results with the mouse[1] it would appear that repeated recrystallization of inositol does not materially alter the anti-alopecia properties of inositol.

Summary. In the rat fed a ration of naturally occurring feeds inositol supplementation gave a growth response and it also cured and prevented a widespread loss of hair. When a folic acid preparation, together with inositol and B_6 was fed a better growth rate was obtained than when the folic acid preparation was omitted.

These data clearly show that a natural ration composed of yellow corn, soybean oil meal, $CaHPO_4$, $CaCO_3$, $MnSO_4$, NaCl, and 5% alfalfa hay contains insufficient inositol. folic acid concentrate and B_6 for normal growth and proper hair and fur development in the rat. It would appear that the phytin present in this ration was either inadequate to supply the required inositol or that it was unavailable.

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Heredity and Distribution of the Rh Blood Types.*

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In a preceding paper[1] a theory of 6 allelic genes was proposed to account for the existence of variants within the Rh-positive type of human blood. The present communication is a preliminary report of investigations on the Rh blood types in families, and their distribution in different races, these studies having been carried out in order to test the 6-gene theory.

In Table I is presented, in simplified form, the 8 Rh blood types and their reactions with the 3 sorts of anti-Rh agglutinins, namely, anti-Rh (standard, and agglutinating the bloods of approximately 85% of white individuals), anti-Rh_1 (giving about 70% positive reactions with bloods of white individuals), and anti-Rh_2 (giving about 35% positive reactions). Among white individuals approximately 95% fall into one of the 4 types listed in the left half of the table, while the 4 types in the right half of the table together make up only 5% of the population. For analyzing data, it is sometimes convenient to disregard the reactions of the standard anti-Rh sera, and in that way the two halves of the table are fused, giving rise to 4 classes, comparable serologically and genetically to the 4 blood groups. Obviously, each class consists of 2 distinct Rh types.

In Table II are summarized our findings to date on the heredity of the Rh blood types in 94 families (83 white, 11 negro). It will be seen that among the 274 children there is only a single apparent exception to the theory. In this case (a negro family) the mother was Rh-negative, her husband Rh′, and the child Rh_2. However, it was also found that the supposed father belonged to type N and the child to type M, so that this was evidently an illegitimate child. Of particular interest are the 4 matings Rh-negative $\times$ Rh_1Rh_2 in which there were 8 Rh_1 and 8 Rh_2 children. These findings are entirely comparable to those obtained in the mating O $\times$ AB in studies on the heredity of the Landsteiner blood groups. In addition to the 94 complete families, tests were carried out on mother-child combinations, and among 73 mothers with 123 children, not once were the combinations Rh-negative mother with type Rh_1Rh_2 child or type Rh_1Rh_2 mother with Rh-negative child encountered. The findings will be discussed in greater detail in the full report[2] which will also contain the complete family

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1 Wiener, A. S., PROC. SOC. EXP. BIOL. AND MED., in press.

2 Wiener, A. S., Sonn, E. B., and Belkin, R. B., in preparation.

TABLE I.
Classification of the Rh Blood Types.

Class	Testing sera: Anti-Rh_1	Anti-Rh_2	Anti-Rh	Type	Testing sera: Anti-Rh_1	Anti-Rh_2	Anti-Rh	Type
U	+	—	+	Rh_1	+	—	—	Rh′
V	—	+	+	Rh_2	—	+	—	Rh″
UV	+	+	+	Rh_1Rh_2	+	+	—	Rh′Rh″
W	—	—	—	Neg.	—	—	+	Rh

TABLE II.
Heredity of the Rh Blood Types in 94 Families.

Mating	No. of families	Children of types: Neg.	Rh_1	Rh_2	Rh_1Rh_2	Rh	Rh′	Totals
Neg. × Neg.	2	9	—	—	—	—	—	9
Neg. × Rh_1	17	13	36	—	—	3	—	52
Neg. × Rh_2	5	1	—	6	—	—	—	7
Neg. × Rh_1Rh_2	4	—	8	8	—	—	—	16
Neg. × Rh	1	3	—	—	—	1	—	4
Neg. × Rh′	1	—	—	(1)	—	—	—	1
Rh_1 × Rh_1	15	4	44	—	—	4	1	53
Rh_1 × Rh_2	15	3	13	7	13	—	1	37
Rh_1 × Rh_1Rh_2	19	—	27	6	22	—	—	55
Rh_2 × Rh_2	1	1	—	1	—	—	—	2
Rh_2 × Rh_1Rh_2	4	—	1	5	3	—	—	9
Rh_2 × Rh	2	—	—	1	—	4	—	5
Rh_1Rh_2 × Rh_1Rh_2	7	—	4	—	17	—	—	21
Rh′ × Rh_1Rh_2	1	—	1	1	1	—	—	3
Totals	94	34	134	36	56	12	2	274

TABLE III.
Racial Distribution of the Rh Blood Types.

Race	No. tested		Neg.	Rh_1	Rh_2	Rh_1Rh_2	Rh	Rh′	Rh″	Rh′Rh″
White	500	Approx. %	12.75	50	15.5	17	2.5	2	0.25	—*
Negroes‡	143†	No.	13	31	30	15	51	1	—	—
		%	9.1	21.9	21.0	10.5	35.7	0.7	—	—
Chinese§	80	No.	1	46	4	28	1	—	—	—
		%	1.25	57.5	5.0	35.0	1.25	—	—	—

* This theoretically possible type has not yet been encountered, which is not surprising considering the extremely low value for the expected frequency of the type.
† Two bloods gave atypical reactions.
‡ Wiener, A. S., Belkin, R. B., and Sonn, E. B., detailed paper in preparation.
§ Wiener, A. S., Sonn, E. B., and Yi, C. L., detailed paper in preparation.

data, including the blood groups, sub-groups, and M-N types.

Studies on the distribution of the Rh blood types among whites, negroes, and Chinese revealed very striking differences among these races. These findings are in sharp contrast to the results of the blood grouping and M-N tests for the same races, since, for example, the distributions of the M-N types among whites, negroes and Chinese do not differ greatly. However, we did observe that all the group A and AB individuals among the Chinese belong to sub-group A_1, so that sub-group A_2 is apparently lacking in this race.

In Table III we have tabulated our findings on the distribution of the Rh blood types among negroes and Chinese as compared with their approximate distribution among white individuals (the latter being based on a series of more than 500 persons). It is of interest to note the very high incidence of the type Rh among negroes, in contrast to its low incidence in whites and Chinese. On the other hand, among Chinese the Rh-negative type is

practically lacking[3] while the frequencies of types Rh_1 and Rh_1Rh_2 are high. In fact, if the single Rh-negative individual and the single individual of type Rh are discounted as possibly due to racial admixture, then the distribution in our series of Chinese reduces itself to a two-factor scheme, quite similar, for example, to the distribution of the M-N types among American Indians.

The application of simplified gene frequency formulæ calculated from the frequencies of classes U, V, UV, and W supplies further support for the hypothesis of multiple allelic genes. However, the family and statistical data are still inadequate to test the theory satisfactorily with regard to the rarer types, Rh′, Rh″, and Rh′Rh″.

3 Levine and Wong found only 0.7% Rh-negative individuals among 150 Chinese (*Am. J. Obst. and Gyn.*, 1943, **45**, 832).

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Comparison of Dextran Synthesis by Leuconostoc Enzyme with Starch Synthesis by Potato Phosphorylase.*

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Earlier papers[1,2] described the synthesis from sucrose of a serologically reactive dextran by cell-free enzymes obtained from a species of streptococcus-like bacteria, *Leuconostoc mesenteroides*. Some evidence was obtained which indicated that the course of the synthesis follows the equation:

$$n \text{ sucrose} \rightarrow \underset{\textit{dextran}}{(\text{glucose anhydride})_n} + n \text{ fructose} \quad (1)$$

However, that mode of dextran formation was not proved and, indeed, analogy with the enzymatic syntheses of glycogen and starch would suggest the possibility that glucose-l-phosphate might participate as an intermediate substance in the conversion of sucrose to dextran. That possibility was made more significant by a recent report[3] that suspensions of *Leuconostoc mesenteroides* can phosphorolyse sucrose with the production of glucose-l-phosphate. If glucose-l-phosphate does serve as an intermediate substance in the formation of dextran, the synthesis of that polysaccharide by the bacterial enzyme would fall in the same class of reactions as the syntheses of glycogen and starch by the phosphorylases of other biological origin.

In order to get experimental data on this question, a comparison was made between leuconostoc enzyme and potato phosphorylase in respect to their action upon sucrose and upon glucose-l-phosphate. The leuconostoc enzyme was obtained from sucrose broth cultures of the strain used in previous studies; it was prepared by an improved method which will be described in another paper, and was of much greater potency than any of the preparations utilized in the earlier studies. The potato phosphorylase was prepared according to the first steps in the procedure described by Green and Stumpf.[4]

A set of 3 test mixtures was prepared with each enzyme. The first consisted of equal parts of enzyme solution and of 0.2 M sucrose in 0.1 M acetate buffer, pH 5.6; the second

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1 Hehre, E. J., *Science*, 1941, **93**, 237.

2 Hehre, E. J., and Sugg, J. Y., *J. Exp. Med.*, 1942, **75**, 339.

3 Kagan, B. O., Latker, S. N., and Zfasman, E. M., *Biokhimiya*, 1942, **7**, 93.

4 Green, D. E., and Stumpf, P. K., *J. Biol. Chem.*, 1942, **142**, 355.