sperms, is the product of a sexual fusion in which two maternal nuclei combine with one pollen nucleus. Consequently, it is triploid in its chromosomal constitution. This has been repeatedly demonstrated, both cytologically and genetically. The triploid nature of the endosperm makes it possible to produce four classes of seed differing in the proportion of dominant and recessive genes. In the case of the factor for yellow pigmentation, the cells of the endosperm may have 0, 1, 2 or 3 genes for yellow pigment with the corresponding factorial composition *y y y*, *y y Y*, *y Y Y* or *Y Y Y*. These four classes differ in color and may be described roughly as white, pale yellow, dilute yellow and deep yellow. If all types occur on the same ears it is often difficult to make an accurate classification, especially between the two intermediate classes. By making appropriate pollinations, however, they can be separated with a high degree of accuracy.

In 1928 pollinations were made to produce these four classes of seeds. Surcropper, a white-seeded variety, and Ferguson's Yellow Dent, a yellow-seeded variety, were pollinated by a mixture of pollen from both sorts. On the ears of the white-seeded variety two types of seeds were produced, white and pale yellow, having, respectively, 0 and 1 factors for yellow endosperm. On the ears of the yellow seeded variety two additional types, dilute yellow and deep yellow, were produced, having, respectively, 2 and 3 factors for yellow endosperm.

The vitamin assay of these four classes was made by feeding to albino rats according to the Sherman-Munsell unit method.[3] The results in 1928 showed a high degree of association between the number of genes for yellow pigment and the number of units of vitamin A per gram of material. The pollinations and vitamin estimates were repeated in 1929 with corn grown under different seasonal conditions. Again, almost complete association was shown. The average results for two years are shown in the following table:

No. of genes for yellow	Factorial composition of endosperm	Units of vitamin A per gram		
		1928	1929	Average
0	*y y y*	0.05	0.05	0.05
1	*y y Y*	2.50	2.00	2.25
2	*y Y Y*	5.00	5.00	5.00
3	*Y Y Y*	7.00	8.00	7.50

These results show, first, that a white-seeded variety of corn, which ordinarily has little or no vitamin A in the endosperm, is capable of forming this substance in its seeds if the gene for yellow pigmentation is introduced. The white and pale yellow seeds were produced by the same plants, the only difference between them being in the microscopic pollen nuclei which entered into the fusion to produce the endosperm.

The next point of interest is that there is a direct quantitative relationship between the number of genes for pigmentation in the cells of the endosperm and the amount of vitamin A in the seed. Each gene for yellow induces the formation of approximately 2.5 units of vitamin A per gram of seed. The obvious conclusion must be that the gene for yellow pigmentation is responsible for the formation of vitamin A, either directly, or indirectly, with the production of carotinoid pigments as an intermediate step.

Finally, these results may have some bearing on the chemical nature of this particular gene. So far as we are aware, this is the first case in which it has been possible to establish, with any degree of exactness, a direct quantitative relationship between different doses of the same gene and their chemical effect. Although even this tells us little of what the gene may be, it does, perhaps, furnish some indirect evidence of what the gene is not. It seems scarcely probable, for example, that this gene functions as an enzyme, since the total reaction resulting from enzyme activity is seldom closely related to the concentration of the enzyme, while each gene for yellow pigment seems to govern the formation of a definite quantity of vitamin A. It is true that the rate of a reaction in which an enzyme is involved varies with the concentration of the enzyme, but the proportion is usually not a direct one like that shown here.

The straight-line relationship between the number of genes for yellow pigmentation and amounts of vitamin A is more indicative of a direct chemical reaction between the gene and some other substance which is present in the cells of the endosperm of both white-seeded and yellow-seeded varieties of corn.

P. C. MANGELSDORF
G. S. FRAPS
TEXAS AGRICULTURAL EXPERIMENT STATION

[3] H. C. Sherman and H. E. Munsell, *J. Am. Chem. Soc.*, 47: 1639.

A DIFFERENTIATION OF THE SO-CALLED ANTIPELLAGRIC FACTOR, VITAMIN G[1]

IN February of 1926 Goldberger and coworkers[2] and Smith and Hendrick[3] have demonstrated the dual nature of vitamin B. In May of the same year Goldberger and Lillie[4] submitted evidence that a deficiency of the stable factor in a diet fortified with an abun-

[1] Research Paper No. 142, Journal Series, University of Arkansas.

[2] Goldberger, Wheeler, Lillie and Rogers, Pub. Health Rep., 1926, 41: 297–318.

[3] Smith and Hendrick, *ibid.*, 1926, 41: 201–207.

[4] Goldberger and Lillie, *ibid.*, 1926, 41: 1025–1030.

dance of the labile, antineuritic substance resulted in a pellagra-like disease in the rat. Following the arrest of growth, alopecia and bilateral symmetrical lesions of the skin were the most noteworthy symptoms noted. Although the work of Goldberger and associates has in the main been substantiated in this country and England, Salmon, Hays and Guerrant,[5] and Chick and Roscoe[6] have suggested the complicity of the antipellagric dietary essential, since occasionally animals deprived of this syndrome remain stunted in growth but exhibit no skin lesions.

During the past two years, while engaged in studies of the biochemistry and pathology[7] of the pellagra-like avitaminosis in the albino rat, we have had occasion to observe various manifestations of the so-called vitamin G deficiency in 125 animals. The disease was produced by one of us (B. S.) on a dietary régime described elsewhere.[8] The optimum ration for the production of the dermatitis was found to be one deficient in the vitamin B complex, supplemented by a daily allowance of 500 mg of rice polishings, irradiated for 10 hours, according to the suggestion of Hogan and Hunter,[9] in order to insure the destruction of the greater portion of vitamin G. To summarize our results of 1929, the majority of the animals in which pellagra-like symptoms were produced showed dermatitis 20 to 50 days preceding the cessation of growth. On the other hand, our experience with other vitamin G deficient diets has been that the majority of animals failed in growth markedly without any accompanying skin lesions; and we would like to point out in this connection that all our rations were amply fortified with the antineuritic, growth-promoting factor. We have repeated our experiments of 1929 and have corroborated our former findings that there is no relation between failure in growth and the incidence of pellagra-like symptoms in the rat, the dermatitis being prevalent in some animals that make normal growth and absent in others that are first stunted in growth for weeks and months, and that finally collapse following great losses of weight. Recently we found accentuated dermatitis in six positive controls, out of 12 studied, accompanying excellent growth, the males having attained a weight of 240 to 270 grams and the females a weight of about 200 grams. The rations of the positive controls contained 10 per cent. autoclaved yeast as a source of vitamin G, and irradiated rice polishings as a source of vitamin B. Since we autoclave our yeast (Northwestern) at 20 pounds pressure for 6 hours, it is quite possible that, under our conditions, we are destroying the greater portion of the antidermatitis factor, at the same time not injuring the relatively stable growth-promoting factor. We, therefore, conclude that the so-called vitamin G is composed of two dietary essentials: one the deficiency of which produces pellagra-like symptoms in the rat; and another the deficiency of which produces a decline of growth.

Since the nomenclature of the so-called antipellagric factor is still in a state of confusion, the English investigators calling it vitamin B_2, while the American biochemists refer to it as vitamin G, the letter F of the vitamin alphabet having been left unrepresented,[10] we suggest a logical home for the letter F and have it indicate the stable growth-promoting factor associated with the vitamin B complex, and that we retain the letter G for the antipellagric factor, the deficiency of which produces the characteristic skin lesions in the rat.

BARNETT SURE
MARGARET ELIZABETH SMITH
M. C. KIK
DEPARTMENTS OF AGRICULTURAL CHEMISTRY AND HOME ECONOMICS,
UNIVERSITY OF ARKANSAS

LIVER EXTRACT AS A SOURCE OF VITAMINS B AND G[1]

SOME of the early investigations of the distribution of vitamins showed that the livers of animals were a good source of vitamin B (vitamin B + G). More recently liver and liver extracts have been widely used in the treatment of pernicious anemia.

Curtis and Newburgh[2] last year reported some feeding tests with liver extract. They found that when 2 per cent. of liver extract (Lilly) was added to a basic diet containing no other source of vitamin B, growth and energy ingestion were approximately normal; the replacement of liver extract by equal amounts of yeast vitamin powder (Harris) caused less growth and less energy ingestion. If the liver extract was autoclaved before feeding, all the experimental animals developed polyneuritis. These tests indicated that liver extract was a good source of both vitamins B and G but gave no information regarding the relative richness of the substance in the separate factors.

[5] Salmon, Hays and Guerrant, *J. Inf. Dis.*, 1928, 43: 426–441.
[6] Chick and Roscoe, *Biochem. J.*, 1927, 21: 698.
[7] Thatcher and Sure, Arch. of Path., in press.
[8] Thatcher, Sure and Walker, *So. Med. J.*, 1930, 23: 143.
[9] Hogan and Hunter, *J. Biol. Chem.*, 1928, 78: 433–445.
[10] Report of Committee on Nomenclature of American Society of Biological Chemists, SCIENCE, 1929, 69: 276.
[1] Read before the Biological Section, Alabama Academy of Science, Auburn, Alabama, April 18, 1930.
[2] A. C. Curtis and L. H. Newburgh, "The Effect of Liver and Liver Extract upon Appetite," *Jour. Clin. Inv.*, 7: 518, 1929.