Schizophrenia
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Description

Schizophrenia is one of the most common psychiatric disorders—it affects roughly 1% of the population. The incidence of the disease increases tenfold among individuals who have a first-degree relative with schizophrenia. Symptoms of schizophrenia usually appear in late teens or early twenties (age 17-29), and it is extremely rare or non-existent after the age of 45.

Schizophrenia is mainly associated with positive symptoms, such as delusions and hallucinations. Examples of delusions include patients with schizophrenia may falsely believe that others are trying to deceive them, or that neighbors want to control their lives. Examples of hallucinations include hearing voices no one else hears. Although rare, patients with schizophrenia may also feel or smell things that do not exist. Patients with schizophrenia also experience negative symptoms, such as apathy, depression, and emotional withdrawal. This includes difficulty experiencing pleasure in everyday life. Lastly, patients with schizophrenia also show cognitive abnormalities, including disrupted thought processes and difficulty paying attention.

The frequency and severity of schizophrenia symptoms vary among patients. All of these three clusters of schizophrenia symptoms—positive symptoms, negative symptoms, and cognitive dysfunction—affect the quality of life of patients, and make it harder for them to lead a normal life.

Neural substrates of Schizophrenia

Many clinical research findings have implicated dopamine in the occurrence of positive symptoms, negative symptoms, and cognitive dysfunction in schizophrenia. Dopamine is produced in the midbrain and is projected to various brain areas, including the basal ganglia, hippocampus, and prefrontal cortex. Specifically, empirical studies that measured protein function associated with dopamine receptors implicates dopamine D1 receptors in the basal ganglia for the occurrence of negative symptoms in schizophrenia. Similarly, using PET scan, researchers found that the distribution of dopamine D1 receptors in the prefrontal cortex correlates with the severity of negative symptoms in schizophrenic patients. Interestingly, existing neural and clinical theories relate the occurrence of psychotic episodes in schizophrenia to either hippocampal, prefrontal, or dopaminergic dysfunction. Some argue that prefrontal dysfunction is the mechanism underlying the occurrence of psychotic symptoms in schizophrenic patients, such that psychosis is related to the maintenance of a large amount of irrelevant information in prefrontal working memory store. In contrast to the dopaminergic and prefrontal explanations for the occurrence of psychosis in schizophrenia, some argue that psychotic symptoms in schizophrenia are caused by hippocampal damage, which leads to excessive retrieval of unrelated information from memory.

Treatment

Antipsychotics are used to treat the psychiatric aspects, including positive symptoms, in schizophrenia, possibly by blocking D2 receptors and decreasing the effect of dopamine in the basal ganglia or hippocampus. It is debatable whether antipsychotics have any effect on negative symptom of schizophrenia. Some antipsychotics can cause Parkinsonism possibly because they inhibit D2 receptors. Interestingly, first-generation (typical) antipsychotics, such as haloperidol, have higher affinity to D2 receptors and more associated with Parkinsonism than second-generation (atypical) antipsychotics, such as clozapine. Interestingly, current research suggests that some novel D1 agonists can be used to treat negative symptoms and cognitive deficits in schizophrenia. This is, however, is in early clinical research investigation.

Prognosis

Because of the genetic link and the multiple neural atrophies, the development of effective drugs for schizophrenia has proved difficult, and currently there are no treatments that prevent schizophrenia.