

AQA Psychology A-level

Option 2: Schizophrenia

Summary Notes

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Part 1: Diagnosing Schizophrenia: Symptoms and Issues

Schizophrenia is characterised by a range of **positive and negative symptoms**. Individuals may experience either type of symptom or a combination of both.

1. Positive Symptoms

Positive symptoms involve an **addition to normal experience or behaviour**.

- **Hallucinations** involve **experiencing sensory perceptions that are not actually present**. The most common type is **auditory hallucinations**, where a person hears voices or sounds that other people cannot hear.
- **Delusions** are **strongly held false beliefs** that are not based on reality and are difficult to change. For example, a person may believe that they are being **followed** or **persecuted** despite having no evidence that this is happening.

2. Negative Symptoms

Negative symptoms involve a **loss or reduction of normal behaviour or functioning**.

- **Speech poverty** involves a **reduction in the amount and quality of speech**. A person may give very short answers and provide little additional information.
- **Avolition** is a **lack of motivation to carry out goal-directed behaviour**. This may make everyday activities, such as getting dressed, working or maintaining personal hygiene, difficult.

Issues in Diagnosis

Diagnosing schizophrenia can be **complex**, as several factors can affect how the disorder is **identified and understood**.

Issue	What it means	What it might look like
Co-morbidity	Experiencing two or more disorders at the same time.	Someone with schizophrenia may also experience depression or substance-use problems.
Culture bias	Cultural beliefs can influence how experiences and symptoms are understood and expressed.	Hearing the voice of a deceased relative may be viewed as a spiritual experience in one culture but interpreted as a hallucination in another.
Gender bias	Differences between males and females may influence how symptoms are experienced or interpreted.	Males tend to have an earlier onset and more negative symptoms, while females tend to have a later onset and more affective symptoms.
Symptom overlap	Different psychological disorders can involve the same or similar symptoms.	Hallucinations and delusions can occur in schizophrenia but can also occur in other psychological disorders such as depression



AO3 - EVALUATION

A strength of distinguishing between positive and negative symptoms is that it has **practical applications for treatment**. **Antipsychotic medication** is generally more effective at reducing **positive symptoms**, such as hallucinations and delusions, while negative symptoms such as **avolition and speech poverty** are often more difficult to treat. This means that identifying the type of symptoms a person is experiencing can help clinicians understand their **treatment needs and likely response to treatment**. Therefore, **distinguishing between positive and negative symptoms has practical value for the treatment of schizophrenia**.

One issue with diagnosing schizophrenia is **co-morbidity, as it can make it difficult to differentiate schizophrenia from other psychological disorders**. **Buckley et al. (2009)** found that **29%** of people with schizophrenia also experienced **PTSD**, while **50%** experienced **depression**. The high levels of co-morbidity suggest that schizophrenia frequently occurs alongside other disorders, meaning some symptoms may be **shared between disorders**. This can make it difficult for clinicians to determine whether symptoms are caused by schizophrenia, another disorder, or both. Therefore, **co-morbidity can make it difficult to distinguish schizophrenia from other psychological disorders**.

One issue with diagnosing schizophrenia is **gender bias, as differences between men and women may make symptoms more difficult to recognise accurately**. **Longenecker et al. (1995)** could not find an explanation for the sudden increase in the number of **male schizophrenia diagnoses after the 1980s**. **Cotton et al. (2003)** suggested that this may be due to gender bias, as there are no major differences in **genetic susceptibility** to schizophrenia between men and women. Women may have better **interpersonal functioning** and may be able to continue working and functioning despite experiencing symptoms, meaning their symptoms may be **masked or appear less severe**. This could make clinicians less likely to recognise their symptoms as schizophrenia. Therefore, **gender bias may result in schizophrenia being underdiagnosed in women and overrepresented in men**.

A further issue with diagnosing schizophrenia is **cultural bias**, as cultural differences can affect whether an **experience is considered a symptom of schizophrenia or a normal cultural experience**. **Escobar et al. (2012)** highlighted that **African Americans are more likely to be diagnosed with schizophrenia** than some other groups. For example, **hearing voices** may be considered a positive experience involving **spirituality or communication with ancestors** in some cultures and may even be encouraged. However, clinicians may interpret this experience as an **auditory hallucination**, particularly when using diagnostic criteria developed within Western cultures. This could lead to culturally normal experiences being **mistaken for symptoms of schizophrenia**, increasing the likelihood of **misdiagnosis**.



Part 2: Biological Explanations of Schizophrenia

The biological approach suggests that schizophrenia is caused by **physical factors** within the body, including **genetic vulnerability**, **neurotransmitter abnormalities** and differences in **brain structure and activity**.

Genetic Explanation

The genetic explanation suggests that schizophrenia has a **heritable component**, meaning that individuals may inherit a biological vulnerability to developing the disorder.

Gottesman (1991) investigated the relationship between **genetic similarity** and the likelihood of developing schizophrenia.

He found that the risk of developing SZ increased as genetic similarity increased:

- **Monozygotic (Identical) twins:** 48%
- **Dizygotic (Fraternal) twins:** 17%
- **Siblings:** 9%
- **Parents:** 6%

This supports a **genetic basis** for schizophrenia because MZ twins share **100% of their genes** and showed the highest concordance rate.

However, concordance rates are never **100%**, suggesting genetics alone cannot explain schizophrenia. This indicates **environmental factors** must also contribute to SZ.

1. Candidate genes and schizophrenia

Schizophrenia is thought to be a **polygenic disorder**, meaning that it is influenced by multiple genes rather than a single “schizophrenia gene”.

Ripke et al. (2014) conducted a large-scale **Genome-Wide Association Study (GWAS)** involving up to **36,989 people with schizophrenia and 113,075 controls**. They identified **108 genetic loci** associated with **schizophrenia risk**. A **genetic locus** is a **specific position on a chromosome where a particular gene or genetic variant is located**.

The findings suggest that **many different areas of the genome** are involved in schizophrenia. However, each genetic variant makes only a **small contribution to an individual's risk**, meaning that no single gene is likely to cause schizophrenia by itself. Instead, vulnerability is likely to result from the **combined effects of many genetic variants**, alongside environmental factors.



2. Neural Correlates

Neural correlates are patterns of **brain structure or activity** that are associated with particular symptoms of schizophrenia. Researchers can use **brain-imaging techniques**, such as fMRI scans, to identify differences between people with schizophrenia and those without the disorder.

Different neural correlates may help explain **different symptoms** of schizophrenia.

Negative symptoms – ventral striatum

Juckel et al. (2006) found **reduced activity in the ventral striatum** in people with schizophrenia. The ventral striatum is involved in **reward and motivation**. Reduced activity may therefore make it harder for individuals to experience pleasure or motivation from everyday activities, which could contribute to **avolition**.

Auditory hallucinations – speech areas

Allen et al. (2007) used **fMRI scans** to investigate people experiencing auditory hallucinations. They found abnormal activity in the **anterior cingulate cortex and left temporal cortex**, areas involved in **monitoring and processing speech**. This may cause individuals to **misattribute their own internal speech as coming from an external source**, contributing to **auditory hallucinations**.

Neural Explanations: The Dopamine Hypothesis

The **dopamine hypothesis** suggests that schizophrenia is linked to abnormalities in **dopamine activity** within different areas of the brain.

1. Original dopamine hypothesis

The original dopamine hypothesis suggested that schizophrenia was caused by **hyperdopaminergia**, meaning **abnormally high levels of dopamine activity**.

- It focused mainly on excessive dopamine activity in **subcortical areas** of the brain.
- This was thought to cause **positive symptoms**, particularly:
 - **Auditory hallucinations**
 - **Delusions**

The explanation was supported by evidence that drugs which **increase dopamine activity** can worsen schizophrenia symptoms, while drugs that **reduce dopamine activity** can improve symptoms.

However, the original hypothesis was considered too **simplistic** because it could not explain the **full range of schizophrenia symptoms**, particularly negative symptoms.



2. Revised dopamine hypothesis

The hypothesis was therefore revised to suggest that schizophrenia is caused by **dopamine dysregulation**, meaning that dopamine activity is **too high in some areas of the brain and too low (hypodopaminergia) in others**.

Type of Dopamine abnormality	Brain Area	Possible Effect
Hyperdopaminergia	Subcortex	Excessive dopamine activity is associated with positive symptoms , such as hallucinations and delusions . Excess dopamine activity in subcortical circuits may affect how information involved in speech, perception and the interpretation of experiences is processed, making internally generated thoughts or sounds more likely to be experienced as external.
Hypodopaminergia	Prefrontal cortex	Reduced dopamine activity is associated with negative symptoms such as avolition and speech poverty . Reduced dopamine activity may disrupt the planning, motivation and decision-making functions of the prefrontal cortex, making it harder to initiate activities or produce speech.

The revised hypothesis therefore suggests that schizophrenia is not simply caused by **too much dopamine**, but by an **imbalance of dopamine activity across different brain regions**.

AO3 - EVALUATION

One strength of the biological approach to schizophrenia is that there is **research evidence supporting a genetic basis for the disorder**. **Brown et al (2002)** found that the risk of developing schizophrenia increased from **under 0.7%** in offspring of fathers **under 25** to **over 2%** when the father was over **50 years old**. This suggests that increased paternal age may lead to **DNA mutations** affecting **candidate genes** involved in neurotransmitter functioning, such as those linked to **dopamine and serotonin**. Therefore, this supports the idea that schizophrenia has a strong **biological basis** and suggests that genetic factors may contribute to an individual's vulnerability to developing the disorder.



Another strength of the biological approach is that the **role of dopamine is supported by evidence from drug treatments**. **Tauscher et al (2014)** found that **antipsychotic drugs**, which act as **dopamine antagonists** by reducing dopamine activity at **post-synaptic receptors**, were effective in reducing symptoms of schizophrenia. This suggests that abnormal **dopamine activity** plays an important role in the development of schizophrenia, as reducing dopamine levels can improve symptoms. Therefore, this provides support for the **dopamine hypothesis** and the biological explanation of schizophrenia.

One limitation of the biological approach is that the **dopamine hypothesis may place too much emphasis on the role of dopamine**. Although dopamine appears to be involved in schizophrenia, **Moghaddam and Javitt (2012)** argued that biological explanations may overlook the role of other neurotransmitters, such as **glutamate and serotonin**. For example, **Meltzer (2012)** found that **clozapine**, an atypical antipsychotic that affects multiple neurotransmitter systems including serotonin, can be more effective than some other antipsychotics in reducing symptoms. Therefore, the biological explanation may be **reductionist** because schizophrenia is likely caused by the interaction of several biological factors rather than dopamine alone.

A key limitation of the biological approach is that **evidence from neural correlates is often correlational, meaning it cannot establish a cause-and-effect relationship**. **Allen et al. (2007)** used **fMRI scans** and found differences in activity in brain areas such as the **anterior cingulate cortex and left temporal cortex**, which were associated with symptoms such as **auditory hallucinations**. However, it is unclear whether reduced activity in these areas causes hallucinations or whether experiencing hallucinations leads to changes in brain activity. There may also be a **third variable** influencing both factors. Therefore, biological explanations may be limited because correlational research cannot prove that brain abnormalities directly cause schizophrenia.

Another limitation of the biological approach is that it may be **too reductionist because it underestimates the role of psychological and environmental influences**. Evidence from treatment research suggests that schizophrenia is best understood through an **interactionist approach**, where biological vulnerability interacts with psychological factors. For example, **Drury et al (1996)** found that patients with schizophrenia who received **CBT alongside antipsychotic medication** showed faster recovery and greater reductions in symptoms compared with those who received medication alone. This suggests that although **biological factors**, such as neurotransmitter abnormalities, play an important role in schizophrenia, psychological factors also contribute to the development and management of the disorder. Therefore, biological explanations may be incomplete because schizophrenia is unlikely to be caused by biological factors alone.



Part 3: Psychological Explanations For Schizophrenia

The **psychological explanation** suggests that schizophrenia develops due to abnormal **family communication patterns**, **stressful environments** and **dysfunctional thought processes**.

These factors are not considered the sole cause of schizophrenia but are thought to act as **contributory factors** involved in the development and maintenance of symptoms.

Family Explanations

1. Schizophrenogenic Mother

Fromm-Reichmann (1948) suggested that schizophrenia could develop due to the influence of a **schizophrenogenic mother**.

She argued that these mothers were often **cold, rejecting and controlling**, creating a tense and unhealthy family environment.

This could increase feelings of **anxiety and paranoia** within the child, which may contribute to the development of **positive symptoms**, particularly **paranoid delusions**.

2. Double-Bind Theory

Bateson et al. (1956) suggested that schizophrenia could develop when children experience repeated **contradictory messages** from their parents.

For example, a child may receive one message verbally but a conflicting message through body language or behaviour.

The child is unable to challenge or clarify these messages, meaning they experience a confusing and unpredictable environment.

Over time, this may affect the child's ability to interpret communication and understand reality, contributing to symptoms such as:

- **Disorganised thinking**
- **Paranoid delusions**

3. Expressed Emotion

Expressed Emotion (EE) refers to the level and type of emotion displayed by family members towards an individual with schizophrenia. High levels of **negative expressed emotion** can include:

- **Criticism**
- **Hostility**
- **Emotional over-involvement**



High EE can act as a source of **stress** for individuals with schizophrenia. This increased stress may make symptoms more difficult to manage and increase the likelihood of **relapse**.

High levels of EE may also reduce **treatment compliance**, as individuals may be less likely to continue taking medication or engaging with psychological therapies.

Cognitive Explanations

Dysfunctional Thought Processes

Frith (1992) suggested that schizophrenia may develop due to **dysfunctional thought processes**, meaning abnormal patterns of thinking that lead to difficulties interpreting experiences and controlling thoughts.

Frith suggested that two cognitive processes are particularly important:

1. Metarepresentation

Metarepresentation is the cognitive ability to recognise the difference between our own thoughts, intentions and actions and those of other people.

It helps us:

- Recognise that **our thoughts belong to us**.
- Understand **where our thoughts and actions come from**.
- Distinguish between **internal and external experiences**.

A dysfunction in metarepresentation may mean that an individual has difficulty recognising their own thoughts as **self-generated**. This can contribute to:

- **Auditory hallucinations** → internal speech may be experienced as coming from an external source.
- **Thought insertion** → the individual may believe that thoughts have been **put into their mind by someone else**.
- **Paranoid delusions** → unusual experiences may be interpreted as being caused by **other people**.

For example, an individual may believe that thoughts in their own mind have been placed there by another person. This confusion between internal and external experiences may contribute to **paranoid delusions**.



2. Central Control

Central control is the cognitive ability to carry out a deliberate action while suppressing an **automatic response**.

This is often measured using the **Stroop test**. The Stroop test requires individuals to identify the colour of a word rather than automatically reading the word itself.

For example:

PURPLE YELLOW RED
BLACK RED GREEN
RED YELLOW ORANGE
BLUE PURPLE BLACK
RED GREEN ORANGE

The individual must suppress the automatic response of reading “Purple” and instead identify the colour of the font.

Individuals with schizophrenia may have dysfunctional **central control**, meaning they struggle to suppress automatic associations.

This may contribute to **derailment**, where speech becomes disorganised because individuals follow unrelated associations between words and move away from the original topic.



AO3 - EVALUATION

One strength of psychological explanations of schizophrenia is that they are **supported by research evidence**. **Stirling et al (2006)** compared individuals with schizophrenia with a healthy control group on a **cognitive task measuring central control skills**. They found that people with schizophrenia made significantly more errors and took twice as long to complete the task compared with the control group. This suggests that **faulty cognitive processing** may contribute to symptoms of schizophrenia, supporting the idea that dysfunctional thought processes are involved in the disorder.

Another strength of psychological explanations of schizophrenia is that they have **practical applications through understanding the role of environmental factors**. Research into **expressed emotion** has shown that high levels of criticism, hostility and emotional over-involvement from family members are associated with increased rates of relapse in individuals with schizophrenia. This has led to the development of treatments such as **family therapy**, which aims to reduce stress within the family environment and improve communication. Therefore, psychological explanations have value because they have contributed to effective interventions that can improve outcomes for individuals with schizophrenia.

One limitation of psychological explanations of schizophrenia is that they **may be incomplete because they fail to account for biological factors**. Biological research has identified the role of **genetics, neurotransmitters such as dopamine and neural abnormalities** in the development of schizophrenia. This suggests that psychological explanations may only explain the **proximal causes** of schizophrenia, such as how symptoms develop, rather than the **distal causes**, which are linked to the origins of the disorder. Therefore, psychological explanations may provide only a partial explanation and are better considered alongside biological factors.

Another limitation of expressed emotion as an explanation of schizophrenia is that it **may not represent a cause of the disorder**. **Brown et al (1962)** found that individuals returning to families with **high expressed emotion** were more likely to relapse than those returning to low EE families. However, this evidence is **correlational**, meaning it cannot determine whether high levels of criticism and emotional involvement cause symptoms to worsen, or whether the difficulties of living with someone with schizophrenia cause families to become more critical and emotionally involved. Therefore, expressed emotion may be better understood as a factor influencing the **maintenance and relapse of schizophrenia** rather than the original cause of the disorder.

A further major limitation of family-based psychological explanations is that they **lack scientific support and may unfairly blame families**. These theories were based on early observations of families of individuals with schizophrenia rather than objective research evidence. **Harrington (2012)** argued that researchers were often searching for “crazy-making characteristics” within families, making these explanations vulnerable to bias. In addition, blaming parents or caregivers may be harmful because it ignores the possibility of a **genetic predisposition** and can increase guilt among families who are already supporting someone with schizophrenia. Therefore, family-based explanations may be both **unreliable and socially sensitive**.



Part 4: Biological Therapies For Schizophrenia: Drug Therapy

There are two main types of **antipsychotic drugs** used to manage schizophrenia:

- **Typical antipsychotics** (first generation)
- **Atypical antipsychotics** (second generation)

Antipsychotics are **dopamine antagonists**, meaning they reduce the activity of the neurotransmitter **dopamine**.

They work by binding to **dopamine receptors** on the **postsynaptic membrane**, preventing dopamine from attaching. This reduces dopamine transmission, helping to return abnormal dopamine activity towards normal levels and reduce symptoms of schizophrenia.

	Typical Antipsychotics	Atypical Antipsychotics
Example	Chlorpromazine	Clozapine and risperidone
How it works	Blocks dopamine receptors , reducing dopamine transmission. This is particularly effective for positive symptoms such as hallucinations and delusions.	Also affects dopamine receptors , but acts on other neurotransmitter systems too. Clozapine affects dopamine, serotonin and glutamate; risperidone affects dopamine and serotonin.
When / Where used	One of the earliest antipsychotics. Its sedative effects also made chlorpromazine useful in psychiatric institutions , as it could reduce agitation and calm highly distressed patients.	Developed later to improve treatment and reduce some of the problems associated with typical drugs. Clozapine may be used when other antipsychotics have not been effective.
Main Benefit	Particularly useful for reducing positive symptoms and agitation.	Can help with positive and negative symptoms , depending on the drug. They were developed partly because of the limitations of typical antipsychotics.
Side effects	Can cause significant side effects, including movement disorders caused by dopamine blockade.	Generally associated with fewer movement-related side effects , although they can cause other serious side effects depending on the drug



Key atypical antipsychotics

Clozapine

- Can improve **cognitive functioning and mood**, as well as reducing schizophrenia symptoms. This is useful because there is a high rate of **comorbidity with depression** in people with schizophrenia.
- However, Clozapine can cause serious side effects, particularly **agranulocytosis** – a dangerous reduction in **white blood cells**.
- This can weaken the immune system and increase the risk of **serious infections**.

Risperidone

- Effective at **lower doses** than Clozapine because it has a stronger effect on **dopamine receptors**.
- This can make it a useful alternative for people who may be at greater risk from **Clozapine's serious side effects**.

AO3 - EVALUATION

One strength of drug therapies for schizophrenia is that there is **research evidence supporting their effectiveness**. **Thornley et al (2003)** conducted a meta-analysis examining the effectiveness of **chlorpromazine**, a typical antipsychotic, and found that it was more effective than a placebo in reducing symptoms of schizophrenia. Similarly, **Meltzer (2012)** found that the atypical antipsychotic **clozapine** was particularly effective for individuals with **treatment-resistant schizophrenia**, who had not improved with other antipsychotic drugs. This is because clozapine acts on several neurotransmitter systems, including **dopamine and serotonin**, which may help explain why it can reduce symptoms when other medications are ineffective. Therefore, the effectiveness of both typical and atypical antipsychotics provides support for drug therapies as a treatment for schizophrenia.

One strength of drug therapies for schizophrenia is that they have **important practical and economic benefits**. Antipsychotic medication can reduce **positive symptoms**, such as **hallucinations and delusions**, allowing individuals to manage their condition more effectively and improve their ability to engage in everyday activities. This may reduce the impact of schizophrenia on individuals, families and wider society by **decreasing** the need for **intensive support**, reducing hospital admissions and helping some individuals return to education or employment. Therefore, drug therapies can be considered **cost-effective** because they may reduce the financial burden on healthcare services while improving quality of life for individuals with schizophrenia.

One major limitation of drug therapies is that antipsychotics can produce **serious side effects, creating ethical concerns about their use**. Typical antipsychotics can cause **tardive dyskinesia**, which involves involuntary facial movements, and **neuroleptic malignant syndrome (NMS)**, which can involve fever, muscle rigidity and changes in mental state. Atypical antipsychotics, such as **clozapine**, can also cause **agranulocytosis**, a dangerous reduction in white blood cells requiring regular blood monitoring. Therefore, although antipsychotics can reduce symptoms, a **cost-benefit analysis** is needed to determine whether the benefits outweigh the risks.



One limitation of drug therapies is that the way **antipsychotics work does not fully match current explanations of schizophrenia**. Antipsychotics act as **dopamine antagonists**, reducing dopamine activity, which supports the original idea that schizophrenia is caused by **excess dopamine activity**. However, the **revised dopamine hypothesis** suggests that schizophrenia involves both **high dopamine activity in subcortical areas** and **low dopamine activity in cortical areas**. This creates a problem because reducing dopamine further should theoretically worsen symptoms linked to low dopamine activity. Therefore, although antipsychotics are effective in reducing symptoms, this raises questions about whether our understanding of their effects and the mechanisms behind schizophrenia is fully accurate.

Another limitation of drug therapies is that **research supporting the effectiveness of antipsychotics may lack validity**. Although evidence supports both **typical antipsychotics** and **atypical antipsychotics**, **Healy (2012)** argues that studies may not accurately measure true symptom improvement. For example, **chlorpromazine** has strong **sedative effects**, meaning that patients may appear calmer and more manageable without there being a genuine reduction in their schizophrenia symptoms. In addition, pharmaceutical companies may **selectively publish research** focusing on **short-term benefits** while failing to highlight long-term risks or using **inappropriate control groups**. Therefore, the effectiveness of antipsychotics may be overstated, reducing the validity of research supporting drug therapies.



Part 5: Psychological Therapies For Schizophrenia

Psychological therapies aim to help individuals with schizophrenia **manage symptoms**, improve their understanding of their experiences and reduce factors that may contribute to **relapse**.

Unlike **drug therapies**, psychological therapies do not primarily target biological abnormalities, such as dopamine activity. Instead, they focus on improving **thought processes**, **coping strategies** and the individual's **social environment**.

The main psychological therapies used for schizophrenia are:

- **Cognitive behavioural therapy for psychosis (CBT-p)**
- **Family therapy**

Cognitive Behavioural Therapy

CBT is based on the idea that schizophrenia symptoms can be influenced by the way individuals **interpret and respond to their experiences**.

CBT does not aim to **cure schizophrenia**. Instead, it helps individuals **understand and manage their symptoms**, reducing the distress they cause.

1. CBT for Psychosis (CBT-p)

CBT-p usually begins with an **initial assessment**, where the therapist identifies the individual's main **symptoms, difficulties and beliefs** about their experiences.

A key aim is to develop the individual's **understanding of their symptoms**. Recognising that **hallucinations and delusions are symptoms of schizophrenia** may reassure individuals that they are not “crazy”, improving **self-awareness** and reducing the distress caused by these experiences.

CBT-p also encourages individuals to **question and challenge unhelpful beliefs** by:

- Examining the **evidence** supporting their thoughts.
- Considering **alternative explanations**.
- Deciding whether their original interpretation is the **most realistic** explanation.

For example, someone who believes they are being targeted by the **Mafia** may be encouraged to consider other explanations for someone staring at them, such as the person being **distracted or lost in thought**.

Through this process, CBT-p aims to help individuals develop **more realistic interpretations** of their experiences and reduce the **distress associated with symptoms**.



2. Family therapy

Family therapy aims to reduce **stress within the family environment** and improve relationships between individuals with schizophrenia and their relatives.

Family therapy is based on the idea that high levels of **expressed emotion (EE)** within families can act as a source of stress and increase the likelihood of **relapse**.

Key features of family therapy include:

- Reducing **negative expressed emotion**, such as:
 - **Criticism**
 - **Hostility**
 - **Emotional over-involvement**
- Helping families develop a better **understanding of schizophrenia**, reducing blame and improving attitudes towards the disorder.
- Developing **communication and problem-solving skills** to reduce conflict and improve relationships.
- Training relatives to recognise **early warning signs of relapse**, allowing support to be provided before symptoms become more severe.

By reducing family stress, family therapy aims to lower the likelihood of **relapse** and improve **treatment compliance**, such as taking medication and engaging with other therapies.

AO3 - EVALUATION

One strength of CBT for schizophrenia is that there is **research evidence supporting its effectiveness**. **Sensky et al (2000)** compared the effectiveness of **CBT** with supportive counselling in patients with schizophrenia who had not responded fully to medication.

Participants who received CBT showed **greater improvements in symptoms** compared with those who received supportive counselling, suggesting that CBT provides benefits beyond simply receiving social support. This is because CBT helps individuals understand their symptoms, challenge unhelpful beliefs and develop coping strategies for experiences such as **hallucinations and delusions**. Therefore, this provides support for CBT as an effective psychological therapy and suggests that psychological approaches can improve outcomes for individuals with schizophrenia.

Another strength of CBT for schizophrenia is that it helps individuals **develop coping strategies without the physical side effects associated with antipsychotic medication**.

While antipsychotic drugs mainly reduce **positive symptoms** such as hallucinations and delusions by altering neurotransmitter activity, CBT helps individuals understand and challenge their interpretations of these experiences. For example, CBT may help a person recognise that hearing voices does not necessarily mean the voice is real or dangerous, reducing the distress caused by hallucinations. This can reduce the distress caused by symptoms and improve **quality of life** without the risks associated with medication, such as **weight gain, tardive dyskinesia and neuroleptic malignant syndrome**. Therefore, CBT has practical value because it provides an alternative or additional method of managing schizophrenia symptoms while avoiding some of the physical costs of drug therapy.



One strength of family therapy for schizophrenia is that it has **research support for reducing relapse**. **Pharoah et al (2010)** reviewed evidence into family interventions and found that family therapy was associated with **reduced relapse rates** and **improved social functioning** compared with standard care alone. This suggests that improving communication and reducing **expressed emotion**, such as criticism and hostility, can help individuals manage schizophrenia more effectively. Therefore, family therapy is a valuable treatment because it can improve long-term outcomes and reduce the likelihood of relapse.

One limitation of CBT is that it may **not be appropriate for all individuals with schizophrenia, particularly those experiencing severe symptoms**. CBT requires individuals to engage with the therapy process, reflect on their thoughts and challenge their beliefs about their experiences. However, some individuals experiencing severe symptoms may struggle to engage due to **reduced insight, low motivation, cognitive difficulties or the severity of their symptoms**. In addition, CBT requires individuals to attend regular therapy sessions, meaning practical barriers such as **difficulty travelling, limited access to services or long waiting lists** may prevent some individuals from receiving treatment. Therefore, CBT may only be suitable for certain individuals, meaning it cannot be considered a **universal treatment** for schizophrenia

One limitation of family therapy is that it **relies on family members being willing and able to engage with the treatment**. Family therapy aims to improve communication and reduce **expressed emotion**, such as criticism and hostility, within the family environment. However, in families where relationships have already become **highly strained**, **members may be unwilling to participate** or may struggle to work together during therapy sessions. This **reduces the effectiveness** of family therapy because it depends on **active cooperation** from both the individual with schizophrenia and their relatives. Therefore, family therapy may not be suitable or effective for all families, particularly those experiencing high levels of conflict.

One limitation of psychological therapies for schizophrenia is that it is **unclear whether improvements are due to the specific techniques used within each therapy**. **Rosenzweig (1936)** proposed the **Dodo bird effect**, suggesting that different psychological therapies may be **equally effective** because improvements are caused by **common factors**, such as the therapeutic relationship, empathy and support from the therapist, rather than the specific approach used. This creates a problem for therapies such as **CBT and family therapy**, as improvements may not necessarily be due to challenging dysfunctional thoughts or reducing expressed emotion, but instead the **general support provided by treatment**. Therefore, it is difficult to determine whether one psychological therapy is more effective than another in managing schizophrenia.



Part 6: The Interactionist Approach To Schizophrenia

The **interactionist approach** suggests that **schizophrenia** develops due to a combination of **biological vulnerability (diathesis)** and **environmental stressors (stress)**. This is known as the **diathesis–stress model**. The approach therefore suggests that **schizophrenia should be treated in a similar way**, using both **biological and psychological therapies**.

Diathesis–Stress Model

The **diathesis–stress model** suggests that schizophrenia develops through an **interaction between a vulnerability (diathesis) and stress**. The model has changed over time, with the modern version recognising that both **diathesis and stress can have biological and psychological causes**.

1. Original Model

The original model, proposed by **Meehl (1962)**, suggested that the diathesis was entirely **biological**, caused by a single ‘**schizogene**’. The schizogene was thought to produce a **schizotypic personality**, which made the individual vulnerable to developing schizophrenia. However, the biological vulnerability alone was not enough to cause the disorder.

A **psychological stressor** was also needed to trigger the development of schizophrenia. **Meehl** particularly focused on **dysfunctional family environments**, such as the **schizophrenogenic mother**, as a potential source of stress.

Therefore:

Single biological diathesis (‘schizogene’) + Psychological stressor = Schizophrenia

2. Modern Model

The modern model recognises that a **diathesis does not have to be genetic or biological**. Instead, a diathesis is **any factor that increases an individual's vulnerability to developing schizophrenia**. This means that **biological and environmental factors can both contribute to vulnerability**, and different factors can interact with each other.



Interaction	How the Factors Interact
Genes x Genes	Different genetic variations can combine to create a greater biological vulnerability . Schizophrenia is polygenic , meaning many genes contribute to risk. → For example, having several risk-related genetic variations may create a greater vulnerability than having only one
Environment x Environment	An early environmental experience can create a psychological vulnerability , making the individual more sensitive to later stress. → For example, childhood abuse may increase sensitivity to stress. If the individual then experiences a continued stressful environment , such as family conflict, the additional stress may increase their risk of developing SZ.
Genes x Environment	A genetic vulnerability may make an individual more sensitive to environmental stress. → For example, someone with genetic risk factors may be more likely to develop SZ when exposed to childhood trauma or cannabis use .

Key idea: The modern model is not saying “genes + stress”. It suggests that different factors can create vulnerability and interact in different ways. A vulnerability can be biological, genetic or environmental.

Treatment

The interactionist approach suggests that schizophrenia should be treated using a **combination of biological and psychological therapies**, rather than relying on one approach alone. For example using:

Antipsychotic medication

- Targets **neurotransmitter abnormalities**, such as **dopamine dysregulation**.
- Helps to **reduce symptoms** and make them more manageable.

AND

CBT

- Helps individuals **manage their thoughts, beliefs and experiences**.
- Can be used to **challenge paranoid beliefs** and develop coping strategies for **hallucinations**.

The treatments can **work together** because reducing symptoms with medication may make it **easier for the individual to engage with CBT**, while CBT can help them **manage any remaining symptoms and cope with their experiences**. This means the two approaches can complement each other rather than treating schizophrenia as purely **biological or psychological**.



AO3 - EVALUATION

One strength of the interactionist approach is that it is **supported by research evidence**.

Tienari et al (2004) conducted an adoption study using Finnish adoptees who had a biological mother with schizophrenia and compared them with adoptees without a genetic risk. They found that **adoptees with a high genetic vulnerability** were more likely to develop schizophrenia-spectrum disorders when they were raised in **dysfunctional adoptive families**, whereas genetic risk alone did not significantly predict schizophrenia. This suggests that schizophrenia develops through an interaction between **biological vulnerability (diathesis)** and **environmental stressors**, supporting the **diathesis-stress model**. Therefore, the interactionist approach provides a more complete explanation than approaches focusing on biological or psychological factors alone.

Another strength of the interactionist approach is **research supporting combined treatments**. **Tarrier et al (2004)** studied **315 patients** who were allocated to different treatment conditions, including groups receiving **CBT alongside standard treatment**. After an 18-month follow-up, they found that those receiving **additional psychological treatments** showed **greater improvements** in symptoms compared with treatment as usual. This suggests that combining **biological treatments**, such as medication, with **psychological therapies** can improve symptom management. Therefore, the interactionist approach is valuable because it recognises that schizophrenia is influenced by multiple factors and may require a combination of treatments.

One limitation of the interactionist approach is that the **diathesis-stress model may oversimplify the development of schizophrenia**. The original model suggested that schizophrenia results from a biological vulnerability interacting with a psychological stressor. However, research suggests that schizophrenia involves many interacting factors. For example, **Ripke et al (2014)** identified over **108 genetic variations** that may contribute to schizophrenia risk, suggesting there is no single genetic cause. In addition, stressors can include **childhood trauma**, **expressed emotion** and **cannabis use**, meaning both the diathesis and stressor can be more complex than originally suggested. Therefore, although the interactionist approach is useful, the original diathesis-stress model may not fully explain the complexity of schizophrenia.

Another limitation of the interactionist approach is that it can be **difficult to determine which factors are responsible for the development of schizophrenia**. The approach suggests that **biological, psychological and environmental factors** interact, but this makes it difficult to establish the **relative importance** of each factor. For example, it is unclear whether **genetic vulnerability** makes individuals more likely to experience **stress**, or whether **stress** increases vulnerability to symptoms. This also creates practical difficulties when deciding **which factors to target first in treatment**, as some individuals may benefit more from **drug therapies**, whereas others may require **psychological therapies or environmental support**. Therefore, although the **interactionist approach** provides a more **complete explanation** of schizophrenia, the complexity of multiple **interacting factors** makes it difficult to identify the most effective treatment approach for each individual.

