NEUROABSTRACTS

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CORTICAL THICKNESS IN CHILDREN OF PARENTS DIAGNOSED WITH BIPOLAR DISORDER

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Bipolar disorder (BD) is associated with reduced cortical thickness in prefrontal and temporal cortical areas responsible for affect regulation. Children of parents diagnosed with bipolar disorder (high-risk (HR) offspring) are at risk of developing a psychiatric illness, and may have cortical abnormalities in affect regulating pathways prior to the onset of any psychiatric disorder. The current study is investigating cortical abnormalities in children at risk of developing bipolar disorder to better understand the developmental trajectories of psychopathology. Twenty-four HR bipolar offspring (average age = 13.7 (2.69) years, 12 females), and 9 age and sex matched healthy controls (HC) (average age = 12.9 (2.70) years, 5 females) were included in this study. Structural brain scans were administered and measures of cortical thickness were extracted and compared between the HR and HC offspring. HR offspring had significantly decreased cortical thickness in the left superior frontal gyrus, BA 6 (t=4.23 and t=3.57), the left posterior cingulate, BA 30 (t=3.49 and t=3.44) compared to HC offspring. Further analysis of subgroups of the HR population found additional areas of cortical abnormalities in the frontal and temporal cortex. The significant cortical differences found between HC children and HR bipolar offspring suggest that neurobiological markers of risk and resilience can be identified in children at risk of developing BD.

UBISOL-QE AS A THERAPEUTIC TREATMENT FOR PARAQUAT INDUCED NEURODEGENERATION IN RAT MODEL OF PARKINSON'S DISEASE

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The standard pathophysiology in neurodegenerative diseases, such as Parkinson's disease (PD), is caused by loss of dopaminergic neurons. Mitochondrial dysfunction and oxidative stress have been implicated in these types of neuronal death. It has been shown that exposure to environmental toxins such as paraquat, a commonly used herbicide, can lead to an increase in the incidence of PD. So far, there is no effective therapeutic treatment that can halt the progression of the neurodegeneration. We have been conducting research on a water soluble formulation of CoQ10 (Ubisol-QE) and are testing its ability to protect neurons in vivo. It has previously been shown that Ubisol-QE prevents progression of neurodegeneration in rat models of PD post-injury. We wanted to determine the duration of Ubisol-QE treatment required to effectively sustain neuroprotection. Therefore, over the 8-week treatment portion of the experiment, one group of paraquat exposed rats were treated for the entire time with Ubisol-QE, while another had their treatment withdrawn after 4 weeks. We evaluated the neuroprotective effects by the number of tyrosine hydroxylase positive neurons in the substantia nigra region of the brain of animals who were given continuous treatment and those of whom who had their treatment withdrawn. The results suggest that Ubisol-QE has a significant effect after 8 weeks of treatment, indicating that longer duration of the treatment is needed. In observing the results from the neuromotor decline experiments, the treatment group had far less hind leg slips than the untreated groups, reflecting the protection that was seen in the immunohistochemical results. Consequently it can be suggested that Ubisol-QE could be an effective treatment to halt the neurodegeneration found in PD patients.

GSK-3 BETA INHIBITION MORE STRONGLY BLOCKS ACQUISITION THAN EXPRESSION OF AMPHETAMINE-PRODUCED CONDITIONED PLACE PREFERENCE IN RATS

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Dopamine (DA) has been implicated in behavioural sensitization and incentive learning. The signaling molecule glycogen synthase kinase-3 (GSK-3), activated downstream of DA D2-like receptors, affects DA-mediated behaviours. For example, inhibition of GSK-3 attenuates locomotor sensitization to psychomotor stimulant drugs. Few studies have examined the effects of GSK-3 inhibition in incentive learning paradigms. We examined the effects of GSK-3 inhibition on incentive learning produced by amphetamine (1.5 mg/kg) in the conditioned place preference (CPP) paradigm. The selective GSK-3 inhibitor SB 216763 was administered intraperitoneally to male Wistar rats at doses of 1.0, 2.0, and 2.5 mg/kg during the acquisition or expression phase. Due to studies more strongly implicating signaling molecules in acquisition rather than expression of learning, we hypothesized a block of acquisition of CPP at doses that fail to block expression of CPP. Results supported this hypothesis: the 1.0 mg/kg dose of SB 216763 failed to block either acquisition or expression of CPP; the 2.0 mg/kg dose blocked acquisition, but not expression of CPP; and the 2.5 mg/kg dose blocked both acquisition and expression of CPP. These results indicate that inhibiting GSK-3 may attenuate incentive learning differentially in acquisition versus expression of conditioning. (Funded by NSERC)