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# SCHIZOPHRENIA- A SYNDROME OF INSULIN IMPAIRMENT

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Introduction: Schizophrenia is a human specific disease, unknown in other primates. Low adiposity during childhood was associated with a greater risk of developing schizophrenia, but this disease has a higher incidence in the obese children and adults, too.
Objectives: We developed a new hypothesis on the human evolution. According to it, changes in the activity of the IGF1 (insulin-like growth factor1)/insulin pathway (altered in human pathological conditions such as the psychiatric disorders) could be responsible for the appearance of certain specific human traits.
Aims: This hypothesis predicts that schizophrenia should result from a deviation of the normal IGF1/insulin signaling in brain in both directions. A drug that increases insulin resistance should improve the condition in the slim patients and worsen them in the obese patients.
Methods: A multi-center double-blind study on palmitate paliperidone (pp), the major active metabolite of the antipsychotic risperidone (that increases insulin resistance and stimulates weight gain), was conducted. 25-150 mg eq pp was injected intramuscular (into the deltoid muscle or the gluteal muscle) at 1 week interval for 1 year in a double blind study in patients with acute symptoms.
Results: Our preliminary observations based on the Positive and Negative Syndrome Scale (PANSS) total scores showed that the slim patients had statistically the best response to pp and the worse effects were observed in the obese patients. The study is still ongoing.
Conclusions: These results suggest that schizophrenia is a polarized disease with 2 major phenotypes- one with either insulin sensitivity or obesity and insulin resistance.

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