

Schizophrenia 2

Subtypes of schizophrenia

Paranoid schizophrenia is the commonest form. It is characterized by • persecutory delusions, often systematized, and by persecutory auditory hallucinations. Thought disorder and affective, catatonic, and negative symptoms .are not prominent. Personality is relatively well preserved

In hebephrenic schizophrenia, also called disorganized schizophrenia, thought • disorder and affective symptoms are prominent. The mood is variable, with behaviour often appearing silly and unpredictable. Delusions and hallucinations are fleeting and not systematized. Mannerisms are common. Speech is rambling and incoherent, reflecting the thought disorder. Negative symptoms occur early, .and contribute to a poor prognosis

In catatonic schizophrenia, the most striking features are motor symptoms, •
formerly common, catatonic schizophrenia is now very rare, at least in
industrialized countries. Possible reasons for this include a change in the nature
of the illness, improvements in treatment, or past misdiagnosis of organic
.syndromes with catatonic symptoms

Simple schizophrenia is characterized by the insidious development of odd •
behaviour, social withdrawal, and declining performance at work. Positive
.symptoms are not apparent

Undifferentiated schizophrenia is the term used for cases that do not fit readily •
into any of the above subtypes, or where there are equally prominent features of
.more than one of them

Differential diagnosis

.Organic syndromes

Schizophrenia-like disorders can also occur, in clear consciousness, in a range of neurological and medical disorders. These conditions are referred to as a psychotic disorder due to another medical condition

Drug-induced states (including substance misuse). Certain prescribed drugs, particularly steroids and dopamine agonists, can cause florid psychotic states. Psychoactive substance misuse, particularly with psychostimulants or phencyclidine, and also alcohol, should always be considered in the presentation .of schizophrenia-like psychoses

.Mood disorders with psychotic features

.Delusional disorders

.Personality disorder

Epidemiology

Prevalence: the frequent statement that about 1 in 100 people develop schizophrenia, but the value of 7 in 1000 is statistically more appropriate

Age at onset: Schizophrenia can begin at any stage of life, from childhood to old age. The usual onset is between 15 and 54 years, most commonly in the mid-twenties; more detailed analyses suggest that there are two peaks, one at 20 years, and a second peak at 33 years

Gender: In addition to possible gender differences in age of onset, meta-analyses show that schizophrenia is more common in men than in women, with a male:female ratio of 1.4:1

Aetiology

Overview

Schizophrenia exemplifies the whole range of biological, psychological, and social factors considered to be important in psychiatric causation and the methods that have been applied to try to identify them

Genetics Family, twin, and adoption studies have all been applied extensively in schizophrenia, and cumulatively provide irrefutable evidence for a major genetic contribution to the syndrome. Such studies set the context for the current wave of genomic and molecular studies designed to identify the individual genes and mechanisms by which the risk for schizophrenia is mediated

Environmental risk factors Despite the prominent genetic component, the twin studies discussed earlier, along with many epidemiological and other studies, show that environmental factors are also important in the aetiology of schizophrenia. A range of factors, especially prenatal and perinatal ones, have been identified

Maternal infections Maternal malnutrition Birth complications Winter birth
Advanced paternal age Urban birth and upbringing Childhood trauma and
adversity Being an immigrant Cannabis smoking Tobacco smoking Life events

Psychological factors

Personality: it is difficult to distinguish between premorbid personality and the .prodromal phase of emerging illness

Psychological theories: In the past, psychological theories of schizophrenia were based on psychodynamic concepts and often centred on a pathogenic role of family. Such theories are now largely of historical interest, since they lack .empirical evidence

Neurobiology

Structural brain changes: Whether there is a neuropathology associated with schizophrenia has been a matter of debate for over a century. The search began with Alzheimer, who spent a decade studying the brains of patients with dementia praecox before he reported the case of presenile dementia with which his name is associated. The failure of Alzheimer and others to identify a neuropathology was central to the view of schizophrenia as a functional disorder rather than an organic one. However, evidence has accrued over the past 40 years which disproves the null hypothesis that there are no structural differences in the brain in schizophrenia, albeit their details and interpretation remain poorly understood, and the findings are not clinically useful in the diagnosis of individual patients

Dopamine: Two early lines of research converged and implicated dopamine as having a key role in schizophrenia. The first concerned the effects of amphetamine which, among other actions, releases dopamine at central synapses. Repeated use of amphetamine at high doses can induce a disorder similar to acute schizophrenia, and acute amphetamine administration worsens psychotic symptoms in people with schizophrenia. The second approach starts from the finding that all antipsychotic drugs are dopamine-receptor antagonists, and that their affinity at dopamine D2 receptors is the property that correlates best with their clinical potency. These observations led to the well-known 'dopamine hypothesis' of schizophrenia, which emerged during the early 1970s and has persisted

The cause of dopaminergic involvement in schizophrenia is unclear. There may be a genetic component, but current theories emphasize dysregulation secondary to glutamatergic and developmental abnormalities, as outlined below. A phenomenological account has been proposed by Kapur (2003). He argues that the known roles of dopamine in cognition and behaviour suggest that it mediates the 'salience' of external events and internal representations, and that in schizophrenia the dopamine abnormalities lead the patient to misattribute stimuli and their meaning. Delusions and hallucinations are viewed as the consequence of these experiences and the patient's attempt to make sense of them

Glutamate: The excitatory neurotransmitter amino acid glutamate is second only to dopamine in neurochemical theories of schizophrenia. The key finding that triggered this interest was that antagonists of the NMDA type of glutamate receptor (e.g. phencyclidine and ketamine) can induce a schizophrenia-like psychosis

Serotonin (5-hydroxytryptamine; 5-HT): A role for serotonin (5-HT) in schizophrenia has long been considered, because the hallucinogen, lysergic acid .diethylamide (LSD), is an agonist at 5-HT₂ receptors



SCHIZOPHRENIA

CAUSES



Genetics

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Structural changes in the brain

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Previous drug use

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Environmental risk

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SCHIZOPHRENIA

CAUSES

Researchers believe that a combination of genetics, brain chemistry and environment contributes to development of the disorder.



Genetics



Brain Chemistry



Environment

SCHIZOPHRENIA is a Serious Mental Disorder in Which People Interpret Reality Abnormally

People with Schizophrenia Require Lifelong Treatment



SYMPTOMS



Delusions



Hallucinations



Disorganized Speech



Lack of Initiative



Reduced Speech



Flattened Affect