Ashbya* was grown on a modification of the medium used by Kögl and Fries which contains thiamine and *i*-inositol.

THE RH FACTOR AND RACIAL ORIGINS[1]

IN 1940 a new factor (Rh) in human blood was described[2] which is present in the blood cells of about 85 per cent. of white individuals (Rh-positive type). This blood property was found to be inherited as a simple mendelian dominant by a pair of allelic genes, *Rh* and *rh*.[3] Investigations on the distribution of the Rh factor among Negroes in New York City revealed a somewhat lower incidence of the Rh-negative type, while in full-blooded American Indians the Rh-negative type appears to be practically absent.[4] The extension of these studies to other races should yield results of significance from the standpoint of racial origins.

To account for the present distributions of the Rh factor in white individuals and in American Indians, a number of hypotheses could be considered, analogous to those proposed to explain the distribution of the four blood groups. Two main possibilities will be discussed: (1) That man was originally Rh-positive and that the present incidence of the Rh-negative type resulted from mutations from gene *Rh* to gene *rh*. While this might conceivably account for the exceptional occurrence of Rh-negative individuals among American Indians, to explain the higher incidence of the *rh* gene (almost 40 per cent.) in white individuals one would have to postulate an improbably high rate of mutation. (2) Another possibility is that there were originally two or more races, some predominately or exclusively Rh-positive, others Rh-negative, and that by crossing the present distribution of the Rh factor resulted.

Of significance with regard to this problem is the relationship that has been demonstrated by Levine *et al.* between the Rh factor and erythroblastosis foetalis, a disease responsible for a certain number of stillbirths and neonatal deaths.[5, 6, 7, 8, 9] In the typical case, the mother is Rh-negative, the father Rh-positive and the fetus Rh-positive, the latter having inherited the Rh factor from the father. Due presumably to some defect in the placenta, fetal blood es-

[1] From the Serological Laboratory of the Office of the Chief Medical Examiner of New York City. Aided by a grant from the Carnegie Foundation and the Committee on Human Heredity of the National Research Council.

[2] K. Landsteiner and A. S. Wiener, *Proc. Soc. Exp. Biol. and Med.*, 43: 223, 1940.

[3] K. Landsteiner and A. S. Wiener, *Jour. Exp. Med.*, 74: 309, 1941.

[4] K. Landsteiner, A. S. Wiener and G. A. Matson, *Jour. Exp. Med.*, 76: 73, 1942.

[5] P. Levine, E. M. Katzin and L. Burnham, *Jour. Am. Med. Assoc.*, 116: 825, 1941.

[6] P. Levine, P. Vogel, E. M. Katzin and L. Burnham, SCIENCE, 94: 371, 1941.

[7] P. Levine, L. Burnham, E. M. Katzin and P. Vogel, *Am. Jour. Obstet. and Gynec.*, 42: 925, 1941.

[8] L. Burnham, *Am. Jour. Obstet. and Gynec.*, 42: 389, 1941.

[9] A. S. Wiener, *Am. Jour. Clin. Path.*, 12: 302, 1942.