SimplyPsychology

Mark scheme — A-level Psychology 7182/3

Paper 3 · Section C option · Schizophrenia

A-level topic mock · 2026 · Maximum mark: 48

Schizophrenia is A-level only (AQA spec 4.3.5) — it is a Paper 3 option and is not assessed at AS. Indicative content is not exhaustive; credit any other valid points. Levels-based questions (Q10 and Q11) require holistic judgement using the descriptors — match the answer to the band that best fits, then fine-tune within it. Specialist vocabulary (positive/negative symptoms, hallucinations, delusions, speech poverty, avolition, co-morbidity, symptom overlap, dopamine hypothesis, hyperdopaminergia, expressed emotion, dysfunctional thought processing, typical/atypical antipsychotics, diathesis-stress) follows AQA's 2025 wording. **Note (2025 spec):** token economies and the "reliability and validity of classification" are **no longer on the specification**; the diagnosis bullet now reads "issues in diagnosis: co-morbidity, culture and gender bias and symptom overlap."

C Schizophrenia

0 1

AO1 · 1 mark · multiple choice

> *Which one of the following is a negative symptom of schizophrenia?*

Answer: C — Avolition.

Avolition (a reduction in goal-directed activity) is a negative symptom — a loss of normal functioning. A (hallucinations), B (delusions) and D (disorganised speech) are all positive symptoms — additions to, or distortions of, normal experience.

0 2

AO1 · 1 mark · multiple choice

> *Which one of the following best describes co-morbidity as an issue in the diagnosis of schizophrenia?*

Answer: A — Two or more disorders occurring together in the same person.

B describes inter-rater reliability; C describes symptom overlap; D describes culture bias. All are genuine diagnostic issues, but only A defines co-morbidity.

0 3

AO1 · 1 mark multiple choice

> *Which one of the following best describes the original dopamine hypothesis of schizophrenia?*

Answer: B — An excess of dopamine activity in subcortical areas of the brain.

The original hypothesis proposed hyperdopaminergia (too much dopamine activity, especially in the mesolimbic pathway). A reverses the direction; C describes other neural correlates (enlarged ventricles / reduced grey matter); D describes serotonin, which is implicated in depression and OCD, not the dopamine hypothesis.

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0 4

AO1 · 3 marks short answer

> *Outline genetics as a biological explanation for schizophrenia.*

Marks for this question: AO1 = 3 marks

- **1 mark** for the core idea: schizophrenia runs in families — the closer the genetic relatedness to a person with schizophrenia, the greater the risk.
- **1 mark** for supporting concordance detail, e.g. **Gottesman's (1991)** family study: ~1% general population, ~9% siblings, rising to ~48% for monozygotic (MZ) twins vs ~17% for dizygotic (DZ) twins. Higher MZ than DZ concordance points to a genetic component.
- **1 mark** for further accurate elaboration, e.g. schizophrenia is **polygenic** (many genes each add a small risk — **Ripke et al. 2014** identified 108 risk loci); candidate genes affect dopamine function; or adoption-study evidence (Tienari) that risk follows biological rather than adoptive parents.

Credit twin, family or adoption evidence. Do not require all three concordance figures — any accurate detail showing increasing risk with relatedness is creditworthy.

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0 5

AO2 · 4 marks application

> *Identify two of Leah's symptoms. For each, state whether it is a positive or negative symptom and explain your answer.*

Marks for this question: AO2 = 4 marks

- **1 mark** for each of two symptoms correctly identified from the stem (max 2).
- **1 mark** for each correct positive/negative classification, with explanation linked to the stem (max 2).

Indicative content (credit any two):

- **Delusion (of persecution) — positive symptom**: "convinced her neighbours are secretly recording her and plotting against her … no evidence". A positive symptom because it is an addition to normal experience — an irrational belief held despite contrary evidence.
- **Hallucination (auditory) — positive symptom**: "hearing a voice that comments on what she is doing". Positive because it is a sensory experience occurring without an external stimulus.
- **Avolition — negative symptom**: "stopped washing, getting dressed and leaving her room … spends most of the day doing nothing". Negative because it is a loss/reduction of normal goal-directed functioning. (Reduced self-care / social withdrawal may also be credited as evidence of avolition.)

Full marks require two correctly classified symptoms with explanations that draw on Leah's behaviour. A strong answer chooses one positive and one negative symptom, but two correctly classified positives (or negatives) can still gain full marks.

0 6

AO2 · 4 marks application

> *Use your knowledge of family dysfunction to explain Sanjay's relapse. Refer to expressed emotion in your answer.*

Marks for this question: AO2 = 4 marks

- **4 marks** — Clear, coherent explanation that defines expressed emotion (EE), identifies the relevant features in the stem, and links high EE to relapse. Accurate terminology.
- **3 marks** — Mostly effective; EE applied to the stem but one element less developed.
- **2 marks** — EE defined or applied, but the link to relapse is limited.
- **1 mark** — Brief or muddled; minimal use of the stem.

Indicative content:

- **Expressed emotion** = the level of negative emotion expressed towards a patient by their family: **criticism, hostility** and **emotional over-involvement**.
- **High criticism / hostility**: Sanjay's mother "frequently criticises him for being lazy and not even trying to get better".
- **Emotional over-involvement**: she "becomes tearful and over-protective, refusing to let him leave the house on his own".
- **Link to relapse**: a high-EE home is a significant source of **stress**. Living in this environment is associated with much higher relapse rates (**Vaughn and Leff 1976**: 51% relapse in high-EE vs 13% in low-EE homes), explaining why Sanjay's symptoms worsened and he was readmitted soon after returning home.

Top-band answers explicitly use the term "expressed emotion", quote at least two features of the mother's behaviour, and connect high EE to stress/relapse.

0 7

AO1 · 4 marks short answer

> *Outline the cognitive explanation for schizophrenia. Refer to dysfunctional thought processing in your answer.*

Marks for this question: AO1 = 4 marks

- **1 mark** for the central premise: schizophrenia is associated with **dysfunctional (faulty) thought/information processing** — abnormal cognition underlies the symptoms.
- **1–2 marks** for **metarepresentation dysfunction (Frith 1992)**: an impaired ability to reflect on and monitor one's own thoughts and intentions. The person cannot recognise their own thoughts/actions as self-generated, which can produce **auditory hallucinations** (experiencing one's own thoughts as external voices) and delusions of control.
- **1–2 marks** for **central control dysfunction**: difficulty suppressing automatic responses while carrying out deliberate actions, so automatic associations intrude — producing **disorganised speech and thought** (e.g. derailment, word salad).

Award up to 4 marks. Reference to Frith's two deficits is the clearest route to full marks, but other accurate accounts of dysfunctional processing (e.g. faulty attention/perception biases) are creditworthy. Stirling et al. (2006) Stroop evidence may be used to illustrate central-control deficits but is not required.

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0 8

AO1 · 3 marks short answer

> *Outline how cognitive behaviour therapy (CBT) is used in the treatment of schizophrenia.*

Marks for this question: AO1 = 3 marks

- **1 mark** for the aim: CBT helps patients **identify and challenge** the irrational/dysfunctional thoughts underlying delusions and hallucinations, using gentle questioning and **reality testing** (examining the evidence for a belief).
- **1 mark** for **normalising**: explaining that hallucinations/unusual experiences lie on a continuum with normal experience, which reduces distress, anxiety and stigma.
- **1 mark** for further accurate detail, e.g. developing **coping strategies** for managing symptoms (e.g. distraction, responding to voices); building **engagement/rapport**; typically **12–24 sessions**; used **alongside** antipsychotic medication, not instead of it.

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0 9

AO1 · 3 marks short answer

> *Outline how family therapy is used in the treatment of schizophrenia.*

Marks for this question: AO1 = 3 marks

- **1 mark** for the aim: family therapy works with the patient **and their family** to reduce **expressed emotion** (criticism, hostility, over-involvement) and so reduce relapse.
- **1 mark** for a recognised technique, e.g. **psychoeducation** about schizophrenia; **communication training**; reducing criticism and emotional over-involvement.
- **1 mark** for further accurate detail, e.g. **problem-solving** training for everyday difficulties; forming a therapeutic alliance to improve treatment adherence; NICE recommends ~10 sessions over several months (Pharoah et al. evidence may be cited).

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AO1, AO3 · 8 marks 8-mark

> *Discuss drug therapy as a treatment for schizophrenia. Refer to typical and atypical antipsychotics, and to at least one strength and one limitation.*

Marks for this question: AO1 = 4 marks, AO3 = 4 marks

Level	Marks	Descriptor
4	7–8	Knowledge of typical and atypical antipsychotics is accurate and well detailed. Evaluation includes at least one strength and one limitation, both effectively explained. Clear, coherent, focused; specialist terminology used effectively.
3	5–6	Knowledge generally accurate; both drug types referenced. Evaluation mostly effective but limited in places. Reasonable structure.
2	3–4	Some accurate knowledge (may cover only one drug type). Evaluation limited; mainly descriptive.
1	1–2	Knowledge limited or muddled. Little or no evaluation.
0	0	No relevant content.

Indicative AO1 content:

- **Typical (first-generation) antipsychotics**, e.g. **chlorpromazine, haloperidol**: act as **dopamine antagonists**, blocking **D2 receptors** in the brain. Effective mainly against **positive symptoms**. Consistent with the dopamine hypothesis. Chlorpromazine also has a sedative effect.
- **Atypical (second-generation) antipsychotics**, e.g. **clozapine, risperidone, olanzapine**: act on dopamine **and serotonin** (and other) receptors; occupy D2 receptors more transiently. Aim to treat **positive and negative symptoms** with **fewer extrapyramidal side effects**. Clozapine is used for treatment-resistant cases but carries a risk of **agranulocytosis** (requires blood monitoring).

Indicative AO3 content (any combination of strengths/limitations):

- **Strength — effectiveness evidence**: **Thornley et al. (2003)** meta-analysis (13 trials, 1,121 participants) found chlorpromazine significantly better than placebo at reducing symptom severity. Around 70% of patients improve, supporting widespread use.
- **Strength — practical/economic value**: antipsychotics are relatively cheap, act faster than psychological therapies, and enable many patients to live in the community rather than in hospital, reducing care costs.
- **Strength — atypicals improve adherence**: fewer extrapyramidal effects than typicals means patients are more likely to keep taking the medication.
- **Limitation — serious side effects**: typicals cause extrapyramidal symptoms and **tardive dyskinesia** (~30% on long-term use); atypicals cause weight gain, metabolic syndrome and (clozapine) agranulocytosis. Side effects reduce quality of life and adherence.
- **Limitation — treats symptoms, not cause**: antipsychotics suppress symptoms only while taken; relapse is common after discontinuation, and they do not address cognitive or social factors.

- **Limitation — challenges to the dopamine rationale / bias**: the effectiveness of clozapine (which acts on several neurotransmitters) suggests dopamine is not the whole story; **Healy (2012)** argued that publication and researcher bias may inflate reported effectiveness.

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AO1, AO2, AO3 · 16 marks 16-mark

> *Discuss the importance of an interactionist approach in explaining and treating schizophrenia. Refer to the case of Daniel as part of your discussion.*

Marks for this question: AO1 = 6 marks, AO2 = 4 marks, AO3 = 6 marks

Level	Marks	Descriptor
4	13–16	Knowledge of the interactionist approach / diathesis-stress model is accurate and generally well detailed. Application to Daniel is effective and integrated across the stem. Discussion is thorough and effective. Clear, coherent and focused; specialist terminology used effectively.
3	9–12	Knowledge evident with some accuracy. Application mostly effective. Discussion mostly effective but limited in places.
2	5–8	Some accurate knowledge of the approach. Application limited. Discussion superficial / mainly descriptive.
1	1–4	Knowledge limited; little or no application or discussion.
0	0	No relevant content.

Indicative AO1 content — should cover both *explanation* and *treatment*:

- **The interactionist approach** argues schizophrenia is best explained by the interaction of **biological** and **psychological/environmental** factors, rather than either alone.
- **The diathesis-stress model**: a **diathesis** (vulnerability) plus **stress** (a trigger) is needed for schizophrenia to develop — neither is sufficient on its own.
- **Original model (Meehl 1962)**: diathesis = a single "schizogene" producing a biological vulnerability; if a person did not have the gene, no amount of stress would lead to schizophrenia.
- **Modern model (e.g. Houston et al. 2008)**: the diathesis can be polygenic and can include early **trauma**; "stress" is broadened beyond parenting to include, for example, **cannabis use** (a risk factor that can act as a trigger in vulnerable individuals).
- **Treatment implication**: an interactionist model justifies **combined treatment** — antipsychotic medication (targeting the biological diathesis) alongside CBT and/or family therapy (targeting psychological vulnerability and environmental stress).

Indicative AO2 content — engagement with Daniel:

- **Diathesis**: Daniel's biological father had schizophrenia → an inherited genetic vulnerability. Crucially, Daniel was adopted into a "settled, happy" home and still developed schizophrenia, suggesting the vulnerability travelled with his genes rather than his upbringing (parallels Tienari's adoption design).
- **Stress**: heavy **cannabis use**, **intense exam pressure** and a **difficult break-up** in his first year at university are environmental stressors that could trigger onset in a vulnerable person.
- **Interaction**: neither the genetic risk (he had a happy childhood with no symptoms) nor ordinary stress alone would be sufficient — it is the **combination** that best explains why psychosis emerged at 19.

- **Treatment**: the psychiatrist's plan of **antipsychotics + CBT** is interactionist treatment in practice — addressing both the biological and the psychological sides of Daniel's illness.

Indicative AO3 content:

- **Strength — research support (Tienari et al. 2004)**: Finnish adoption study — children of schizophrenic mothers adopted into **disturbed** families had much higher rates of schizophrenia (≈11%) than those adopted into **healthy** families (≈1%), and a healthy family was protective. Strong evidence that genetic vulnerability and environmental stress *interact*.
- **Strength — explains why MZ concordance is below 100%**: identical twins do not always both develop schizophrenia; the model accounts for this through differences in exposure to stress — an advantage over purely genetic accounts.
- **Strength — supports combined treatment**: studies (e.g. Tarrier) show patients receiving medication *plus* psychological therapy often show greater improvement than either alone, validating the interactionist treatment approach Daniel is offered.
- **Limitation — the original model is oversimplified**: treating diathesis as a single gene and stress as purely environmental is outdated; vulnerability is polygenic, stress can be biological (e.g. prenatal), and epigenetics blurs the gene–environment divide (Houston et al.).
- **Limitation — diathesis and stress are hard to operationalise/measure**: quantifying "vulnerability" and "stress" is difficult, and family-environment ratings (e.g. Tienari's) are necessarily subjective.
- **Limitation — treatment-causation fallacy**: the success of combined treatment does not, by itself, prove an interactionist *cause* (Jarvis & Russell) — successful treatment cannot be used as direct evidence for the model of causation.

Top-band answers will (1) describe the interactionist approach AND the diathesis-stress model (ideally original vs modern); (2) map Daniel's biological father onto the diathesis and his cannabis use / exam stress / break-up onto the stressors, noting the adoption detail; (3) connect his antipsychotics-plus-CBT plan to interactionist treatment; (4) evaluate using named evidence (Tienari, MZ-concordance logic, combined-treatment studies) with at least one limitation; and (5) reach a clear conclusion — typically that the interactionist/diathesis-stress model is the current consensus, is well supported by adoption studies, and best explains why combined treatment is used.

END OF MARK SCHEME · Maximum mark: 48