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SCHIZOPHRENIA Factsheet

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What is oxidative stress?

While oxygen is a vital component of life, some oxygen-based compounds called free radicals can be toxic due to their highly unstable nature. The key free radical classes are the reactive oxygen species (ROS) and reactive nitrogen species (RNS), and they are formed as by-products of normal metabolism. Under normal conditions, these free radicals are tightly monitored and controlled by stringent protective barriers, such as their rapid removal from cells; and antioxidant enzymes that break them down. At these tightly maintained concentrations, free radicals play an important role in cellular signalling, immune responses and cell growth. However, excess free radicals can result from interruption of the antioxidant defense barrier, or from excess production. This can cause oxidative stress, resulting in structural damage to cellular proteins, fats, carbohydrates and nucleic acids (DNA and RNA). Severe oxidative stress can result in failure of cell growth, apoptosis and cell necrosis.

What is the evidence for oxidative stress?

High quality evidence finds medium-sized reductions in glutathione and glutathione peroxidase levels in people with schizophrenia compared to controls, and no differences in glutathione disulfide or gluthatione reductase levels.

In people with first-episode psychosis, moderate quality evidence finds large reductions in total antioxidant levels in plasma or serum compared to controls.

In people with acute relapse of psychosis there are decreases in catalase, superoxide dismutase, and glutathione peroxidase in red blood cells.

In stable outpatients with schizophrenia, moderate quality evidence finds medium-sized reductions in superoxide dismutase in red blood cells, and medium to large increases in superoxide dismutase in serum. There are also increases in thiobarbituric acid reactive substances in serum, catalase in red blood cells, and nitrate in plasma of stable outpatients. There are no differences in thiobarbituric acid reactive substances in plasma or glutathione peroxidase in red blood cells.

In chronic inpatients, moderate quality evidence suggests a large effect of increased malondialdehyde in plasma. There are medium to large effects of decreased catalase in both red blood cells and plasma, decreased vitamins C and E in plasma, decreased glutathione peroxidase in both red blood cells and plasma, and decreased superoxide dismutase in plasma. There are no differences in superoxide dismutase in red blood cells, or thiobarbituric acid reactive substances or uric acid in plasma.

Moderate quality evidence finds no differences in catalase, glutathione, glutathione peroxidase, superoxide dismutase, total antioxidant status or cell/DNA oxidative damage in people with early-onset schizophrenia (<18 years) compared to age-matched controls.

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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 enefits 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.

For more information see the technical table

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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