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BOOK OF ABSTRACTS

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ALTERATIONS OF GLUCOSE METABOLISM IN NEONATAL HYPOXIC ISCHEMIC ENCEPHALOPATHY IN NEWBORN RATS

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Neonatal asphyxia is one of the most serious causes of morbidity and mortality of newborns, also known as hypoxic-ischemic encephalopathy (HIE), accounting for nearly a quarter of all neonatal fatalities worldwide.[1] Neonatal HIE has incidence of about 1-6 per 1000 term births in the developed countries. The neonatal brain hypoxic ischemic (HI) injury occurs as the result of a permanently or temporarily restriction in blood supply and impaired oxygen delivery to the brain. Infants who survive may develop severe neurological sequels such as cerebral palsy, seizures, epilepsy, mental retardation, behavioural and/or learning disorders.[2] As the consequence, there is a notable socioeconomic burden. Thus, the urgent need for further investigation and understanding of pathogenic processes to develop effective intervention procedures is comprehensible.

The main goal of our work was to study the alterations of glucose metabolites in neonatal HIE in newborn rats.

Perinatal HI insult was induced on 7th day old rat pups according to Rice - Vanucci model[3] in HIE group. Briefly, in isoflurane aneasthetized rat pups a left common carotid artery was isolated and ligated. After short recovery animals were incubated in normobaric hypoxic chambre (pO2 8%) for 90%. CONT animals underwent only surgical procedure without hypoxia and served as controls. The serum levels of glucagon, glucagon-like peptide-1 (GLP-1), grehlin, leptin and plasminogen activator inhibitor 1 (PAI-1) were throughly assessed by magnetic bead–based immunoassays on Bio-Plex 200 systems (Bio-Rad, U.S.) and interpreted. We observed the relevant changes in cluster of those five glucometabolic hormones measured and compared HIE vs CONT experimental groups.

Our data indicates, in accordance with published data, that the encephalopathy leads to significant alterations in glucose metabolism regulation in immature rats.[4] The role of these changes is not well understood yet, although several mechanisms interacting with seizures, such as anticovulsant effect of ghrelin, has been already described.[5]

In conclusions, our data indicates the important role of systemic glucose metabolism regulation and suggests its importance within encephalopathy. Interactions with glucose metabolism thus represent the potential pharmacological target to focus on in further research strategies.

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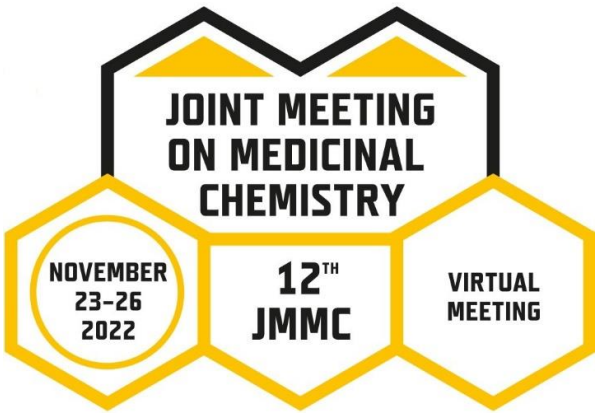

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