Schizophrenia

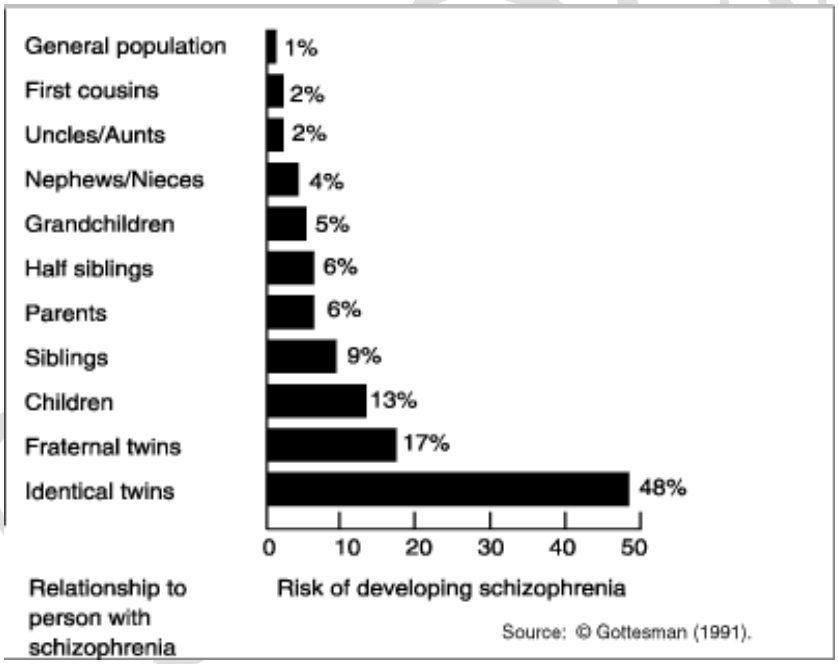
- It is a severe psychiatric disorder with symptoms of emotional instability, detachment from reality and withdrawal into self.
- It is an umbrella term used to outline a range of different psychiatric disorders that affect thoughts, emotions and behaviours. It is a psychotic disorder which involves a loss of contact with reality.
- It was first called "dementia praecox" (premature dementia).
- There are two main types of schizophrenia:
    - Type 1- Episodic with mainly positive symptoms
    - Type 2- Chronic with mainly negative symptoms
- There are 5 diagnostic subtypes:
    - Simple- People gradually withdraw themselves from reality
    - Paranoid- When people have delusional thoughts and hallucinations and may experience delusions of grandeur
    - Catatonic- When people have motor activity disturbances that may involve them sitting or standing in the same position for hours
    - Disorganised- When people have disorganized behaviour, thoughts and speech patterns. They may also experience auditory hallucinations
    - Residual- Patients who have once suffered from more extreme schizophrenia but who now only have milder symptoms such as bizarre thoughts, withdrawal and affective flattening.
    - Undifferentiated- When an individual does not fit into one of the types above but is still experiencing affected thoughts and behaviours
        - *DSM V- "The DSM-IV subtypes of schizophrenia (i.e., paranoid, disorganized, catatonic, undifferentiated, and residual types) are eliminated due to their limited diagnostic stability, low reliability, and poor validity. These subtypes also have not been shown to exhibit distinctive patterns of treatment response or longitudinal course. Instead, a dimensional approach to rating severity for the core symptoms of schizophrenia is included in Section III to capture the important heterogeneity in symptom type and severity expressed across individuals with psychotic disorders."*
- Diagnosis according to the DSM:
    - Shows two of the following for at least a month:
        - Delusions
        - Hallucinations
        - Disorganised speech
        - Disorganised or catatonic behaviour
        - Flattening of emotions
        - Continual voices in the head giving a running commentary of what is happening
    - Social and/or occupational dysfunction for at least 6 months
    - No evidence that medical factors are causing the behaviours
- Symptoms can be split into +ve and –ve:
    - +ve refers to the addition of certain behaviours such as hallucinations, delusions of grandeur or control and insertion of thoughts

- –ve refers to the removal of certain behaviours such as poverty of speech, withdrawal from society and flattening of mood and avolition (lack of will)

- Risk factors are thought to include low socio-economic status, minority ethnicity and urban residence
- Prognosis is better in non-industrialised areas
- Shows "rule of the thirds" in that 1/3 recover more or less completely; 1/3 have episodic impairment; 1/3 have a chronic decline
- Biological explanations include genetics, neurotransmitters and hormones, structural brain abnormalities
- Psychological explanations include family dynamics, life stress and urbanisation (Pedersen and Mortensen, 2001- As population density rises, so does prevalence of schizophrenia; 2 hypotheses: Causation and Migration; evidence points to causation)

EXPLANATIONS OF SCHIZOPHRENIA

1. Genetic- There is a link between schizophrenia and inherited genetic material and so the closer our genetic link is to someone diagnosed with schizophrenia, the more likely we are to be diagnosed ourselves.



Appears to support the genetic link. However, the highest risk was 48% (not 100%, indicating a wholly genetic trait) so it looks as if people may be born with a predisposition to develop schizophrenia and it is some environmental influence that ultimately causes it.

Fraternal= dizygotic twins

Identical= monozygotic twins

	Prevalence amongst biological relatives	Prevalence among adoptive relatives
Kety et al (1968) schizophrenia only	13%	2%
Tienari et al (1994) all 'severe' psych. diagnoses	30%	15%

- The study by Gottesman and Shields (1972) aimed to review research on genetic transmission of schizophrenia. They wrote a review article on 3 adoption studies and 5 twin studies into schizophrenia between 1967 and 1976 with a total of 711 participants in the adoption studies and 210 MZ and 319 DZ twin pairs were studied. They found 58% chances in MZ twins and 12% for DZ twins. Less in adoption and more in twins. The study had a large sample (reliable), was valid (cause-effect) but did not eliminate researcher bias.
- Studies of children adopted away at birth, where they're adopted away and their real parent was schizophrenic, but their adoptive parents do not have schizophrenia, show the same likelihood of developing schizophrenia in their life as if they were raised by a schizophrenic parent. So this has confirmed the role of genes.
- Yang et al (2013) analysed ten "candidate" genes that could be responsible for schizophrenia in a sample of 1,512 participants. While there was no single gene that appeared to be associated with schizophrenia, the DAO (D-Amino acid Oxidase) gene was strongly associated with schizophrenia in comparison to all of the other candidate genes. These results suggest that DAO, which is involved in the N-methyl-d-aspartate receptor regulation, signalling and glutamate metabolism, is the master gene of the genetic associations and interactions underlying schizophrenia. Besides, the interaction between DAO and RASD2 has provided an insight in integrating the glutamate and dopamine hypotheses of schizophrenia.
- Roofeh et al (2013) noted that the human leukocyte antigen region of a genome could well be a plausible cause for some types of schizophrenia.
- A study conducted by Tienari et al (1991) showed that a mother with schizophrenia increased the chance of it in the adoptees, but only if the adoptive family were themselves psychologically disturbed in some way. So there does appear to be a genetic basis to schizophrenia BUT even vulnerable individuals can be protected from schizophrenia if their upbringing is healthy. This shows the importance of an interaction approach to the condition, such as a diathesis-stress model.
- The genetic explanation is deterministic and reductionist.

2. Biochemical- The Dopamine Hypothesis

    - This theory suggests that an excess of dopamine is implicated in schizophrenia.
    - Evidence was presented when people who did not have the illness experimented with drugs such as cocaine, amphetamine or methamphetamine which are drugs that

stimulate the release of dopamine in the brain and trigger the symptoms of schizophrenia. This is called amphetamine psychosis.

- Drug treatment such as prescribing "phenothiazines" does help to treat some of the symptoms of schizophrenia but these drugs bring about symptoms similar to Parkinson's disease which is caused by LOW levels of dopamine.
- Dopamine is a neurotransmitter and hormone released by the brain and is involved in the functions of movement, memory, pleasurable rewards, sleep, cognition, etc. It is also a precursor to other hormones such as adrenaline.
- Linstroem et al (1999) used a PET scan to test out the dopamine hypothesis. 10 schizophrenics and 10 healthy controls were injected with a radioactively labelled chemical called L-DOPA which is used in the production of dopamine. The PET scan could trace its usage in all participants and was taken up significantly faster in the schizophrenics, pointing towards them producing more dopamine.
- The evidence linking dopamine and schizophrenia is mostly correlational and so we don't know which causes which and this problem of "direction of causality" is there with the genetics link as well.
- This explanation is reductionist and over-simplistic as new treatments show glutamate and serotonin link to schizophrenia as well.
- The success of treatments which block dopamine receptors doesn't necessarily mean that dopamine caused the condition in the first place. This is the **treatment aetiology fallacy**! Taking paracetomol relieves a headache but does not mean the headache was cause by a lack paracetomol. This is an issue for **ALL DRUG TREATMENTS**!

3. Cognitive approach- This idea states that schizophrenia is caused by faulty information processing.
    - These include misattributed speech and behaviour, faulty self-monitoring and deficits in short term memory and semantic memory
    - He believes that most symptoms can be explained by cognitive impairment by the following three factors which form the "metarepresentation" system:
        i. Inability to generate voluntary action→ avolition
        ii. Inability to monitor voluntary action
        iii. Inability to monitor beliefs and intentions of others(lacks theory of mind)→ paranoid delusions
    - This is due to functional disconnection between frontal areas of the brain concerned with action and more posterior areas of the brain that control perception. He has produced evidence for his ideas by detecting changes in cerebral blood flow in the brains of schizophrenics when engaged in specific cognitive tasks.
    - This shows a nice overlap between cognitive and biological explanations and so it is a bit more holistic as compared to the other explanations but is still reductionist as he does not consider environmental factors.
    - Johnson et al (2013) tested the cognitive abilities of 99 schizophrenics and 77 healthy controls on a battery of cognitive tests. It was seen that the schizophrenics performed worse across all cognitive tests including those for working memory (which involve tasks such dealing with inner speech) and that this might be the core determinant of overall cognitive impairment in schizophrenics.