

T. Nathan Mundhenk, Michael A. Arbib
University of Southern California
Computer Science Department

Modeling schizophrenia and agency recognition. Understanding how it is we know what we did.

We have developed a model of pre-frontal cortical (PFC) areas that attempts to explain the seemingly diverse and unusual symptoms observed in patients diagnosed with schizophrenia. The present presentation will focus on how the model explains why such patients at times attribute their actions to another agent. The model's first basic assumption is that since dopaminergic and serotonergic neurons seem to have a normal morphology but are directly affected by drugs used to treat schizophrenia, their role is secondary but important. However, given recent findings of abnormally decreased functioning in GABAergic Chandelier neurons in schizophrenics we have hypothesized that their role may be more primary. The model explains the effects of the strong dopamine antagonists usually referred to by the name 'typical' neuroleptics as influencing the gain of dopaminergic neurons that correlate agent and action. It is hypothesized that such neural correlating activities bind what action we execute to what action we expect. Further, the dopaminergic neurons are hypothesized to be responsible for modulating Chandelier neurons, which regulate or clear memory fields in PFC. The model also suggests why the symptoms of schizophrenia are reduced by the newer 'atypical' neuroleptics as they act to strongly inhibit memory fields via strong serotonin antagonism. Thus, they inhibit neurons that would have otherwise been inhibited by faulty Chandelier neurons. From this, auditory verbal hallucinations and alien hand sign are explained as a result of residual memory left from a previous action that should have been wiped by Chandelier neuron suppression. This creates a mismatch between memory fields for agency and action that causes an individual with schizophrenia to attribute an action that they executed to another agent.