## **POSTER PRESENTATION**



# Evaluating computational models of language disturbance in schizophrenia

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Abnormal brain processes that underlie schizophrenia are incompletely understood, and no current laboratory or imaging technique can reliably identify individuals with schizophrenia. Instead, diagnosis of this disorder relies on symptoms observed in clinical interviews using conversational language. The symptoms of schizophrenia are complex and span a wide range of altered behavior and perception, including delusions (fixed false beliefs) and derailment (fluent speech that fails to follow a coherent discourse plan).

Establishing the brain processes underlying the symptoms of schizophrenia would greatly advance our understanding of this disorder. Yet after almost a century of clinical research, they remain incompletely understood. Only recently, theories have begun to emerge that have the potential to explain how abnormalities at the brain level might lead to the emergence of symptoms. One recent theory that has been widely endorsed in the psychiatric literature, put forth by Kapur [1], is based on the view that dopamine (DA) mediates the significance, or salience, of subjective experience. Kapur proposes that in schizophrenia, an overabundance of midbrain DA activity leads to a pathological enhancement of salience, which in turn causes psychotic symptoms. Delusions, for example, are explained as secondary reactions to an altered experience of the world - i.e. as an attempt by the brain to make sense of the excessive significance assigned to insignificant events.

The theory that some symptoms of schizophrenia are caused by excessive salience forms the theoretical basis of the present computational study. Using DISCERN [2], a neural network-based model of human story understanding and recall, excessive salience (and thus excessive DA release) was simulated using artificially high network learning rates. This "hyperlearning" hypothesis converges with other recent evidence linking abnormal response to prediction error to psychosis and delusions [3,4]. See [5] for a detailed description of both DIS-CERN and the hyperlearning simulation.

The language disturbances caused by the hyperlearning simulation were compared to those caused by other simulated pathologies designed to model alternative hypotheses on the causes of schizophrenia, including loss of connectivity, working memory distortion, and overactivation of semantic memory, among others. The different simulated pathologies were evaluated quantitatively with respect to their ability to match patient data from a parallel clinical study of story recall in patients with schizophrenia-spectrum disorders.

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