Neural Correlates in Childhood-onset Obsessive Compulsive Disorder

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Abstract:

Obsessive-compulsive disorder (OCD) is a serious neuropsychiatric disease with complex genetic underpinnings and associated neural correlates. It affects approximately 1-3% of Canadians (1) and has been identified by the World Health Organization as a leading global cause of non-fatal illness burden (1). Although effective treatments such as cognitive-behaviour therapy (CBT) and serotonergic medications exist, these are often delayed for years (2) due to OCD under-recognition and barriers such as limited access to CBT and associated therapy costs. Moreover, treatment response rates and outcomes worsen as OCD duration increases (3, 4).

Childhood-onset OCD (CO-OCD) is the most heritable known form of the disorder (5) such that siblings of OCD-affected youth sharing 50% of the same genes have a 10-fold increased risk for OCD (6). But predicting which children will develop OCD and who will respond to CBT remains challenging. No OCD vulnerability genes have been confirmed despite costly family, linkage and candidate gene research (6), and most recently, the first OCD genome-wide association study including 1,865 OCD-affected individuals, led by the Nominated Principal Applicant (7). *Thus, new strategies are crucial for identification of OCD risk markers, to enhance early identification and to develop and prioritize treatment.*

The **main objective** is to compare neural correlates in CO-OCD and unaffected, at-risk sibling (SIB) groups with matched healthy controls (HC), in order to evaluate both their suitability as trait markers of genetic risk (to better understand the disorder) and their potential utility in clinical settings (to better manage the disorder). T**hree specific aims** are to identify potential OCD endophenotypes by comparing groups with respect to: 1) neurocognitive functioning and 2) NSS prevalence; and 3) to examine their knowledge translation (KT) potential as CBT response predictors.