labelled whole mouse brain, and the DNA was extracted and then chromatographed on hydroxy-apatite. In the preparations from scrapie-affected brain a small peak eluted at about 50°C was found. This may represent a small single-stranded specific scrapie-agent DNA.

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Deoxyglucose and Brain Glycolysis

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Injection or infusion of large doses of 2-deoxyglucose is known to cause elevated plasma concentrations of glucose and free fatty acids in man and laboratory animals, probably as a result of increased secretion of adrenaline. The deoxy sugar has also been suggested to act as an inhibitor of glycolysis in the brain. It is phosphorylated by brain hexokinase, so that both deoxyglucose and its phosphate might be expected to interact with glycolytic enzymes. Samples of guinea-pig cerebral cortex were homogenized in 0.32M-sucrose and centrifuged at 100000g for 1 h. The supernatant fraction was examined for activities of enzymes involved in the normal metabolism of glucose and glucose 6-phosphate. Enzymic specific activities ($\mu\text{mol/min per g}$) at 37°C and 2 mM substrate concentrations were: hexokinase (glucose: 5.9); glucose 6-phosphate dehydrogenase (glucose 6-phosphate: 4.1); 6-phosphogluconate dehydrogenase (6-phosphogluconate: 0.75); phosphoglucomutase (glucose 1-phosphate: 40.8); hexose phosphate isomerase (glucose 6-phosphate: 169; fructose 6-phosphate: 284). Inclusion in the reaction mixtures of 2-deoxyglucose or its phosphate at 2–4 mM had no effect on any activity except that of hexokinase. Deoxyglucose inhibits hexokinase by competition with glucose (K_i 0.25 mM; Bachelard *et al.*, 1971), but the phosphate was found to be inactive. Deoxyglucose thus has considerably less affinity for hexokinase than has glucose (K_m 0.04 mM) and also a lower affinity for the uptake process (Bachelard, 1971). Hexose phosphate isomerase is inhibited by deoxyglucose 6-phosphate in kidney and ascites-tumour cells (Wick *et al.*, 1957; Nirenberg & Hogg, 1958), but not in diaphragm (Kipnis & Cori, 1959). In the brain, only the transport and phosphorylation of

glucose seem to be affected. In order for accumulation of concentrations of deoxyglucose sufficient to cause significant inhibition of cerebral hexokinase, with resultant decreased glucose utilization and depletion of ATP, the circulating deoxyglucose concentration would have to be maintained at least one order higher than glucose. The possibility of action on enzymes acting at stages of metabolism remote from glucose 6-phosphate, although unlikely, has not been excluded. The recognition of deoxyglucose by the glucose-binding site on hexokinase, and the non-recognition of deoxyglucose 6-phosphate by the glucose 6-phosphate-binding site, provided further evidence for the allosteric properties of the enzyme.

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Increased Activity of the Sodium-plus-Potassium Ion-Stimulated Adenosine Triphosphatase in Rat Brain during Electrically Induced Convulsions

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Increased activity of the $\text{Na}^+ + \text{K}^+$ -stimulated adenosine triphosphatase of rat brain during convulsions induced by lethal doses of Metrazol (6,7,8,9-tetrahydro-5-azepotetrazole) has been reported by Bignami *et al.* (1966) and De Robertis *et al.* (1969). During an investigation of the effect of experimentally induced thyroid disorders on energy utilization in rat brain we have observed a similar increase in $\text{Na}^+ + \text{K}^+$ -stimulated adenosine triphosphatase activity in brain tissue from rats made to convulse by electric currents.

Male Wistar rats (100g) were stimulated through electrodes applied to the ears by monophasic square waves (85 V; 150/s; duration 1.5 ms) applied for 1 s. The animals were killed after 10 s during the tonic phase by total immersion in liquid N_2 . Homogenates (10%, w/v) of cerebral-cortex tissue and whole cerebellum were prepared in water (Samson & Quinn, 1967). $\text{Na}^+ + \text{K}^+$ -stimulated adenosine triphosphatase activity was determined in the unfractionated homogenates and in the total particulate fraction (obtained by centrifuging the homogenate at 10⁶g for 60 min) by the procedure of Rodnight & Lavin (1966).