

Novel Biomarker for Schizophrenia (Ramot)

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Novel Biomarkers for Schizophrenia

RNA editing is thought to be a major contributor to brain development and function [1]. A-to-I RNA editing is an enzymatic conversion of genomically encoded Adenosine to Inosine that is recognized as Guanosine by the cellular translational machinery. Such RNA editing events can “re-code” the pre-mRNA message and may lead to modifications of the biochemical properties of the expressed proteins. Studies have shown that proper RNA editing is essential for sustaining neural functioning mediated by the fine tuning of proteins involved in brain neurotransmission. In the current study, we aimed to detect and determine the levels of A-to-G RNA editing of target sites located in coding DNA sequence of genes that are highly associated with neuronal plasticity and function in RNA samples extracted from BA10 area of schizophrenia patients compared to non-affected controls.

Target enrichment and Library preparation: The method can be described as low input multi sample targeted DNA enrichment system that is coupled to Next-generation sequencing machine for in-parallel targeted re-sequencing. This platform enables the parallel amplification and sequencing of over a hundred of RNA editing sites, most of which are located in the coding sequence in a single experiment- facilitating high throughput profiling of RNA editing patterns.

Using a high throughput DNA sequencing platform, we were able to show a general decrease in brain RNA editing levels in schizophrenia samples compared to controls, and identify novel genes that are subjected to deregulated RNA editing that may be relevant to the pathophysiology of schizophrenia.

These results suggest that aberrant A-I RNA editing may play a role in the etiology of schizophrenia and that specific RNA editing pattern might serve as a biomarker for the disease.

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