THE ASSESSMENT OF GLUCOMETABOLIC HORMONES IN IMMATURE RATS AFTER HYPOXIC-ISCHEMIC INSULT

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Sonam Kapoor M.Sc. *Biochémia* *2022*

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Sonam Kapoor[1,2] Lucia Rackova[1] Jan Svoboda[2] David Kala[2] Jakub Otahal[2] Zuzana Brnoliakova[1]

[1]Institute of Experimental Pharmacology and Toxicology, Center of Experimental Medicine, Slovak Academy of Sciences, Dubravska cesta 9, 841 04 Bratislava, SR
[2]Institute of Physiology, Czech Academy of Sciences, Videnska 1083, 142 20 Prague, CR
sonam.kapoor@savba.sk

Neonatal hypoxic-ischemic encephalopathy (HIE) is one of the main devastating causes of morbidity and mortality in new-borns. Infants who survive such severe hypoxic-ischemic insult (HII) frequently develop neurological sequelae later in life, such as cerebral palsy, epilepsy, developmental delay, cognitive impairment, and behavioral.[1] Although the glucose metabolism in the adult brain has been studied

pilocarpine (Li-Pilo) intraperitoneally (*i.p.*) (35 mg /kg *b.w.*, n=6) or saline (0,9% NaCl, n=5) as for control group (Control). Serum was collected on the 19th postnatal day.[3] The serum levels of glucagon, glucagon-like peptide-1 (GLP-1), ghrelin, leptin, and plasminogen activator inhibitor 1 (PAI-1) were thoroughly assessed by magnetic bead-based immunoassays on Bio-Plex 200 systems (Bio-Rad, U.S.) and interpreted. The relevant changes in the set of five glucometabolic hormones were observed: the levels of GLP-1, glucagon, leptin, and PAI-1 were significantly elevated in Li-Pilo experimental group as compared to the Control.

Our data confirmed former observations that encephalopathy causes major modifications in glucose metabolism disposition in immature rats.[4] Though different mechanisms interacting with seizures, such as the anticonvulsant impact of ghrelin, have already been reported, the relevance of these changes is still about to be elucidated.[5] In conclusion, this work broadens knowledge of the alterations in glucose metabolism in immature rats after HII. The data demonstrated the significance of systemic glucose metabolism regulation in encephalopathy. Interactions with glucose metabolism thus represent a potential pharmaceutical target for future scientific research.

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Zdroje:

1. A.B. Edwards; R.S. Anderton; N.W. Knuckey; B.P. Meloni. Brain sciences. 2018. 8, 147.

2. E. Brekke; T.S. Morken; U. Sonnewald. Neurochemistry International. 2015. 82, 33–41.

3. J. Folbergrová; P. Ješina; J. Otáhal. Front. Neurosci. 2021, 15, 1-10.

4. M. Grieco; A. Giorgi; M.C. Gentile; M. D'Erme; S. Morano; B. Maras; T. Filardi. Front. Neurosci. 2019, 13.

5. J. Portelli; Y. Michotte; I. Smolders. Epilepsia. 2012, 53, 585-595.

preveda@preveda.sk;
konferencia.preveda@gmail.com

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