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## INTERRELATIONSHIPS OF NUTRIENTS

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### Interrelationships of nutrients: Chairman's opening remarks


By H. N. MUNRO, *Department of Biochemistry, University of Glasgow*


When the basic principles of nutrition were being established, it was clearly necessary to consider nutrients independently in order to identify their significance and to determine quantitative needs. This approach has tended to overshadow the obvious fact that individual nutrients are not consumed in isolation but as part of a diet providing a large number of variable components. For example, the nutritive values of proteins are usually determined under conditions ensuring their maximal utilization, but such studies do not adequately represent the fate of protein in natural diets, in which not only are the absorbed amino acids derived from several foods, but the nutritive value of the mixture is affected by the amounts of other dietary constituents, such as the energy-yielding nutrients, the minerals and the vitamins. The assessment of protein utilization under these practical conditions has been particularly studied by Platt and his colleagues, who have recently devised methods of predicting the nutritive values of the protein in human diets as they are consumed (Platt, Miller & Payne, 1961). During this Symposium, I am sure that we shall hear more of these interesting studies which emphasize the real importance of interrelationships of nutrients in the field of applied nutrition.

There is, however, another and equally important aspect of the interrelationships of nutrients, namely the exploration of mechanisms underlying such interactions. The reasons for interactions between nutrients can be varied, and are not confined to intermediary metabolism. This is illustrated by the known action of dietary carbohydrate on utilization of food protein. In the first place, carbohydrates present in foodstuffs can react chemically with free amino groups in the food proteins (Lea & Hannan, 1950), rendering amino acids, particularly lysine, unavailable to the body (Henry & Kon, 1950). Secondly, the type of carbohydrate in a meal probably influences the efficiency of absorption of protein consumed in the same food. Harper & Katayama (1953) found that a low-protein diet was better utilized by rats when sucrose was replaced by starch as the dietary carbohydrate, which was attributed to

retardation of the transit of food by the polysaccharide, so that the amino acids liberated from the protein were utilized more efficiently. The presence of carbohydrate in a meal can also influence the subsequent metabolic fate of the amino acids after absorption. Some years ago we noted that nitrogen balance underwent a temporary deterioration when the protein and carbohydrate in a diet were consumed in separate meals, whereas no such change in nitrogen balance followed separation of dietary protein and fat (Cuthbertson & Munro, 1939; Munro, 1949). Subsequent studies (Munro, Black & Thomson, 1959) suggest that the presence of carbohydrate in a meal causes release of insulin which then induces deposition of some of the incoming amino acids in muscle in the form of labile protein. Changes in the amount of this limited store of protein appear to be the cause of the temporary fluctuations in nitrogen balance when the times of consuming protein and carbohydrate are varied. However, under experimental conditions in which only essential amino acids were provided in the diet, Geiger & Nimni (1958) observed that rats did not grow unless the diet contained some carbohydrate, which was needed for the synthesis of non-essential amino acids. This represents yet another action of carbohydrate on amino acid metabolism. Lastly, dietary carbohydrate acts non-specifically as an energy source which, like other energy sources, is a factor in the utilization of dietary protein. Thus there are at least five independent mechanisms by which the carbohydrate of the diet can influence the fate of dietary protein.

Finally, it must be acknowledged that limitation of the study of interactions to pairs of nutrients is unrealistic. Some at least of the contradictory evidence concerning the role of diet in the production of arterial degenerative disease may be attributed to multiple unrecognized interactions. For example, there is a conflict of evidence about whether a high intake of protein augments or decreases susceptibility to arterial degenerative disease. Lofland, Clarkson & Goodman (1961) gave cholesterol to pigeons and observed that deposition of cholesterol in the wall of the aorta was increased by a high intake of protein and that this was further accentuated at the highest level of protein intake by using coconut oil as the dietary fat, whereas maize oil reduced cholesterol deposition on the high-protein diet. However, when the diet contained no added cholesterol, combination of a high level of protein with coconut oil reduced the concentration of cholesterol in the wall of the aorta and made the birds less susceptible to atherosclerosis. The above studies were further complicated by differences in the susceptibility of different breeds of pigeon to atherosclerosis. These complex interrelationships warn us that, in order to characterize the action of a diet on the health of the recipient, we must be prepared to find interactions not only between nutrients but also between factors in the diet and in the host, a situation which was appreciated by Gillman & Gillman (1951) when they wrote some years ago: 'Interpretations based on single factor relations do not adequately take into account the multiplicity of other factors, both in the organism and in the experimental diet, which obviously condition the results of the experiment.'

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## Dietary factors affecting energy utilization

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The most common nutritional experiment is the comparative feeding trial. Two groups of animals are given free access to two diets and their gains in weight recorded and compared. Differences in weight gain in such experiments can be very large. In thiamine deficiency in rats, for example, deficient animals may weigh less at the end than at the beginning of the trial though the control rats have doubled or trebled their weight. In such an experiment the heats of combustion of the thiamine-deficient diet and the control diet per unit weight are virtually identical. It is thus possible to state that thiamine deficiency has prevented the animals making full use of the calories supplied as food. As the deficiency proceeds, however, thiamine-deficient animals eat less food. Although their diets may be isocaloric per unit weight, deficient and control animals do not consume the same number of calories. By pair-feeding the animals, thus restricting the intake of food by the control animals to that consumed by the thiamine-deficient animals, it is found that both the control animals and the deficient ones lose weight or, if the deficiency is mild, gain weight only slowly. The thiamine-deficient animals usually but not invariably still weigh less than the control animals (Voris, Black, Swift & French, 1942; Pecora & Highman, 1953; van Eys, 1961).

Table 1. *Effect of mild thiamine deficiency on weight gain and energy retention by male rats* (*Voris* et al. 1942; *Voris & Moore*, 1943)

| | | | Gain of carcass | | | Fat in gain | |
|---|---|---|---|---|---|---|---|
| Group | Food eaten in 10 weeks (g) | Carcass weight gain (g) | Fat (g) | Protein (g) | Energy (kcal) | % of body-weight | Ratio, fat calories: total calories |
| Thiamine-deficient | 549 | 145 | 11·9 | 32·7±1·6 | 294 | 8·2 | 38 |
| Pair-fed controls | 550 | 144 | 15·2 | 31·8±1·4 | 319 | 10·5 | 44 |
| Unrestricted controls | 755 | 166 | 40·7 | 30·4±0·3 | 549 | 24·7 | 69 |

The results of a pair-feeding experiment in which a mild thiamine deficiency was produced are given in Table 1. In these experiments all the animals gained about the same amount of protein. Protein metabolism was thus unimpaired, a conclusion supported by the fact that thiamine-deficient rats can still manufacture antibodies (Axelrod & Hopper, 1960). The amount of fat gained by the thiamine-deficient rats