sumed that muscular tissue represents 50% of the live weight of the rat and that glycogen is uniformly distributed, it can be calculated from the above data that the normal rats deposited as muscle glycogen 20.8% of the absorbed glucose while the hypophysectomized rats deposited as muscle glycogen only 11.3% of the absorbed glucose. While the assumptions involved in this method of calculation of muscle glycogen increases may be open to question in the hypophysectomized rat, it appears probable that hypophysectomized rats form proportionately less glycogen from the absorbed glucose than do normal animals.

8588 P

Carbohydrate Levels in Fasted and Fed Hypophysectomized Rats.*

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In an inquiry as to the nature of the hypoglycemia and low glycogen stores found in completely hypophysectomized rats, the following experiments have been undertaken: (1) the comparison under standard conditions of the carbohydrate levels of normal, hypophysectomized† and partially hypophysectomized rats when fully fed and when fasted 8 and 18 hours; and (2) the preparation of curves showing the changes in carbohydrate levels in these animals following the feeding of uniform carbohydrate meals.

All rats used were young males 50-70 days of age, the experimental animals 20-30 days post operative. Sodium amytal was used as anesthetic for obtaining samples. Muscle glycogen figures for each animal were the averages of 2 separate determinations, and the liver glycogen values included only fermentable reducing substances. The carbohydrate meals were given in the form of cornstarch, in 50% suspension in water, fed by stomach tube without anesthesia.

It was found that when the hypophysectomized animals were

* Aided by the Presnell Fund for the study of the metabolic relations of the anterior hypophysis contributed by Robert R. Presnell, Frank Tuttle, Arthur Stebbins and Mrs. Gordon Kahn, of Los Angeles.

† The parapharyngeal approach was used and the completeness of the operation was checked at autopsy in all cases.

fully fed (taken directly from their cages) all carbohydrate levels were within normal limits, but that when they were fasted, these levels fell rapidly, as shown in Table I. Each figure in this table represents the decrease in carbohydrate content as per cent of the average levels found in the fully fed animals.‡

TABLE I.

Decrease in Carbohydrate Levels During Fasting of Normal (N) and Hypophysectomized (H) Rats.

	8 hr. Fast		18 hr. Fast	
	N	H	N	H
Liver Glycogen	27	95	96	99
Muscle "	8	24	6	41
Blood Glucose	20	49	32	54

Values for similar series of operated controls, in which one-third or more of the anterior pituitary remained *in situ* and including several cases with various degrees of brain injury, did not differ from the normal; they are not included in this table.

That chronic inanition did not produce similar results was shown by the fact that normal rats kept on the same amounts of daily rations as were consumed by the hypophysectomized rats had fasting carbohydrate levels within normal limits.

When 1 gm. of starch was fed to rats fasted 18 hours, it was found 4 hours later that none of the levels in the completely hypophysectomized rats were as high as those in the normal animals so treated. When, however, an additional gram of starch was fed 4 hours following the first, thus providing enough carbohydrate and allowing sufficient time to overcome the initial fasting deficits, muscle glycogen and blood glucose values were easily brought to normal; but liver glycogen values even in this case increased much more slowly. This failure to form liver glycogen in the usual amounts from ingested carbohydrate may be explained in large part by low absorption rates, the rates of digestion and absorption of starch, as well as of glucose having been determined to be about 30% below normal in hypophysectomized rats. Calculation§ of the approximate increase in body carbohydrates due to feeding equal amounts of starch shows that the figures obtained in these series do not by themselves indicate the participation of other factors than

‡ Six to twelve animals were used in every group. The average coefficient of variation (standard deviation in % of the mean) in all groups was for blood glucose 12%, for muscle glycogen 13% and for liver glycogen 50%; the differences between the average values for normal and hypophysectomized animals were significant in all fasted series.

§ Based on averages for groups of 8-13 animals in each case.

those mentioned above; but their operation may not be ruled out. If, as Fisher and Pencharz[1] have found, a larger proportion of carbohydrate is oxidized by hypophysectomized rats following glucose feeding, this process also would be expected to decrease the amounts of carbohydrate stored in the liver under these conditions.

Although normal high carbohydrate levels in blood and muscle and moderately high liver glycogen values (maximum average 49% of normal) were obtained by feeding standard carbohydrate meals, these levels differed from the normal in that they were not maintained for any length of time, falling even while absorption was still continuing. The levels were followed for 36 hours after the initial feedings of one gm. of starch to normal and 1 and 2 gm. to hypophysectomized rats, determinations being made at 4, 8, 12, 24 and 36 hours, and 6-10 animals being used for each point on the several curves; the results amply confirm the conclusion previously drawn from work on fasted animals that hypophysectomized rats lose body carbohydrates at a much greater rate than do normal animals.

8589 C

Cataract- and Dermatitis-Producing Nutritional Factors.

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Cataract has been produced experimentally by parathyroidectomy, pancreatectomy, vitamin G-low diets,[1] lactose-rich[2] and galactose-containing[3] diets. In human subjects dinitrophenol has been reported as likewise cataract-producing. The difficulty of reconciling these diverse precipitating conditions is great, and in fact may not be possible. However, it is interesting to speculate upon the possible interrelation of the 2 obviously nutritional factors, vitamin G (B_2) and lactose.

The delay in, or interference with, complete absorption accompanying the presence of large amounts of lactose or galactose in the

[1] Fisher and Pencharz, PROC. SOC. EXP. BIOL. AND MED., 1936, **34**, 106.

[1] Day, P. L., and Langston, W. C., *J. Nut.*, 1934, **7**, 97.

[2] Mitchell, H. S., and Dodge, W. M., *J. Nut.*, 1935, **9**, 37.

[3] Mitchell, H. S., PROC. SOC. EXP. BIOL. AND MED., 1935, **32**, 971; Yudkin, A. M., and Arnold, C. H., *ibid.*, 1935, **32**, 836.