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SUMMARY

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SUMMARY

Previous studies in this laboratory showed protein deficiency in the immediate postweaning period to be associated with decreased activities of brain glutamate dehydrogenase and decarboxylase. The activity of GABA transaminase was not found to be altered.

Diets composed of poor quality protein such as kodri (Paspalum Scorbiculatum))and the poor Gujarati diet produced effects similar to a low protein diet.

In most of these studies decreased enzyme levels were associated with impaired performance in behavioural measures such as performance on the water maze, the Hebb-Williamsmaze, visual discrimination and reversal learning, locomotion scores and tasks involving motor coordination.

The present studies were undertaken as an extension of these studies and were aimed at :

- (1) identifying the amount of good quality protein needed in the diet to prevent the effects of deficiency and to reverse the effects of previous deficiency;
- (2) studying the effects of different degrees of protein deficiency;
- (3) investigating the time course of changes with protein deficiency; and

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 (4) comparing the effects of neonatal and postweaning undernutrition and of protein and calorie deficiencies during the postweaning period.

Diets containing 7-8% of good quality protein were found to prevent the deficits in brain glutamate dehydrogenase and decarboxylase found with a 5% protein diet. This is about half the amount of protein required for obtaining maximum growth and is consistent with the expectation that the amount of protein needed for conserving central nervous system function may be less than that needed for maximum body growth. A 10% protein diet was found to be sufficient and appeared to be necessary to restore brain enzyme activities to normal levels although recoveries of body and liver weights and liver glutathione were greater with a 20% protein diet.

The effects of deficiency on brain enzymes were not magnified by diets containing less than 5% protein. A surprising and rather unexpected observation was that brain GDH showed a decrease with a low protein diet but no change with diets containing little or no protein. Clear cut affects of deficiency on brain GDH and GAD were evident in low protein animals with 3 weeks of treatment. GAD activity showed a similar pattern with complete protein deprivation.

The studies also suggested that liver glutathione may be a better index of protein nutritional status than liver protein.

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With different levels of protein in the diet, changes in liver glutathione were more consistent with body weight changes than those in liver protein. Similarly the recovery in liver glutathione in rehabilitated rats also showed a direct relation with the level of protein used.

Neither moderate nor severe (50 and 67% restriction) undernutrition during the postweaning period was found to affect the activities of brain GDH in spite of the fact that the body weight deficits in the severely undernourished animals were greater than those found in protein deficient animals in an earlier experiment. These observations confirmed earlier impressions that the effects of undernutrition may be different from those of protein deficiency in the postweaning period.

Neonatal undernutrition produced by rearing pups in large litters was found to result in decreased activities of brain enzymes. While both continued undernutrition or protein deficiency after weaning in neonatally undernourished rats led to the persistence of these deficits, the same were found to be fully reversed on rehabilitation after weaning.

The results suggest that effects of nutritional deficiency on the metabolism of the brain depend on the nature, severity and duration of deficiency and the age of the animal of at the time_deficiency cours as well as the metabolic parameters investigated. These results have been discussed in relation to the situation in man.

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