**Schizophrenia**

**Classification and diagnosis:**

*Clinical characteristics according to DSM:*

* Positive symptoms – excess or distortion of normal functions, e.g. experience of control, delusions, hallucinations, and disordered thinking.
* Negative symptoms – reflect a diminution or loss of normal functions, often persistent during periods of low or absent positive symptoms, e.g. affective flattening, alogia, avolition.
* Diagnosis of SZ – under DSM-IVR, requires at least one-month duration of two or more positive symptoms.

*Issues with reliability:*

Inter-rater reliability – extent to which two independent psychiatrists give consistent diagnosis for an individual. Rosenhan (1973) ‘on being sane in insane places’ demonstrated problems with reliability in diagnosis – ‘normal’ people presented themselves to psychiatric hospitals claiming they heard an unfamiliar voice saying ‘empty’, ‘hollow’, and ‘thud. They were all admitted for SZ. Took staff two weeks to recognise they were ‘normal’. Follow-up study, Rosenhan – warned hospitals of his intention to send out more ‘pseudo patients’. This resulted in a 21% detection rate, although he had sent nobody.

Issue – as need a reliable diagnosis to provide effective and tailored treatments.

DSM-III designed to provide an objective and reliable system for classifying psychiatric disorders. Therefore, should lead to greater agreement over the diagnosis of SZ.

* - Despite claims for increased reliability in DSM-III, over 30 years there has been little evidence that the DSM-III has been routinely used with high-reliability by mental health clinicians.
* - Whaley (2000) – found inter-rater reliability correlations in the diagnosis of SZ to be as low as +.11.
* - ICD + DSM – used to improve reliability. However, inconsistencies between the two criteria change over means not everyone uses them. Some still use outdated criteria, e.g. Schneider criteria.
* - Scheff (1966) – using the DSM/ICD can lead to labelling and stigmitisation, therefore patients may experience self-fulfilling prophecy and difficulty maintaining relationship/jobs.
* - Cultural differences – reliability questioned by variations in diagnosis across country and culture. E.g. Copeland (1971), gave 134 US and 194 British psychiatrists a description of a patient. 69% of US diagnosed SZ compared to only 2% of British.

*Issues with validity:*

Comorbidity – the extent that two (or more) conditions co-occur. Psychiatric comorbidities are common among patients with SZ, e.g. substance abuse, anxiety, and symptoms of depression. Comorbidity creates difficulty in the diagnosis and treatment of a disorder.

* + Buckley et al. (2009) – estimates that comorbid depression occurs in 50% of patients, and 47% have a lifetime diagnosis of comorbid substance abuse.
* - Problem of being diagnosed with SZ – can lead to patients receiving a lower standard of medical care for other comorbid (physical/psychological problems, this in turn adversely affects their prognosis).
* - Comorbidity and suicide risks – persons with SZ poses a high risk of suicide, with comorbid depression being a major cause for suicidal behaviour. E.g. Kessler et al. (1994), rate for attempted suicide rose from 1% for those with SZ alone to 40% for those with at least one lifetime comorbid mood disorder.
* - Large range of symptoms, sub-types, and mixed disorder categories – DSM/ICD make it difficult to define and diagnose when a person actually has SZ, and might suggest it is not one distinct disorder. E.g. Ellason and Ross (1995), pointed out that people with dissociative identity disorder (DID) actually have ore SZ symptoms than people diagnosed with SZ.
* - Some e.g. SZAZ question with SZ exists at all. However, has been widely discredited.

**Biological explanations of SZ:**

*Genetic factor:*

SZ is caused by underlying faulty genetics. Passed on through generations.

Family studies evidence – find individuals who have SZ and determine whether their biological relatives are similarly affected more often than non-biological relatives, SZ more common among biological relatives and the closer the degree of genetic relatedness, the greater the risk.

* + Gottesman (1991) – concordance rates of SZ, 46% for children with two SZ parents, 13% for one SZ parent and 9% for one SZ sibling.
* - Issues with family studies - concordance rates may be due to common rearing patterns and environment (negative emotional climate, EE families) rather than hereditary. Correlational study. Retrospective. Concordance rates for two SZ parents not 100%.

Twin studies evidence – the greater the concordance rates in MZ twins (twins who share 100% of their genes) the greater the influence of genetics over environment.

* + Joseph (2004) – concordance rates for MZ twins 40.4% compared to 7.4% for DZ twins.
* - Issues with twin studies – MZ twins are more likely to be treated more similarly, encounter more similar environments, and experience more ‘identity confusion’, so are more likely to produce higher concordance rate. MZ not 100%.

Adoption studies evidence – compare concordance rates of SZ in biologically related individuals who have been reared apart, so have not experienced the same environment.

* + Tienari et al. (2000) – Finland, of 164 adoptees whose biological mothers had been diagnosed with SZ, 6.7% also received a diagnosis of SZ compared to just 2% of the control group (197 control adoptees).
* - Wahlberg et al. (2000) – re-examined data, found only children adopted into families with poor communication have been found to be at increased risk.
* - If schizo-spectrum series not included, no significant results found.
* - Yet to identify a specific gene – therefore difficult to understand and prevent the precise mechanism of genetic transmission.
* - Reductionist.

*Dopamine hypothesis:*

Messages from neurons that transmit dopamine fire too easily or too often, leading to characteristic symptoms of SZ. SZs are thought to have abnormally high numbers of D2 receptors on receiving neurons, resulting in more dopamine binding and therefore more neurons firing. Dopamine plays a key role in guiding attention, so disturbances in this process may well lead to the problems relating to attention perception.

* + Antipsychotic drugs – block the activity of dopamine in the brain. Davis et al. (1980), met-analysis of 29 studies that analysed the effectiveness of antipsychotic drugs compared with a placebo. Relapse occurred in 55% of the placebo patients compared to only 19% of the antipsychotic group.
* + Post-mortem studies – Seeman (1987), SZ patients show an increase of dopamine in parts of the brain. However, PET scans (of live people) haven’t found any evidence.
* + Parkinson’s disease – low levels of dopamine activity are found in people who suffer from Parkinson’s disease, those how took L-Dopa to increase dopamine levels developed schizophrenic-type symptoms.
* + Amphetamines – dopamine antagonist, stimulating nerve cells containing dopamine causing the synapses to be flooded with this neurotransmitter. Large doses can cause characteristics of SZ, e.g. hallucinations and delusions.
* + Strong empirical support.
* - Cause and effect issues.
* - Aetiology fallacy – just because a drug reduces the symptom, doesn’t mean it causes it (increase of dopamine).
* - Diathesis stress model.

**Biological therapies for SZ:**

Anti-psychotic drugs – Conventional antipsychotics (chlorpromazine), dopamine antagonists in that they bind to D2 receptors but do not stimulate them, thus blocking their action and reducing positive symptoms, e.g. hallucinations and delusions. Atypical antipsychotics (chlozapine), only temporarily occupy D2 receptors and then rapidly dissociate to allow normal dopamine transmission, it is this characteristic which is believed to be the cause of less side-effects.

* + Relapse rates – Davis et al. (1980), meta-analysis of 29 studies that analysed the effectiveness of antipsychotic drugs compared with a placebo. Relapse occurred in 55% of the placebo patients compared to only 19% of the antipsychotic group. However, Ross and Read (2004) point out that 45% of placebo group did benefit, so from the 81% of antipsychotic group who did benefit a large proportion (45%) would have benefited from placebo, so less effective than believed.
* + Cheap, fast acting, easy to take (less motivation/time needed).
* - Tardive dyskinesia – Conventional drugs in particular have many dangerous and degrading side effects (e.g. tardive dyskinesia). Hill (1986), about 30% develop tardive dyskinesia, and it is irreversible in 75%. However, Atypical psychotics provide an alternative with less side effects.
* - Motivational deficits – Ross and Read (2004), argue that being prescribed medication enforces the view that there is ‘something wrong with you’. Prevents individuals from thinking about possible stressors which may trigger condition, reducing motivation to look for possible solutions that may alleviate stressor and reduce suffering.
* - Treatment of symptoms, not cure – many on drugs long-term.
* Ethical issues – critics argue that if side-effects, deaths, and psychological consequences were taken into account, a cost-benefit analysis would be negative. In the US, an out-of-court settlement was awarded to a tardive dyskinesia sufferer on the basis of the Human Rights Act of 1988, ‘no one shall be subjected to inhuman or degrading treatment or punishment’.

ECT – electrode placed above temple of non-dominant side of brain and middle of forehead (unilateral ECT). Patient is injected with a short-acting barbiturate, so they are unconscious before shock. They are given a nerve-blocking agent, paralysing the muscles to prevent contraction and causing fractures. Small amount of current (0.6amps), lasting half a second, passes through the brain, producing a seizure lasting one minute. Needs 3 to 15 treatments.

* - Effectiveness – American Psychiatric Association review in 2001 listed 19 studies that compared ECT with stimulated ECT, it concluded that ECT produced results that were no different from or worse than antipsychotic medication. However, Sarita et al. (1998), found no difference in symptom reduction between 36 SZ patients given either ECT or simulated ECT.
* - Appropriateness – significant risks with ECT, e.g. memory dysfunction, brain damage, and death. The use of ECT for SZ had declined and has only been used historically in the UK.

**Psychological explanations of SZ:**

*Social explanation: (dysfunctional family)*

*Cause* - Fromm-Reichmann (1948), ‘schizophrenogenic family’ who have high emotional tension and many secrets. The influence of the ‘schizophrenogenic mother’ in particular may lead to a lack of trust in offspring. This led to Bateson et al.’s (1956) ‘double-bind hypothesis’, repeated exposure to faulty communication in the family (e.g. contradictory behaviour –caring yet critical at the same time) puts the child in doubt, confusion and withdrawal. These interactions prevent the development of an internally coherent construction of reality, manifesting itself into symptoms of SZ.

* - Very little supporting evidence – whilst those which claim to demonstrate a link are mainly retrospective (past events), used poorly operationalised definitions of SZ, and made little use of control groups. This makes it difficult to draw a conclusion about cause and effect. Dysfunctional family maybe an effect of SZ rather than cause.

*Maintenance* - Vaughn and Leff (1976), ‘expressed emotion’ refers to a pattern of criticism and hostility in relatives of people with schizophrenia which is strongly linked to relapse and maintenance of SZ. It appears that the negative emotional climate in these families arouses the patient and leads to stress beyond their already impaired coping mechanism.

* + Brown (1972) -showed patients who returned to families with high EE (e.g. hostility, criticism, over-involvement) demonstrated higher levels of relapse (58%) than those who returned to low EE settings (10%).
* + Treatment implication – led to family intervention therapies which have had some success in reducing relapse rates.
* - Places too much responsibility/blame on family, mothers in particular. Living and coping with an SZ individual is distressing enough, as family struggles it is unhelpful to suggest they are the cause.
* - Many patients estranged from family, yet relapse – suggests not just dysfunctional family, e.g. dysfunctional friends or high EE work environment.
* - Reductionist – ignores biological factors for which there is considerable empirical evidence (e.g. genetics), suggests diathesis-stress model.

*Cognitive explanation:*

Frith (1992) – SZ occurs due to faulty information processing leading to cognitive overload. The person is unable to distinguish effectively between their thoughts and outside stimuli hence experience hallucinations and passivity symptoms.

* + Empirical support – from studies demonstrating specific changes in blood flow to areas of the brain when SZs perform cognitive tasks. And, Neufeld (1978), compared cognitive processes of SZs and a control group. Sz took longer to encode stimuli and showed ST memory problems, suggests that their ability to process info was impaired
* Bentall (2005) – schizophrenia occurs due to deficits and biases in information processing which over emphasise threatening interpretations, and bias sensory information (delusions, paranoia).
* - Very little supporting empirical evidence.
* + Allowed for the development of CBT – effective and appropriate treatment.
* - Cause or effect?
* - Cognitive explanations alone are descriptive of symptoms, rather than cause – however, assumes underlying biological abnormalities lead to cognitive malfunctions.

**Psychological therapies for SZ:**

* CBT – adjust faulty thinking patterns and beliefs. Taught to trace back origin of symptoms, evaluate content of delusions, and rationally test validity of faulty beliefs. Patients encouraged to come up with their own alternatives to previously maladaptive beliefs.
* + Outcome studies – measures the extent to which a patient has recovered compared to an accepted form of treatment. Suggest that patients receiving CBT experience fewer hallucinations and delusions, and recover to a greater extent compared to those on antipsychotics. Drury et al. (1996), reduction of positive symptoms and 25-50% reduction in recovery time. Kuipers et al. (1997), lower patient drop-out rates and greater satisfaction.
* - Financial constraints, can take time for treatment to work.
* - Most studies (including the two above) of effectiveness of CBT have been conducted with patients treated at the same time with antipsychotic medication. It has been difficult to access the effectiveness of CBT independent of antipsychotic medication.
* - Works by trying to generate less distressing explanations for psychotic experience, rather than trying to eliminate them completely. Also, doesn’t treat negative symptoms as they may be useful serving as ‘safety behaviours’, which may prevent positive symptoms getting worse. Therefore, CBT offers some hope of alleviating maladaptive process, but is inappropriate as a form of cure.
* - Not appropriate for everyone – Kingdon and Kirschen (2006), study of 142 SZ patients, many not deemed suitable for CBT due to a lack of fully engagement with the therapy. In particular, older aged deemed not suitable more than younger patients.
* - Ethical issues – studies must be carried out in a way that does not increase the probability that the participants would come to any form of harm, especially when dealing with vulnerable groups like SZs. Potential of harm in outcome studies are associated with the medication discontinuation, use of placebo groups, and capacity for informed consent.
* Family intervention - aims to reduce EE in the family (and thus relapse rates) by educating family members about disorder, how to manage it etc., and improving communication styles and discussing problems between family members and the patient.
* + NICE (2009) – meta-analysis of 29 studies, when compared to patients receiving standard care alone, there was a reduction in hospital admissions during treatment and severity of symptoms both during and up to 24 months following the intervention. Relapse rate of family intervention was 26% compared to 50% of control condition.
* + Why is it effective? – although Pharoah et al.’s (2012) meta-analysis established that family intervention maybe effective in improving clinical outcomes such as mental state and social functioning, psychologists also believe that effectiveness is more to do with increases in medical compliance. Patients more likely to reap the benefits of medication because they are more likely to comply, having been educated about the disorder.
* - Not useful for those with families unwilling to help.