# TITLE: A Neurobiological Pathway That Connects Polygenic Risks With Behavioral Changes Shared Between Schizophrenia And Bipolar Disorder

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## ABSTRACT (upto 300 words)

Schizophrenia (SZ) and bipolar disorder (BP) are highly heritable major psychiatric disorders that share a substantial portion of genetic risk as well as their clinical manifestations. This raises a fundamental question of whether, and how, common neurobiological pathways translate their shared polygenic risks into shared clinical manifestations. This study shows the miR-124-3p-AMPAR pathway as a key common neurobiological mediator that connects polygenic risks with behavioral changes shared between these two psychotic disorders. We discovered the upregulation of miR-124-3p in neuronal cells and the postmortem prefrontal cortex from both SZ and BP patients. Intriguingly, the upregulation is associated with the polygenic risks shared between these two disorders. Seeking mechanistic dissection, we generated a mouse model that upregulates miR-124-3p in the medial prefrontal cortex. We demonstrated that the upregulation of miR-124-3p increases GRIA2-lacking calcium-permeable AMPARs and perturbs AMPAR-mediated excitatory synaptic transmission, leading to deficits in the behavioral dimensions shared between SZ and BP.

## BIOGRAPHY (upto 200 words)

Ho completed his PhD at The Johns Hopkins University School of Medicine, where he worked under the supervision of Dr. Akira Sawa. His presentation draws on the research conducted during his doctoral studies. He is now a postdoctoral fellow at the California Institute of Technology (Caltech). His research endeavors are funded by a Neuroscience Research Fellowship awarded by the American Academy of Neurology.

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