A NEW INSIGHT INTO PSYCHIATRIC DISORDERS AND DEMENTIA

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Introduction: Major psychiatric disorders (schizophrenia, bipolar disorder - BPD) and Alzheimer's disease (AD) are human-specific. Many people who develop symptoms of schizophrenia around 50+ years decline rapidly into dementia. Symptoms of older schizophrenic patients who do not develop this rapid decline in their mental capacities improve over time. Dementia is a complication of both schizophrenia and old age. Similar genetic defects are involved in both schizophrenia and BPD. Our hypothesis might explain the appearance and mechanism of specific human brain diseases.

Aims: Our hypothesis suggests the insulin/IGF1 pathway as a key biochemical instance which, due to alterations in its regulation undergone throughout human evolution, may be responsible for the appearance of pathological changes characteristic for certain psychiatric disorders.

Methods: We examined the explanatory character of this hypothesis for certain abnormal biochemical aspects characteristic for dementia and/or the improvement in old patients, as well as the resulting predictions of the insulin/IGF1 profile which may anticipate the evolution of the disease in these patients.

Results: Decreased insulin/IGF1 activity is associated with schizophrenia and BPD. But obese patients have higher incidences of psychiatric disorders. Patients with higher insulin sensitivity may improve over time, and the late onset of the disease as well as the mental decline may be associated with higher insulin resistance (risk factor for AD).

Conclusions: This hypothesis may provide new insights for human neurodegenerative diseases together with new therapeutic approaches.