

Accentuation of Dietary Amino Acid Deficiency by Raw Soybean Growth Inhibitor. (20637)

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The present report is an extension of our studies on the inhibition of chick growth by raw soybean meal in the diet, as affected by partial dietary deficiencies of methionine, lysine, arginine, and isoleucine(1). The last 3 examples were shown to be analogous to the well known case of methionine which has long been recognized as the amino acid inadequately provided in whole soybean protein when fed as the only source of protein(2). The general nature of this interaction between the essential amino acid level and the growth inhibitor has now been demonstrated in the additional case of tryptophan.

Methods. The 20% protein chick diet for Exp. 1 was the same as used previously, except that meat scrap provided 15% protein in the diet in addition to the 5% quota of raw soybean meal protein. This amount of raw soybean protein has been shown to impart nearly all of the growth inhibition that may be observed with higher levels of raw soybean meal(1,3). Methionine at a level of 0.3% was also added. Duplicate groups of nine carefully selected day-old New Hampshire chicks were placed on each diet and raised to 28 days age.

The results are given in Table I. In a second experiment we employed the 30% protein diet of Hill, *et al.*(4) who have reported that amino acids would not prevent or reduce any of the growth retardation brought about by feeding raw soybeans to chicks. It was indicated by calculation that the possible deficiencies to be encountered would be limited

TABLE I. Effect of Tryptophan Addition to a Raw Soybean Meal and Meat Scrap Diet.

Supplement or treatment of diet	Avg wt of chicks at 28 days, g
None	131.3
L-Tryptophan, 0.2%	254.5
Soybean meal portion heated	354.5
Soybean meal portion heated, plus L-Tryptophan 0.2%	327.3

TABLE II. Effect of Methionine and Arginine Additions to a Raw Soybean, Casein and Fish Meal Diet.

Supplement or treatment of diet	Avg wt of chicks at 28 days, g
None	247.8
DL-Methionine, 0.4% plus L-Arginine, 0.3%	307.6
Soybean meal portion heated	316.5

to methionine and arginine. Only these amino acids were added, instead of the large total amount of some 11 amino acids added to the diet by Hill *et al.* The amino acids replaced an equivalent amount of casein. The results of this experiment are given in Table II.

Discussion. The diet used in Exp. 1 was expected to be slightly deficient or barely adequate in tryptophan content. When the soybean growth inhibitor was destroyed by heating, the chicks grew well and there was no increased growth response from added tryptophan. However, the diet containing the soybean protein in raw meal state permitted only relatively poor growth. The growth was distinctly improved by the addition of tryptophan (Table I). This example may be added to those which show that the increased growth inhibition, caused by interaction of the raw soybean inhibitor with a partial deficiency or a barely adequate level of an essential amino acid, is a general phenomenon not limited specifically to methionine(1). The fact that the growth was not fully restored by the added tryptophan is of incidental importance only, and may indicate that yet another essential amino acid was involved, secondarily to tryptophan.

In the experiment conducted with the diet of Hill, *et al.* it is evident that the addition of the amino acid supplements did increase growth rate (Table II). In this case the amino acid supplements were almost as effective as the destruction of the growth inhibitor by heat. It would appear that no essential

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