ABSTRACTS

THE LONG-TERM EFFICACY AND COGNITIVE EFFECTS OF ELECTROCONVULSIVE THERAPY IN INDIVIDUALS WITH DEPRESSION AND ANXIETY

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Electroconvulsive therapy (ECT) is a recognized intervention for treatment-resistant depression (TRD). However, uncertainty remains regarding the severity and duration of ECT-related cognitive impairments and whether psychiatric co-morbidities such as anxiety contribute to residual impairments. This study investigates the efficacy and cognitive outcomes of bilateral, brief-pulse ECT for TRD up to 6 months post-ECT. Additionally, it explores whether patients with co-morbid anxiety respond differently to ECT compared to individuals with only depression. Out of 118 outpatients receiving ECT, 17 (5-Depression; 12-Anxiety & Depression) fit the inclusion criteria. Assessment measures included: Beck Depression Inventory-II (BDI-II), Personality Assessment Inventory (PAI), Repeatable Battery for the Assessment of Neuropsychological Status (RBANS), and Squire Subjective Memory Questionnaire (SSMQ). Assessments were performed at baseline, following the 4th ECT, 2-4 weeks post-ECT, and 6 months post-ECT. BDI-II results indicated that bilateral, brief-pulse ECT is an effective acute treatment for TRD. In patients diagnosed with comorbid depression and anxiety, benefits are maintained up to 6 months post-ECT. Additionally, PAI anxiety levels decreased significantly with treatment of depression. The patients' objective cognitive functioning measured by the RBANS was within the average range, and no significant cognitive changes were detected over time. The SSMQ results indicated that patients' reported subjective memory impairment did not change significantly over time. Contrary to this, the subrgoup of patients with co-morbid depression and anxiety had significantly lower RBANS scores overall. However, the small sample size and medication differences provide substantial impetus for further investigation.

SEX DIFFERENCES IN STRESS ADAPTATION: NEUROPLASTICITY WITHIN THE PARAVENTRICULAR NUCLEUS OF THE HYPOTHALAMUS

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The hypothalamic-pituitary-adrenal (HPA) axis is sexually dimorphic. However, little is known about sex differences in the HPA axis' adaptation to chronic stress. Here, we studied sex-dependent neural plasticity mechanisms relevant to the habituation of the HPA axis to repeated stress. This habituation manifests as decreased excitability of HPA axis output neurons, which are neuroendocrine neurons that express corticotropin releasing hormone (CRH) in the paraventricular nucleus of the hypothalamus (PVN). Mice were first subjected to 1 hour of daily restraint stress for 3 weeks, and were then challenged with the same stressor after either 1 day, 1 week, or 4 weeks of no-stress recovery periods. As controls, one group of mice received 1 hour of restraint without prior stress, and another control group received no stress. Using immunohistochemistry, we quantified the induction of the immediate early gene (IEG) c-Fos, a marker of neuronal activation, in PVN-CRH neurons. We found that, in both sexes, 3 weeks of repeated stress decreased restraint-induced c-Fos expression in the PVN-CRH neurons. Furthermore, this habituation was fully reversed by 4 weeks without stress. However, females showed greater habituation and a slower recovery; significant attenuation persisted after 1 week without stress whereas males had fully recovered by that time. In summary, we found sex differences in the neural plasticity associated with the HPA axis habituation to and recovery from repeated stress. These findings should be further explored to elucidate the mechanistic causes for these differences in c-Fos expression.

Acknowledgements: Supported by CIHR, NSERC and OMHF.

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MORPHOLOGICAL EFFECTS OF DEVELOPMENTAL ETHANOL EXPOSURE ON MEDIAL PREFRONTAL LAYER VI NEURONS IN JUVENILE MICE

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The teratogenic effects of chronic prenatal alcohol exposure have been well documented and are classified as fetal alcohol spectrum disorders (FASD). Of the cognitive deficits associated with FASD, attention deficits are among the most prevalent and persistent, yet there is a limited understanding of the neural mechanisms underlying these deficits. It has been shown that pyramidal neurons in layer VI of the medial prefrontal cortex (mPFC) are critical for optimal attention. The morphological development of these neurons is driven by nicotinic acetylcholine receptors, which are vulnerable to dysregulation by developmental ethanol exposure in rodents. To date, studies of developmental ethanol exposure on mPFC neuronal morphology have been quite limited. In this study, a quantitative Sholl analysis of mPFC layer VI neuron morphology was performed on biocytin-filled neurons using Neurolucida software to produce three-dimensional neuron tracings. Mice of both sexes were exposed to either ethanol vapour or air during the human 2nd and 3rd trimester equivalents, and assessed for neuron morphology at the juvenile-equivalent age of postnatal day 15. Morphological measures analyzed included apical and basal dendritic matter, complexity, and cell body area. Compared to the controls, developmental ethanol exposure decreased the length and complexity of apical dendrites in male mice, but increased the length and complexity of basal dendrites in female mice. This demonstrates a sex difference in developmental ethanol-induced morphological alterations to mPFC layer VI neurons. These findings suggest that exposure to ethanol during development cause structural changes within the mPFC early in postnatal life, which may alter the trajectory of normal morphological refinements during mPFC maturation. Alterations to dendrite morphology may influence the function of the mPFC through changes to the location and type of afferent axonal inputs from other brain areas to layer VI pyramidal cell dendrites. Furthermore, these results provide a potential morphological mechanism underlying attention deficits associated with FASD.

EXPRESSION OF OBJECT RECOGNITION MEMORY AND THE ONTOGENY OF TEMPORAL ORDER RECOGNITION MEMORY IN C57BLK/6J X 129S1/SVIMJ MICE

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Mental disorders with adolescent onset, such as schizophrenia, are theorized to be a consequence of the abnormal maturation of brain circuits implicated in learning and memory. To understand how developing brain circuits become pathological, it is important to investigate how these neural circuits mature to support behaviour. This study investigated the behavioural expression of object recognition (OR) and temporal order recognition (TOR) memory in C57BLK/6J x 129S1/SvImJ mice and identified the age at which they expressed TOR memory. We found that: 1) C57BLK/6J x 129S1/SvImJ mice had an innate preference toward select objects; 2) they only expressed OR memory during the first 20 seconds of object exploration and required a long-term, 24-hour consolidation period; and 3) C57BLK/6J x 129S1/SvImJ mice are able to express TOR at postnatal day (PD) 32 but not at PD14, PD16 or PD23. These data suggest that TOR memory, which is known to depend on the medial prefrontal cortex (mPFC), features a late onset consistent with the delay in the maturation of the mPFC. Additionally, this indicates that behaviours such as TOR have a later onset than more survival-critical behaviours such as fear learning.