Diagnosis and classification

Classification – ICD10 and DSM-5 differ slightly in their classification of SZ. DSM-5 = one positive symptom must b present. ICD=two or more neg symptoms sufficient.

Positive symptoms – additional experiences beyond those of ordinary existence. Hallucinations and delusions. *Hallucinations* – sensory experiences that have either no basis in reality or are distorted perceptions of things that are there. *Delusions* – irrational beliefs. E.g. belief that you are Jesus or Napoleon. Delusions of persecution – government or aliens are after you.

Negative symptoms – *Avolition* – finding it difficult to begin or keep up with goal-directed activity. Reduce in motivation. Poor hygiene, grooming. *Speech poverty* – changes in patterns of speech. Reduction in the amount and quality of speech. Speech disorganisation – incoherent or speaker changes topic mid-sentence.

Biological explanations

Genes – SZ runs in families. Candidate genes – these confer a small increased risk of developing SZ. Polygenic – requires a number of factors to work in combination. Ripke – 108 separate genetic variations. Genes associated with increased risk included those coding for the functioning of a number of neurotransmitters inc. dopamine. **Dopamine hypothesis-** Hyperdopaminergia in the subcortex – high levels of dopamine in subcortex. E.g. excess in Broca's area associated with poverty of speech and auditory hallucinations. Hypodopaminergia in the cortex – low levels of dopamine in the prefrontal cortex in negative symptoms of SZ. **Neural correlates –** measurements of the structure or function of the brain that correlate with an experience. **Negative symptoms** – avolition – ventral striatum abnormalities. Lower levels of activity than control participants. Neg correlation between activity levels in the ventral striatum and the severity of overall negative symptoms. **Positive symptoms –** auditory hallucinations. Lower levels in the superior temporal gyrus and anterior cingulate gyrus. ☹mixed evidence for the dopamine hypothesis – agonists - increase dopamine and makes SZ worse BUT other neurotransmitters are involved e.g. glutamate. ☹ possible that SZ symptoms mean less info passes through the striatum, resulting in reduced activity.☺support for genetics.

Biological treatments

Typical antipsychotics – Chlorpromazine – work by acting as antagonists. They block dopamine receptors in the synapses of the brain, reducing the action of dopamine. It is also an effective sedative therefore can be used to calm patients. **Atypical antipsychotics –** Clozapine – used when other treatment fail. People need regular blood tests to ensure they are not developing agranulocytosis. Binds to dopamine receptors but also acts on serotonin and glutamate receptors. It reduces depression and anxiety. Risperidone – developed in the 90s. binds more strongly than clozapine. ☺ evidence for effectiveness – relapse rate is lower, ☹side effects –from mild to fatal. Tardive dyskinesia - involuntary facial movements ☹ dopamine hypotheses not full explanation and drug treatments are based on this hypothesis.

Psychological explanations

Family dysfunction

Schizophrenogenic mother – schizophrenogenic means 'schizophrenia-causing'. A mother who is cold, rejecting and controlling and tends to create a family climate characterized by tension and secrecy. Leads to distrust and then paranoid delusions and then SZ. **Double-bind –** communication style in the family. Child trapped in situations where they fear they are doing the wrong thing, receives mixed messages about what this is and unable to comment on the unfairness of the situation. When wrong they are punished by withdrawal of love. World is confused and dangerous ad leads to disorgnised thinking and paranoid delusions. Just a risk factor. **Expressed emotion -** level of negative emotion by carers. Verbal criticism (occasional violence), hostility (anger and rejection), emotional over-involvement. These cause stress. Can cause relapse. ☹support for family relationships – 69% of women with SZ had history of abuse in childhood ☹ parent blaming.

Cognitive explanations

Disruption to normal thought processing in the ventral striatum – negative symptoms. Reduced processing of the temporal and cingulate gyri is linked with hallucinations. **Dysfunctional thought processing – metarepresentation –** disrupts our ability to recognise our own actions and thought as being carried out by ourselves rather than someone else. **Central control –** ability to suppress automatic response. Disorganized speech and though disorder could result from inability to suppress automatic thoughts. ☺ Evidence – stroop task – patients took twice as long.

Psychological therapies

CBT: 5-20 sessions. Aim to identify irrational thoughts and trying to change them. Therapist to suggest less threatening possibilities. Useful for patients to see where their symptoms are coming from. This does not get rid of symptoms but helps someone with SZ hope this them. **Family therapy:** families involved in treatment. Help reduce stress within the family that may contribute to relapse. Strategies include – improving ability of the family to anticipate and solve problems, helping the family to balance between looking after person with SZ and maintaining their own lives and improving family's beliefs about SZ. **Token economies –** reward systems used to manage the behaviour of patients. Tokens are given immediately to patients when they have carried out a desirable behaviour e.g. getting dressed. These are then swapped for rewards e.g. sweets, magazines or a walk outside of the hospital. ☺ effective – CBT sig but fairly small effect on positive and neg symptoms. ☺☹treatments improve quality of life but do not cure ☹ethics – token economies are controversial.

Reliability and validity

Reliability – consistency. Inter-rater reliability – extent to which different assessors agree in their assessments. Extent to which two or more mental health professional arrive at the same diagnosis for the same patients. **Validity –** measuring what we intend to measure. Criterion validity – do different assessment systems arrive at the same diagnosis for the same patient? Research – more likely to be diagnose by ICD than DSM. Therefore poor validity. **Co-morbidity:** two or more conditions occurring together. SZ patients have also been diagnosed with depression (50%), substance abuse (47%), PTSD (29%) and OCD (23%). Are we bad at telling the difference or are they a single condition? **Symptom overlap –** overlap between symptoms of SZ and other conditions. E.g. bipolar and SZ both have delusions and avolition. ICD -patient would have SZ, DSM – bipolar. **Gender bias** – men may be more genetically vulnerable or gender bias. Some women may not be diagnosed with SZ as they are higher functioning. Underdiagnosed in women. **Cultural bias –** African Americans and afro-Caribbean origin are several times more likely than white people to be diagnosed. Not genetic vulnerability but culture bias. Hearing voices acceptable in African cultures – seen as bizarre in different cultures.

Interactionist approach –

Diathesis-stress model suggests that both a vulnerability to SZ and a stress-trigger are necessary in order to develop SZ. **Meehl's model:** original model, suggested diathesis was entirely genetic as a result of a schizogene. **Modern understanding of diathesis** – many genes increase the vulnerability slightly. It can also include trauma. HPA system can become over-active, making the person more vulnerable to later stress. **Modern understanding of stress –** cannabis is a stressor because it increases the risk of SZ by up to 7 times. **Treatment –** combining both medication and CBT. ☺evidence – adoptive parents study ☹original model is over simple – stress can come in many forms, including biological ☺ combination treatments are effective.

Schizophrenia