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Sensory gating deficits, pattern completion, and disturbed fronto-limbic balance, a model for description of hallucinations and delusions in schizophrenia

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Abstract

View references (55)

Schizophrenia, if not the most difficult, is one of the most difficult mysterious puzzles for psychiatrists, psychologists, and neuroscientists to solve. In this paper, based on the previously known pathologies of schizophrenia, a new model is proposed for explanation of the formation of positive psychotic symptoms of hallucinations and delusions. This model can be used for understanding psychotic or psychotic-like positive symptoms of bipolar mood disorder, posttraumatic stress disorder, obsessive compulsive, and amphetamine and drug-induced psychotic disorders. Based on the postulated model, a spectral view on these disorders with psychotic features is also proposed. These pathologies include auditory sensory gating deficits in hippocampus, abnormal emotional coding in amygdala, pattern completion in thalamic and cortical areas, and disturbed fronto-limbic balance. This model includes anatomical and neurotransmitter defects of hippocampus, amygdala, thalamus, cingula, and prefrontal cortex and their interconnections. A role for hippocampal sensory gating deficits in the pathogenesis of positive psychotic symptoms and interrelation between amygdala and its dopamine level with hippocampus is speculated. This model also hires the interesting function of pattern completion in thalamus and cortical areas for a better explanation of the pathogenesis of hallucinations and delusional psychotic symptoms. Furthermore, there is also explanation for the polygenic etiology of the schizophrenic and psychotic disorders and relation between schizophrenia and bipolar mood disorder in anatomy and neural systems involved. A spectral view is proposed that explains the absence of clear cut border between different psychotic or psychotic-like disorders in their form and severity based on the involved genes and brain functional systems. Including excessive prefrontal pruning, there is also explanation for the appearance of positive psychotic symptoms in early adulthood. An explanation for the high dopamine level of amygdala despite its decreased size and abnormal anatomy is also suggested as a compensatory function which might explain the decline in positive psychotic symptoms when schizophrenics age according to amygdala burn out. Based on this model, speculations are provided for: late onset of the effects of antipsychotics on positive psychotic symptoms, mechanism for the therapeutic effect of serotonin type 2A receptor blockers and GABAergic medications in psychosis, role of smoking in diminution of psychotic symptoms, and relationship between biological and psychological issues in the formation of psychotic symptoms. Finally, based on this model, a new role for nicotinic cholinergic drugs (such as galantamine) for treatment of schizophrenia and other psychotic or psychotic-like disorders is proposed.

Reaxys Database Information

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Indexed Keywords

EMTREE drug terms: amphetamine; dopamine; dopamine receptor blocking agent; galantamine; haloperidol; neuroleptic agent; neurotransmitter; nicotinic agent; noradrenalin; serotonin 2A antagonist; serotonin 2A receptor

EMTREE medical terms: amygdaloid nucleus; anxiety disorder; article; auditory hallucination; auditory nervous system; bipolar disorder; bipolar mania; brain metabolism; cingulate gyrus; clinical feature; delusion; disease severity; dopamine brain level; drug mechanism; emotion; frontal lobe; GABAergic system; heredity; hippocampus; human; limbic system; model; neuroanatomy; neurologic disease; obsessive compulsive disorder; positive syndrome; posttraumatic stress disorder; prefrontal cortex; priority journal; psychosis; schizophrenia; sensory gating deficit; stimulus response; thalamus; visual hallucination