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YOU ARE INVITED TO ATTEND THE

DEFENSE OF THE DOCTORAL

DISSERTATION

"Neurochemical and behavioral analysis of a high-risk schizophrenia mouse model"

by

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Cell Biology, Neuroscience and Physiology Program
M.S. 2020, Thomas Jefferson University, Philadelphia, PA
B.A. 2017, Rutgers University, New Brunswick, NJ

Thesis Advisor:

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Department of Pharmacology, Physiology & Neuroscience

Thursday, September 3rd, 2026
1:00 pm.
Medical Science Building, H609b

Join Zoom Meeting:
https://rutgers.zoom.us/my/ss2032?pwd=Fqvg0fU3BvC9nlM9SDMaYEIa3bjTaQ.1&omn=98123394285

Meeting ID: 985 555 3976
Password: 441677

Abstract

Schizophrenia (SZ) is a severe and debilitating psychiatric disorder for which current treatments remain inadequate. Although dopamine dysregulation is well established as a key component of SZ pathophysiology, the therapeutic gap is driven, in part, by an incomplete understanding of the neurophysiological mechanisms underlying the disorder. The use of genetically informed models of schizophrenia to investigate dopaminergic alterations has been on the rise. This study utilizes a mouse model of the 3q29 deletion, a 1.6 Mb de novo mutation that confers a greater-than-40-fold increased risk of SZ, the single largest known molecular risk factor for the disorder to date. Investigating this model revealed a hyperdopaminergic phenotype characterized by elevated evoked dopamine levels in the dorsal striatum and a hypolocomotive behavioral phenotype. Mechanistically, we have evidence that elevated activity of dopamine D3 autoreceptors is driving a PKC-mediated reduction in dopamine transporter (DAT) availability, thereby leading to insufficient clearance and prolonged dopaminergic signaling. To test this potential mechanism, a chronic PKCβ inhibitor was administered to measure biological and behavioral outcomes. This study establishes a potential new treatment target for 3q29 deletion schizophrenia by modulating elevated dopamine levels in the dorsal striatum and alleviating downstream behavioral phenotypes in the 3q29 microdeletion mouse model.