The Effect of Galactose Metabolic Disorders on Rat Brain Na\(^+\),K\(^+\)-ATPase Activity

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To evaluate the effect of galactose metabolic disorders on the brain Na\(^+\),K\(^+\)-ATPase in suckling rats. Separate preincubations of various concentrations (1–16 mm) of the compounds galactose-1-phosphate (Gal-1-P) and galactitol (galtol) with whole brain homogenates at 37 °C for 1 h resulted in a dose dependent inhibition of the enzyme whereas the pure enzyme (from porcine cerebral cortex) was stimulated. Glucose-1-phosphate (Glu-1-P) or galactose (Gal) stimulated both rat brain Na\(^+\),K\(^+\)-ATPase and pure enzyme. A mixture of Gal-1-P (2 mm), galtol (2 mm) and Gal (4 mm), concentrations commonly found in untreated patients with classical galactosemia, caused a 35% (p < 0.001) rat brain enzyme inhibition. Additionally, incubation of a mixture of galtol (2 mm) and Gal (1 mm), which is usually observed in galactokinase deficient patients, resulted in a 25% (p < 0.001) brain enzyme inactivation. It is suggested that: a) The indirect inhibition of the brain Na\(^+\),K\(^+\)-ATPase by Gal-1-P should be due to the presence of the epimer Gal and phosphate and that the pure enzyme direct activation by Gal-1-P and Glu-1-P to the presence of phosphate only. b) The observed brain Na\(^+\),K\(^+\)-ATPase inhibitions in the presence of toxic concentrations of Gal-1-P and/or galtol could modulate the neural excitability, the metabolic energy production and the catecholaminergic and serotoninergic system.