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Schizophrenia: Clinical, neuropsychological and genetic correlations

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Schizophrenia is a complex genetic disorder involving dysfunction in several brain regions, particularly the prefrontal and mesial temporal cortices and neurotransmitter systems such as glutamate. Evidence for cortical dysfunction has been observed by a variety of techniques. In prefrontal cortex, for example, patients exhibit cognitive, physiological and molecular alterations. Glutamatergic abnormalities have been inferred from several observations. In postmortem prefrontal cortex, alterations in several glutamate-related measures have been reported, including reduced mRNA levels of excitatory amino acid transporter 2 (EAAT2) also known as the glial glutamate transporter which plays an essential role in synaptic glutamate removal. These convergent findings suggest that some aspects of glutamate neurotransmission may be altered in prefrontal and mesial temporal regions in patients with schizophrenia and that these abnormalities could be partly genetic. Significant associations for complex genetic disorders such as schizophrenia often are difficult to replicate. This difficulty is not surprising given their presumed heterogeneity and the small effects of individual genes. Support for such associations can be enhanced by providing convergent biological and molecular evidence for functional effect of genes on pathophysiological mechanisms.

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Analysis of iPSC-derived dopaminergic neuron susceptibility to influenza and excitotoxicity in non-affective psychosis

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H1N1 virus susceptibility of iPSC-derived DA neurons from schizophrenia patients and controls will be compared. C57/BL-6 fibroblasts were reprogrammed into iPSCs using a lenti-viral vector containing SOKM genes. Pluripotency verification with the AP assay and immunocytochemistry ensured iPSC presence. The experimental outcome of ISPCs from DA neuron differentiation will be discussed in the Results section. Fibroblasts from patients and controls will be reprogrammed into iPSCs using a sendai-virus vector containing SOKM. IPSCs will be characterized using the AP assay, immunocytochemistry, and RT-PCR. IPSCs will then be differentiated into DA neurons. Gene methylation will be compared for both groups with custom-designed microarrays.

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