Abnormal Psychology (HL Option A)

IB HL Study Guide

Contents

- What is Abnormal Psychology? HL
- Concepts of Normality and Abnormality
  - Four Definitions of Abnormality
  - Comparing the Four Definitions
- Validity and Reliability of Diagnosis
  - Reliability
  - Validity
  - Rosenhan (1973) — "On Being Sane in Insane Places"
  - DSM-5 vs ICD-11
- Etiology of Abnormal Psychology — Major Depressive Disorder (MDD) HL
  - Biological Etiology of Depression
  - Cognitive Etiology of Depression
  - Sociocultural Etiology of Depression
- Etiology of Abnormal Psychology — Schizophrenia HL
  - Positive and Negative Symptoms
  - Biological Etiology of Schizophrenia
  - Cognitive Etiology of Schizophrenia
  - Sociocultural Etiology of Schizophrenia
- Treatment of Abnormal Psychology HL
  - Biological Treatments
  - Cognitive Treatment — CBT
  - Sociocultural Treatments
- IB Exam Technique — Paper 2 (HL Options)
  - SAQ Structure (9 marks, ~220 words)
  - ERQ Structure (22 marks, ~800 words)
  - Common Errors
- May 2026 Exam Predictions — HL Option A Abnormal Psychology
- Practice Questions
  - Short-Answer Questions (SAQs — 9 marks each)
  - Extended Response Questions (ERQs — 22 marks each)

Videos on this page: Watch: Psychological Disorders Overview · Watch: Depression and Treatment

What is Abnormal Psychology? HL

Abnormal psychology is an **HL-only option** examined in **IB Psychology Paper 2**. It is not assessed at SL. Paper 2 has two sections: SAQs (9 marks each) and ERQs (22 marks each), both drawn from the HL option you have studied. Option A — Abnormal Psychology — covers three major areas: concepts of normality and abnormality, the etiology (causes) of abnormal behaviour, and the treatment of psychological disorders.

Defining what counts as "abnormal" in psychology is far more contested than the word implies. Behaviour that is statistically rare, functionally disabling, culturally deviant, or personally distressing can each be labelled abnormal — yet none of these criteria alone is sufficient, and each brings its own conceptual problems. This complexity is itself examinable content.

> **IB TIP**
>
> Paper 2 SAQs are worth 9 marks (same as Paper 1). The structure is identical — define, study, link — and the same word target (~220 words) applies. For ERQs, aim for approximately 800 words; a tight thesis and genuine evaluation outweigh padding.

Concepts of Normality and Abnormality

Four Definitions of Abnormality

IB examiners require you to know at least four distinct definitions of abnormality, each with at least one strength and one limitation.

1. Statistical definition

Abnormality is defined as behaviour that deviates significantly from the statistical norm — that is, from what the majority of people in a population do. Behaviour lying at the extreme ends of a normal distribution (beyond roughly two standard deviations from the mean) is considered statistically abnormal.

- **Strength:** Objective and empirically measurable; does not rely on subjective judgement.
- **Limitation:** Does not distinguish between harmful and benign deviations. Extraordinary intelligence and exceptional athletic performance are statistically rare but are not disorders. Rarity alone tells us nothing about whether a behaviour is problematic.

2. Functional definition

Abnormality is defined as an inability to perform the activities necessary for everyday functioning — working, maintaining relationships, looking after oneself, and meeting social obligations.

- **Strength:** Practically relevant; grounds the definition in observable impairment rather than abstract norms.
- **Limitation:** What counts as adequate functioning varies significantly between cultures and social contexts. A behaviour that impairs functioning in one environment may be manageable in another.

3. Cultural definition

Abnormality is defined as behaviour that violates the norms, expectations, and values of the culture in which it occurs.

- **Strength:** Context-sensitive; recognises that behaviour cannot be judged independently of its social setting.
- **Limitation:** There is no universal cultural standard. Behaviour considered disordered in one culture may be accepted or valued in another (e.g., certain trance states are seen as signs of spiritual possession in some cultures and psychosis in others). This definition risks ethnocentrism if applied without awareness.

4. Subjective experience (personal distress)

Abnormality is defined by the presence of significant personal distress — the individual themselves feels that something is wrong or that they are suffering.

- **Strength:** Respects the lived experience of the person; avoids imposing external judgements; aligns with a patient-centred model of mental health.
- **Limitation:** Not all disorders involve subjective distress. Individuals in a manic episode or with certain personality disorders may not feel distress even when their behaviour is severely disruptive. Conversely, distress is a normal part of many life experiences (grief, stress) without indicating disorder.

Comparing the Four Definitions

Definition	Criterion	Key strength	Key limitation
Statistical	Deviation from population norm	Objective, measurable	Does not distinguish harmful from benign deviations
Functional	Inability to perform daily activities	Practically grounded	Culturally relative — functioning varies by context
Cultural	Violation of cultural norms	Context-sensitive	No universal standard; risks ethnocentrism
Subjective experience	Personal distress	Respects lived experience	Not all disorders involve distress

MEMORISE THIS

A mnemonic for the four definitions: S-F-C-S — Statistical, Functional, Cultural, Subjective experience. Each answers a different question: How rare? How disabling? How norm-violating? How distressing?

Validity and Reliability of Diagnosis

Psychological diagnosis involves assigning a label from a classification system — primarily the **DSM-5** (Diagnostic and Statistical Manual of Mental Disorders, 5th edition, American Psychiatric Association) or the **ICD-11** (International Classification of Diseases, World Health Organization). The scientific quality of any diagnostic system must be evaluated against two standards: reliability and validity.

Reliability

Test-retest reliability: The same clinician reaches the same diagnosis for the same patient at two different times.

Inter-rater reliability: Two different clinicians independently examining the same patient reach the same diagnosis. Earlier versions of the DSM had poor inter-rater reliability for many categories. DSM-5 field trials showed moderate inter-rater reliability for major disorders (e.g., kappa values of around 0.46–0.86 for major depressive disorder, varying by site).

Validity

Criterion validity: Does the diagnosis predict outcomes (treatment response, prognosis) that it theoretically should? A diagnosis with good criterion validity should help determine effective treatment.

Cross-cultural validity: Do diagnostic criteria identify the same disorder meaningfully across different cultural contexts? Evidence suggests that many DSM categories were developed primarily in Western, industrialised samples and may not translate without modification to other cultural contexts.

Rosenhan (1973) — “On Being Sane in Insane Places”

This is the most commonly assessed study for the validity and reliability of psychiatric diagnosis.

Aim: To investigate whether psychiatric hospitals could distinguish the sane from the insane.

Method: Eight pseudo-patients (including Rosenhan himself — a psychiatrist, three psychologists, a paediatrician, a painter, a housewife, and a graduate student) presented to 12 different psychiatric hospitals in five US states complaining of a single symptom: hearing a voice that said “empty,” “hollow,” and “thud.” Beyond this presenting complaint, they behaved completely normally. If admitted, the pseudo-patients ceased

reporting any symptoms and behaved as they ordinarily would. They took notes about their experiences openly.

Findings:

- All 8 pseudo-patients were admitted. 7 were diagnosed with **schizophrenia**; 1 with **bipolar disorder with psychotic features**.
- None of the hospitals detected the pseudo-patients as sane. All 8 were eventually discharged "in remission" — the 7 diagnosed with schizophrenia as "schizophrenia in remission"; the 8th (diagnosed with manic-depressive psychosis) with that label in remission.
- Average hospitalisation was 19 days (range 7–52 days).
- Note-taking was recorded in patient charts as evidence of psychopathological behaviour.
- Other patients (not staff) were often suspicious: "You're not crazy. You're a journalist or professor."
- In a follow-up condition, a hospital informed that pseudo-patients would be sent was later found to have identified 41 suspected pseudo-patients (out of 193 admissions) — yet Rosenhan sent none.

Conclusion: Psychiatric hospitals in 1973 were unable to detect sanity. Once a psychiatric label is applied, all subsequent behaviour is interpreted through that lens (labelling effect). The diagnosis of schizophrenia says more about the context than about the individual.

Evaluation:

Strength	Limitation
Ecological validity — conducted in real psychiatric settings with real admission procedures	Deception-based research raises ethical concerns; hospital staff could not consent to being studied
Highlights the real-world consequences of psychiatric labelling, with direct policy implications	The study is dated — conducted in 1973; DSM criteria and clinical training have substantially changed since
Cross-hospital replication (12 hospitals, 5 states) increases generalisability within its context	Presenting with auditory hallucinations is a clinically significant symptom; admission was arguably appropriate and cautious, not erroneous

⚠ **EXAM ALERT**

Rosenhan (1973) is one of the most tested studies in the IB Abnormal Psychology option. It appears in SAQs on validity of diagnosis ("describe one study related to the validity of psychological diagnosis") and in ERQs evaluating diagnostic classification systems. Know: 8 pseudo-patients, 12 hospitals, 7 schizophrenia diagnoses, none detected, discharged "in remission."

DSM-5 vs ICD-11

The DSM-5 (categorical system; 947 pages; primarily used in the USA) and ICD-11 (used globally for all medical conditions, including mental health; adopted 2022) share most major diagnostic categories but differ in some criteria thresholds and organisational structure. Both have been criticised for:

- **Medicalising** normal human distress (e.g., removing the bereavement exclusion from major depression in DSM-5).
- **Cultural bias** — diagnostic criteria were largely developed on Western, male samples.
- **Labelling effects** — psychiatric diagnosis can become a self-fulfilling prophecy that shapes identity and affects how others respond to the individual (Rosenhan, 1973).

Etiology of Abnormal Psychology — Major Depressive Disorder (MDD) HL

Major Depressive Disorder is characterised by persistent depressed mood or loss of interest/pleasure, accompanied by cognitive, somatic, and behavioural symptoms for at least two weeks. IB requires you to explain depression from at least two levels of analysis.

Biological Etiology of Depression

Monoamine hypothesis

The monoamine hypothesis proposes that depression is caused by a deficit in monoamine neurotransmitters — particularly **serotonin** and **norepinephrine** — at central synapses. Evidence comes primarily from the mechanism of antidepressant drugs: monoamine oxidase inhibitors (MAOIs) prevent the breakdown of monoamines; tricyclic antidepressants block reuptake of serotonin and norepinephrine; SSRIs selectively block serotonin reuptake — all resulting in elevated monoamine availability and, in many patients, reduced depression symptoms.

Limitation: This is pharmacological evidence, not direct measurement of neurotransmitter levels in depressed individuals. The “medication-as-evidence” fallacy applies: the fact that raising serotonin often reduces depression does not prove that low serotonin causes depression. Many patients do not respond to SSRIs, suggesting the relationship is more complex.

HPA axis dysregulation

Chronic stress activates the hypothalamic-pituitary-adrenal (HPA) axis, producing elevated cortisol. Hypercortisolaemia damages hippocampal neurons (reducing hippocampal volume) and dysregulates the feedback mechanism that normally

terminates the stress response. This creates a self-perpetuating cycle of cortisol elevation associated with depressive symptoms. Luby et al. (2012) demonstrated that early parenting quality affects hippocampal volume via cortisol mediation — linking stress biology directly to structural brain changes.

Genetic factors — Kendler et al. (1993)

Aim: To estimate the heritability of major depression in women and to examine gene-environment interactions.

Method: Kendler et al. studied a population-based sample of **female twin pairs** (Virginia Twin Registry), assessing lifetime diagnosis of major depression through structured psychiatric interviews. The sample included both monozygotic (MZ) and dizygotic (DZ) twin pairs.

Findings: The concordance rate for major depression was approximately **46% for MZ twins** and approximately **20% for DZ twins**.

Conclusion: The substantially higher concordance in MZ pairs (who share ~100% of DNA) compared to DZ pairs (~50% shared DNA) indicates a significant genetic component to major depression. However, the MZ concordance is well below 100%, confirming that genes alone do not determine depression — environmental factors are also necessary. The authors estimated heritability at approximately 40–50%.

Evaluation of biological etiology:

Strength	Limitation
Twin study methodology (Kendler et al.) allows estimation of heritability while controlling for shared environment	Correlation between neurotransmitter levels and depression does not establish causation — the monoamine hypothesis remains contested
HPA axis research links stress, brain structure, and depression through measurable biological mechanisms	MZ concordance of ~46% means environment accounts for more than half the variance — purely biological accounts are insufficient
Genetic evidence motivates pharmacological treatments with documented efficacy	Female-only sample (Kendler et al.) limits generalisability to men

Cognitive Etiology of Depression

Beck's Cognitive Triad (1967)

Aaron Beck proposed that depression is maintained by a pattern of systematic negative thinking he called the **cognitive triad** — three interlocking negative schemas about the self, the world, and the future.

Triad component	Content
Negative view of self	”I am worthless, inadequate, defective”
Negative view of the world	”The world makes impossible demands; I fail everywhere”
Negative view of the future	”Things will never improve; I expect failure and suffering”

These negative schemas produce characteristic **cognitive distortions** — systematic errors in thinking that maintain the depressive cognitive pattern:

- **Overgeneralisation:** Drawing sweeping conclusions from a single event (“I failed this exam, therefore I am a failure at everything”).
- **Catastrophising:** Interpreting minor setbacks as disasters.
- **Personalisation:** Attributing external events to oneself without evidence (“The team failed because of me”).
- **Black-and-white thinking:** Viewing situations in all-or-nothing terms with no middle ground.

Beck’s original clinical research (Beck et al., 1979) involved systematic observation of depressed patients in psychotherapy. He found that depressed patients showed consistent, repetitive patterns of negative automatic thoughts that were not present in non-depressed patients — supporting the view that depression involves a distinct cognitive mode rather than simply more of the negative thoughts everyone has.

Evaluation of cognitive etiology:

Strength	Limitation
Based on clinical observation — high ecological validity; directly informed CBT, one of the most evidence-based treatments available	Chicken-and-egg problem: do negative cognitive patterns cause depression, or does depression cause negative thinking? Causality is difficult to establish
Specific, testable predictions (e.g., depressed people show more cognitive distortions on assessment tools like the BDI — Beck Depression Inventory)	The model may describe depression rather than explain it; it does not account for why some people develop negative schemas and others do not
Powerful therapeutic implications: CBT (derived from Beck’s model) has robust RCT evidence	Cognitive factors do not explain biological markers of depression (hippocampal changes, cortisol) without additional integration

Sociocultural Etiology of Depression

Brown and Harris (1978) — “The Social Origins of Depression”

Aim: To investigate how social factors contribute to the onset of depression, with particular attention to vulnerability factors that mediate the effect of stressful life events.

Method: Brown and Harris conducted a community survey of **539 working-class women** in Camberwell, London. They used structured interviews to assess: recent stressful life events, long-term social difficulties, clinical depression (using a standardised interview), and the presence of four proposed vulnerability factors.

Findings: Women who experienced a severe life event (e.g., bereavement, serious illness) were significantly more likely to develop depression — but only if they also had one or more **vulnerability factors**:

1. Loss of mother before age 11
2. Three or more children under the age of 14 at home
3. Absence of a close, confiding relationship (e.g., no intimate partner or friend)
4. Lack of paid employment

Women with all four vulnerability factors who experienced a severe life event had a depression onset rate of approximately 79%. Those without vulnerability factors who experienced the same life events had a much lower onset rate.

Conclusion: Sociocultural vulnerability factors do not cause depression directly, but they remove the psychological and social resources that buffer individuals against the depressogenic impact of stressful life events. The study supports a **diathesis-stress model** at the social level: vulnerability + stressor = disorder.

Social causation vs social drift

The **social causation hypothesis** holds that lower socioeconomic status, social isolation, and chronic stress generate depression (i.e., poverty and disadvantage cause disorder). The **social drift hypothesis** holds that the reverse is true: people with depression are less able to maintain employment and social standing and thus drift downward socioeconomically. Brown and Harris's data supported social causation — the vulnerability factors preceded the depression onset.

Cultural variation in depression

Cross-cultural research indicates that depression is expressed differently across cultures. In many non-Western contexts, depressive experiences are predominantly **somatic** (bodily complaints: headaches, fatigue, physical pain) rather than **cognitive** (sadness, hopelessness, worthlessness) — the pattern more common in Western populations. This challenges the universality of DSM/ICD diagnostic criteria and suggests that cultural context shapes not just help-seeking but the phenomenology of depression itself.

Evaluation of sociocultural etiology:

Strength	Limitation
Large community sample (n=539); high ecological validity — real women, real life events, real depression	Retrospective self-report: participants recalled life events and childhood history; vulnerable to recall bias
Identifies modifiable risk factors with clear public health implications (social support, employment, maternal care)	Causal direction is not certain — some factors (e.g., absence of confiding relationship) could be consequences as well as causes of depression
Diathesis-stress model integrates sociocultural factors with biological/cognitive accounts	Working-class London sample — generalisability across cultures, gender, and class is limited

Etiology of Abnormal Psychology — Schizophrenia HL

Schizophrenia is characterised by a breakdown in thought, perception, emotion, and behaviour. Diagnosis requires two or more of the following symptoms for at least one month, with significant functional impairment: delusions, hallucinations, disorganised speech, grossly disorganised or catatonic behaviour, and negative symptoms.

Positive and Negative Symptoms

Category	Symptoms	Description
Positive symptoms	Hallucinations	Sensory experiences without external stimulus — most commonly auditory ("hearing voices")
	Delusions	Fixed false beliefs resistant to evidence — e.g., persecutory ("being followed"), referential ("TV broadcasts are about me"), grandiose
	Disorganised thinking	Incoherent speech, loose associations, tangentiality
Negative symptoms	Flat affect	Reduced emotional expression in face and voice
	Alogia	Poverty of speech — brief, empty responses
	Avolition	Reduced motivation and goal-directed behaviour

MEMORISE THIS

Positive vs negative symptoms: Positive symptoms are things added to normal experience (hallucinations, delusions). Negative symptoms are things subtracted from normal functioning (flat affect, alogia, avolition). Biological treatments (antipsychotics) are more effective for positive than negative symptoms — this distinction has treatment implications.

Biological Etiology of Schizophrenia

Dopamine hypothesis

The dopamine hypothesis proposes that excess dopamine activity in the **mesolimbic pathway** (the limbic system's reward circuit) causes the positive symptoms of schizophrenia. Evidence: typical antipsychotics (e.g., haloperidol) are D2 receptor antagonists — they block dopamine receptors and reduce positive symptoms. Drugs that increase dopamine activity (amphetamines, cocaine) can induce psychotic symptoms in healthy individuals.

Limitation: The dopamine hypothesis is now considered an oversimplification. The **glutamate system** is also implicated: phencyclidine (PCP), a glutamate antagonist, produces both positive and negative symptoms. Atypical antipsychotics act on serotonin as well as dopamine. Negative symptoms are not well explained by dopamine excess alone.

Genetic factors — Gottesman (1991)

Gottesman compiled data across multiple family and twin studies to produce one of the most cited tables in abnormal psychology: concordance rates for schizophrenia as a function of genetic relatedness.

Key concordance figures:

Relationship	Shared DNA	Concordance rate
Identical (MZ) twin	~100%	~48%
Fraternal (DZ) twin	~50%	~17%
Sibling	~50%	~9%
Child of one parent with schizophrenia	~50%	~13%
General population	—	~1%

Conclusion: The stepwise increase in concordance with genetic relatedness provides strong evidence for a heritable component to schizophrenia. The MZ concordance of ~48% also shows that genes alone are not deterministic — environmental factors account for the remaining variance.

Evaluation:

Strength	Limitation
Systematic compilation across multiple studies increases reliability of estimates	MZ twin concordance of ~48% is far from 100% — environment is a major contributor; purely biological accounts are insufficient
Consistent with adoption study data (children of parents with schizophrenia raised by non-affected families retain elevated risk)	Equal environment assumption in twin studies is contested — MZ twins may be treated more similarly, inflating genetic estimates
Data motivated successful search for candidate genes and chromosomal loci	The dopamine hypothesis does not explain negative symptoms or treatment-resistant cases

Cognitive Etiology of Schizophrenia

Cognitive accounts of schizophrenia focus on the specific deficits and processing anomalies that may give rise to symptoms.

- **Impaired source monitoring:** Difficulty distinguishing between self-generated and externally generated speech — a cognitive model of auditory hallucinations. If internal speech is misattributed to an external source, the result is the subjective experience of "hearing voices."
- **Working memory and executive function deficits:** Impaired working memory is among the most robustly replicated cognitive findings in schizophrenia, associated with disorganised thinking and inability to maintain goal-directed behaviour.
- **Attention deficits:** Impaired ability to filter irrelevant stimuli (impaired latent inhibition) — may contribute to the flooding of consciousness with normally filtered information that underlies some positive symptoms.

Limitation: Cognitive deficits in schizophrenia could be consequences of the disorder (or its treatment) rather than causes. It is methodologically difficult to study pre-onset cognitive function in clinical populations.

Sociocultural Etiology of Schizophrenia

Urbanicity — Krabbendam and van Os (2005)

A meta-analysis by Krabbendam and van Os (2005) examined the relationship between urban living and schizophrenia risk across multiple studies. They found that individuals raised in urban environments have approximately twice the risk of developing schizophrenia compared to those raised in rural areas, and that this risk increases with the degree of urbanisation.

Two competing explanations are proposed:

- **Social causation:** Urban environments involve chronic stress (noise, crowding, social fragmentation, poverty, discrimination) that directly increases vulnerability to psychosis.
- **Social drift:** Individuals with prodromal symptoms (early-stage schizophrenia) drift toward urban, deprived areas because of difficulty maintaining stable employment and housing.

Krabbendam and van Os's analysis of prospective data (following individuals before and after any diagnosis) supported social causation as the primary mechanism — urban exposure preceded diagnosis.

Expressed emotion (EE) and relapse

Expressed emotion refers to the emotional climate in a patient's family environment, measured by three dimensions: **criticism, hostility**, and **emotional over-involvement**.

Research consistently shows that patients with schizophrenia returning to high-EE family environments have significantly higher relapse rates than those returning to low-EE environments. This is relevant both to etiology (family stress as a maintaining factor) and to treatment (family psychoeducation to reduce EE).

▸Watch: Psychological Disorders Overview VIDEO

Treatment of Abnormal Psychology HL

IB Paper 2 requires you to evaluate treatments from at least two levels of analysis. Treatments must be linked to a specific disorder (depression or schizophrenia).

Biological Treatments

Drug therapy for depression — SSRIs

Selective serotonin reuptake inhibitors (SSRIs) are the first-line pharmacological treatment for major depressive disorder. The most well-known SSRI is **fluoxetine** (brand name Prozac).

Mechanism: SSRIs block the serotonin transporter protein (SERT) on the presynaptic neuron, preventing the reuptake of serotonin from the synaptic cleft back into the releasing neuron. This increases the concentration of serotonin available at postsynaptic receptors.

Efficacy: Multiple RCTs demonstrate that SSRIs are more effective than placebo for moderate-to-severe depression (effect sizes moderate: d ≈ 0.3–0.5 in comprehensive meta-analyses, e.g., Cipriani et al., 2018). Response typically begins after 2–4 weeks of treatment.

Side effects: Nausea, sexual dysfunction, sleep disturbance, emotional blunting. Risk of discontinuation syndrome if stopped abruptly.

Drug therapy for schizophrenia — antipsychotics

- **Typical antipsychotics** (first generation, e.g., haloperidol, chlorpromazine): D2 receptor antagonists. Highly effective for positive symptoms (hallucinations, delusions). Side effects include **tardive dyskinesia** (involuntary repetitive movements, potentially irreversible) and **extrapyramidal side effects** (Parkinsonism, akathisia).
- **Atypical antipsychotics** (second generation, e.g., clozapine, risperidone): act on both dopamine and serotonin receptors; more effective for negative symptoms and treatment-resistant cases. Clozapine has significant side effects including agranulocytosis (requiring regular blood monitoring).

Evaluation of biological treatments:

Strength	Limitation
Rapid onset relative to psychological therapies; accessible and scalable through primary care	Treats symptoms, not underlying causes — relapse rates are high when medication is discontinued
RCT evidence supports efficacy for both depression (SSRIs) and schizophrenia (antipsychotics)	Side effects affect adherence; non-adherence is a major factor in schizophrenia relapse
Allows patients who cannot engage in psychotherapy (due to severity) to stabilise	Medication-as-evidence fallacy: efficacy of serotonergic drugs does not confirm the monoamine theory of depression

Cognitive Treatment — CBT

CBT for depression

Cognitive Behavioural Therapy (CBT) for depression is directly derived from Beck's cognitive triad model. The core goal is to identify and modify the negative automatic thoughts and cognitive distortions that maintain depression.

Key stages:

1. **Psychoeducation:** Patient learns the cognitive model — the relationship between thoughts, emotions, and behaviours.
2. **Identifying automatic thoughts:** Patient keeps a thought diary; negative automatic thoughts are recorded and examined.
3. **Cognitive restructuring:** Challenging the evidence for and against negative automatic thoughts; developing more balanced, realistic alternatives.
4. **Behavioural activation:** Scheduling pleasant activities to break the withdrawal-low-mood cycle.
5. **Relapse prevention:** Identifying warning signs and strategies for maintaining gains.

CBT for schizophrenia

CBT for psychosis focuses on: challenging the reality status of delusions (not confronting them directly but examining the evidence); developing coping strategies for distressing hallucinations; and addressing negative symptoms through activity scheduling.

DeRubeis et al. (2005) — CBT vs antidepressants

Aim: To compare CBT and antidepressant medication (paroxetine) for moderate-to-severe major depression.

Method: A large RCT (n=240) randomly assigned patients with moderate-to-severe depression to 16 weeks of CBT or paroxetine (a SSRI-type antidepressant). Response rates were assessed at 8 and 16 weeks, and at one-year follow-up.

Findings: Response rates at 16 weeks were equivalent for CBT and antidepressant medication — approximately 50% response in each condition. At one-year follow-up, patients who had received CBT had significantly lower relapse rates than those who had taken medication and then stopped.

Conclusion: CBT is as effective as antidepressant medication for moderate-to-severe depression and produces more durable effects, suggesting that it addresses underlying cognitive patterns rather than only managing symptoms.

Evaluation of CBT:

Strength	Limitation
Addresses the cognitive root causes of depression — lower relapse rates than medication alone (DeRubeis et al., 2005)	Requires patient motivation, insight, and capacity for self-reflection — not suitable for severe psychosis without stabilisation
RCT evidence (highest level of evidence) supports efficacy for both depression and psychosis	Therapist skill and therapeutic alliance influence outcomes — hard to standardise and scale
Develops transferable skills for self-management — patient gains long-term coping tools	Access is limited by cost and availability of trained therapists; waiting lists can be lengthy

Sociocultural Treatments

Community-based care and assertive community treatment (ACT)

Assertive community treatment involves multidisciplinary mental health teams (psychiatrists, psychologists, social workers, nurses) providing intensive, community-based support to individuals with severe mental illness. ACT teams make outreach visits, assist with medication management, provide social support, and coordinate with housing and employment services. Research indicates that ACT reduces hospitalisation and improves community functioning compared to standard outpatient care.

Reducing expressed emotion — Leff et al. (1982)

Leff and colleagues implemented a family intervention for patients with schizophrenia returning to high-EE family environments. The intervention included psychoeducation about schizophrenia, relatives' groups (where family members shared experiences and strategies), and individual family sessions. The key goal was reducing critical comments, hostility, and emotional over-involvement.

Findings: At nine-month follow-up, the intervention group had significantly lower relapse rates (8%) compared to the control group (50%). Expressed emotion in families in the intervention group decreased significantly.

Conclusion: Psychosocial family intervention targeting expressed emotion is an effective maintenance treatment for schizophrenia, operating through a sociocultural mechanism (interpersonal family climate).

Stigma and structural barriers

Mental health stigma — both public stigma (negative societal attitudes toward those with mental illness) and self-stigma (internalisation of stigma by the person with the disorder) — is a major barrier to treatment access and adherence. Structural barriers include unequal access to mental healthcare across socioeconomic groups, cultural mistrust of mental health systems, and lack of culturally competent services for minority populations.

Evaluation of sociocultural treatments:

Strength	Limitation
High ecological validity — treats patients in their actual social environment; addresses maintaining factors, not just symptoms	Resource-intensive: ACT and family interventions require trained staff, coordination, and sustained funding
Leff et al. (1982) demonstrates that family-level intervention significantly reduces schizophrenia relapse	Difficult to isolate which component of a multi-component intervention is the active ingredient
Addresses the social determinants of mental illness and treatment-seeking, not just individual pathology	Effectiveness may vary by family structure, cultural context, and specific diagnosis

▸**Watch: Depression and Treatment** VIDEO

IB Exam Technique — Paper 2 (HL Options)

SAQ Structure (9 marks, ~220 words)

Paper 2 SAQs ask you to "describe" or "explain" a study or concept related to the HL option. The 9-mark structure is:

1. **Define** the relevant concept (1–2 sentences).
2. **Describe the study** — aim, method (briefly), findings, conclusion.
3. **Link** the study explicitly to the specific concept in the question (1–2 sentences). Never describe the study and leave the connection implicit.

ERQ Structure (22 marks, ~800 words)

Paper 2 ERQs use command terms: **evaluate, discuss, to what extent**. The structure is:

1. **Thesis** — one sentence stating your overall argument (e.g., "Biological explanations for depression are supported by genetic and neurochemical evidence but are insufficient alone because environmental factors account for more than half the variance").
2. **Study 1** — full description (aim, method, findings, conclusion), ~200 words.
3. **Evaluation of Study 1** — at least one strength and one limitation, explicitly linked to the essay question.

4. **Study 2** — full description, ~200 words.
5. **Evaluation of Study 2** — strength and limitation.
6. **Counter-argument** — a genuinely different perspective or an alternative explanation (e.g., "However, a cognitive account of depression…").
7. **Balanced conclusion** — restate your thesis in light of the evidence; acknowledge what remains uncertain.

Common Errors

- **Generic study descriptions:** Describing Rosenhan or Kendler accurately but not connecting the findings to the specific concept in the question (e.g., validity of diagnosis, heritability). Examiners penalise disconnected study descriptions regardless of accuracy.
- **One-sided evaluation:** Listing only limitations without genuine engagement with strengths, or vice versa. A top-band ERQ must engage with both sides of the argument.
- **Confusing etiology and treatment:** Paper 2 questions specify a topic — do not drift from etiology to treatment or vice versa unless the question invites it.

WORKED EXAMPLE

Model SAQ plan — "Explain one etiology of depression" (9 marks):

1. Identify the level and theory: Sociocultural etiology — Brown and Harris's vulnerability model.
2. Define: The sociocultural approach explains depression through social vulnerability factors that amplify the impact of stressful life events.
3. Study — Brown and Harris (1978):
    - Aim: investigate how social factors and vulnerability contribute to depression onset.
    - Method: community survey of 539 working-class women in London; structured interviews assessing life events, depression, and four vulnerability factors.
    - Findings: women with severe life events AND vulnerability factors had depression onset rates of ~79%; women without vulnerability factors had much lower rates despite similar life events.
    - Conclusion: social vulnerability factors remove protective resources, making individuals more susceptible to the depressogenic effects of stressful life events.
4. Link: this supports a sociocultural etiology because it shows that depression is not simply caused by individual biology or cognition but by the interaction between social circumstances (vulnerability factors) and life events — placing the cause of depression partly outside the individual.

⚠ EXAM ALERT

The most common Paper 2 SAQ error is describing a study accurately but leaving the link implicit. Write the link explicitly: "This demonstrates a sociocultural etiology because…" The link sentence is what converts a description into an explanation — which is what the command term requires.

May 2026 Exam Predictions — HL Option A Abnormal Psychology

Based on IB Paper 2 question patterns across recent exam sessions, the following topics carry the highest probability for May 2026.

High-probability SAQs:

- Rosenhan (1973) — "describe one study related to the validity of psychiatric diagnosis" (very high probability; appeared multiple sessions)
- Brown and Harris (1978) — "explain one sociocultural etiology of depression"
- Beck's cognitive triad — "explain one cognitive etiology of depression"
- Gottesman concordance data — "describe one study related to the biological etiology of schizophrenia"

High-probability ERQs:

- "Evaluate biological and cognitive explanations for one psychological disorder" — requires Kendler (1993) + Beck/DeRubeis, with genuine comparative evaluation
- "Compare the effectiveness of two treatments for a psychological disorder" — requires CBT (DeRubeis et al., 2005) + SSRIs or antipsychotics, with RCT evidence and relapse rate data
- "Evaluate sociocultural explanations for one psychological disorder" — requires Brown and Harris (1978) + Krabbendam and van Os (2005) or Leff et al.

IB TIP

For ERQs comparing treatments, structure your answer around a common evaluation criterion: speed of onset (biological faster), relapse rates (CBT lower), side effects (biological treatment), accessibility (medication more scalable), and suitability for different severities. This shows analytical command rather than a list of individual study summaries.

Practice Questions

Short-Answer Questions (SAQs — 9 marks each)

SAQ 1. Explain one concept of abnormality.

Model answer framework:

- Choose one definition — e.g., statistical definition.
- Define clearly: abnormality as deviation from the statistical norm (behaviour beyond ±2 SD from the population mean).
- Evaluate the concept: strength — objective, empirically measurable; limitation — does not distinguish harmful from benign deviations (extraordinary intelligence is statistically rare but not a disorder).
- Conclude: the statistical definition is useful as an objective criterion but insufficient on its own as it ignores the social and personal meaning of behaviour.

SAQ 2. Describe one study related to the validity of psychiatric diagnosis.

Model answer framework:

- Name the study: Rosenhan (1973), "On Being Sane in Insane Places."
- Aim: to investigate whether psychiatric hospitals could distinguish sanity from insanity.
- Method: 8 pseudo-patients presented with single symptom (auditory hallucinations) to 12 hospitals; once admitted, behaved normally and ceased reporting symptoms; recorded experiences.
- Findings: all 8 admitted; 7 diagnosed with schizophrenia, 1 with manic-depressive psychosis; none detected as sane; all discharged "in remission" (schizophrenia or manic-depressive label respectively); average hospitalisation 19 days.
- Conclusion: psychiatric labels persist and distort interpretation of all subsequent behaviour; hospitals failed to detect sanity.
- Link: this directly challenges the validity of psychiatric diagnosis — the diagnosis reflected the context and labelling system, not the actual mental state of the pseudo-patients.

SAQ 3. Explain one biological etiology of schizophrenia.

Model answer framework:

- Identify: genetic factors (Gottesman, 1991).
- Define: heritability of schizophrenia refers to the proportion of variance in risk explained by genetic factors.
- Study — Gottesman (1991): compilation of family and twin study data; MZ concordance ~48%, DZ ~17%, sibling ~9%, general population ~1%.
- Conclude: stepwise increase with genetic relatedness confirms a significant genetic component; MZ concordance below 100% confirms environmental contribution.
- Link: this supports a biological etiology — shared genetics increase schizophrenia risk — but the incomplete MZ concordance means genes alone cannot be the sole cause.

SAQ 4. Outline one treatment for depression.

Model answer framework:

- Choose CBT or SSRIs.
- For CBT: name the treatment (Cognitive Behavioural Therapy; derived from Beck's 1967 cognitive triad model); describe the mechanism (identifying and challenging negative automatic thoughts; behavioural activation); evidence: DeRubeis et al. (2005) — CBT equivalent to antidepressants in acute phase; superior at one-year follow-up (lower relapse rates).
- Link: CBT treats the cognitive cause of depression (negative automatic thoughts and distortions) rather than only symptoms, explaining its superior relapse prevention.

Extended Response Questions (ERQs — 22 marks each)

ERQ 1. Evaluate biological and cognitive explanations for depression.

Answer guidance:

- **Thesis:** Both biological and cognitive explanations for depression are supported by substantial evidence but are incomplete — a full account requires integration, as the two levels of analysis address different dimensions of the disorder.
- **Biological explanation:** Genetic factors — Kendler et al. (1993). Describe fully: female twin registry, MZ ~46%, DZ ~20%, heritability ~40–50%. Evaluate: twin methodology allows heritability estimation; but MZ <100% means environment is essential; female-only sample limits generalisability.
- **Cognitive explanation:** Beck's cognitive triad — describe the three components (self, world, future), cognitive distortions, and Beck et al. (1979) clinical observations. Evaluate: high ecological validity (clinical research); directly generated CBT evidence; but causal direction unclear.
- **Counter-argument / integration:** Caspi et al. (2003) (GxE) demonstrates that biological vulnerability (5-HTT gene) interacts with stressful life events — bridging biological and sociocultural levels; suggests neither level is sufficient alone. Brown and Harris (1978) similarly shows that social vulnerability amplifies the impact of life events, paralleling the cognitive model's focus on vulnerability schemas.
- **Conclusion:** Biological and cognitive explanations each capture real aspects of depression. Genetic studies confirm heritability; cognitive models explain the maintenance of symptoms and drive evidence-based treatment. Integration across levels provides the most complete account.

ERQ 2. Evaluate the effectiveness of two treatments for a psychological disorder.

Answer guidance:

- Choose depression; treatments: SSRIs and CBT.

- **Thesis:** Both SSRIs and CBT are evidence-based treatments for major depressive disorder, but they differ in speed of onset, relapse prevention, and suitability — CBT produces more durable outcomes, while SSRIs are more accessible and appropriate for severe cases.
- **Treatment 1 — SSRIs:** Mechanism (block serotonin reuptake). Evidence: Cipriani et al. (2018) meta-analysis — SSRIs more effective than placebo; moderate effect sizes ($d \approx 0.3–0.5$). Evaluate: widely accessible; rapid onset relative to CBT; but high relapse rates when discontinued; side effects; symptom management rather than cause treatment.
- **Treatment 2 — CBT:** Mechanism (cognitive restructuring, behavioural activation, targeting Beck's triad). Evidence: DeRubeis et al. (2005) RCT — CBT equivalent to paroxetine in acute phase; lower relapse rates at one-year follow-up. Evaluate: targets cognitive etiology; durable outcomes; lower relapse; but requires patient motivation; therapist-dependent; resource-intensive.
- **Counter-argument:** Combined treatment (CBT + antidepressant) may be more effective than either alone for severe depression — biological stabilisation enables engagement in psychotherapy. This limits an either/or comparison.
- **Conclusion:** CBT and SSRIs are comparably effective in the acute phase but SSRIs carry higher relapse risk when stopped. For patients who can engage, CBT is preferable for long-term outcomes. A stepped-care approach — medication to stabilise, then CBT for relapse prevention — may be optimal.