amino acids other than methionine and arginine were at marginal or slightly deficient levels. Unfortunately, growth data were given by Hill *et al.* for only the first 7 days of the chick's life. Growth data were not provided for a second series of tests conducted with a lower protein level, but it was stated that the results supported those in the first series. It is well known that for at least half of this time, the baby chick is still assimilating the unabsorbed yolk. Experimental diets during this period could not be expected to show very consistent differences since a large part of the total nutrients (unabsorbed yolk) is not controllable. Aside from the shortness of the growth period employed by Hill *et al.*, it is possible that their failure to observe a growth stimulation on the addition of amino acids to a diet containing raw soybeans may have been a manifestation of a very general principle, which holds in amino acid nutrition, as well as elsewhere. Hill *et al.* added 11 amino acids in balanced proportions intended to meet all requirements. The principle involved in this case is that adding a balance to an imbalance does not correct the latter. An imbalance may be corrected only by adding the specific components that are inadequately provided for balance, or by adding a complementary imbalance.

The hypothesis that impairment of proteolysis in the gut is a primary factor which appears to accentuate partial amino acid deficiencies or to convert a marginal supply of an amino acid to a deficiency in effect, has been discussed elsewhere(1,5,6). Recently, it has been shown that highly purified trypsin in the diet will reverse the effects of the raw soybean growth inhibitor, in support of the view that an anti-tryptic action is the primary mechanism of the increase in growth inhibition in this case(7).

Summary. 1. The presence of raw soybean protein in the chick diet was found to develop or accentuate a deficiency of tryptophan in the case of a chick diet marginal in this amino acid. 2. A diet deficient or marginal in arginine and methionine was rendered more acutely deficient when the soybean protein component was present as raw meal. 3. The effects of these accentuated deficiencies on growth were reversed by adding the specific amino acids involved or by heat destruction of the growth inhibitor. 4. These results are in agreement with previous results in the cases of methionine, lysine, arginine and isoleucine.

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Level of Liver Glycogen in Rats Having Steroid Diabetes. (20638)

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It has been generally believed that a deficiency of insulin limits the storage of glycogen in liver(1). This is not true of the rat having moderate alloxan diabetes(2) although it is noted in severely diabetic animals. The relationship of insulin action to the storage of liver glycogen requires further study with careful exploration of each experimental variable. Many studies employ the level of blood glucose as the sole criterion of the degree of diabetes. This is inadequate for the purpose. The amount of urinary glucose as related to

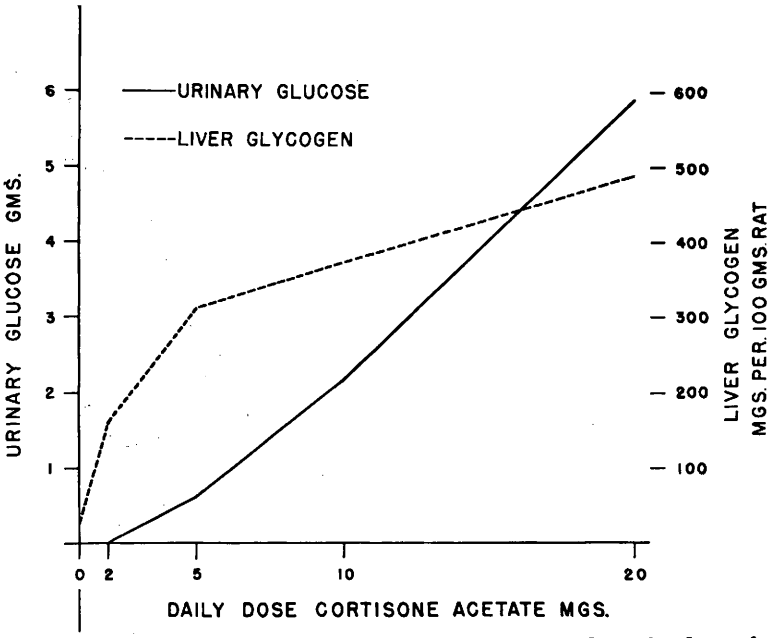

FIG. 1. Levels of urinary glucose and of liver glycogen as related to the dose of cortisone acetate. Averages.

food-intake permits a more quantitative appraisal of the severity of the diabetes.

The positive effect of cortisone and related steroids upon the level of liver glycogen is established. What is the level of liver glycogen in animals having steroid diabetes? Does the animal excrete glucose only after it has reached the limit of its capacity for storage, or does the administration of diabetogenic amounts of steroids interfere with glycogen storage? We find a very high level of liver glycogen in rats having steroid diabetes.

Methods. Male rats of the Sprague-Dawley strain having initial weights of approximately 300 g were placed in metabolism cages and fed 13 ml of a medium carbohydrate diet(3) by stomach tube each morning and late afternoon. The temperature was 74° to 78°C. Twenty-four-hour samples of urine were collected at 8:00 to 8:30 a.m. and were preserved with toluene for the quantitative determination of glucose(4). A micro-suspension of cortisone acetate was injected subcutaneously following each morning feeding. At the end of the injection period each rat was anesthetized with cyclopal; the liver was rapidly excised, weighed and dropped into hot KOH. Glycogen was determined by the use of anthrone(5).

Experiments and results. In Exp. 1 the rats were injected with cortisone for 7 days and the liver excised at the end of a 24-hour fast. The dosage was 2 mg per rat per day, 6 rats; 5 mg, 13 rats; 10 mg, 18 rats; and 20 mg, 16 rats of which 3 died. The dose of 2 mg per rat per day did not cause glycosuria but all of the rats on higher doses excreted measurable amounts of glucose. With each increase in dosage of cortisone there was a significant increase in the average level of liver glycogen and of urinary glucose (Fig. 1). The values for liver glycogen are expressed in relationship to body-weight. Cortisone causes a loss of weight to an extent which is proportional to the dose. These differences in weight loss by 7 days are small and do not significantly effect the shape of the dose-response curve for liver glycogen.

In Exp. 2 the level of liver glycogen was determined in rats which had received 7 daily injections of cortisone. In some of the rats the livers were removed either 4 or 6 hours after the last feeding. The effect of feeding upon the level of liver glycogen is maximal at about

TABLE I. Liver Glycogen of Fasted and Non-Fasted Rats Given Cortisone Acetate.

Daily dose cortisone, mg	Hr post-feeding	No. rats	Days on cortisone	Avg liver glycogen, % of liver wt
5	24	6	7	7.17
	4	2	6⅙	9.23
10	24	6	7	8.06
	6	4	6¼	9.97
20	24	8	7	9.06
	6	7	6¼	8.92

this time. In the remaining animals the livers were removed 24 hours after feeding. Although the numbers of animals are small, the data (Table I) do suffice to show that the peak level of liver glycogen in rats treated with large doses of cortisone acetate is about as high in the fasted as in the fed rat. The highest individual value was 11.84% glycogen in the liver of a fed rat given 10 mg of cortisone acetate daily for 7 days.

Discussion. Liver glycogen is very high in rats having steroid diabetes induced by cortisone acetate. At each higher dose there is an increase in both the amounts of urinary glucose and of liver glycogen. However, the slopes of the dose-response curves for urinary glucose and for liver glycogen are not parallel and at any given dosage level the correlations between the 2 values for individual animals are low. We have not measured these 2 indices of altered carbohydrate metabolism under identical conditions. Urinary glucose is excreted following each feeding and the urine is sugar-free by the end of a 24-hour fast. The deposition of liver glycogen is cyclic. If it were important to study more closely the interrelationship between these 2 indices it would be necessary to measure them simultaneously and at frequent intervals following feeding and during fasting.

Summary. Normal male rats were force-fed a medium carbohydrate diet. Steroid diabetes was induced by the administration of cortisone acetate in doses of 2, 5, 10, and 20 mg/rat/day for 7 days. The level of liver glycogen reached very high values in both fed and fasted animals. The glycogen level was proportional to the dose of cortisone acetate and was correlated with the level of urinary glucose.

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Excretion of Urinary Mucoprotein Following Parathyroid Extract in Rats.* (20639)

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Following the administration of parathyroid extract to rats, the levels of serum mucoprotein rise(1). When doses of 1000 or more units of the hormone are given over a short period of time, striking changes are seen in the kidneys. The tubules become filled with a water-soluble, alcohol-insoluble material which gives a positive periodic acid-leucofuchsin reaction, and mucoprotein granules appear in tubular cells. These observations suggested that in such animals the urinary mucoprotein levels also might be enhanced.

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