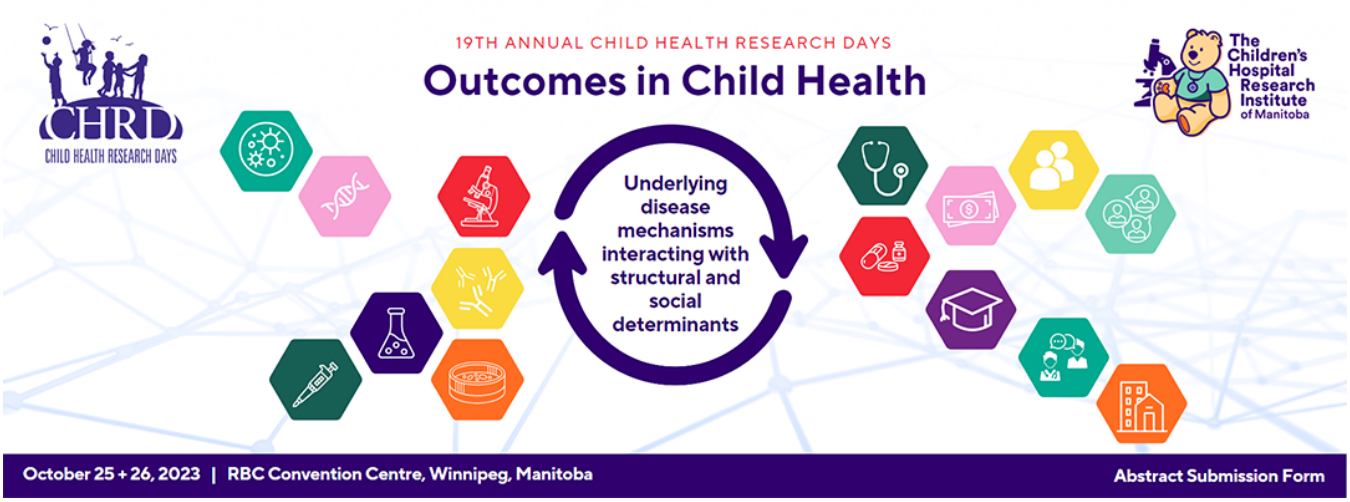

CHRD 2023: Abstract Submission Form

Submitter Name
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Presenter Name
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Presenter Status
Masters Student

Research Category
Basic Science

Role in the project
Perform Experiments
Analyze Data
Write Abstract

Title
Characterizing the neuronal role of CK2 using Drosophila melanogaster

Background
Variants in CSNK2A1 or CSNK2B encoding respective α and β subunits CK2 (casein kinase 2), cause pediatric neurological disorders predominantly characterized by different levels of developmental delay, intellectual disability, and seizures. The two disorders associated with CSNK2A1 or CSNK2B are Okur-Chung Neurodevelopmental Syndrome (OCNDS) and Poirier-Bienvenu Neurodevelopmental Syndrome (POBINIDS), respectively. Combined, there are over 100 cases published for these two rare disorders. However, the functional impact of disease variants has yet to be assessed in vivo and the role of CK2 in neurons is not understood.

Objective
We aim to assess the role of Ck2 in the central nervous system of Drosophila in both neurons and glia. Secondly, we will determine the effect of expressing CSNK2A1 and CSNK2B variants in the fruit fly to assess function.

Methods
We will use the GAL4/UAS system to knockdown Ck2 via RNA interference in neurons (nSyb- GAL4) or glia (Repo-GAL4) and assess motor and seizure induction. We will tandemly express both human CSNK2A1 and CSNK2B (and disease variants) in various fly tissues to assess function.

Results
We observed lethality upon neuronal knockdown of Ck2. Some escapers show wing deficits and exhibit

lifespan defects. We then conducted adult-specific neuronal knock-down with elav- GAL4 GeneSwitch that revealed lifespan and motor defects in flies upon neuronal knockdown. Glial knockdown of Ck2 caused minimal phenotypes. The overexpression of human CK2 ubiquitously in the fly caused lethality and disease variants are currently being assessed.

Conclusion
Given the neuronal knockdown of Ck2 results in organismal lethality, neuronal CK2 is therefore critical for the development of Drosophila melanogaster. Adult-specific knockdown of Ck2 causes lifespan and climbing defects indicating a role for Ck2 in long-term neuronal function.

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