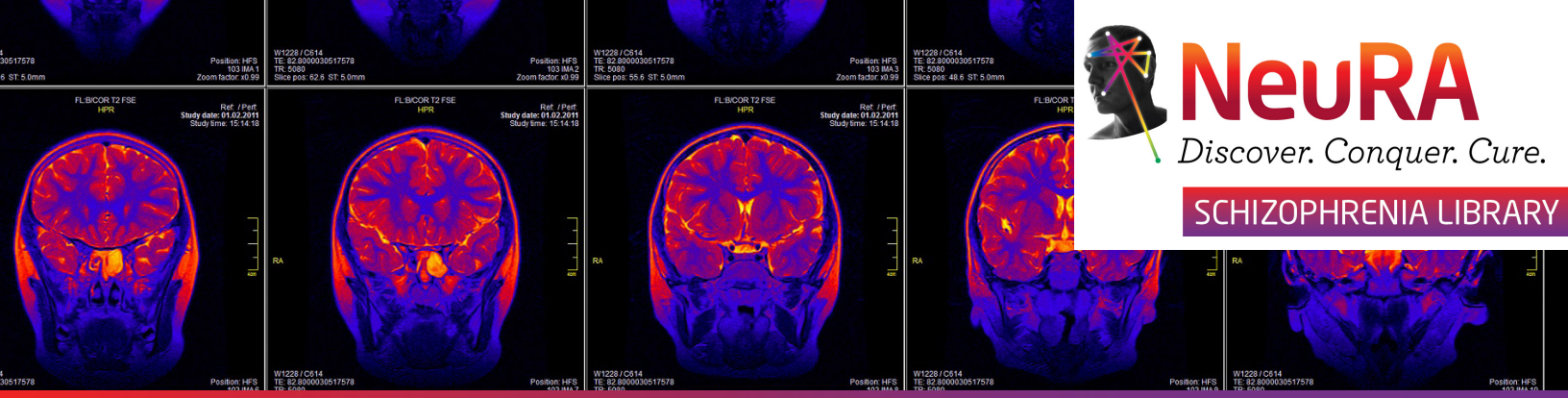

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SCHIZOPHRENIA Factsheet

October 2020

What are neurometabolites?

Products of normal chemical metabolism may be altered in schizophrenia. Changes in metabolite levels may be indicative of altered biochemical activity. Magnetic resonance spectroscopy has been used to measure levels of neurometabolites, such as N-acetylaspartate, creatine, choline, myo-inositol and glutamine. These derivatives are indirect indicators of biochemical activity. Alterations in the N-acetylaspartate/creatine ratio is associated with the protective myelin sheath surrounding neurons, which is used as a marker of neural cell viability. Decreased levels of N-acetylaspartate are associated with neuron death, or injury to the part of the neuron that connects to other cells, the axon.

What is the evidence for neurometabolites?

High quality evidence found a small decrease in myo-inositol levels in the medial prefrontal region in people with schizophrenia compared to controls. Moderate to high quality evidence found N-acetylaspartate levels were reduced in the frontal lobe, temporal lobe, thalamus, hippocampus, cerebellum, and cingulate cortex. Lower quality evidence also found reduced N-acetylaspartate in the parietal lobe, basal ganglia, and occipital lobe (white matter only). N-acetylaspartate may be increased in the striatum and the lenticular nucleus. There were small to medium-sized reductions in glutamate, and increases in glutamine, in the frontal cortex of people with schizophrenia, which may progress with age.

In unmedicated people with schizophrenia (drug naive or drug free), there were reductions in N-acetylaspartate in the thalamus and in frontal white matter (using <3T MRI scanners only), and medium-sized increases in glutamate+glutamine in the medial prefrontal cortex, and increases in choline in the basal ganglia. There were no changes in glutamate, creatine or myo-inositol in unmedicated patients.

In first-degree relatives of people with schizophrenia, there were N-acetylaspartate reductions in the anterior cingulate and in the hippocampus. In people at either clinical or genetic high-risk of schizophrenia, there were N-acetylaspartate reductions in the thalamus and in the N-acetylaspartate/creatine ratio in the prefrontal cortex.

For more information see the technical table

NeuRA
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NeuRA (Neuroscience Research Australia) is one of the largest independent medical and clinical research institutes in Australia and an international leader in neurological research.

Diseases of the brain and nervous system pose the greatest health, economic and social burden of any disease group because they are chronic, debilitating and have no known cures.

Medical research is the cornerstone of efforts to advance the health and wellbeing of families and the community. Our dedicated scientists are focussed on transforming their research into significant and practical benefits for all patients.

While we hope you find this information useful, it is always important to discuss any questions about schizophrenia or its treatment with your doctor or other health care provider.

HOW YOUR SUPPORT HELPS

We are able to make significant advances due to the generosity of countless people. Your donation allows us to continue to work towards transforming lives. For information on how you can support our research, phone **1300 888 019** or make a secure donation at **neura.edu.au/donate/schizophrenia**.

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