SCHIZOPHRENIA WORKBOOK

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Classification of Schizophrenia

Symptoms

1) What is the literal meaning of the work schizophrenia, who coined the term and when?

2) A common error when talking about schizophrenia is thinking that it means 'split personality'. What is the actual split which schizophrenics experience?

3) List the 5 main symptoms of schizophrenia below. For each one give an example.

-
-
-
-
-

The prevalence of schizophrenia is approx **1%** of the general population (lifetime risk). Interestingly it is more common in males from teenage years up to middle age, and more common in women around the time of the menopause. It is also more common in the lower classes although this may be a result of having schizophrenia rather than a cause of it.

4) Wing (1992) argued for a distinction between **primary** and **secondary** impairments in schizophrenia. **Primary** impairments are an **intrinsic** part of the disorder; **secondary** impairments are **social** and **psychological** problems that result from the disorder.

Research and draw Wing's diagram of primary and secondary impairments below:

Subtypes of Schizophrenia

Schizophrenia is not thought to be a 'single' condition but instead a label applied to a collection of disorders. This means that it can be broken down into different subtypes.

5) One way of dividing schizophrenia up is to talk about **positive symptoms** and **negative symptoms** and the **syndromes** (collection of symptoms) which relate to these symptoms. Outline below what is meant by:

Positive symptoms:

Negative symptoms:

6) In which type of schizophrenia is the prognosis worse?

A further way of dividing schizophrenia up is in terms of 'types' I and II.

7) Show below how these types differ:

Type I:

Type II:

Evaluation

8) Explain why it may be too simplistic to talk about schizophrenia simply in terms of positive and negative symptoms. Which third type exists that is unrelated to the others?

9) Positive and negative symptoms exist on a continuum rather than being a dichotomy. Explain what this means.

10) Why might having one term 'schizophrenia' for these different disorders be misleading?

Diagnosing Schizophrenia

The DSM IV can be used to diagnose schizophrenia. Fill in the table below showing the DSM criteria for schizophrenia

A. Characteristic symptoms	
B. Social / occupational dysfunction	
C. Duration	

The DSM lists 5 *types* of schizophrenia (N.B. this therefore breaks down type I and type II into more specific types)

11) Give a description for each of the types of schizophrenia listed below

Paranoid

Catatonic

Disorganised

Undifferentiated

Residual

Explanations of Schizophrenia

Biological (medical) Model

The biological (medical) model views schizophrenia as a **physical** condition arising from defects in **genes**, **neurotransmitters**, and **the neuro-anatomy**. It therefore believes that the best treatment for it is drugs.

Genes
Family studies:

1) What is the risk of getting schizophrenia in the general population?

2) What is the risk in the following cases:

a) Parent has schizophrenia:

b) Sibling has schizophrenia:

c) Both parents have schizophrenia:

Evaluation

3) Explain one criticism of using family studies to ascertain the genetic contribution for schizophrenia

Twin studies

If schizophrenia is genetic then concordance rates should be higher for MZ than DZ twins, given that they share 100% rather than 50% of their genes.

4) Give the average concordance rate for:

MZ twins:

DZ twins:

Evaluation

There are a number of problems with using twin studies as evidence for the genetic contribution to schizophrenia:

MZ twins are always the **same sex** whereas DZ twins can be different sexes. This is this particularly important as schizophrenia is more common in males up to middle age and more common in females after the menopauase. Same sex children will also have more similar environments which may have an effect on the concordance rates.

Further ways in which the environment for MZ twins may be more similar than for DZ twins is that MZ twins are more likely to be treated the same way as they look the same. MZ twins are known to mimic each other,

Twin studies are also blighted by very small samples and many of the earlier studies used unreliable zygosity tests e.g. going on looks rather than a blood test.

Finally the 'proband' method favoured by many researchers' means that if both twins have schizophrenia then they may be counted twice (as each twin will be a counted as a proband who has a twin with schizophrenia). This leads to an exaggeration in both the MZ and DZ rates.

However if concordance rates include related disorders to schizophrenia e.g. **schizoid personality disorder** then the concordance rates increase by a further 50% more.

Adoption studies

5) Explain why adoption studies have an advantage over twin studies:

6) Explain the findings from Heston (1966). What can we conclude from this study?

Evaluation

Adoption studied provide more reliable data than other research methods, however these again use very small samples. This still doesn't account for the 66% of schizophrenics who don't appear to have a biological relative with the disorder.

A ground breaking study by Tienari in Finland showed the importance of the environment once a child has been adopted out of a schizophrenic family. If the child's adoptive family is high stress then the chances of the child developing schizophrenia increase considerably.

However if the adoptive family is very low stress then the child has roughly the general populations chance of developing schizophrenia. This relationship is called a **diathesis-stress** relationship (diathesis = genetic predisposing factors, stress = environmental stressors which trigger the disorder).

Neuro-chemical Explanations

7) List below the three techniques used to investigate neurotransmitter dysfunction in schizophrenia:

Dopamine hypothesis

Dopamine is a neurotransmitter; it is one of the chemicals in the brain that causes neurons to fire. The original dopamine hypothesis stated that schizophrenia is caused by an excess of dopamine, causing neurons to fire too often and send too many messages around the brain (hence the disorganisation and other symptoms).

8) How does the use of drugs (amphetamines and anti-psychotic) support the idea that schizophrenia is linked to high levels of dopamine?

Further evidence for the role of dopamine comes from Parkinson's disease. This is caused by a lack of dopamine (opposite of schizophrenia). L-Dopa which is used to treat it works by increasing dopamine levels. This can produce schizophrenic symptoms in individuals with no history of the disorder.

Evaluation

However A number of post-mortem studies failed to find evidence that schizophrenia is caused by too much dopamine. The hypothesis was then changed to 'too many dopamine receptors' – autopsies of dead people found an unusually large number of dopamine receptors (however this increase in dopamine receptors may have been due to the anti-psychotic drugs they were taking rather than the schizophrenia itself). Subsequent PET scans have indicated that schizophrenics have considerably more dopamine receptors in the brain than normal people (and these were carried out on individuals who had not taken medication this ruling out the problem above).

There remain problems with the dopamine theory though:

Antipsychotic drugs which treat Schizophrenia by reducing dopamine levels are only really effective on type I schizophrenia i.e. the **positive** symptoms rather than the **negative** symptoms. Furthermore, when these drugs do have an effect, despite blocking dopamine receptors immediately they only become clinically effective after weeks or even months.

Newer anti-psychotic drugs such as Clozapine actually work less on dopamine and more on serotonin – they are also more effective in treating type II schizophrenia i.e. the **negative symptoms**. This has caused some psychologists to suggest that schizophrenia rather than being due to too much dopamine is due to a faulty interaction of dopamine and seretonin.

The treatment **aetiology fallacy** states that just because drugs which decrease schizophrenia also reduce dopamine does not mean that schizophrenia was cause by too much dopamine.

Overall the evidence for a relationship between neurotransmitters and schizophrenia is conflicting and inconclusive due to the widespread symptoms it is unlikely that any single neurotransmitter is to blame.

Neuro-anatomical Explanations of Schizophrenia

9) Describe techniques which can be used to uncover neuroanatomical differences in people with schizophrenia:

10) Explain the abnormalities found in the following areas of the brain:

Reduced cerebral cortex

Enlarged ventricles

Reduced blood flow in cerebral cortex

Evaluation

Two main problems exist with neuro-anatomical evidence:

Firstly the findings are inconsistent. A number of studies have shown no consistent differences in schizophrenic's brains i.e. no foolproof way of genuinely differentiating the schizophrenic from the normal brain. Secondly even if anatomical differences do exist these may be an effect of the disorder not its cause e.g. caused by faulty cognitions or some other biological mechanism e.g. high dopamine levels.

Cognitive Explanations of Schizophrenia

The cognitive approach focuses on information processing deficiencies in the schizophrenic's mind. In particular it focuses on cognitive biases and distortions. In particular the approach is helpful in trying to understand some of the positive symptoms of schizophrenia.

Hallucinations

73% of schizophrenics experience hallucinations

1) How does Bentall (1990) explain the hallucinations experienced by schizophrenics?

5 Factor model (Slade & Bentall)

Slade & Bentall suggest that schizophrenics have a tendency to engage in what they call 'sensory deception' whereby they fail to discriminate between external sources of information (i.e. things that are real) and internal sources of information (i.e. things that are imagined). They do not generate internal sources of information differently from the rest of us; they simply **interpret** these stimuli differently.

2) Draw below the 5 factor model proposed by Slade & Bentall

Evaluation of 5 factor model

The idea that hallucinations are reinforcing because they bring relief from anxiety (stage 4) has been challenged; Close and Garety (1998) found that there is an *increase* in anxiety not a *relief* in it following hallucinations which suggests that they would not be reinforcing but quite the opposite!

Whilst the 5 factor model is based on some sound clinical observations and experimental evidence, there is very little *direct* evidence for their theory.

The main problem with this model is that it only explains hallucinations where schizophrenia has a number of other important aspects (see DSM criteria).

Delusions

3) Explain what is meant by a delusion and why they are difficult to define:

There are 2 main types of cognitive theory on delusions:

Delusions are the result of abnormal cognitions in reasoning

This theory views delusions as stemming from the schizophrenics attempts to defend themselves from low self-esteem and depression.

4) Explain below Bentall's (1991) explanation for delusions and give an example:

Delusions are the product of abnormal perceptions

This theory proposes that delusions are an adaptive and rational response to the hallucinations the schizophrenic is experiencing.

5) Draw below Maher's (1974) Anomolous Experience Model:

Evaluation of cognitive theories of schizophrenia

Maher's model (above) has provided a useful therapeutic treatment (see treatments section later on).

Cognitive theories are useful in uncovering biases and distortions in the schizophrenic but they are not so effective at stating where these biases come from in the first place. For this the cognitive model often has to turn to the biological model. This is essentially a cause and effect problem - are the cognitive biases the cause of schizophrenia or are they in turn caused by something else e.g. genes and neurotransmitters.

It is difficult to discard much of the biological evidence e.g. adoption studies which point to important inherited factors and neuro-anatomical evidence which shows that brain damage may cause they cognitive distortions.

Sociocultural Explanations of Schizophrenia

Sociocultural explanations look at processes going on outside the individual, at their environment and family factors which may cause schizophrenia

Labelling

1) Explain Szasz's (1979) view of schizophrenia. Szasz wrote a famous book called 'The myth of mental illness' - what do you think he meant by this title?

2) Explain Scheff's **Labelling theory**. Give an example of **'residual rule'** breaking and explain what is meant by a **self-fulfilling prophecy**.

3) Outline the key aims, method, results and conclusion of Rosenhan's study

4) How does Rosenhan's study demonstrate the effects of labelling?

Evaluation of Labelling Theory

Rosenhan's study has a number of ethical issues: deception, informed consent and harm. Critics have also claimed that it lacks validity as doctors can only diagnose the behaviours they see and do not expect to be tricked.

Labelling theory only accounts for how symptoms of schizophrenia are maintained; it does not explain the initial cause nor does it offer any kind of treatment (other than abolishing labels altogether).

Labelling theory ignores the compelling biological evidence for schizophrenia and has been accused of trivialising a very serious disorder with seriously ill people who badly need help and medication.

Family dysfunction

5) Explain below **Bateson's Double Bind** theory:

6) Explain what is meant by the following

Schismatic families:

Skewed families:

7) Explain what is meant by **high expressed emotion.** How did Brown et al (1958) discover this as an important factor in schizophrenia?

8) Outline below the aim, method, results, conclusion and evaluation of Bebbington and Kuiper's (1994) study.

Evaluation of family dysfunction

Tienari's adoption study in Finland provides some generalised support for family theories as those children who were adopted into high-stress families were far more likely to develop schizophrenia than those who were adopted into low stress families. Although this study does not provide support for any of the *specific* theories shown above, recent studies have shown that high EE families are common with anorexia and depression as well which suggests that they may create an environment which fosters mental disorders in general.

There is a problem with how high EE is measured. Often it will involve a single observation or interview which may not give an accurate representation of how that family normally functions (for instance due to the demand characteristics of being observed).

All of the family theories proved popular in the 1960s but as medical evidence increases these theories have lost favour. Whilst there is some evidence that family systems contribute to onset or relapse of schizophrenia, it could be that the reverse is true: - having a schizophrenic in the family causes the family to deteriorate and become dysfunctional. He or she may create the family problems – in which case the family is not the cause! It is likely that it in fact works in both directions like a vicious cycle.

Treatments for Schizophrenia

Anti-psychotic Drug Treatment

Drug treatment is obviously based on a biological model of schizophrenia, assuming at least a partial biochemical cause for schizophrenia. The antipsychotic drugs revolutionised the treatment of schizophrenia in the 1950s. Previously the only way to treat schizophrenia had been to institutionalise the patient, but the discovery of effective drugs to control schizophrenic symptoms meant that institutionalisation was very often not necessary.

1) Outline below how **chlorpromazine** works. Which symptoms does it treat? What is its effectiveness rate and how has its effectiveness been demonstrated?

Since the advent of conventional anti-psychotics, a group of drugs called **atypical anti-psychotics** have been developed and have proven particularly effective in treating those who are resistant to the likes of chlorpromazine. These drugs differ from conventional APs by a) blocking the action of dopamine only temporarily, which allows for more natural functioning and fewer side effects e.g. tardive dyskinesia (involuntary mouth movements) and b) blocking serotonin function as well. An example of atypical anti-psychotics is Clozapine.

Evaluation of drug treatment

2) list some of the **side effects** associated with *typical* neuroleptic medication e.g. chlorpromazine:

3) What is the most important side-effect of the *atypical* drug clozapine?

4) What is the main problem with atypical drugs?

5) Why can it be said that medication only 'controls' rather than 'cures' the patient?

6) Has a drug been found yet which is effective in controlling negative symptoms?

Behavioural Treatments for Schizophrenia

Token economy

7) What is meant by a 'token economy'? When and why is it used? Give an example of how it might be used.

Social Skills training

This attempts to modify the behaviour of people with schizophrenia on the basis that they lack and therefore need to learn the skills necessary for successful interactions and relationships with others. Social Learning and behaviourist techniques are used such as *modelling* good behaviour, reinforcement by the therapist and role-playing to help acquire verbal and non-verbal skills.

8) List below some of the activities that a typical programme might involve:

9) How often will patients have social skills training and what ratio of trainers to patients are there?

Evaluation of behavioural therapy

There is lots of empirical evidence to suggest that patients find social skills training useful however it does not always generalise well to real life settings where the patient may just resort to maladaptive behaviours again.

Some consider behavioural therapy to be dehumanising - the patient is being treated a bit like an animal to be conditioned.

The therapy is expensive but less so that cognitive-behavioural as the ration of therapists to patients is 1:5. If it help to prevent relapse then it is worth the expense.

Behavioural therapy alone is never enough to tackle schizophrenia and therefore it is always used in combination with other therapies; e.g. drugs and cognitive-behavioural therapy.

Psychotherapy for schizophrenia

10) Which range of therapies does the term 'psychotherapy' cover?

11) What is the main reason why psychotherapies have often been ineffective in treating schizophrenia?

Cognitive-behavioural therapy

12) Fill in the grid below of Tarrier et al's study

Aim:

Method:

Results:

Conclusion:

Evaluation:

13) Explain below what is meant by '**coping strategy enhancement**' (CSE) and how it is used:

14) Outline below the six steps used in CSE

1.

2.

3.

4.

5.

6.

15) Explain how the following 2 components are used to progress CSE therapy further:

Education / Raport training

Symptom targeting

Evaluation of CSE

16) In Tarrier's study what % improvement did patients show using CSE compared to a control group?

17) In the same study how did CSE compare to problem-solving therapy? When was the follow up study done and what did they find?

18) What was the drop out rate in Tarrier's study and why is a problem?

Cognitive therapy

19) What is meant by 'reality testing' and how is it used in cognitive therapy?

20) Why was it thought originally that 'reality testing' would not work on schizophrenic patients?

21) What are the two key principles that underline cognitive therapy

22) Describe below Chadwick's (1996) case study of 'Nigel' which supports the use of cognitive therapy

Evaluation of cognitive therapy

Evidence suggests that cognitive therapy works best when combined with drug treatment and that patients respond better to drug treatment when it is combined with CBT. However on its own, cognitive therapy is rarely as effective as drug treatment for schizophrenia.

Psychotherapy generally is certainly not as effective on schizophrenia as it is on depression which suggests that schizophrenia's origins may be more biological. One of the problems with assessing the success of psychotherapy is controlling important extraneous variables; a number of studies have shown that improvements in mental disorders might occur simply because the therapist becomes a form of social support rather than because of any key technical ingredient which the therapy offers.

Community care

23) Explain how the following led to a decrease in people being hospitalized with schizophrenia and an increase in the number being cared for in the community:

The anti-psychiaty movement

Discovery of neuroleptic drugs

The current approach to community care

24) Complete the grid below for the study by Stein and Test (1980)

Aim:

Method:

Results:

Conclusion:

Evaluation:

Evaluation of community care

Those who favour community care argue that quality of life can be improved at little extra cost, although Stein and Test's study above indicates that in fact continual time and effort is required to keep the schizophrenic in the community and therefore there is an ongoing cost. In many cases the burden falls on the families of individuals since the level of care provided by the NHS is not good enough - this therefore carries major costs for them.

Ethically it is far better to treat schizophrenics in the community as many argue that mental hospitals are dehumanising, stigmatizing and actually make the condition worse (as patients become helpless and dependent) - remember One Flew Over the Cuckoos Nest.

However there are risks to community care - if it is not done properly, occasionally tragedies can happen which are often highlighted in media reports such as the murder of Jonathon Zito on the tube in London by a schizophrenic who had paranoid delusions. Often these killings take place by schizophrenics who are 'self medicating' (i.e. drugs and alcohol) - which suggests a failing in the measures that have been set up to help them.