

The metabolic syndrome in schizophrenia: is inflammation a contributing cause?

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Abstract

This non-systematic review of the literature summarizes the evidence that inflammation plays a major role in the psychopathology of schizophrenia and in the mechanisms that contribute to physical ill health that is commonly associated with schizophrenia. The impact of prenatal infections on the developing brain, the possible genetic link between the human lymphocyte antigen gene, inflammation, heart disease and diabetes, together with the increase in pro-inflammatory cytokines in the blood and cerebrospinal fluid provide convincing evidence that inflammation is a major factor in the pathology of this disorder. The changes in immune-related markers and specific neurotransmitters associated with the positive symptoms of schizophrenia are described. In addition, the possible mechanism whereby structural changes occur in the brain is associated with the neurotoxic effects of pro-inflammatory cytokines, together with the neurotoxic metabolites from the tryptophan–kynurenine pathway that is activated by pro-inflammatory cytokines, is also discussed. The role of effective antipsychotic drug treatment in attenuating the inflammatory response is described. However, evidence is limited regarding the causal connection between atypical antipsychotic drugs and the changes in glucose and lipid metabolism that could trigger the onset of physical ill health, including diabetes and heart disease. Indeed, there is evidence that there is a metabolic predisposition to diabetes in patients with schizophrenia that is exacerbated by obesity and thereby contributes to cardiovascular disease and other co-morbid illnesses. It is concluded that the effects of inflammatory mediators on the brain causally contribute to the pathology of schizophrenia and the ill health that accompanies the disorder.

Keywords

Antipsychotics, cytokines, diabetes, ill health, kynurenine pathway, schizophrenia