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

Stylianos Tsakiris[a*], Kyriakoula Marinou[a] and Kleopatra H. Schulpis[b]

[a] Department of Experimental Physiology, Medical School, University of Athens, P.O. Box 65257, GR-15401 Athens, Greece. Fax: 003-010-7775295. E-mail: stsakir@cc.uoa.gr
[b] Inborn Errors of Metabolism Department, Institute of Child Health, Aghia Sophia Children's Hospital, GR-11527 Athens, Greece

* Author for correspondence and reprint requests

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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.