SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Schizophrenia

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.3.5 · A-level Paper 3

Compiled by **Simply Psychology** · simplypsychology.org/schizophrenia.html

Contents

2025 spec changes: "Classification, reliability and validity" of diagnosis has been **removed** (the broader "issues in diagnosis" wording remains). **Token economies** as a treatment have been **removed**. Past-paper questions on token economies and the reliability/validity of diagnosis are now obsolete.

AQA 2025 SPECIFICATION — SCHIZOPHRENIA (4.3.5)

- **Positive symptoms** (hallucinations, delusions) and **negative symptoms** (speech poverty, avolition). **Issues in diagnosis**: co-morbidity, culture and gender bias, symptom overlap.
- **Biological explanations**: genetics and neural correlates including the dopamine hypothesis.
- **Psychological explanations**: family dysfunction and cognitive explanations (dysfunctional thought processing).
- **Drug therapy**: typical and atypical antipsychotics.
- **Cognitive behaviour therapy and family therapy** as treatments.
- The importance of the **interactionist approach**; the **diathesis-stress model**.

1 Symptoms and Diagnosis of Schizophrenia

Schizophrenia is a severe mental disorder affecting around 1% of the population. It usually emerges in late adolescence or early adulthood and disrupts thought, perception, emotion and behaviour. Diagnosis uses the **DSM-5** (USA) or **ICD-11** (WHO/UK).

Positive Symptoms

Positive symptoms are additions to normal experience:

- **Hallucinations** — unusual sensory experiences without an external stimulus. Most commonly *auditory* (hearing voices), but can be visual, tactile or olfactory.
- **Delusions** — irrational beliefs held with strong conviction despite contrary evidence. Examples: *delusions of grandeur* ("I am Jesus"), *delusions of persecution* ("the government is watching me"), *delusions of reference* (a TV presenter is sending personal messages).

Negative Symptoms

Negative symptoms are losses or reductions of normal functioning:

- **Speech poverty (alogia)** — reduced amount and quality of speech. The person speaks less, in shorter sentences, with fewer thoughts expressed.
- **Avolition** — reduced motivation to carry out goal-directed activity. The person may not get dressed, attend appointments or maintain hygiene.

Issues in Diagnosis

Issue	What it means
Co-morbidity	The presence of two or more disorders simultaneously. Around 50% of schizophrenia patients also meet criteria for depression, substance abuse or anxiety disorders. Makes diagnosis difficult — are the symptoms schizophrenia, the other disorder, or both?
Symptom overlap	Symptoms of schizophrenia overlap with those of other disorders. Bipolar disorder, severe depression and schizoaffective disorder can all involve hallucinations or disordered thinking — making boundaries between disorders blurry.
Culture bias	Diagnosis rates vary across cultures and ethnicities — for example, Black Caribbean people in the UK are diagnosed with schizophrenia at substantially higher rates than white British people. Some symptoms (hearing voices) are valued spiritually in some cultures but pathologised in Western psychiatry.
Gender bias	Differential diagnosis rates — historically men are diagnosed earlier and at higher rates. Loring and Powell (1988) found clinicians more likely to diagnose schizophrenia when patients were described as male.

Evaluation

A serious limitation of diagnosis is co-morbidity, which is well-evidenced. Buckley et al. (2009) found that around half of people diagnosed with schizophrenia also had a diagnosis of depression, and 47% a co-morbid substance-abuse disorder. The implication is significant: if these conditions occur together so often, they may not be genuinely separate disorders, and symptoms attributed to schizophrenia could in fact belong to the co-morbid condition. This undermines the **validity** of schizophrenia as a distinct diagnostic category and makes it harder to select an appropriate treatment.

A further limitation is the poor reliability of diagnosis. Cheniaux et al. (2009) had two psychiatrists independently diagnose 100 patients: one diagnosed 26 with schizophrenia using the DSM and 44 using the ICD, while the other diagnosed 13 and 24 respectively. Such low **inter-rater reliability** means clinicians frequently disagree, so a diagnosis can depend more on who is assessing the patient, and which manual they use, than on the patient themselves. Because research and treatment both rest on diagnosis, unreliable classification weakens the entire field.

Diagnosis is also subject to culture bias, making it socially sensitive. Black Caribbean and African men in the UK are diagnosed with schizophrenia at several times the rate of white British men, yet rates are not similarly elevated in their countries of origin — pointing to clinician bias rather than a genuine difference in prevalence. Applying Western criteria to other groups (an **imposed etic**) risks pathologising culturally normal experiences such as hearing the voice of an ancestor. The real-world consequence is over-diagnosis, inappropriate medication and stigma for already-marginalised groups, which is why this research must be handled carefully.

Finally, symptom overlap challenges the validity of schizophrenia as a unique category. Because schizophrenia shares symptoms — delusions, avolition, disorganised thinking — with bipolar disorder and severe depression, a patient may simultaneously qualify for several diagnoses. This blurring suggests classification systems may not "carve nature at its joints", and that schizophrenia might be better understood as a cluster of overlapping difficulties rather than a single discrete disease. Taken together with co-morbidity and reliability problems, this casts real doubt on whether schizophrenia is being validly diagnosed at all.

2 Biological Explanations: Genetics and Neural Correlates

Genetics

Schizophrenia runs in families. **Gottesman (1991)** reviewed family studies and reported the following lifetime risks:

- General population: ~1%
- Sibling of a schizophrenia patient: ~9%
- Child with one schizophrenic parent: ~13%
- Child with two schizophrenic parents: ~46%
- Dizygotic (DZ) twin: ~17%
- Monozygotic (MZ) twin: ~48%

The much higher concordance for MZ vs DZ twins suggests a substantial genetic contribution. However, MZ concordance is well below 100% — environment also matters.

Polygenic: schizophrenia is influenced by many genes (Ripke et al. 2014 identified 108 risk loci), not a single "schizophrenia gene". Candidate genes include those affecting dopamine function (e.g. *COMT*, *DRD2*).

Neural Correlates — The Dopamine Hypothesis

The **original dopamine hypothesis** proposed that schizophrenia is caused by an excess of dopamine activity in subcortical areas of the brain (**hyperdopaminergia** in the mesolimbic pathway). Evidence:

- Drugs that increase dopamine (amphetamines, L-DOPA) can produce schizophrenia-like symptoms in non-schizophrenic users.
- Drugs that block dopamine D2 receptors (antipsychotics) reduce positive symptoms — see Section 5.
- Post-mortem studies show higher densities of D2 receptors in schizophrenia patients.

The **revised dopamine hypothesis** (Davis et al. 1991) added that *hypo*dopaminergia in the prefrontal cortex contributes to negative symptoms, while hyperdopaminergia in subcortical areas contributes to positive symptoms.

Other Neural Correlates

- **Enlarged ventricles** — fluid-filled spaces in the brain are larger in schizophrenia patients, suggesting reduced brain tissue.
- **Reduced grey matter** in the frontal and temporal lobes.

- **Glutamate** dysfunction — emerging research suggests reduced NMDA-receptor function may also play a role.

Evaluation

A major strength of the genetic explanation is the convergent evidence from twin, family and adoption studies. Gottesman's figures show risk rising with genetic relatedness (1% general population → 48% for MZ twins), and Tienari et al.'s Finnish adoption study found high risk in children of schizophrenic mothers even when raised in healthy adoptive families. Because adoption studies separate genes from the rearing environment, they rule out the obvious confound that families share *environments* as well as genes. Together this strongly supports a genetic vulnerability and shows the pattern is not simply learned from a disturbed home.

The dopamine hypothesis is also supported by powerful drug evidence. Antipsychotics that block D2 receptors reduce positive symptoms, whereas dopamine-releasing drugs such as amphetamines can induce psychosis-like symptoms in healthy people. This "double dissociation" — turning symptoms down by lowering dopamine activity and up by raising it — is exactly what the hypothesis predicts, giving it strong explanatory power and, crucially, a basis for effective treatment.

However, a key limitation is that MZ concordance is far below 100%. If schizophrenia were purely genetic, identical twins (who share 100% of their DNA) would always be concordant, yet only around 48% are. The implication is that genes create a *vulnerability* rather than a certainty, and environmental factors must trigger the disorder. This is the central argument for the **diathesis-stress model** (Section 7) and shows a purely genetic account is incomplete.

A further limitation is that the original dopamine hypothesis is now seen as oversimplified. Clozapine, one of the most effective antipsychotics, acts on serotonin and glutamate as well as dopamine, and newer research implicates glutamate/NMDA dysfunction. This suggests dopamine is part of a more complex neurochemical picture rather than the sole cause. More broadly, reducing a disorder shaped by family, cognitive and social factors to "genes and dopamine" is **biologically reductionist** — an interactionist account that integrates these levels of explanation has greater validity.

3 Psychological Explanations: Family Dysfunction

Several family-based explanations propose that dysfunctional family communication contributes to schizophrenia.

The Schizophrenogenic Mother (Fromm-Reichmann 1948)

An early psychoanalytic explanation proposed that the "schizophrenogenic mother" — cold, rejecting and controlling — created the family environment in which schizophrenia develops. The theory is now largely discredited and is socially harmful (placing blame on mothers).

The Double-Bind Theory (Bateson et al. 1956)

Bateson proposed that schizophrenia develops in children who repeatedly receive **contradictory communication** from caregivers — e.g. a mother says "I love you" while withdrawing affection. The child cannot respond correctly to either message, leading to confused thinking, paranoia and delusions.

Expressed Emotion (EE)

Expressed emotion describes the level of negative emotion expressed within a family — specifically high levels of **criticism, hostility** and **emotional over-involvement**. High-EE families are associated with much higher relapse rates in schizophrenia patients.

- **Vaughn and Leff (1976)**: 51% of patients in high-EE families relapsed within 9 months vs 13% in low-EE families.
- EE is now seen mainly as a factor in *relapse* rather than initial onset.

Evaluation

A strength of the expressed-emotion explanation is robust, replicated research support. Vaughn and Leff (1976) found a 51% relapse rate in high-EE homes versus 13% in low-EE homes, and this association has since been replicated across many studies and cultures. The implication is that the family environment plays a real, measurable role in the *course* of schizophrenia, and — importantly — because EE is modifiable, it identifies a clear target for intervention. This directly justifies family therapy (Section 6), giving the explanation strong applied value.

However, a limitation is the problem of cause and effect. Living with someone experiencing a severe psychotic illness is extremely stressful, so high expressed emotion may be a *response* to the patient's symptoms rather than a cause of them. Because the research is largely correlational, the direction of the relationship cannot be established, and it would be unfair — and socially insensitive — to assume families have caused the disorder.

This links to a broader limitation: the older family explanations are socially sensitive and poorly supported. The schizophrenogenic-mother and double-bind theories were never reliably demonstrated empirically, yet they had the damaging effect of blaming parents (especially mothers) for a serious biological illness. Modern psychology has largely abandoned them, and their history is a reminder that explanations of mental illness can cause real harm to families if adopted without strong evidence.

A further limitation is that family dysfunction offers an incomplete account. EE explains relapse far better than it explains onset, and many people develop schizophrenia without any obvious family dysfunction while many high-EE families never produce the disorder. This suggests family factors act as a *stressor* operating on a pre-existing vulnerability rather than as a sole cause — again pointing towards the interactionist diathesis-stress model.

4 Psychological Explanations: Cognitive (Dysfunctional Thought Processing)

Cognitive explanations focus on **dysfunctional thought processing** — the way schizophrenia patients process information differently from healthy controls.

Frith's (1992) Two Cognitive Deficits

Frith proposed two cognitive processing failures associated with schizophrenia:

- **Metarepresentation dysfunction** — the ability to reflect on one's own thoughts and behaviour is impaired. The person cannot recognise their own thoughts as their own, leading to *hallucinations* (e.g. hearing one's own thoughts as external voices) and certain delusions.
- **Central control dysfunction** — difficulty suppressing automatic responses while performing actions deliberately. Leads to disorganised speech and behaviour as automatic associations intrude.

Stirling et al. (2006) — Stroop Task

Stirling compared 30 schizophrenia patients with 18 controls on the Stroop task (naming the colour of words while ignoring the word itself). Patients took twice as long, supporting the central-control deficit hypothesis.

Evaluation

A strength of the cognitive explanation is direct experimental evidence of dysfunctional processing. Stirling et al. (2006) found that schizophrenia patients took around twice as long as controls on the Stroop task, which requires suppression of an automatic response. This is exactly the deficit Frith's central-control account predicts, so the finding provides objective, replicable support that the thinking of people with schizophrenia really does differ at the level of information processing — not just in what they report experiencing.

The approach also has clear practical application, which strengthens its credibility. If symptoms arise from faulty thought processing, then therapies that target thinking should help — and CBT for schizophrenia (Section 6) does reduce the distress caused by hallucinations and delusions. The success of a treatment derived from the theory provides indirect support for the cognitive account, and brings economic benefit by helping patients function with fewer or shorter hospital admissions.

However, a key limitation is that cognitive explanations describe a proximate cause rather than an ultimate one. They tell us *how* thinking goes wrong but not *why* the dysfunction arises in the first place. The cognitive deficits may themselves be the product of the dopamine abnormalities or genetic vulnerability described in Section 2. This means the cognitive account is best seen as one link in a causal chain rather than a complete explanation, and is most powerful when combined with biological accounts.

A further limitation is the problem of cause and effect. Because studies like Stirling's are conducted *after* diagnosis, the cognitive deficits could be a consequence of having schizophrenia (or of antipsychotic medication) rather than a cause of it. Without longitudinal evidence showing the deficits precede the illness, the causal claim of the cognitive explanation remains uncertain.

5 Drug Therapy: Typical and Atypical Antipsychotics

Typical Antipsychotics (First Generation)

Developed in the 1950s. Examples: **chlorpromazine, haloperidol**. Work primarily by **blocking D2 dopamine receptors** in the brain — supporting the dopamine hypothesis. Effective at reducing positive symptoms (hallucinations, delusions) but cause significant side effects.

Side effects: **tardive dyskinesia** (involuntary movements), **extrapyramidal symptoms** (Parkinson-like tremor and rigidity), **neuroleptic malignant syndrome** (rare but potentially fatal). Sedation, weight gain, sexual dysfunction.

Atypical Antipsychotics (Second Generation)

Developed from the 1970s onward. Examples: **clozapine, risperidone, olanzapine**. Bind to D2 receptors but also affect serotonin receptors and other neurotransmitter systems. Generally more effective for **negative symptoms** and produce fewer extrapyramidal side effects.

Clozapine is particularly effective for treatment-resistant schizophrenia but carries the risk of **agranulocytosis** (a potentially fatal blood condition) — requires regular blood monitoring.

Side effects: weight gain, metabolic syndrome, diabetes (with olanzapine), agranulocytosis (clozapine).

Evaluation

A major strength of antipsychotics is strong evidence of effectiveness. Thornley et al. (2003) reviewed 13 trials with 1,121 participants and found chlorpromazine was associated with significantly better symptom reduction than placebo, and around 70% of patients show improvement. The implication is that, for most people, antipsychotics genuinely reduce the severity of distressing positive symptoms, enabling many to live in the community rather than in hospital — a substantial benefit for patients and a significant economic saving for the NHS.

However, a serious limitation is the burden of side effects. Typical antipsychotics frequently cause extrapyramidal symptoms, and long-term use produces tardive dyskinesia in roughly 30% of patients; atypicals carry risks of weight gain, diabetes and (with clozapine) potentially fatal agranulocytosis. These effects reduce quality of life and are a major reason patients stop taking medication, which in turn raises relapse rates. This shows drug treatment is far from a benign intervention and creates a genuine cost–benefit dilemma.

A further limitation is that antipsychotics treat symptoms rather than causes. The drugs suppress symptoms only while they are being taken, and relapse rates are high after discontinuation, because the underlying genetic, cognitive and social factors remain unaddressed. This is a strong argument for combining medication with psychological therapies (Section 6) rather than relying on drugs alone — consistent with the interactionist approach.

A final issue concerns possible bias in the evidence base. Healy (2012) argued that many effectiveness trials are funded by pharmaceutical companies and that publication bias inflates the apparent benefit of antipsychotics, with some apparent "effectiveness" reflecting their calming/sedative action rather than a true treatment of schizophrenia. If the published evidence overstates real-world benefit, then clinical decisions and the cost–benefit balance may be skewed — a reminder to evaluate the source of treatment evidence critically.

6 Psychological Therapies: CBT and Family Therapy

Cognitive Behaviour Therapy (CBT) for Schizophrenia

CBT for schizophrenia helps patients identify and challenge dysfunctional thinking patterns underlying delusions and hallucinations. Key techniques:

- **Engagement and rapport building** — establishing trust with a clinician (vital given paranoia).
- **Reality testing** — examining the evidence for and against delusional beliefs.
- **Normalising experiences** — explaining that many people without schizophrenia hear voices or have unusual experiences, reducing distress and stigma.
- **Coping strategies** — developing techniques for managing voices (e.g. talking back to them, scheduling listening times).
- Typically 12–24 sessions; used alongside (not instead of) drug therapy.

Family Therapy

Family therapy works with the patient and their family to reduce **expressed emotion** and improve communication. Approaches include:

- **Psychoeducation** about schizophrenia for family members.
- **Reducing criticism and emotional over-involvement**.
- **Communication training** — clearer, more constructive exchanges.
- **Problem-solving training** for everyday difficulties.
- Typically 9–12 months; NICE recommends 10+ sessions over 3 months.

Evaluation

A strength of CBT is meta-analytic research support. Jauhar et al. (2014) reviewed 34 studies and found that CBT produced small but significant reductions in both positive and negative symptoms, and CBT is recommended for schizophrenia by NICE. The implication is that, even though schizophrenia has a biological basis, addressing how patients interpret and respond to their symptoms produces measurable clinical benefit — supporting the cognitive explanation and giving patients tools to manage distress rather than simply suppressing symptoms with drugs.

Family therapy has even stronger evidence of effectiveness, with clear economic value. Pharoah et al. (2010) reviewed 53 studies and found that family therapy reduced relapse rates by around 50% and lowered hospital admissions. Because in-patient care is extremely expensive, reducing relapse and re-admission produces substantial savings for health services as well as improving patients' and carers' quality of life — a strong "psychology and the economy" argument for funding these interventions.

However, a limitation is that psychological therapies are not suitable at every stage and may have modest effects. CBT requires reflection, engagement and reasonably intact communication, which is very difficult during an acute psychotic episode — so most patients must first be stabilised on medication. Moreover, the effect sizes for CBT are small (Jauhar et al.), so it should be seen as a helpful adjunct rather than a stand-alone cure.

A further limitation is the difficulty of isolating the therapies' specific benefit. Because psychological therapies are almost always delivered *alongside* antipsychotic medication, it is hard to know how much improvement is due to the therapy itself rather than the drugs. While this complicates evaluation, it also reflects best practice: the evidence consistently points to **combined** biological and psychological treatment as most effective, which is exactly what the interactionist approach (Section 7) recommends.

7 The Interactionist Approach: The Diathesis-Stress Model

> **THE DIATHESIS-STRESS MODEL**
>
> The **diathesis-stress model** proposes that schizophrenia develops when an individual with a **diathesis** (a vulnerability — usually genetic) is exposed to **stress** (environmental triggers). Neither alone is sufficient — both are needed.

Key Research — Tienari et al. (2004)

Tienari studied 145 Finnish adoptees whose biological mothers had schizophrenia, plus a matched 158 adoptees without this genetic risk. Adoptive families were rated for healthy vs disturbed dynamics. Key findings:

- 11% of high-genetic-risk children adopted into **disturbed** families developed schizophrenia.
- Only ~1% of high-genetic-risk children adopted into **healthy** families developed schizophrenia.
- Children without genetic risk had very low rates of schizophrenia regardless of family environment.

This is a textbook demonstration of diathesis-stress — both genetic vulnerability AND a disturbed family environment were needed to produce schizophrenia. A healthy family had a protective effect against genetic risk.

Modern Versions

Houston et al. (2008) proposed an updated model in which the diathesis is itself influenced by experience (e.g. childhood trauma) as well as genes — diathesis and stress are more deeply intertwined than the original model suggested.

Treatment Implications

The diathesis-stress model implies that **combined treatment** is most effective:

- **Drug therapy** targets the biological diathesis (e.g. dopamine dysregulation).
- **CBT** targets cognitive vulnerability.
- **Family therapy** reduces environmental stress (EE).

NICE guidelines reflect this — recommending drug therapy alongside CBT and (where appropriate) family therapy for best outcomes.

Evaluation

A major strength of the interactionist approach is powerful supporting evidence from Tienari et al. (2004). In this adoption study, children of schizophrenic mothers developed schizophrenia far more often when raised in disturbed adoptive families (11%) than in healthy ones (1%), whereas children without genetic risk were largely unaffected regardless of family environment. Because neither the diathesis nor the stress alone was sufficient, this is a near-textbook demonstration that genes and environment *interact* — something neither a purely biological nor a purely psychological account can explain.

The model also resolves a problem that defeats purely biological explanations: why MZ concordance is below 100%. If genetically identical twins do not always share schizophrenia, genes alone cannot be the whole story; the diathesis-stress model explains this elegantly by proposing that the second twin may simply not have encountered sufficient environmental stress. This greater explanatory power is a clear advantage over reductionist single-factor accounts.

A further strength is the model's direct application to treatment. Because it locates the disorder in an interaction of biological vulnerability and environmental stress, it justifies **combined** treatment — antipsychotics for the diathesis plus CBT and family therapy to reduce stress — which is exactly what NICE recommends and what produces the best outcomes in practice. This gives the approach real clinical and economic value.

However, a limitation is that the original diathesis-stress model was oversimplified. Treating the diathesis as purely genetic and stress as purely environmental ignores evidence that experience can alter biology (e.g. epigenetic changes following childhood trauma) and that genes can influence the environments people encounter. As Houston et al. (2008) argue, diathesis and stress are far more intertwined than the original "two separate factors" model implied, so the model needs continual refinement.

A second limitation is the difficulty of measuring diathesis and stress. Quantifying "genetic vulnerability" and "environmental stress" is hard: Tienari's ratings of family disturbance were necessarily subjective, and polygenic risk scores are still developing. This makes precise predictions about who will develop schizophrenia difficult, even if the general principle of interaction is well supported.

Conclusion. Despite measurement difficulties, the diathesis-stress model is the current consensus framework for understanding schizophrenia. It is well supported by adoption research, it explains findings (such as sub-100% twin concordance) that single-factor theories cannot, and it underpins the combined biological-plus-psychological treatment used in modern clinical practice.

These notes were prepared for Simply Psychology and cover spec section 4.3.5 of the AQA Psychology 2025 specification (A-level only, Paper 3). Token economies and reliability/validity of classification were **removed** in 2025. For deeper coverage, see simplypsychology.org/schizophrenia.html.