DCX labeled neurons and statistical differences were assessed by repeated measures ANOVA.

Results: EE increased Ki-67 positive cells in WT mice and prevented the occurrence of depressive-like behavior in YAC128 mice. EE increased neuronal differentiation in both WT and YAC128 mice. Dendritic arborization was also increased by EE exposure in WT mice when compared to WT animals housed in standard housing conditions.

Conclusion: Our results indicate that exposure to EE is able to modulate hippocampal neuroplasticity in WT and YAC128 mice. EE may have a potential therapeutic role in reversing the deficits in neuroplasticity seen in HD.

https://doi.org/10.1016/j.ibror.2019.07.1500

P32.16

Hemispherical asymmetry in reports gene or protein expression related to mood disorders in the brain of rodents: A pilot systematic review

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Expression of genes or proteins has been an important tool in elucidating the neurochemical functioning underlying mood disorders, especially in rodent models. The region-specificity of such expressions has been largely acknowledged, but most of the studies consider these brain regions as a single functional unit, failing to considerate hemispherical asymmetry that could alter the results and their interpretation. The aim of this review is to verify the influence of cerebral laterality on the expression of genes or proteins in reports investigating mood disorders in rodent models. Papers studying mood disorders (depression or anxiety) that assessed the gene or protein expression in the brain of rodents, measuring and comparing this expression between the two hemispheres were included. This study followed the PRISMA protocol. A search was conducted on PubMed using keywords designed to retrieve papers that met the inclusion criteria. Fifty-six articles were screened by title and abstract and nine of them were eligible. Two papers were excluded which resulted in the final seven papers. Data extraction and analysis were conducted by two independent reviewers, with a third resolving discrepancies. Results points to an asymmetrical role of the main brain areas related with stress evoked disorders, such as depression and anxiety, with neurotrophic factors and some neurotransmitters receptors expression. Hemisphere asymmetry factor was mainly studied in the prefrontal cortex, hippocampus, amygdala, and olfactory bulb. Neurochemical parameters such as $GABA_A$ receptor subunits, serotonin, dopamine and BDNF are altered in an asymmetrical way. That dissimilarity is linked with both the administration of substances and with animal models of stress. One main question raised by this review is whether few experimental protocols are designed to verify hemisphere differences or whether non-significant hemispherical analyses fail to be published. We suggest that hemisphere differences should be emphasized in further studies that explore psychiatric disorders.

https://doi.org/10.1016/j.ibror.2019.07.1501

P32.17

Galectin-1 improves cognition and reduces amyloid-β deposits in an animal model of Alzheimer's disease possibly by modulating microglia phenotype and increasing Aβ clearance

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Alzheimer's disease (AD) is the most common form of dementia associated with an imbalanced production and clearance of amyloid-β peptides (Aβ). Amyloid deposition and neuroinflammation are recognized hallmarks in AD, affecting mainly brain cortex and hippocampus, in addition to microvascular alterations and dysfunction of the blood-brain barrier (BBB). The glycan-binding protein galectin-1 (Gal1) modulates immune and endothelial cells in nervous system compartments, where a neuroprotective role was proposed in autoimmune encephalomyelitis. We study the impact of Gal1 on the cognitive and histopathological state of AD mice. We administered Gal1 (9 i.p. injections of 100 ug/dose) or vehicle during 3 weeks to 12 months-old PDAPPJ20 transgenic mice, or non-transgenic controls. The Gal1 treated group significantly improved cognitive response in the Novel Object Location Recognition test ($p < 0.05$). Amyloid+ area in the hippocampus was decreased by 53,5%. Microglia is actively involved in Aβ phagocytosis. Gal1 treatment induced a reduction in the microglial activation score employing morphological analysis in the dentate gyrus. The integrity of the BBB is essential to Aβ clearance via the glymphatic system but could be altered by perivascular Aβ deposits-mostly Aβ1–40. Using tomato lectin to label the hippocampal vasculature coupled with immunofluorescence against Aβ peptides, we found a 30% decrease of perivascular Aβ ($p < 0.05$) in Gal1 treated mice, without affecting vascular density, which could indicate augmented clearance. We are currently working to determine mice BBB integrity employing Evans Blue intravenous injections and exploring its permeability to cerebral parenchyma, and using an in vitro BBB model to determine whether Aβ1–40 alters it at non toxic concentrations. Human brain microvascular endothelial cells on a transwell membrane are used, monitored by Transendothelial Electrical Resistance (TEER) and permeability essays. We are also investigating possible protective effects of Gal1 on barrier's integrity.

https://doi.org/10.1016/j.ibror.2019.07.1502