suggests that HE is an autoimmune disorder instead of thyroid disease.

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Brain metabolic abnormalities in schizophrenia patients

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Introduction Main schizophrenia symptoms result from abnormalities in brain function, such as hypofrontality and structural deficits on the prefrontal-thalamic-cerebellar circuit, as shown in brain imaging studies in first-episode SCZ patients. Whether metabolic alterations may be underlying these events is being studied thoroughly.

Objectives/aims To assess brain metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin, brain. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Lower metabolic activity in the frontal regions of the brain is associated to an increase in norepinephrine transmission and decrease in dopaminergic transmission with reduced dopamine efflux in the frontal cortex. This seems to lead to cellular changes resulting in resulting lower blood flow and glucose demand. Molecular analysis of postmortem SCZ patients' brains has indicated alterations in glucose metabolism and insulin signalling pathways, showing evidence for prefrontal cortex decreased expression of glucose metabolism, namely glycolytic enzymes such as glyceraldehyde 3-phosphate dehydrogenase, hexokinase, phosphoglycerate mutase, enolase and pyruvate kinase and decreased levels and phosphorylation of the insulin receptor and insulin signalling proteins AKT1 and GSK3β. Significantly elevated glucose concentrations in cerebrospinal fluid were observed in SCZ patients, but with no serum levels differences. A SCZ brain specific increased glucose could be explained by preferential utilization of lactate, predominantly produced by astrocytes, over glucose as an energy substrate.

Conclusions Abnormalities in brain glucose metabolism and insulin signalling seem to appear in early stages of SCZ, suggesting a role in SCZ onset and pathophysiology.

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Peripheric metabolic abnormalities in schizophrenia patients

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Introduction Schizophrenia (SCZ) is frequently associated with metabolic symptoms including dyslipidaemia, hyperinsulinemia, type 2 diabetes and obesity. In fact, SCZ patients have been reported to present higher prevalence of these conditions than general population, commonly associated to second generation antipsychotic therapy. Recent studies, however, have demonstrated that peripheral metabolic disturbances can appear at disease onset or drug-naïve patients.

Objectives/aims To assess metabolic disturbances in first episode and/or drug-naïve SCZ patients.

Methods We conducted a literature review through Pubmed search for MeSH: schizophrenia, metabolism, glucose, insulin. Controlled studies on first episode and/or drug-naïve SCZ patients were included.

Results Several studies showed no change in SCZ patients' fasting blood glucose, while others found increased glucose levels and impaired glucose tolerance in SCZ patients compared to healthy controls in several recent studies. Hyperinsulinemia and insulin resistance have also been identified in antipsychotic-naïve SCZ patients and it has been suggested that early onset patients are more likely to present insulin resistance. In addition, there's evidence of increased circulating levels of chromogranin A, pancreatic polypeptide, prolactin, cortisol, progesterone, thus emphasising that multiple components of the hypothalamic-pituitary-adrenal-gonadal axis may be affected in SCZ. These elevations were associated to normal glycaemia suggesting there may be insulin intolerance during early stages of SCZ, requiring an increased secretion from pancreatic Bcells to maintain normal glucose levels.

Conclusions Recent studies of first onset and/or drug-free schizophrenia patients have shown impaired fasting glucose tolerance, hyperinsulinemia and insulin intolerance, suggesting that metabolic abnormalities may play a role in SCZ onset and pathophysiology.

Disclosure of interest The authors have not supplied their declaration of competing interest.

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Systemic review: High dose olanzapine treatment for treatment resistant schizophrenia

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Objectives Schizophrenia is a major mental illness with a progressive course. Thirty percent of cases of patients with schizophrenia do not respond to adequate trials of at least 2 different groups of antipsychotics,are currently classified as having treatment resistant schizophrenia (TRS). Clozapine remains the gold standard, treatment of choice for TRS. However, clozapine does not come without its own challenges. Its risk profile, particularly agranulocytosis, reported in 1% of cases, has led to the necessity of weekly blood counts within the first 18 weeks of treatment and subsequently every month with slow dose titration. Clinically, sedation, weight gain and hypersalivation may further hamper the compliance of patients. Non-compliance has been reported to cause rebound psychosis. Recent studies have raised questions as to which antipsychotic is most efficacious for TRS. Thus, we conducted a systematic review of high dose olanzapine treatment for people with TRS.

Method A systematic review of prospective studies found through search of PubMed, Scopus and hand-searched key papers which included randomized controlled trials and open-label studies which looked at high dose of olanzapine treatment response for TRS.