SCHIZOPHRENIA

REVISION

COMPLETE REVISION POWERPOINT
CLASSIFICATION OF SZ
SCHIZOPHRENIA - FACTS

• Sz is a type of psychosis, a severe mental disorder in which thoughts (cognition) and emotions are so impaired that contact is lost with external reality

• This will affect a person’s language, thought, perception and sense of self
SCHIZOPHRENIA - FACTS

• Sz is the most common psychotic disorder that affects about 1% of the population at some point in their lifetime
  – Many continue to lead normal lives after diagnosis and subsequent treatment
• Sz is most often diagnosed between 15-35 years old (men and women affected equally)
• There are many symptoms of Sz (including delusions and hallucinations) but not every patient displays all the symptoms
MYTHS OF SCHIZOPRENEIA

• Schizophrenia is neither “split personality” nor “multiple personality”
• People with Schizophrenia do not constantly display psychotic behaviour (loss of contact with reality)
SCHIZOPHRENIA

• On your phones investigate:
  – DSM-V (2013)
  – Positive Symptoms of Sz (Hallucinations, Delusions, Disorganised Speech, Grossly Disorganised or Catatonic Behaviour)
  – Negative Symptoms of Sz (Speech Poverty, Avolition, Affective Flattening, Anhedonia)
DIAGNOSING SCHIZOPHRENIA

• Clinicians use a diagnostic manual like the DSM-V (the most recent manual)
• The DSM (Diagnostic and Statistical Manual of Psychiatric Disorders) is a classification and description of over 200 mental disorders, grouped in terms of their common features
• The DSM is used mainly in the US, whereas Europe mainly use the ICD (International Classification for Diseases)
  – The most recent is the ICD-X with the ICD-XI being published in 2017
DIAGNOSING SCHIZOPHRENIA

Here are the characteristics necessary for a diagnosis of Sz using the DSM-V:

Criterion A

Two (or more) of the following symptoms

- Delusions
- Hallucinations
- Disorganised Speech (e.g. frequent derailment or incoherence)
- Grossly disorganised or catatonic behaviour
- Negative symptoms, i.e. affective flattening, alogia or avolition

Only one Criterion A symptom is required if delusions are bizarre or hallucinations consist of a voice keeping up a running commentary on the person’s behaviour or thoughts, or two or more voices conversing with each other.
DIAGNOSING SCHIZOPHRENIA

Criterion B – Social/Occupational Dysfunctions
• For a significant portion of the time since the onset, one or more major areas of functioning such as work, interpersonal relations or self-care are markedly below the level achieved prior to the onset

Criterion C - Duration
• Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms (or less if successfully treated) that meet Criterion A
• During non-active periods, disturbance may be limited to negative symptoms or two or more symptoms in Criterion A in weakened form (e.g. odd beliefs, unusual perceptual experiences)
POSITIVE SYMPTOMS

• These are symptoms that appear to reflect an excess or distortion of normal functions

• They include:
  – HALLUCINATIONS
  – DELUSIONS
  – DISORGANISED SPEECH
  – GROSSLY DISORGANISED OR CATATONIC BEHAVIOUR
POSITIVE SYMPTOMS

HALLUCINATIONS

• Bizarre, unreal perceptions of the environment that are usually auditory, visual, olfactory or tactile

• Many schizophrenics report hearing voice(s) telling them to do something (harm themselves or others) or are commenting on their behaviour
POSITIVE SYMPTOMS

DELUSIONS

• Bizarre beliefs that seem real to the person but are not real
• These can be paranoid in nature; where they believe a person is following or spying on them
• They may believe that their phone is tapped or video cameras are hidden in their home
• They may also be inflated beliefs about having special powers or are important/famous (delusions of grandeur)
• They may also experience delusions of reference; where they feel the events in the environment are directly related to them (e.g. personal messages coming to them from the TV)
POSITIVE SYMPTOMS

DISORGANISED SPEECH

• This is the result of abnormal thought processes, where the individual has problems organising his/her thoughts and this shows up in their speech.

• They may slip from one topic to another (derailment), even in mid-sentence; and in extreme cases their speech may be so incoherent that it sounds like complete nonsense.
  – This is referred to as “word salad”
POSITIVE SYMPTOMS

GROSSLY DISORGANISED OR CATATONIC BEHAVIOUR

• This includes the lack of ability or motivation to initiate a task, or to complete it once it is started, which leads to difficulties in daily living and can result in decreased interest in personal hygiene

• The individual may dress or act in ways that appear bizarre to other people, such as wearing heavy clothes on a hot summer’s day

• Catatonic behaviours are characterised by a reduced reaction to the immediate environment, rigid postures or aimless motor activity
NEGATIVE SYMPTOMS

• These are those that appear to reflect a reduction or loss of normal functions, which often persist even during periods of low (or absent) positive symptoms.

• About 1 in 3 Sz patients suffer from significant negative symptoms.

• Negative symptoms weaken the person’s ability to cope with everyday activities, affecting their quality of life and their ability to manage without significant outside help.

• Individuals with Sz are often unaware of the extent of their negative symptoms, and are typically less concerned about them than their relatives may be.
NEGATIVE SYMPTOMS

• Enduring negative symptoms are sometimes referred to as “deficit syndrome”, characterised by the presence of at least two negative symptoms for 12 months or longer.

• Individuals with deficit syndrome have been found to have more pronounced cognitive deficits and poorer outcomes than patients who do not have this syndrome.

• Negative symptoms respond poorly to traditional antipsychotic treatment, but the newer atypical antipsychotics claim to be more superior.
NEGATIVE SYMPTOMS

• Negative symptoms include:
  – ALOGIA (SPEECH POVERTY)
  – AVOLITION
  – AFFECTIVE FLATTENING
  – ANHEDONIA
NEGATIVE SYMPTOMS

ALOGIA (SPEECH POVERTY)

• This is characterised by the lessening of speech fluency and productivity, this is thought to reflect slowing or blocked thoughts.

• These patients may produce fewer words in a given time on a task of verbal fluency (e.g. naming as many animals as you can in one minute).

• This is not a matter of not knowing as many words as non-schizophrenics, but more a difficulty of spontaneously producing them.

• This type of speech appears to be associated with long illness and earlier onset of the illness.
EXAMPLE OF ALOGIA

- In conversation, alogic patients will reply very sparsely and their answers to questions will lack spontaneous content; sometimes, they will even fail to answer at all. Their responses will be brief, generally only appearing as a response to a question or prompt.
EXAMPLE OF ALOGIA

- ALOGIA
  - Q: Do you have any children?
    A: Yes.
  - Q: How many?
    A: Two.
  - Q: How old are they?
    A: Six and sixteen.
  - Q: Are they boys or girls?
    A: One of each.
  - Q: Who is the sixteen year old?
    A: The boy.
  - Q: What is his name?
    A: Edmond.
  - Q: And the girl's?
    A: Alice.

- NORMAL SPEECH
  - Q: Do you have any children?
    A: Yes, a boy and a girl.
  - Q: How old are they?
    A: Edmond is sixteen and Alice is six.
NEGATIVE SYMPTOMS

AVOLITION

• This is a reduction of interests and desires as well as an inability to initiate and persist in goal-directed behaviour (e.g. sitting in the house for hours every day not doing anything)

• This is distinct from poor social function or disinterest, which can be the result of other circumstances
  – E.g. they may have no social contact with friends as they do not have any, they are far away

• This would not be considered as avolition, which is specified as a reduction in self-initiated involvement in activities that are available to the patient
NEGATIVE SYMPTOMS

AFFECTIVE FLATTENING

- This is a reduction in the range and intensity of emotional expression, including facial expressions, voice tone, eye contact and body language.
- When speaking, patients may also show a deficit in prosody (i.e. paralinguistic features) that provide extra information that is not explicitly contained in a sentence, and which gives cues to the listener as to emotional or attitudinal content and turn-taking.
NEGATIVE SYMPTOMS

ANHEDONIA

• This is a loss of interest or pleasure in all or almost all activities, or lack of reactivity to normally pleasurable stimuli

• It may be pervasive (i.e. all-embracing) or it may be confined to a certain aspect of experience

• Physical anhedonia is the inability to experience physical pleasures such as from food, bodily contact, etc

• Social anhedonia is the inability to experience pleasure from interpersonal situations like interacting with other people
  – Social anhedonia overlaps with other disorders (like depression), whereas physical anhedonia does not
  – Physical anhedonia is considered a more reliable symptom of Sz (Sarkar et al, 2010)
DIAGNOSIS OF SCHIZOPHRENIA

• To be diagnosed with Schizophrenia a person requires to exhibit at least one-month duration of two or more positive symptoms

• [http://www.youtube.com/watch?v=H_jYqSA_fJk](http://www.youtube.com/watch?v=H_jYqSA_fJk)
RELIABILITY AND VALIDITY IN DIAGNOSIS AND CLASSIFICATION
RELIABILITY
RELIABILITY

• Diagnostic reliability means that a diagnosis of Sz must be **repeatable**
  – I.e. clinicians must be able to reach the same conclusions at two different points in time (test-retest reliability), or different clinicians must reach the same conclusions (inter-rater reliability)
    • Inter-rater reliability is measured by a statistic called a kappa score (a score of 1 indicates perfect inter-rater agreement and a score of 0 indicates zero agreement)
    • A kappa score of 0.7 or above is generally considered good
    • In the DSM-V field trials, the diagnosis of Sz had a kappa score of only 0.46
CULTURAL DIFFERENCES IN DIAGNOSIS

• Research suggests that there is a significant variation between countries when it comes to diagnosing Sz
  – I.e. culture has an influence on the diagnostic process
• Copeland (1971) gave 134 US and 194 British psychiatrists a description of a patient
  – 69% of the US psychiatrists diagnosed Sz compared to 2% of the British psychiatrists
CULTURAL DIFFERENCES IN DIAGNOSIS

• One of the main characteristics of Sz, “hearing voices”, is also influenced by cultural environment
  – Luhrmann et al (2015) interviewed 60 adults diagnosed with Sz (20 each from US, India and Ghana)
  – Each was asked about the voices they heard
    • Many of the African and Indian subjects reported positive experiences with their voices (describing them as playful, or giving advice), not one American did (reported the voices to be violent and hateful)
VALIDITY
This occurs when the accuracy of diagnosis is dependent on the gender of the individual.

The accuracy of diagnostic judgements can vary for a number of reasons, including gender-biased diagnostic criteria or clinicians basing their judgements on stereotypical beliefs held about gender.

- E.g. critics of the DSM diagnostic criteria suggest that some diagnostic categories are biased toward pathologising one gender than the other.

Broverman et al (1970) found that clinicians in the US associated mentally healthy “adult” behaviour with mentally healthy “male” behaviour.

As a result, there was a tendency for women to be perceived as less mentally healthy.
Many of the positive and negative symptoms of Sz are also found in many other disorders like depression and bipolar disorder.

This problem is referred to as symptom overlap.

E.g. Ellason and Ross (1995) point out that people with dissociative identity disorder (DID) actually have more schizophrenic symptoms than people diagnosed as being schizophrenic.

Most people who are diagnosed with Sz have sufficient symptoms of other disorders that they could also receive at least one other diagnosis (Read, 2004).
CO-MORBIDITY

• This is an important issue for the validity of the diagnosis of mental illness
• It is the extent that two (or more) conditions co-occur
• Psychiatric co-morbidities are common among patients with Sz
  – These include, substance abuse, anxiety and symptoms of depression
  – E.g. Buckley et al (2009) estimate that co-morbid depression occurs in 50% of patients, and 47% of patients also have a lifetime diagnosis of co-morbid substance abuse
CO-MORBIDITY

- Sz and OCD are two distinct psychiatric conditions
- Roughly 1% of the population develop Sz and about 2-3% develop OCD
- Since both are fairly uncommon, we would expect that only a few people with Sz would develop OCD and vice versa
- However, evidence suggests that the two conditions appear together more often than chance would suggest
- A meta-analysis by Swets et al (2014) found at least 12% of patients with Sz also fulfilled the diagnostic criteria for OCD and about 25% displayed significant OCD symptoms
EVALUATION
RELIABILITY
LACK OF INTER-RATER RELIABILITY

• Despite claims for increased reliability in DSM-III (and later versions), over 30 years later there is still little evidence that DSM is routinely used with high reliability by mental health clinicians

• Whaley (2001) found inter-rater reliability correlations in the diagnosis of Sz as low as 0.11

• Further problems with the inter-rater reliability of the diagnosis of Sz are shown in the study by Rosenhan
In Rosenhan’s study (1973) “normal” people presented themselves to psychiatric hospitals in the US claiming they heard an unfamiliar voice in their head saying the words “empty”, “hollow” and “thud”

They were all diagnosed with Sz and admitted

Throughout their stay, none of the staff recognised that they were actually normal

In a follow-up study, Rosenhan warned hospitals of his intentions to send out more “pseudopatients”

This resulted in a 21% detections rate (of pseudopatients), although none actually presented themselves
UNRELIABLE SYMPTOMS

• For a diagnosis of Sz, only one of the characteristic symptoms is required “if delusions are bizarre”

• However, this creates problems for reliability of diagnosis
  – When 50 senior psychiatrists in the US were asked to differentiate between “bizarre” and “non-bizarre” delusions, they produced inter-rater reliability correlations of only around 0.40
  – Forcing the researchers to conclude that even this central diagnostic requirement lacks sufficient reliability for it to be a reliable method of distinguishing between schizophrenic and non-schizophrenic patients (Mojtabi and Nicholson, 1995)
CULTURAL DIFFERENCES IN THE DIAGNOSIS OF SZ

• Research (e.g. Barnes, 2004) has established cultural, and particularly racial differences in the diagnosis of Sz

• However, the prognosis for members of ethnic minority groups may actually be more positive than for majority group members
  – The ethnic culture hypothesis predicts that ethnic minority groups experience less distress associated with mental disorders due to the protective characteristics and social structures that exist in most ethnic minority cultures
CULTURAL DIFFERENCES IN THE DIAGNOSIS OF SZ

• Brekke and Barrio (1997) found evidence to support this hypothesis in a study of 184 individuals diagnosed with Sz or a Sz-spectrum disorder
  – The sample was drawn from two non-white minority groups (African-Americans and Latinos) and a majority group (white Americans)
  – They found that the non-minority group members were consistently more symptomatic than members of the two ethnic minority groups, findings which support the ethnic culture hypothesis
EVALUATION
VALIDITY
Loring and Powell (1988) randomly selected 290 male and female psychiatrists to read two case description of patients’ behaviour. They were then asked to offer their judgement on these individuals using standard diagnostic criteria. When patients were described as “males” or no information was given about their gender, 56% of the psychiatrists diagnosed Sz. When patients were described as “female”, only 20% were given the diagnosis of Sz. This gender bias was not as evident among female psychiatrists, suggesting that diagnosis is influenced not only by the gender of the patient, but also the gender of the clinician.
THE CONSEQUENCES OF CO-MORBIDITY

• A number of studies have examined single co-morbidities with Sz, but these studies have usually involved only relatively small sample sizes.

• A US study (Weber et al, 2009) looked at nearly 6 million hospital discharge records to calculate co-morbidity rates.

• Psychiatric and behaviour-related diagnosis accounted for 45% of co-morbidity.
THE CONSEQUENCES OF CO-MORBIDITY

• However, the study also found evidence of many co-morbid non-psychiatric diagnosis
• Many patients with a primary diagnosis of Sz were also diagnosed with medical problems including asthma, hypertension and type 2 diabetes
• They concluded that the very nature of a diagnosis of a psychiatric disorder is that patients tend to receive a lower standard of medical care, which in turn adversely affects the prognosis for patients with Sz
DIFFERENCES IN PROGNOSIS

• In the same way that people are diagnosed as schizophrenic rarely share the same symptoms, there is no evidence that they share the same outcomes.

• The prognosis for patients diagnosed with Sz varies with about 20% recovering their previous level of functioning; 10% achieving significant and lasting improvements and about 30% showing some improvement with intermittent relapses.
DIFFERENCES IN PROGNOSIS

• A diagnosis of Sz has little predictive validity, some people never recover, but many do

• What does influence the outcome is more to do with gender and psycho-social factors like social skills, academic achievement and family tolerance of schizophrenic behaviour
BIOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA
FAMILY STUDIES

• These studies find people that have schizophrenia and determine if their biological relatives are similarly affected more than non-biological relatives (e.g. Gottesman, 1991)

• These have established that schizophrenia is more common in biological relatives of a schizophrenic and the closer the genetics the greater the risk

• E.g. a child with two schizophrenic parents = 46% chance; children with one schizophrenic parent = 13%; sibling with schizophrenia = 9%
GENETIC FACTORS

TWIN STUDIES

• Monozygotic (identical) twins = more similar in terms of traits like schizophrenia than dizygotic twins (only share 50% genes)
• Joseph (2004) calculated that all twin studies prior to 2001 show a concordance rate of 40.4% for MZ twins and 7.4% for DZ twins
• Recent research using “blind” diagnosis (where researchers do not know if the twin they are assessing is MZ or DZ), suggests that the concordance rate for MZ twins is lower than this (40.4%) but still many times higher than DZ twins
GENETIC FACTORS

ADOPTION STUDIES

• These studies involve individuals who share genetics, but have been reared apart

• Tienari et al (2000), in Finland, found that from 164 adoptees who’s mothers had been diagnosed with schizophrenia, 11 (6.7%) were also diagnosed with schizophrenia, compared to 4 (2%) of the 197 control adoptees (non-schizophrenic mother)

• This shows that there is a strong link between genetics and schizophrenia
DOPAMINE HYPOTHESIS

• This is one of the many neurotransmitters in the brain and is associated with positive symptoms of Sz

• The Dopamine Hypothesis states that messages from neurons that transmit dopamine, fire too easily or too often leading to characteristics of schizophrenia (hallucinations and delusions)

• Schizophrenics tend to have more D2 receptors on receiving neurons, leading to more dopamine binding and more neurons firing
vesicles with the transmitter dopamine

tyrosine

L-dopa

dopamine

dopamine receptor

the message is passed on

receiving cell

cell nucleus

synapse
DOPAMINE HYPOTHESIS

- Dopamine neurons help with guiding attention
- Disturbances in this process could lead to attention, perception and thought problems (Comer, 2003)
- The key role played by dopamine in schizophrenia is highlighted in 3 sources of evidence
  - Amphetamines
  - Anti-psychotic drugs
  - Parkinson’s disease
AMPHETAMINES

• These are dopamine **agonists** that stimulates nerve cells containing dopamine, causing the **synapse** to be flooded with dopamine

• “Normal” individuals that are exposed to large doses of amphetamines could develop hallucinations and delusions of a schizophrenic episode

• This generally disappears when the drug is no longer taken
PARKINSON’S DISEASE

• Parkinson’s is a degenerative neurological disorder
• People who suffer with Parkinson’s disease often have low levels of dopamine activity
• They can take the drug L-dopa to raise their dopamine levels and have also been found to develop schizophrenic-type symptoms (Grilly, 2002)
ANTI-PSYCHOTIC DRUGS

• These block the activity of dopamine in the brain and are known as **dopamine antagonists**

• By reducing the stimulation of dopamine they will get rid of symptoms like hallucinations and delusions

• These drugs alleviated many symptoms of schizophrenia and so strengthened the case for the important role of dopamine in this disorder
Davis and Kahn (1991) proposed that the positive symptoms of Sz are caused by an excess of dopamine in subcortical areas of the brain (especially in the mesolimbic pathway).

The negative and cognitive symptoms of Sz are thought to come from a deficit of dopamine in areas of the prefrontal cortex (the mesocortical pathway).

Evidence for this revised hypothesis comes from various sources:

- **Neural Imaging** (PET scans found lower levels of dopamine in the dorsolateral prefrontal cortex of Sz patients compared to “normal” controls)

- **Animal Studies** (induced dopamine depletion in the prefrontal cortex in rats led to cognitive impairment (memory deficits) and researchers were able to reverse this with atypical antipsychotics)
KEY TERMS

• **BIOLOGICAL EXPLANATIONS** – emphasise the role of inherited factors and dysfunction of brain activity in the development of a behaviour or mental disorder

• **DOPAMINE HYPOTHESIS** – claims that an excess of the neurotransmitter dopamine in certain regions of the brain is associated with the positive symptoms of Sz

• **GENETICS** – inherited factors make certain individuals more likely to develop a behaviour or mental disorder

• **NEURAL CORRELATES** – changes in neuronal events and mechanisms that result in the characteristic symptoms of a behaviour or mental disorder
BIOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA EVALUATION
GENETIC FACTORS
COMMON REARING PATTERNS MAY EXPLAIN FAMILY SIMILARITIES

• Research shows that Sz appears to run in families (supporting the genetic basis for Sz)

• However, many researchers feel that this may be to do with common rearing patterns or other factors that have nothing to do with heredity

  – E.g. research on expressed emotion shows that negative emotional climate in some families may lead to stress beyond a person’s coping mechanisms and so trigger a schizophrenic episode
MZ TWINS ENCOUNTER MORE SIMILAR ENVIRONMENTS

• A crucial assumption underlying all twin studies is that the environments of MZ and DZ twins are equivalent.

• So the greater concordance for Sz between MZ twins is due to genetic similarity rather than greater environmental similarity.

• Joseph (2004) says that it is widely accepted that MZ twins are treated more similarly, face more similar environments (are more likely to do things together) and experience more “identity confusion” (being treated as “the twins” rather than as two individuals) than DZ
ADOPTEES MAY BE SELECTIVELY PLACED

• An assumption of adoption studies is that adoptees are not “selectively placed”

• Joseph (2004) claims that this is unlikely to have been the case, particularly in early studies

• In Denmark and US, potential adoptive parents would have been informed of the genetic background of children prior to selection for adoption

• Kringlen (1987, cited in Joseph, 2004) “because the adoptive parents evidently received information about the child’s biological parents, one might wonder who would adopt such a child”
DOPAMINE HYPOTHESIS
EVIDENCE FROM TREATMENT

• Much of the evidence supporting the dopamine hypothesis comes from the success of drug treatments that attempt to change levels of dopamine activity in the brain.

• The basic mechanism of antipsychotic drugs is to reduce the effects of dopamine and so reduce the symptoms of SZ.
  – Leucht et al (2013) carried out a meta analysis of 212 studies that analysed the effectiveness of different antipsychotic drugs compared with a placebo.
  – They found that all drugs tested were significantly more effective than placebo in treating positive and negative symptoms, achieved through the normalisation of dopamine.
INCONCLUSIVE SUPPORTING EVIDENCE

• Moncrieff (2009) claims that evidence for the dopamine hypothesis of Sz is far from conclusive
  – E.g. although stimulant drugs such as cocaine and amphetamine have been shown to induce schizophrenic episodes. These drugs affect many neurotransmitters other than dopamine

• Evidence for dopamine concentrations in post-mortem brain tissue has either been negative or inconclusive

• Moncrieff also says that other confounding sources of dopamine release, such as stress and smoking, have rarely been considered
  – Therefore, she suggests, the idea that the symptoms of Sz are caused
CHALLENGES OF THE DOPAMINE HYPOTHESIS

• Noll (2009) claims that there is strong evidence against both the original dopamine hypothesis and the revised dopamine hypothesis

• He argues that antipsychotic drugs do not alleviate hallucinations and delusions in about one-third of people experiencing these symptoms

• Noll says that in some people, hallucinations and delusions are present despite levels of dopamine being normal

• Blocking the D2 receptors of these individuals has little or no effect on their symptoms

• This suggests that, rather than dopamine being the sole cause of positive symptoms, other neurotransmitter systems, acting independently of the dopaminergic system, may also produce the positive symptoms associated with Sz
PSYCHOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA
FAMILY
DYSFUNCTION
DOUBLE BIND THEORY

Bateson et al (1956) suggest that children who frequently receive contradictory messages from their parents are more likely to develop Sz

- E.g. if a mother tells her son that she loves him, but at the same time she turns her head away in disgust
- Here the child receives two conflicting messages on communication about their relationship (i.e. verbal and non-verbal messages are different)
- As the messages invalidate each other; the child is unable to respond to the mother due to this contradiction
- These contradictory messages prevent the development of an internally coherent construction of reality, and in the long run this manifests itself as schizophrenic symptoms
EXPRESSED EMOTION (EE)

- A high degree of EE is a very negative emotional climate.
- EE is a family communication style where members of a family of a psychiatric patient talk about the patient in a critical or hostile manner or there is emotional over-involvement or over-concern with the patient or their behaviour.
- Kuipers et al (1983) found that high EE relatives talk more and listen less.
- Patients returning to a family with high EE are 4 times more likely to relapse than to a family with low EE (Linszen et al, 1997).
- This suggests that people with Sz have a lower tolerance for intense environmental stimuli.
- Negative emotional climate arouses the patient and leads to stress beyond coping mechanisms and so triggers a Sz episode.
- A family environment that is supportive and emotionally undemanding may help the person with Sz reduce their dependence on medication and reduce the likelihood of relapse (Noll, 2009).
COGNITIVE EXPLANATIONS
COGNITIVE EXPLANATIONS

• Research has found evidence of **dysfunctional thought processing** in people with Sz
  – I.e. they process information differently

• **Cognitive explanations** of Sz emphasise the role of dysfunctional thought processing, especially evident in those who display hallucinations and delusions (positive symptoms)
COGNITIVE EXPLANATIONS OF DELUSIONS

• During the formation of delusions, the patient’s interpretations of their experiences are controlled by inadequate information processing.

• A critical characteristic of delusional thinking is the degree to which the individual perceives themselves as the central component in events (egocentric bias) and so jumps to conclusions about external events.

• This is manifested in the patient’s tendency to relate irrelevant events to themselves and so arrive at false conclusions.

• Muffled voices are interpreted as people criticising them, and flashes of light are a signal from God.

• Delusions in Sz are relatively resistant to reality testing, in that patients are unwilling or unable to consider that they may be wrong (Beck and Rector, 2005).

• They are considered to have “impaired insight”, an inability to recognise cognitive distortions and substitute more realistic explanations for events.
COGNITIVE EXPLANATIONS OF HALLUCINATIONS

• Hallucinating individuals focus excessive attention on auditory stimuli (hypervigilance) and so have a higher expectancy for the occurrence of a voice than normal individuals.

• Aleman (2001) suggests that hallucination-prone individuals find it difficult to distinguish between imagery and sensory-based perception.
  – For these individuals, the inner representation of an idea (e.g. “what other people think of me”) can override the actual sensory stimulus and produce auditory image (“he is not a good person”) that is every bit as real as the transmission of actual sound.

• Hallucinating patients with Sz are significantly more likely to misattribute the source of a self-generated auditory experience to an external source than are non-hallucinating patients with Sz (Baker and Morrison, 1998).

• These errors are not corrected by disconfirming evidence because patients with Sz do not go through the same processes of reality testing (such as checking external sources) that others would do.
PSYCHOLOGICAL EXPLANATIONS FOR SCHIZOPHRENIA EVALUATION
FAMILY RELATIONSHIPS

– Tienari et al (1994) found that adopted children who had schizophrenic biological parents were more likely to develop Sz than children with non-schizophrenic biological parents.

– However, this difference only emerged in situations where the adopted family was rated as disturbed.

– Therefore, the illness is only revealed under appropriate environmental conditions.

• GENETIC VULNERABILITY ALONE WAS NOT SUFFICIENT.
DOUBLE BIND THEORY

– Evidence support
  • Berger (1965) found that schizophrenics reported a higher recall of double bind statements by their mothers than non-schizophrenics
    – However, this evidence may not be reliable as patients’ recall may be affected by their Sz

– Less support
  • Liem (1974) measured patterns of parental communication in families with a schizophrenic child and found no difference when compared to “normal” families
  • Hall and Levin (1980) analysed data from a variety of previous studies and found no difference between families with and without a schizophrenic member in the degree to which verbal and non-verbal communication were in agreement
INDIVIDUAL DIFFERENCES IN VULNERABILITY TO EE

• Not all patients who live in high EE families relapse, and not all patients who live in low EE families avoid relapse

• Research has found individual differences in stress response to high EE-like behaviours

• Altorfer et al (1998) found that ¼ of the patients they studied showed no physiological response to stressful comments from their relatives

• Vulnerability to the influence of high EE may also be psychologically based
  – E.g. Lebell et al (1993) suggests that how patients judge the behaviour of their relatives is important
  – In cases where high EE behaviours are not perceived as being negative or stressful, they can do well regardless of how the family environment is objectively rated
  – This shows that not all patients are equally vulnerable to high levels of EE within the family environment
COGNITIVE EXPLANATIONS
SUPPORTING EVIDENCE FOR THE COGNITIVE MODEL OF SZ

- Sarin and Wallin (2014) found supporting evidence for the claim that the positive symptoms of Sz have their origins in faulty cognition
  - E.g. delusional patients were found to show various biases in their information processing, like jumping to conclusions and lack of reality testing
- Likewise, those with Sz with hallucinations were found to have impaired self-monitoring and also tended to experience their own thoughts as voices
- In addition they found that patients with negative symptoms also displayed dysfunctional thought processes such as having low expectations regarding pleasure and success
SUPPORT FROM THE SUCCESS OF COGNITIVE THERAPIES

• The claim that the symptoms of Sz have their origin in faulty cognition is supported by the success of cognitive-based therapies for Sz

• In CBT for psychosis (CBTp), patients are encouraged to evaluate the content of their delusions or of any voices, and to consider ways in which they might test the validity of their faulty beliefs

• The effectiveness of this approach was demonstrated in the NICE review of treatments for Sz (NICE, 2014)
  – This review found consistent evidence that, when compared to treatment by antipsychotics, CBT was more effective in reducing symptom severity and improving levels of social functioning

• NICE = The National Institute for Health and Care Excellence
AN INTEGRATED MODEL FOR SZ

• A problem with any psychological model of Sz is that it deals with one aspect of the disorder (e.g. cognitive impairment) but fails to explain, or ignores, another aspect (e.g. neurochemical changes)

• Howes and Murray (2014) addressed this problem with an integrated model of Sz
  – They argue that early vulnerability factors (e.g. genes, birth complications, etc), together with exposure to significant social stressors (e.g. social adversity), sensitises the dopamine system, causing it to increase the release of dopamine

• This increased dopamine activity results in cognitive processing of paranoia and hallucinations, and eventually the development of psychosis (a loss of contact with reality)
  – This contributes to the stress experienced by the individual, leading to more dopamine released, more symptoms and so on
DRUG THERAPY FOR TREATING SCHIZOPHRENIA
**ANTIPSYCHOTIC MEDICATION**

- Antipsychotic medication helps the person with the disorder to function as well as possible in their life, whilst increasing their feelings of well-being.
- Antipsychotics are usually recommended as the initial treatment for Sz, then clinicians tend to use a combination of medication and psychological therapy.
- All antipsychotics work by reducing the transmission of Dopamine in the areas of the brain associated with the symptoms of Sz.
- **Typical Antipsychotics** (like *chlorpromazine*) are used primarily to combat the positive symptoms of Sz like hallucinations.
- **Atypical Antipsychotics** (like *clozapine*) also combat these positive symptoms, but in addition there are claims that they have beneficial effects on negative symptoms too.
Typical Antipsychotics

- These are also known as “conventional” or “first generation” antipsychotic and were developed in the 1950s.
- The basic role is to reduce the effects of dopamine and so reduce the symptoms of Sz.
- Typical Antipsychotics are dopamine antagonists as they bind to but do not stimulate the dopamine receptor (especially the D2 receptors).
  - This blocks their action and so reducing the stimulation of the dopamine system.
  - Antipsychotic drugs eliminate the hallucinations and delusions experienced by people with Sz.
- Hallucinations and delusions tend to reduce within a few days, although other symptoms may take weeks before a significant improvement is seen.
TYPICAL ANTIPSYCHOTICS

• The effectiveness of the dopamine antagonists in reducing these symptoms led to the development of the dopamine hypothesis of Sz

• Karpur et al (2000) estimated that between 60-75% of D2 receptors must be blocked for these drugs to be effective

• Unfortunately, by doing this, there are a number of other D2 receptors in other parts of the brain that must also be blocked (which could lead to undesirable side effects)

• This represents the “high cost” of using typical antipsychotics to treat Sz

• There are several dopamine pathways in the brain, and it appears that blocking dopamine receptors in only one of them is useful, whereas blocking all of them may be harmful for the person

• This problem has been addressed by the development of atypical antipsychotics
ATYPICAL ANTIPSYCHOTICS

- These are also known as “second generation” antipsychotic and were called this because of three main differences to the “first generation” typical antipsychotics.
- They carry a lower risk of extrapyramidal side effects, have a beneficial effect on negative symptoms and cognitive impairment, and are suitable for treatment-resistant patients.
- As with the Typical Antipsychotics, these drugs also act on the dopamine system by blocking the D2 receptors.
  - However, they only temporarily occupy the D2 receptors and then rapidly dissociate to allow normal dopamine transmission.
ATYPICAL ANTIPSYCHOTICS

• It is this characteristic of atypical antipsychotics that is thought to be responsible for the lower levels of extrapyramidal side effects compared to conventional antipsychotics.

• Because the atypical antipsychotics have very little effect on the dopamine systems that control movement, they tend not to cause the movement problems associated with typical antipsychotics.

• Other ways where atypical and conventional drugs differ are that the typical antipsychotics block only D2 receptors, however, atypical antipsychotics have a stronger affinity for serotonin receptors and a lower affinity for D2 receptors.
What is happening in the brain:

‘Normal’ Dopamine Synaptic Event

Dopamine  Dopamine Receptors

‘Excessive’ Dopamine Synaptic Event

More Dopamine than Receptors
Antipsychotics block Dopamine Receptors
EVALUATION OF DRUG THERAPY
ANTIPSYCHOTICS VS PLACEBO

Leucht et al (2012) meta-analysis 1959-2011 (65 studies involving 6000 patients). All patients stabilised on typical or atypical antipsychotics

Some were taken off their medication and given a placebo instead. Others remained on their medication

Within 12 months, 64% given placebo relapsed, 27% who stayed on medication relapsed
EXTRAPYRAMIDAL SIDE EFFECTS

Antipsychotics can impact the extrapyramidal area of the brain, which controls motor activity.

- Most common symptoms are Parkinson-related symptoms.

More than ½ patients on typical antipsychotics experience this.

Extended use of antipsychotics can lead to tardive dyskinesia.

This can be distressing so other drugs need to be taken OR they stop taking their medication.
ETHICAL PROBLEMS WITH TYPICAL ANTIPSYCHOTICS

If the side effects, death and psychosocial consequences were taken into account, a cost-benefit analysis would be negative.


“No one shall be subjected to inhumane or degrading treatment or punishment.”
ADVANTAGES OF ATYPICAL OVER TYPICAL ANTIPSYCHOTICS

A key advantage of atypical antipsychotics is that patients experience fewer side effects.

Newer atypical antipsychotics less likely to produce extrapyramidal effects.

This means that patients are more likely to continue with their medication.

So more likely to reduce symptoms.
ARE ATYPICAL ANTIPSYCHOTICS BETTER?

Crossley et al (2010) meta-analysis (15 studies)

Found no significant differences between typical and atypical drugs in terms of the effect on symptoms but differences in the type of side effects.

Atypical = gained weight

Typical = extrapyramidal side effects
Ross and Read (2004) = when people are prescribed medication it reinforces the view that there is something wrong with them

This prevents them thinking about possible stressors that might be reasons for their condition

This reduces their motivation to look for solutions to alleviate the stressors
COGNITIVE BEHAVIOURAL THERAPY (CBT) FOR TREATING SCHIZOPHRENIA
HOW DOES CBT WORK?

• **ASSESSMENT** – patient expresses thoughts and feelings to therapist, realistic goals discussed, current distress used as motivation for change

• **ENGAGEMENT** – therapist empathises with patient’s perspective and their feelings of distress

• **THE ABC MODEL** – patient explains their Activating Event (A) that causes their distress, which produces their Behaviour (B), and the Consequence (C) of that behaviour

• **NORMALISATION** – information that many people have unusual experiences (e.g. hallucinations) in extreme stressful situations reduces patients own anxiety and sense of isolation. Therapist puts psychotic experiences on a continuum with normal experiences to show the patient and so the possibility of recovery seems more likely
HOW DOES CBT WORK?

• **CRITICAL COLLABORATIVE ANALYSIS** – therapist uses **gentle questioning** to help patient understand illogical deductions and conclusions, e.g. “why is it that no one else can hear your voices?”. Although there needs to be trust between patient and therapist for this questioning to work (therapist remains empathetic)

• **DEVELOPING ALTERNATIVE EXPLANATIONS** – patient develops their own alternative explanations for previous unhealthy assumptions. New ideas can be made by therapist if the patient is not forthcoming with their own ideas
CBT FOR PYCHOSIS (CBTp)

• The basic assumption of CBTp is that people often have distorted beliefs, which influence their feelings and behaviours in maladaptive ways
  – E.g. someone with Sz may believe that their behaviour is being controlled by someone/something else

• Delusions are thought to result from faulty interpretations of events. CBTp is used to help the patient identify and correct these faulty interpretations

• CBTp can be delivered in groups, but usually 1-to-1

• NICE recommend at least 16 sessions to treat Sz
CBT FOR PYCHOSIS (CBTp)

• The aim of CBTp is to help people establish links between their thoughts, feelings or actions and their symptoms and general level of functioning.

• By monitoring their thoughts, feelings or behaviours with respect to their symptoms, patients are better able to consider alternative ways of explaining why they feel and behave in the way that they do.

  – This reduces distress and so improves functioning.
NATURE OF CBTp

• In CBTp, patients are encouraged to trace back the origins of their symptoms in order to get a better idea of how they might have developed.

• They are encouraged to evaluate the content of their delusions and consider ways in which they might test the validity of their faulty beliefs.

• Patients may also be set behaviour assignments to improve their general level of functioning.
NATURE OF CBTp

• The learning of maladaptive responses to life’s problems is often the result of distorted thinking by the schizophrenic or mistakes in assessing cause and effect
  – E.g. assuming that something terrible has happened because they wished it

• During CBTp, the therapist lets the patient develop their own alternatives to these previous maladaptive beliefs (ideally looking for alternative explanations and coping strategies that are already present in the patient’s mind)
EVALUATION OF CBT
ADVANTAGES OF CBTp OVER STANDARD CARE

NICE (2014) found consistent evidence that CBTp was effective in reducing rehospitalisation rates compared to standard care (antipsychotic drug).

CBTp also reduces symptom severity, compared to drugs.

HOWEVER, most studies on effectiveness of CBTp have been with patients also on medication at the same time.

THEREFORE, it is difficult to assess effectiveness of CBTp separate from drugs.
EFFECTIVENESS OF CBTp IS DEPENDENT ON STAGE OF THE DISORDER

CBTp appears more effective when made available at specific stages of the disorder and when delivery is adjusted to the stage of the individual.

Addington and Addington (2005) claim that CBTp would not be appropriate at the start of Sz where self-reflection happens.

Becoming more cost effective when drugs have stabilised the condition, group-based CBTp can be used.

Also helps to normalise patients as they are meeting other people with similar issues.

Research = Sz patients with more experience of their Sz = greater realisation of their problems from CBTp.
LACK OF AVAILABILITY OF CBTp

In the UK only 1 in 10 patients get access to CBTp

In North West England, only 6.9% of patients had been offered CBTp

Of those offered CBTp, a significant number refuse the therapy or fail to turn up to sessions (Freeman et al, 2013)

THEREFORE, limiting the effectiveness of CBTp even more
PROBLEMS WITH META-ANALYSIS OF CBTp AS A TREATMENT FOR SZ

Meta-analysis in this area can reach unreliable conclusions about CBTp effectiveness is failure to take into account the study quality.

E.g. fail to randomly allocate participants to either CBTp or control.

Despite this, all studies are grouped together for the meta-analysis.

Juni et al (2001) = clear evidence that methodological issues with a meta-analysis led to biased findings about the effectiveness of CBTp.
THE BENEFIT OF CBTp MAY HAVE BEEN OVERSTATED

More recent and methodologically sound meta-analysis of effectiveness of CBTp as sole treatment for Sz shows it may be less effective than originally thought.

Jauhar et al (2014) = only a “small” therapeutic effect on key symptoms of Sz

HIGHERVER, these small effects disappeared when symptoms were assessed “blind” (assessor not aware of control or therapy condition).

This creates uncertainty of whether CBTp is more effective than drugs AND so has led to conflicting recommendations within the UK (Taylor and Perera, 2015).

England and Wales emphasise non-drug therapies like CBTp, BUT Scotland emphasise antipsychotics.
FAMILY THERAPY (CBT) FOR TREATING SCHIZOPHRENIA
HOW DOES FAMILY THERAPY WORK?

• By reducing levels of expressed emotion (EE) and stress, and by increasing the capacity of relatives to solve related problems, family therapy tries to reduce the incidence of relapse for the person with Sz

• Family Therapy uses a number of strategies:
  – **Psychoeducation** – helping the person and their carers to understand and be better able to deal with the illness
  – **Forming an alliance** with the relatives who care for the person with Sz
  – **Reducing the emotional climate** within the family and the burden of care for family members
  – **Enhancing relatives’ ability to anticipate and solve problems**
  – **Reducing expressions of anger and guilt** by family members
  – **Maintaining reasonable expectations** among family members for patient performance
  – **Encouraging relatives** to set appropriate limits whilst maintaining some degree of separation when needed
HOW DOES FAMILY THERAPY WORK?

• Family therapy is often used alongside drug treatment

• During family therapy sessions, the individual with Sz is encouraged to talk to their family and explain what sort of support they find helpful (and what makes things worse)
FAMILY THERAPY

- These are a range of interventions aimed at the family of someone with Sz
- In their guidance on the treatment and management of Sz, NICE recommended that family therapy should be offered to “all individuals diagnosed with Sz who are in contact with or live with family members”
- They also stress that these interventions should be considered a priority where there are persistent symptoms or a high risk of relapse
- Research has shown that Sz in families that express high levels of criticism, hostility or over-involvement had more frequent relapses than other people with Sz in families with less EE
NATURE OF FAMILY THERAPY

• Family therapy is usually offered for a period of between 3 and 12 months and at least 10 sessions

• Family-based interventions are aimed at reducing the level of EE within the family
  – As EE has shown to increase the likelihood of relapse

• Garety et al (2008) estimate that relapse rates for individuals who receive family therapy as 25% compared to 50% who receive standard care alone
NATURE OF FAMILY THERAPY

- Family therapy involves providing family members with information about Sz, finding ways of supporting an individual with Sz and resolving any practical problems.
- It should also involve the person with Sz if practical.
- A characteristic of Sz is that individuals are often suspicious about their treatment.
  - Involving the individual more actively in their treatment overcomes this problem.
- Family therapy improves relationship within the household because the therapist encourages family members to listen to each other and openly discuss problems and negotiate potential solutions together.
PHAROAH ET AL (2010)

- Procedure
  - 53 studies published between 2002 and 2010 that investigated the effectiveness of family intervention were reviewed.
  - Studies chosen were conducted in Europe, Asia and North America.
  - They compared outcomes from family therapy to "standard care" (i.e. antipsychotic medication) alone.
  - The researchers concentrated on studies that were randomised controlled trials (RCT’s).
• Findings
  – The main results were:
    • **MENTAL STATE** – The overall impression was mixed. Some studies reported an improvement in the overall mental state of patients compared to those receiving standard care (others did not)
    • **COMPLIANCE WITH MEDICATION** – The use of family intervention increased patients’ compliance with medication
    • **SOCIAL FUNCTIONING** – Although appearing to show some improvement on general functioning, family intervention did not appear to have much of an effect on more concrete outcomes such as living independently or employment
    • **REDUCTION IN RELAPSE AND RE-ADMISSION** – There was a reduction in the risk of relapse and a reduction in hospital admission during treatment and in the 24 months after
EVALUATION OF FAMILY THERAPY
WHY IS FAMILY THERAPY EFFECTIVE?

Pharoah et al (2010) meta-analysis = family therapy can be effective in improving clinical outcomes like mental state and social functioning.

HOWEVER, others suggest that the main reason for its effectiveness may be more to do with the fact it increases medication compliance LESS to do with clinical outcomes.

SO, patients are more likely to be successful on medication as they are more likely to take them.
METHODOLOGICAL LIMITATIONS OF FAMILY THERAPY STUDIES

Problem with Random Allocation = a large number of studies said to have used random allocation (in Pharoah et al’s meta-analysis) were from China

It appears they had not used random allocation

Lack of Blinding = possible observer bias where raters were not “blinded” to the condition (family therapy or drugs) to which people were allocated
ECONOMIC BENEFITS OF FAMILY THERAPY (FT)

NICE review = FT shows significant cost savings **IN ADDITION TO** medication

Extra cost of FT is balanced by a reduction in costs of hospitalisation due to the lower relapse rates associated with FT

FT reduces relapse rates for a significant period after completion

**THEREFORE,** the cost savings of FT would be even higher
IMPACT ON FAMILY MEMBERS

Lobban et al (2013) analysed the results of 50 FT studies

60% reported a significant positive impact of FT on at least one outcome category for relatives

E.g. coping and problem-solving skills, family functioning and relationship quality (including EE)

HOWEVER, the methodological quality of the studies was generally poor

Making it difficult to distinguish between effective and ineffective interventions
IS FAMILY THERAPY WORTHWHILE?

Garety et al (2008) found no better outcomes for patients given sessions of FT compared to those who had carers but no FT.

They also found that most carers displayed relatively low rates of EE.

This could reflect widespread culture changes in carers’ knowledge and attitudes of Sz.

Garety et al suggest that FT may not improve outcomes of many patients further than a good standard.
TOKEN ECONOMY FOR TREATING SCHIZOPHRENIA
HOW DOES TOKEN ECONOMY WORK?

• The principle of TE is based on the theory of Operant Conditioning
  – This is the relationship between a behaviour and environmental events; particularly positive reinforcement (i.e. an increase in the frequency of a particular behaviour when it is followed by a desirable event)

• There are two types of positive reinforcer:
  – **Primary reinforcers** are anything that gives pleasure (e.g. food, comfort) or remove unpleasant states (e.g. that alleviate boredom). They do not depend on learning in order to gain their reinforcing value
  – **Secondary reinforcers** initially have no value to the individual, but gain reinforcing properties as a result of being paired with primary reinforcers. In TE, the tokens are given to the patients when they engage in a target behaviour (e.g. taking care of their personal hygiene, tidying up, cleaning their room). I.e. the token is the secondary reinforcer
HOW DOES TOKEN ECONOMY WORK?

• To be effective, the token has to be given immediately after a target behaviour
• If not, then another behaviour (e.g. an argument) may have been performed in the intervening period which will be reinforced, not the desired behaviour
THE TOKEN ECONOMY

• This is a form of behavioural therapy where clinicians target behaviours that they believe will improve the patient’s engagement in daily activities

• These may be as simple as dressing themselves, or helping another patient (socially oriented behaviour)

• Tokens are awarded whenever the patient engages in one of the target behaviours, and these tokens can later be exchanged for various rewards and privileges

• The idea is that the patient will engage more often with desirable behaviours as they become associated with these rewards and privileges
THE TOKEN ECONOMY

- Ayllon and Azrin (1968) used TE on a ward of female schizophrenic patients.
- They were given plastic tokens, with “one gift” written on them, for behaviours like making their bed or carrying out domestic chores.
- These were then exchanged for privileges such as being able to watch a movie.
- The researchers found that the use of TE with these patients increased dramatically the number of desirable behaviours that the patients performed each day.
ASSIGNING VALUE TO THE TOKENS

• To give the neutral tokens some “value”, it needs to first be repeatedly presented alongside or immediately before the reinforcing stimulus
• The reinforcing stimulus may be food, privileges or other incentives
• By pairing the neutral tokens with the reinforcing stimulus, the neutral token eventually acquires the same reinforcing properties
• This results in classical conditioning (as the neutral tokens become secondary reinforcements and can therefore be used to modify behaviour)
REINFORCING TARGET BEHAVIOURS

• When patients perform the desirable target behaviours, the clinician awards them tokens.
• When a token can be exchanged for a variety of different privileges and rewards, it is referred to as a generalised reinforcer.
• The more items or rewards that the token can be exchanged for, the more powerful the token becomes.
• Sran and Borrero (2010) compared behaviours reinforced by tokens that could be exchanged for one single highly preferred edible item with tokens that could be exchanged for a variety of preferred edible items.
• They found that all participants had higher rates of responding in those sessions where tokens could be exchanged for a variety of items.
THE “TRADE”

• An important part of TE is the exchange of tokens for backup rewards chosen by the clinician
  – These may include food, sweets, being able to watch a movie, etc

• During the early stages of TE, frequent exchange periods mean that patients can be quickly reinforced and target behaviours can then increase in frequency

• The effectiveness of the TE may decrease if more time passes between presentation of the token and exchange for the backup reinforcers (Kazdin, 1977)
EVALUATION OF TOKEN ECONOMY
Dickerson et al (2005) meta-analysis on effectiveness of TE in a psychiatric setting

11 out of 13 studies reported beneficial effects of TE

THEREFORE, these studies provide evidence of the effectiveness of TE in increasing adaptive behaviours of patient with SZ

HOWEVER, they did acknowledge that **many of these studies had methodological flaws** that limited their impact in the overall assessment of TE in this context
DIFFICULTIES ASSESSING THE SUCCESS OF A TOKEN ECONOMY

Major problem in assessing the effectiveness of TE is that studies of their use tend to be uncontrolled (Comer, 2003)

I.e. there is no control group to compare to (a group that does not have TE)

So patients’ improvements can only be compared to their past behaviours

As patient improvements could be down to extraneous variable (like staff attention) rather than TE
LESS USEFUL FOR PATIENTS LIVING IN THE COMMUNITY

TE has only been shown to be effective in a hospital setting. In a psychiatric ward, inpatients receive 24-hour care, so staff can control and monitor the patients appropriately.

HOWEVER, outpatients living in the community only receive day treatment for a few hours a day (the only time the TE could be used in a day).

THEREFORE, even if TE did produce positive results in a ward setting, the results may not be maintained beyond this environment.
ETHICAL CONCERNS

In order to make reinforcement effective, clinicians may exercise control over important primary reinforcers like food or privacy.

Patients may then exchange tokens if they display target behaviours (e.g. better personal hygiene).

HOWEVER, all humans have basic rights (to food, privacy, etc) that cannot be violated regardless of the positive consequences achieved by manipulating them within TE.
DOES IT ACTUALLY WORK?

There is no conclusive answer to this

Very few randomised trials have been carried out to support the effectiveness of TE in managing Sz

This lack of support is unacceptable and so TE programmes have been used less in much of the developed world

HOWEVER, TE may still be an important treatment if randomised trials were carried out and supported it (McMonagle and Sultana, 2000)

They suggest that this is only likely to be possible in developing countries where some of TE is still used
AN INTERACTIONIST APPROACH
The Diathesis-stress model sees Sz as a result of an interaction between biological (diathesis) and environment (stress) influences.

People have a varying levels of genetic vulnerability to Sz, however, whether or not they develop Sz is partly dependent by this vulnerability but also partly by the amount of stresses they experience over their lifetime.
DIATHESIS

• There are findings that support the fact that there is a genetic role for Sz
  – An identical twin of a person with Sz is more likely to develop Sz than a sibling or non-identical twin
  – Adoptive relatives do not share the increased risk of biological relatives (Tienari et al, 2004)

• However, 50% of identical twins where one twin has Sz, the other never develops it
  – This discordance among identical twins indicates that environmental factors must also play a role in determining whether a biological vulnerability for Sz actually develops into the disorder
STRESS

• Stressful life events that can trigger Sz takes a variety of forms (e.g. childhood trauma, stresses associated with living in a highly urbanised environment)
  – Varese et al (2012) found that children who experienced severe trauma before the age of 16 were three times more likely to develop Sz later in life compared to the general population
  – There was a link between the level of trauma and the likelihood of developing Sz (more severely traumatised the more risk)
STRESS

- A meta-analysis by Vassos et al (2012) found the risk for Sz in the most urban environments was estimated to be 2.37 times higher than in the most rural environments.
  - The reason for this link is not clear, although it may be possible that the more adverse living conditions of densely populated urban environments may contribute.
  - Lots of people live in densely populated areas, but only a few will develop Sz.
  - The relationship between urban stress and Sz is conditional on some other factors (i.e. a pre-existing genetic risk of Sz or some other biological vulnerability for Sz).
ADDITIVE NATURE OF DIATHESIS AND STRESS

• There are several ways in which a combination of diathesis and stress can lead to the onset of Sz
  – E.g. relatively minor stressors may lead to the onset of Sz for an individual who has a high vulnerability, or a major stressful event might cause a similar reaction in a person who has a low vulnerability

• Whatever the combination, diathesis and stress add together in some way to produce the disorder
This study tested the hypothesis that genetic factors moderate susceptibility to environmental risks associated with adoptive family functioning.
TIENARI ET AL (2004)

PROCEDURE

- Hospital records were reviewed for nearly 20,000 women admitted to Finnish psychiatric hospitals between 1960 and 1979, identifying those who had been diagnosed at least once with schizophrenic or paranoid psychosis.
- The list was checked to find those mothers who had one or more of their offspring adopted away.
- The resulting sample of 145 adopted-away offspring (the high-risk group) was then matched with a sample of 158 adoptees without this genetic risk (the low-risk group).
Both groups of adoptees were independently assessed after a median interval of 12 years, with a follow-up after 21 years.

Psychiatrists also assessed family functioning in the adoptive families using the OPAS (“Oulu Family Rating Scale” – translated into English).

This scale measures families on various aspects of functioning such as parent-offspring conflict, lack of empathy and insecurity.

The interviewing psychiatrists were kept blind as to the status of the biological mother (i.e. Sz or no Sz).
FINDINGS

- Of the 303 adoptees, 14 had developed Sz over the course of the study.
- Of these 14, 11 were from the high-risk group and 3 were from the low-risk group (control group).
- However, being reared in a “healthy” adoptive family (low OPAS ratings) appeared to have a protective effect even for those at high genetic risk for Sz.
FINDINGS

• High-genetic-risk adoptees reared in families with low OPAS ratings were significantly less likely to have developed Sz than high-genetic-risk adoptees reared in families with high OPAS ratings.

• In adoptees at high genetic risk of Sz, adoptive-family stress was a significant predictor of the development of Sz. This was not the case in those at low genetic risk.
EVALUATION OF THE INTERACTIONIST APPROACH
DIATHESIS MAY NOT BE EXCLUSIVELY GENETIC

Most diathesis-stress models emphasise “vulnerability”

Which assume causes neurochemical abnormalities that result in an increased risk for Sz

HOWEVER, increased risk could be due to brain damage caused by environmental factors

A vulnerability to Sz if they experience birth complications

This risk of developing Sz later in life is 4 times greater if an individual has prolonged labour (leading to O2 deprivation)
URBAN ENVIRONMENTS ARE NOT NECESSARILY MORE STRESSFUL

Not all research agrees with Vassos et al’s (2012) findings (living in densely populated urban environment was a significant stress factor for Sz)

Romans-Clarkson et al (1990) found no urban-rural differences in mental health in woman (New Zealand)

Paykel et al (2000) found urban-rural difference, BUT noted that these disappeared after adjusting the socioeconomic differences for the two groups

SUGGESTING that, social adversity may be a significant trigger for onset of Sz, BUT claims that social adversity and urbanisation are identical is likely to be an over-simplification
DIFFICULTIES IN DETERMINING CAUSAL STRESS

Diathesis-stress models refer to stressful events that occur close to the onset of Sz.

However, it is possible that stressors earlier in life can influence how people respond to later stressors and so increase susceptibility to Sz.

Hamman (1992) individuals fail to develop effective coping skills when they have used maladaptive methods in childhood.

Therefore, compromising their resilience and increases vulnerability as ineffective coping skills may make life generally more stressful.
LIMITATIONS OF THE TIENARI ET AL STUDY

Researchers in Tienari et al (2004) study identified limitations of their study, particularly in the assessment of adoptive families.

E.g. when psychiatrists assessed stress in adoptive families using the OPAS scale, they were assessing family functioning only at one point in time.

Tienari et al acknowledged that this fails to reflect development changes in family functioning over time.

Observing mutual interactions between the adoptive family and the adoptees makes it impossible to determine how much of the stress observed is assigned to the family and how much is actually caused by the adoptee.
IMPLICATIONS FOR TREATMENT

If the onset of Sz is a result of genetic vulnerability and environmental stress combined, then **this has implications for the treatment**

Although genetic vulnerability is difficult to control, **other factors known to interact with genetic vulnerability can be addressed**

E.g. Børglum et al (2014) found that women infected with cytomegalovirus during pregnancy were more likely to have a child who developed Sz, but only if mother **and** child carried a particular gene defect

This suggests that anti-viral medicine during pregnancy may prevent the onset of Sz in children who’s mother is known to have this gene defect.