The distribution of both taurine and cysteine-sulphinatase decarboxylase has been studied in subcellular fractions of adult rat brain. Much of the taurine (70%) was found in the soluble fraction, as has been found for glutamate and γ -aminobutyrate (Ryall, 1964; Mangan & Whittaker, 1966). Some 20% of the total taurine was present in the crude mitochondrial fraction, of which two-thirds was localized in synaptic endings and the rest in myelin. After 45% of the cysteine-sulphinatase decarboxylase was localized in the crude mitochondrial fraction, where its distribution in synaptosomes and myelin is similar to that of taurine. Both taurine and cysteine-sulphinatase decarboxylase activity could be released from the crude mitochondrial fraction by osmotic shock. The distribution of taurine, cysteine-sulphinatase and glutamate decarboxylases was examined in different anatomical regions of the brain. No direct relationship was found in each area between the concentration of taurine and that of cysteine-sulphinatase decarboxylase, as has also been observed for the γ -aminobutyrate-glutamate decarboxylase system (Roberts, 1962).

There are some structural resemblances between taurine, γ -aminobutyrate and glycine, and in addition there is physiological evidence to suggest that taurine, like γ -aminobutyrate, may have depressant action on neurones (Curtis & Watkins, 1960). The presence of cysteine-sulphinatase decarboxylase and taurine in the nerve-ending particles and the increase in decarboxylase activity during development raises the question as to whether or not taurine is a neurotransmitter or a modulator.

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Undernutrition and the Development of Certain Enzymes in Rat Brain

By B. P. F. ADLARD, J. DOBBING and J. L. SMART.
(Department of Child Health, University of Manchester, Manchester 13, U.K.)

There is much current interest in the possible effects of experimental undernutrition on the chemistry and metabolism of the developing brain. Previous studies have mainly involved measurement of content of nucleic acids, lipids or total protein after growth retardation of suckling animals (Dobbing, 1964; Howard, 1965; Culley & Mertz, 1965; Winick & Noble, 1966; Dobbing, 1968). Apart from two reports (Sereni, Principi, Perletti & Sereni, 1966; Chase, Dorsey & McKhann, 1967), the first of which was based on small numbers of animals, there are no results on brain enzyme activities under such nutritional conditions.

The developmental profiles of four enzymes representative of different metabolic pathways or subcellular organelles have been determined in the homogenates of whole brains of control and undernourished rats aged 0-21 days. Undernutrition was achieved by underfeeding the dams during gestation and lactation such that the weight of the young at 21 days was only 34% of that of controls.

The enzyme activities (per g wet wt. of tissue) rose three- to five-fold between birth and 21 days in control animals. The development of succinate dehydrogenase (EC 1.3.99.1; acceptor ferricyanide), fructose diphosphate aldolase (EC 4.1.2.13) and acetylcholinesterase (EC 3.1.1.7) was retarded by undernutrition. The activity deficits at 21 days were respectively 21, 14 and 11%, compared with a deficit in brain weight of 27%. The developmental profile of β -N-acetylglucosaminidase (EC 3.2.1.30), which is probably a lysosomal enzyme (Sellinger, Rucker & Verster, 1964), was unaffected by undernutrition.

The question is being investigated whether these enzymic deficits persist into adult life after feeding *ad libitum* beyond weaning.

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