SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Clinical Psychology and Mental Health

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.1.4 · A-level Paper 1

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Contents

How to use these notes. Clinical Psychology and Mental Health is an **A-level only** topic (AQA 7182, Paper 1) — it is not assessed at AS. Key terms are in **bold**; tables summarise definitions, characteristics and treatments; "exam tip" boxes call out the most common errors. Each section ends with PEEL evaluation that can be deployed as ready-made paragraphs in extended writing.

Note on the 2025 specification: the topic was renamed from *Psychopathology* to **Clinical Psychology and Mental Health**. "Deviation from social norms" is now **"deviation from social/cultural norms"**, and "deviation from ideal mental health" has been moved to the first definition listed. **No content was removed** — earlier past-paper material remains valid.

AQA 2025 SPECIFICATION — CLINICAL PSYCHOLOGY AND MENTAL HEALTH CONTENT (A-LEVEL ONLY)

- **Definitions in the field of mental health**: deviation from ideal mental health, deviation from social/cultural norms, failure to function adequately and statistical infrequency.
- The **behavioural, emotional and cognitive characteristics** of phobias, depression and obsessive-compulsive disorder (OCD).
- The **behavioural approach to explaining and treating phobias**: the two-process model, including classical and operant conditioning; systematic desensitisation, including relaxation and use of hierarchy; flooding.
- The **cognitive approach to explaining and treating depression**: Beck's negative triad and Ellis's ABC model; cognitive behaviour therapy (CBT), including challenging irrational thoughts.
- The **biological approach to explaining and treating OCD**: genetic and neural explanations; drug therapy.

1 Definitions in the Field of Mental Health

Psychologists have proposed four key ways of defining abnormality. Each captures something useful, but each also has significant limitations — together they form a more complete picture than any one alone.

The Four Definitions

Definition	What it means	Example
Deviation from Ideal Mental Health	Abnormality is the absence of criteria for psychological wellbeing. **Jahoda (1958)** proposed six criteria of ideal mental health: *positive self-attitudes, self-actualisation, autonomy, accurate perception of reality, environmental mastery* and *resistance to stress*.	A person unable to cope with daily stress, with low self-esteem and poor reality testing, would be considered abnormal.
Deviation from Social/Cultural Norms	Behaviour is abnormal if it deviates from the implicit or explicit rules of a society or culture. Norms vary between cultures and across time.	Hearing voices is treated as a symptom of schizophrenia in Western psychiatry but a culturally valued spiritual experience in some African cultures. Homosexuality was classified as a mental disorder in the DSM until 1973.
Failure to Function Adequately	Abnormality is the inability to cope with the demands of everyday life. **Rosenhan and Seligman (1989)** proposed seven features: *personal distress, maladaptive behaviour, unpredictability, irrationality, observer discomfort, violation of moral standards, unconventional behaviour*.	A person who cannot maintain personal hygiene, hold a job or sustain relationships due to severe depression.
Statistical Infrequency	Behaviour is abnormal if it occurs rarely in the population — typically more than two standard deviations from the mean on a normal distribution.	An IQ below 70 (around 2% of the population) is used as one criterion for intellectual disability.

Evaluation

Strength of statistical infrequency — clear, objective criterion. A key strength of the statistical-infrequency definition is that it provides an objective, quantitative cut-off. IQ scores below 70 are used by clinicians to diagnose intellectual disability because they fall more than two standard deviations below the mean. This is important because it removes subjective judgement and allows consistent classification between practitioners. This therefore strengthens its reliability as a diagnostic tool, particularly for traits that can be precisely measured.

Limitation of statistical infrequency — desirable rarity is not abnormal. However, a major limitation is that being statistically rare is not necessarily a sign of mental disorder. An IQ above 130 is just as statistically rare as one below 70 but is considered desirable rather than abnormal. Similarly, very low rates of depression

would be classified as "abnormal" on this definition. This is important because it shows the definition cannot distinguish desirable from undesirable rarity, weakening its validity as a clinical criterion.

Strength of failure-to-function — recognises the patient's perspective. A genuine strength of the failure-to-function definition is that it puts the individual's experience at the centre of diagnosis. Rosenhan and Seligman's criteria — particularly personal distress and inability to cope — are exactly what most people would intuitively associate with "needing help". This is important because it ensures that diagnosis serves the individual's wellbeing rather than imposing external judgements. This strengthens the validity of the definition as a practical guide for clinical decisions about who needs treatment.

Limitation of failure-to-function — abnormality without dysfunction. However, a limitation is that some serious disorders do not impair daily functioning, while some non-disorders do. People with psychopathy can function in society and even thrive professionally despite serious antisocial traits, while parents of newborns may temporarily "fail to function adequately" without being mentally ill. This is important because it shows the definition over- and under-includes, limiting its diagnostic precision.

Limitation of deviation from social/cultural norms — culture-bound and historically variable. A serious limitation of the social-norms definition is that norms vary across cultures and time. Homosexuality was classified as a mental disorder in the DSM until 1973; hearing voices is pathologised in the West but valued spiritually in some African societies. This is important because it shows the definition risks **cultural bias** (an imposed etic) and can be used to pathologise minority or non-conforming groups. This is also an issues-and-debates concern — definitions of abnormality can have serious social consequences for the groups labelled as "abnormal".

Limitation of ideal mental health — sets an unrealistic standard. A limitation of Jahoda's ideal-mental-health definition is that her six criteria are very demanding — few people would meet all six all of the time. This is important because if "normality" is defined as a state most people never achieve, almost everyone is technically "abnormal", which undermines the practical usefulness of the definition. The criteria also reflect Western, individualist values (e.g. autonomy, self-actualisation), which limits their cultural validity.

Conclusion. No single definition adequately captures abnormality. A combined approach — using statistical and functional criteria alongside cultural awareness and ideal-mental-health standards — provides the most defensible basis for clinical assessment, while requiring careful attention to cultural and historical context.

2 Characteristics of Phobias, Depression and OCD

The AQA specification requires knowledge of the **behavioural, emotional and cognitive** characteristics of three disorders: **phobias**, **depression** and **obsessive-compulsive disorder (OCD)**.

Phobias

> **PHOBIA**
>
> A **phobia** is an *irrational*, persistent fear of a specific object, situation or activity that is *out of proportion* to the actual threat posed. Phobias cause significant distress and interfere with daily life. The DSM-5 distinguishes **specific phobias** (e.g. spiders, heights), **social anxiety disorder** and **agoraphobia**.

Domain	Characteristics
Behavioural	**Avoidance** — actively avoiding the phobic stimulus, often disrupting daily life. **Endurance** — sometimes the person remains in the presence of the stimulus, experiencing high anxiety. **Panic response** — crying, screaming, running away or freezing.
Emotional	**Anxiety** — an unpleasant high-arousal state that prevents the person from relaxing. **Fear** — an immediate, intense and unpleasant response. The emotional response is *disproportionate* to the actual danger.
Cognitive	**Selective attention** to the phobic stimulus — the person finds it hard to look away. **Irrational beliefs** about the stimulus (e.g. "all spiders are deadly"). **Cognitive distortions** — the stimulus may be perceived as larger, faster or more threatening than it is.

Depression

> **DEPRESSION**
>
> A **mood disorder** characterised by low mood, loss of interest in activities, and behavioural and cognitive changes lasting at least two weeks. The DSM-5 distinguishes **major depressive disorder**, **persistent depressive disorder (dysthymia)** and **premenstrual dysphoric disorder** among others.

Domain	Characteristics
Behavioural	**Reduced activity** levels — fatigue, withdrawal from social and pleasurable activities. **Disrupted sleep and eating** — insomnia or hypersomnia; significant weight loss or gain. **Aggression and self-harm** — depression can produce irritability, verbal or physical aggression and, in severe cases, self-harm.
Emotional	**Low mood** — persistent sadness or emptiness. **Loss of interest or pleasure (anhedonia)** in formerly enjoyable activities. **Anger** — often directed inward as self-loathing, sometimes outward as irritability.
Cognitive	**Poor concentration** and difficulty making decisions. **Absolutist (black-and-white) thinking** — situations are seen as wholly good or wholly bad. **Negative self-schemas** — pervasive negative thoughts about the self, the world and the future (see Beck's negative triad in Section 5).

Obsessive-Compulsive Disorder (OCD)

> **OCD**
> OCD is an anxiety-related disorder characterised by recurrent, intrusive **obsessions** (thoughts) and repetitive **compulsions** (behaviours) that the person feels driven to perform to reduce anxiety. The cycle of obsessions → anxiety → compulsions → temporary relief is a defining feature.

Domain	Characteristics
Behavioural	**Compulsions** — repetitive, ritualistic behaviours (e.g. hand-washing, checking, counting) performed to neutralise an obsession. **Avoidance** of situations that trigger obsessions (e.g. avoiding public toilets to avoid germs).
Emotional	**Anxiety and distress** — the obsessions cause severe and persistent anxiety, which the compulsions only temporarily relieve. **Depression** — accompanying low mood is common, partly because the person recognises that their behaviour is irrational. **Guilt and disgust** are also frequently reported.
Cognitive	**Obsessions** — recurrent, intrusive, unwanted thoughts (e.g. contamination, harm to others, symmetry). **Awareness of irrationality** — the person recognises their thoughts and behaviours are excessive but cannot easily stop them. **Hypervigilance** — selective attention to potential triggers.

> **EXAM TIP — THE "THREE DOMAINS"**
> For 4–6 mark "Outline the characteristics of X" questions, structure the answer in three clearly labelled paragraphs: **Behavioural**, **Emotional**, **Cognitive**. Give one or two specific examples for each domain. Generic answers ("they feel sad") cap at the lower bands.

3 The Behavioural Approach to Explaining Phobias

Mowrer's (1947) two-process model explains how phobias are *acquired* and *maintained*. It combines two behaviourist mechanisms: **classical conditioning** (how phobias are learned) and **operant conditioning** (how they are maintained over time).

1. Acquisition — Classical Conditioning

A phobia is **acquired** when a previously neutral stimulus becomes associated with something that already produces a fear response. After repeated pairing, the previously neutral stimulus alone triggers fear.

Stage	Stimulus	Response
Before conditioning	Loud noise (UCS)	Fear (UCR)
Before conditioning	White rat (NS)	No fear response
During conditioning	White rat + Loud noise (NS + UCS)	Fear (UCR)
After conditioning	White rat (CS)	Fear (CR) — phobia acquired

Classic study — Watson and Rayner (1920): Little Albert

Procedure: 9-month-old "Little Albert" was shown a white rat (NS) which initially produced no fear. The researchers then paired the rat with a sudden loud noise (UCS) produced by striking a steel bar behind Albert's head. After several pairings, Albert showed fear at the sight of the rat alone — the rat had become a conditioned stimulus.

Generalisation: Albert's fear extended to other furry stimuli — a rabbit, a fur coat and even a Santa Claus mask. This demonstrates **stimulus generalisation**, an important feature of phobias.

2. Maintenance — Operant Conditioning

Once acquired, the phobia is **maintained** through **negative reinforcement**. When the phobic person *avoids* the feared stimulus, the unpleasant anxiety is reduced or eliminated — and this reduction in anxiety *rewards* the avoidance behaviour, making it more likely to be repeated. The phobia therefore strengthens over time rather than fading.

> **EXAMPLE — FEAR OF DOGS**
>
> A child is bitten by a dog (UCS) and experiences fear (UCR). The dog becomes a CS that produces fear (CR). The child then avoids dogs whenever possible — crossing the street, refusing to visit friends with pets. Every avoidance reduces anxiety (negative reinforcement) and strengthens the avoidance habit. The phobia persists for years despite no further negative experiences with dogs.

Evaluation

Strength — supporting evidence from Little Albert (Watson and Rayner 1920). A major strength of the two-process model is direct experimental evidence that phobias can be acquired through classical conditioning. Watson and Rayner's Little Albert study showed that a previously neutral stimulus (a white rat) could produce a conditioned fear response after repeated pairing with a loud noise. This is important because it provides a controlled demonstration of the exact mechanism Mowrer proposed for phobia acquisition. This therefore strengthens the validity of the classical-conditioning element of the two-process model.

Strength — real-world success of behavioural therapies (application). A further strength is the model's applied value. Behavioural therapies derived directly from the model — systematic desensitisation and flooding (Section 4) — have proven effectiveness in treating specific phobias, with success rates often exceeding 75% (McGrath et al. 1990). This is important because the therapeutic success of treatments based on the model is strong indirect evidence that the underlying mechanism is real. This strengthens the model's validity through the convergence of theory and practice.

Limitation — not all phobias follow a traumatic experience. A significant limitation is that many people with phobias cannot recall a traumatic conditioning event. DiNardo et al. (1988) found that some people with dog phobias had never had a frightening experience with dogs, and conversely many people who experience traumatic dog encounters never develop phobias. This is important because if phobias can develop without classical conditioning — and conditioning can occur without producing a phobia — the two-process model is incomplete. This weakens the explanation as a comprehensive account.

Limitation — biological preparedness (Seligman 1971). A further limitation is the existence of **biological preparedness**. Seligman (1971) noted that humans develop phobias of evolutionarily threatening stimuli (snakes, spiders, heights) far more easily than of evolutionarily neutral stimuli (cars, electrical sockets) — even though the latter are far more dangerous in modern life. This is important because the two-process model predicts conditioning should work equally for any stimulus, but it does not. This supports an interactionist **nature–nurture** view: classical conditioning operates on top of evolutionarily prepared fear responses.

Limitation — ignores cognitive factors. A further limitation is that the two-process model is purely behavioural and ignores the cognitive aspects of phobias. Phobic people typically hold irrational beliefs ("all spiders are deadly") that go beyond conditioned associations. This is important because cognitive therapies that target these beliefs — particularly cognitive-behavioural variants of treatment — improve treatment outcomes. This limits the model's completeness and explains why purely behavioural treatments are now often combined with cognitive elements.

Conclusion. Overall, the two-process model captures something important about how phobias are acquired and maintained, and underpins effective treatments. However, biological preparedness, the existence of phobias without traumatic onset and the role of cognitive factors mean the model is best treated as an important *part* of the explanation rather than a complete account.

4 The Behavioural Approach to Treating Phobias

Two behavioural treatments are specified by the AQA syllabus: **systematic desensitisation (SD)** and **flooding**. Both rely on the principle that conditioned fear responses can be unlearned — but they use very different mechanisms.

Systematic Desensitisation (Wolpe 1958)

Systematic desensitisation uses **counterconditioning** — replacing the fear response with a relaxation response that is incompatible with anxiety. Because a person cannot be both relaxed and anxious at the same time (a principle called **reciprocal inhibition**), gradual exposure paired with deep relaxation extinguishes the phobic response.

The three stages

1. **Relaxation training.** The therapist teaches deep relaxation techniques — progressive muscle relaxation, controlled breathing, visualisation.
2. **Constructing an anxiety hierarchy.** The therapist and client jointly construct a graded list of feared situations, from least anxiety-provoking (e.g. "thinking about a spider") to most anxiety-provoking (e.g. "holding a tarantula").
3. **Graded exposure.** The client works up the hierarchy step by step. At each level, the client remains in the situation while practising relaxation until the anxiety subsides. Only when one level is mastered does the client progress to the next.

> **EXAMPLE ANXIETY HIERARCHY — FEAR OF SPIDERS**
>
> **1. Lowest:** Looking at a cartoon picture of a spider.
>
> **2.** Looking at a photograph of a real spider.
>
> **3.** Watching a video of a spider walking.
>
> **4.** Being in the same room as a sealed jar containing a spider.
>
> **5.** Standing within one metre of an uncovered spider in a tray.
>
> **6. Highest:** Allowing the spider to walk on the participant's hand.

Flooding

Flooding involves **immediate, prolonged exposure** to the phobic stimulus with no opportunity to escape. Because the person cannot avoid the stimulus, they cannot reinforce avoidance (no negative reinforcement) and the fear response eventually extinguishes — a process called **extinction**. The person learns that the feared stimulus is harmless because nothing bad happens during prolonged exposure.

- **Method:** A single, long session (often 2–3 hours) — much faster than SD's multiple sessions.
- **Setting:** Can be *in vivo* (real-life exposure) or *in vitro* (imagined, virtual reality).
- **Informed consent** is essential because the experience is highly distressing.

Evaluation

Strength of SD — strong effectiveness evidence (Gilroy et al. 2003). A major strength of SD is robust effectiveness evidence. Gilroy et al. (2003) followed 42 people with spider phobia treated with SD over three 45-minute sessions; at 3 months and 33 months, they showed significantly less fear than a control group treated with relaxation alone. This is important because it shows SD's benefits last well beyond the immediate treatment period, justifying its routine use in clinical practice. This strengthens the case for SD as an evidence-based treatment for specific phobias.

Strength of SD — suitable for diverse patients (application). A further strength is SD's broad applicability. Because it is gradual and gives patients control over the pace, SD is suitable for people who would find flooding too distressing — including children, people with learning disabilities and those with severe anxiety. This is important because the alternative (flooding) is unsuitable for many of the patients who most need treatment. This widens SD's applied value and supports its preferred status in clinical guidelines.

Strength of flooding — fast and cost-effective. A genuine strength of flooding is efficiency. A single 2–3 hour session can achieve what SD takes weeks to deliver, making flooding considerably cheaper for the NHS and providing rapid relief for the patient. This is important because flooding's economic advantage matters in healthcare systems with limited resources. This strengthens flooding's case as a cost-effective treatment, particularly for highly motivated patients.

Limitation of flooding — traumatic for patients (ethical). However, a major limitation of flooding is the trauma it inflicts. Patients experience intense fear during the exposure period and many cannot complete the full session — high **attrition rates** are a known issue. This is important because if patients drop out before extinction occurs, the phobia may actually be strengthened (the partial avoidance is negatively reinforced). This is also an ethical limitation: the procedure causes significant short-term distress, requiring careful informed consent and clinical safeguards.

Limitation of both — symptom substitution and complex phobias. A further limitation of both treatments is that they may not address the underlying cause of the phobia. Psychodynamic critics argue that behavioural treatments produce **symptom substitution** — removing one symptom causes another to appear. This is more relevant for complex social phobias and agoraphobias, where cognitive and developmental factors play a larger role than simple conditioning. This is important because evidence for symptom substitution is weak for specific phobias but stronger for more complex disorders, suggesting behavioural treatments work best for simple phobias.

Limitation — limited effectiveness with social phobias. Both SD and flooding are most effective for *specific* phobias (e.g. spiders, heights, dogs) where the feared stimulus is concrete and identifiable. They are less effective for **social phobia** and **agoraphobia**, which involve more complex cognitive and interpersonal elements. This limits the applicability of behavioural treatments and supports the use of cognitive-behavioural variants for these conditions.

Conclusion. SD and flooding are both effective evidence-based treatments for specific phobias, with SD generally preferred for vulnerable patients and flooding offering cost-effective rapid treatment. Both are most powerful when the phobia is concrete and circumscribed; complex phobias usually require cognitive-behavioural adaptations.

5 The Cognitive Approach to Explaining Depression

The cognitive approach explains depression as the product of **faulty thinking**. Two influential cognitive theories are specified: **Beck's cognitive triad** and **Ellis's ABC model**.

Beck's Cognitive Theory of Depression (1967)

Beck proposed that depressed people develop three faulty thinking patterns that combine to produce and maintain low mood: **(1) cognitive bias**, **(2) negative self-schemas**, and **(3) the negative triad**.

(1) Cognitive biases (faulty information processing)

Depressed people systematically distort information in negative ways:

- **Selective abstraction** — focusing on a single negative detail and ignoring positives ("everyone laughed at one joke I made — I'm useless").
- **Overgeneralisation** — drawing sweeping conclusions from one event ("I failed one test, so I'll fail at everything").
- **Magnification and minimisation** — exaggerating negatives and minimising positives.
- **Personalisation** — attributing negative events to oneself when they were not personal.

(2) Negative self-schemas

A **self-schema** is a mental framework about the self. Depressed people develop **negative self-schemas** through early childhood experiences (rejection, criticism, loss), and these schemas filter all incoming information in a self-defeating way.

(3) The negative triad

The depressed person holds simultaneous negative views of:

Domain	Negative view	Example
The self	"I am worthless / unlovable / a failure."	"I can't do anything right."
The world	"The world is unfair / hostile / against me."	"Everyone is judging me."
The future	"Things will never get better; there is no hope."	"I'll be alone forever."

Ellis's ABC Model (1962)

Ellis proposed that depression is not caused by activating events themselves but by the *irrational beliefs* we hold about them. He called these "**mustabatory thinking**" — beliefs that contain absolute demands like "must", "should" and "ought".

Stage	What it is	Example
A — Activating event	An external event that triggers a response.	"I didn't get the job I applied for."
B — Belief	The interpretation of the event. Can be *rational* ("This is disappointing — I'll try again") or *irrational* ("I'm a complete failure; I'll never get a job").	Irrational belief: "I should have got that job — I'm worthless because I didn't."
C — Consequence	The emotional and behavioural outcome — healthy or unhealthy.	Healthy: disappointment, motivation to try again. Unhealthy: depression, withdrawal.

Ellis later added **D — Disputing** (challenging irrational beliefs) and **E — Effects** (the new healthier consequences) as treatment stages.

> **THREE COMMON IRRATIONAL BELIEFS (ELLIS)**
> **"I must do well and win the approval of others or I am worthless."** · **"Other people must treat me fairly and kindly or they are bad."** · **"The world must give me what I want or life is unbearable."** These absolute demands are at the heart of depressive thinking.

Evaluation

Strength of Beck — strong supporting evidence (Grazioli and Terry 2000). A major strength of Beck's theory is supporting evidence. Grazioli and Terry (2000) assessed 65 pregnant women for cognitive vulnerability before and after birth and found that those identified as high in cognitive vulnerability were more likely to develop post-natal depression. This is important because it shows negative thinking patterns *precede* depression rather than being merely a symptom, supporting Beck's causal claim. This strengthens the validity of the negative-triad model as an explanation rather than a description of depression.

Strength — practical applications (CBT). A further strength is the model's practical value. Beck's theory directly led to **cognitive behaviour therapy (CBT)** — see Section 6 — now one of the most widely used evidence-based treatments for depression worldwide. This is important because the therapeutic success of CBT provides indirect evidence that the underlying cognitive mechanisms Beck identified are real and modifiable. This strengthens the theory's overall credibility and applied value.

Limitation — doesn't explain all aspects of depression. A significant limitation is that Beck's theory does not explain every feature of depression. Some patients show severe biological symptoms (extreme fatigue, sleep disruption, somatic pain) that cannot easily be reduced to cognitive distortion. Others experience hallucinations or delusions of total worthlessness that go beyond the negative triad. This is important because it shows the model captures the cognitive but not the full clinical picture of depression. This limits its completeness as an explanation.

Strength of Ellis — successful in some types of depression. A genuine strength of Ellis's model is its therapeutic success, particularly in cases where depression is triggered by clearly identifiable life events (job loss, relationship breakdown). The ABC model gives clients a clear framework for identifying irrational beliefs and challenging them. This is important because applied effectiveness is strong indirect evidence the model captures something real.

Limitation of Ellis — partial explanation (reactive vs endogenous depression). A limitation is that Ellis's model best fits **reactive** depression (triggered by clear events) but struggles with **endogenous** depression that emerges without an obvious activating event. This is important because if no clear "A" can be identified, the ABC framework offers little guidance. This restricts the model's range and supports a multi-factorial account in which biological and developmental factors also matter.

Limitation — direction of causality (Lewinsohn 1973). A further limitation, raised by Lewinsohn (1973), is that the link between negative thinking and depression may be reversed — depression may *cause* negative thinking rather than the other way round. This is important because the cognitive explanations claim a causal direction (cognition → depression) that cannot be established from correlational data alone. Grazioli and Terry's prospective design partially addresses this, but the question of direction remains open.

Limitation — blames the patient (ethical / socially sensitive). A further limitation is that cognitive explanations risk attributing the cause of depression to the patient's own faulty thinking, potentially adding guilt and self-blame to existing distress. This is important because how an illness is explained shapes how the patient relates to it. The biopsychosocial model — combining cognitive, biological and social factors — avoids this blame while still using cognitive interventions where appropriate.

Conclusion. Both Beck and Ellis provide useful frameworks for understanding the cognitive components of depression, and their applied derivatives (CBT, REBT) remain among the most effective evidence-based treatments. Their key limitations — failure to explain biological symptoms, restricted range for endogenous depression and uncertainty about causal direction — mean they are best understood as part of a multi-factorial biopsychosocial account.

6 The Cognitive Approach to Treating Depression (CBT)

Cognitive Behaviour Therapy (CBT) is the leading evidence-based treatment for depression. It combines the cognitive element (identifying and challenging negative thoughts) with the behavioural element (changing behaviour to test new beliefs). CBT was developed by Beck (1960s) and Ellis (REBT — Rational Emotive Behaviour Therapy).

> **CBT — AQA DEFINITION**
>
> CBT is a type of therapy based on the principles of the cognitive and behaviourist approaches. It aims to identify negative and irrational thoughts and beliefs and to challenge these and replace them with more rational and positive thoughts and beliefs. It is a directive approach to therapy that involves the client as an active figure in the process.

The Process

1. **Assessment.** The therapist and client identify the negative thoughts, beliefs and core schemas associated with the depression.
2. **Goal-setting.** The client and therapist agree clear, measurable goals (e.g. "reduce daily negative thoughts about work by 50% in 8 weeks").
3. **Challenging irrational thoughts.** Using Ellis's **disputing** techniques and Beck's **thought-recording**, the therapist helps the client question the evidence for their negative beliefs.
4. **Behavioural activation.** The client is encouraged to schedule pleasurable, achievement-oriented activities — counteracting depressive withdrawal and providing evidence to disprove negative beliefs.
5. **Homework.** Between sessions the client completes thought diaries, behavioural experiments and graded tasks. This integrates therapy into everyday life.

Challenging Irrational Thoughts — Ellis's Disputes

Dispute type	Question	Example
Logical	"Does this thought logically follow from the evidence?"	"Does failing one exam logically mean you'll fail at everything?"
Empirical	"Where is the evidence that this is true?"	"What evidence do you have that no one likes you?"
Pragmatic	"How useful is it to hold this belief?"	"Even if it were true that you 'must' be perfect, how does that belief help you?"

Behavioural Activation

Behavioural activation is a core CBT technique. Depressed people often withdraw from pleasurable activities, which reinforces low mood. CBT counters this by scheduling enjoyable and achievement-oriented activities, generating positive experiences that disprove negative beliefs about the self and the future. This is the behaviourist side of CBT — using changes in behaviour to drive changes in thinking.

Evaluation

Strength — strong effectiveness evidence (March et al. 2007). A major strength of CBT is robust effectiveness evidence. March et al. (2007) compared CBT, antidepressants and a combined treatment in 327 adolescents with depression and found 81% in both the CBT and antidepressant conditions improved, rising to 86% in the combined-treatment group. This is important because it shows CBT is at least as effective as drugs and produces synergistic benefits when combined. This strengthens the case for CBT as a first-line, evidence-based treatment for depression.

Strength — addresses underlying causes (durability). A further strength is that CBT teaches durable cognitive skills. Unlike drug therapy, which addresses symptoms without changing thinking patterns, CBT teaches patients to identify and challenge negative thoughts themselves — skills that persist after therapy ends. This is important because it reduces relapse rates compared with drug-only treatment. This strengthens CBT's long-term applied value.

Limitation — may not be suitable for severe cases. A significant limitation is that CBT requires active engagement, motivation and a degree of cognitive functioning. Patients with severe depression may lack the energy, concentration or motivation to participate fully — many cannot complete homework or attend regular sessions. This is important because the patients who most need treatment may not benefit most from CBT. This is why severe depression is often treated initially with drugs to stabilise mood, followed by CBT once the patient can engage.

Limitation — overemphasis on the patient as cause (ethical / socially sensitive). A further limitation is that CBT can imply that depression is caused by the patient's faulty thinking, ignoring the role of genuine life circumstances. A patient experiencing poverty, abuse or chronic illness has rational reasons to feel hopeless, and challenging those thoughts may feel invalidating. This is important because over-applied CBT risks blaming the patient for distress that has real external causes. This is a socially sensitive limitation and supports the use of CBT alongside (rather than instead of) social interventions where appropriate.

Limitation — high attrition and therapist effects. A further limitation is high **dropout rates** (around 30% in many trials) and substantial variation in effectiveness depending on therapist skill. This is important because trial-level success rates conceal substantial individual variation in real-world clinical practice. This limits the generalisability of effectiveness figures from controlled trials to routine NHS services.

Application — economic value. A clear strength of CBT is its measurable economic value. NHS guidelines (NICE) recommend CBT as a first-line treatment for moderate depression. By reducing reliance on long-term medication and shortening treatment courses, CBT reduces NHS costs and improves return-to-work rates. This is important because applied economic benefits are themselves evidence the underlying treatment mechanism is real. The Department of Health's IAPT programme (Improving Access to Psychological Therapies) is a direct application of CBT research at national scale.

Conclusion. CBT is one of the most extensively evidenced psychological treatments and a cornerstone of NHS mental-health provision. Limitations regarding severe depression, social context and dropout are real but well-known, and best addressed by combining CBT with appropriate biological and social interventions in a stepped-care model.

7 The Biological Approach to Explaining OCD

The biological approach explains OCD as the product of **genetic vulnerability** and **abnormalities in brain structure and neurochemistry**. Both elements are required by the AQA spec.

Genetic Explanations

OCD runs in families, suggesting a genetic component. **Lewis (1936)** found that 37% of OCD patients had parents with OCD and 21% had siblings with OCD — much higher than the population rate of around 2%. Modern twin studies provide more precise estimates.

Candidate genes

Two genes are most strongly implicated:

- **SERT (5-HTT)** — affects the transport of serotonin across synapses. A variant of this gene has been linked to OCD and is also implicated in depression — explaining the high **comorbidity** between the two disorders.
- **COMT** — affects the breakdown of dopamine. A variant produces higher dopamine levels, which has been linked to OCD.

Polygenic — not monogenic

OCD is **polygenic**, meaning it is influenced by many genes acting together rather than a single "OCD gene". Taylor (2013) found that up to **230 genes** may be involved. This is consistent with the diverse symptom profile across patients.

Twin studies

Nestadt et al. (2010) reviewed twin studies and concluded that 68% of variance in OCD is attributable to genetic factors. MZ twin concordance rates are roughly twice those of DZ twins.

Neural Explanations

Two main neural mechanisms are implicated in OCD:

(1) Neurotransmitter abnormalities

OCD is associated with **low serotonin levels**, which disrupt mood regulation. Drugs that increase serotonin (SSRIs — see Section 8) reduce OCD symptoms, providing indirect evidence for the role of serotonin. Dopamine has also been implicated, with elevated dopamine activity linked to the compulsive element of OCD.

(2) Abnormal brain structures — the "worry circuit"

OCD is associated with abnormal functioning in a brain network sometimes called the **worry circuit**:

- **Orbitofrontal cortex (OFC)** — sends "worry" signals when something is wrong (e.g. "your hands are dirty").
- **Caudate nucleus** (part of the basal ganglia) — normally suppresses minor worry signals. In OCD it is under-active, allowing trivial worries to flood through to the next region.
- **Thalamus** — relays signals back to the OFC, creating a self-reinforcing loop in which the worry intensifies rather than fading.

Brain-imaging studies (PET, fMRI) consistently show increased activity in this circuit in OCD patients, particularly when their obsessions are triggered.

Evaluation

Strength — strong family and twin evidence (Nestadt et al. 2010). A major strength of the genetic explanation is converging evidence from family and twin studies. Nestadt et al.'s (2010) review of twin studies concluded that 68% of variance in OCD is attributable to genetic factors, while Lewis (1936) found 37% of patients had a parent with OCD. This is important because the convergence of independent designs across different decades makes the genetic contribution highly robust. This strengthens the validity of the genetic explanation as a real part of OCD's aetiology.

Strength — neural evidence supported by drug effectiveness. A further strength is that the neural explanation is supported by the success of drug treatments. SSRIs, which increase available serotonin, significantly reduce OCD symptoms (see Section 8). This is important because if increasing serotonin reduces symptoms, low serotonin is likely to be part of the cause. This is converging evidence from a different methodology — pharmacological intervention — that strengthens the validity of the neurotransmitter explanation.

Limitation — concordance rates are not 100% (environment matters). A significant limitation is that MZ twin concordance rates for OCD, while high, are not 100% — meaning genes alone cannot account for OCD. This is important because if OCD were purely genetic, identical twins should always share the disorder. This supports an **interactionist** (diathesis–stress) view in which genetic vulnerability requires environmental triggers (e.g. trauma, life stress) to produce OCD.

Limitation — neural correlates may be effects, not causes. A further limitation is that brain-imaging findings showing OFC and caudate abnormalities in OCD patients are *correlational*. The abnormal activity could be a *consequence* of OCD rather than its cause — repeatedly performing compulsive behaviours could reshape brain function (neuroplasticity) rather than the abnormal brain causing the behaviour. This is important because correlation cannot establish causal direction, weakening the causal claim of the neural explanation.

Limitation — biological reductionism. A further limitation is that the biological explanation is **reductionist**. It reduces a complex disorder involving thought content (the specific content of obsessions), personal history, family context and culture to genes and neurotransmitters. This is important because the specific *content* of obsessions — fears of contamination, harm or symmetry — cannot be explained at the neural level alone. This limits the explanation's completeness and supports the use of complementary cognitive and behavioural accounts.

Limitation — diathesis–stress alternative. A more balanced alternative is the diathesis–stress model: genetic vulnerability provides the diathesis, but environmental stressors (trauma, life transitions) are required to trigger OCD. Cromer et al. (2007) found that over half of OCD patients in their sample had experienced a traumatic event before the onset of symptoms. This is important because it shows the pure biological account is incomplete — genes set risk, but experience determines who develops the disorder.

Conclusion. The biological approach captures a real and substantial contribution to OCD — particularly through robust twin-study evidence and converging pharmacological data. However, the moderate (not 100%) heritability, the correlational nature of brain-imaging findings, and reductionism mean a diathesis–stress model — biology plus environment — provides the most defensible account.

8 The Biological Approach to Treating OCD (Drug Therapy)

The biological approach treats OCD primarily through drugs that adjust brain chemistry. The first-line treatment is **SSRIs**, with **tricyclics** and **SNRIs** used when SSRIs are ineffective.

How SSRIs Work

SSRIs (Selective Serotonin Reuptake Inhibitors) — e.g. **fluoxetine (Prozac)** — increase the availability of serotonin in the synaptic gap by blocking its reuptake into the presynaptic neuron. More serotonin remains in the synapse, available to bind to postsynaptic receptors and normalise the disrupted serotonin system in the worry circuit.

Stage	What happens
1. Presynaptic release	Serotonin is released into the synaptic gap.
2. Normal reuptake	Most serotonin would normally be reabsorbed (reuptake) by the presynaptic neuron and broken down.
3. SSRI action	The SSRI selectively blocks the reuptake transporters for serotonin.
4. Net effect	Serotonin remains in the synapse longer, increasing postsynaptic activation and improving mood/anxiety regulation.

Typical dose: 20 mg of fluoxetine daily, with effects taking 3–4 months to reach full clinical impact. SSRIs are often combined with CBT for best results.

Alternative Drug Treatments

- **Tricyclics (e.g. clomipramine)** — older drugs that affect both serotonin and noradrenaline. Equally effective for OCD but with more severe side effects (dry mouth, weight gain, cardiac effects), so reserved for patients who do not respond to SSRIs.
- **SNRIs (e.g. venlafaxine)** — affect both serotonin and noradrenaline. Used as a second-line treatment when SSRIs fail.
- **Benzodiazepines** — sometimes used short-term for severe anxiety, but not a treatment for OCD itself due to dependence risk.

Evaluation

Strength — strong effectiveness evidence (Soomro et al. 2009). A major strength of drug therapy is robust effectiveness evidence. Soomro et al.'s (2009) review of 17 randomised controlled trials comparing SSRIs with placebo found significantly better outcomes for SSRIs — typically around 70% of patients show some symptom improvement, with around half experiencing a major reduction in symptoms. This is important because the systematic review combines data from many high-quality trials, giving robust evidence of effectiveness. This strengthens the case for SSRIs as a first-line, evidence-based treatment for OCD.

Strength — cost-effective and accessible. A further strength is the economic and practical advantages of drug therapy over psychological therapies. Drugs are cheap to produce, easy to prescribe and require minimal practitioner time. This is important because for healthcare systems with limited mental-health resources, drug therapy is far easier to scale than CBT (which requires trained therapists and weekly appointments). This strengthens the applied case for drug therapy as a routine treatment, particularly when CBT is unavailable.

Limitation — significant side effects. A significant limitation is that SSRIs cause meaningful side effects. Common side effects include nausea, headache, insomnia, sexual dysfunction and emotional blunting. Around 25% of patients experience side effects severe enough to discontinue treatment. This is important because if patients stop taking the medication, the treatment cannot work. This is more serious for tricyclics, which have severe side effects in around 1 in 10 patients. This limits the long-term effectiveness and acceptability of drug therapy.

Limitation — does not address underlying causes (symptom suppression). A further limitation is that drug therapy treats *symptoms* rather than causes. Once the medication is stopped, symptoms typically return — relapse rates are high (around 50% within 6 months of discontinuation). This is important because long-term reliance on medication can be costly, carries cumulative side-effect risks, and does not teach the patient skills to manage their condition. This limits drug therapy as a stand-alone solution and supports its combined use with CBT.

Limitation — publication bias. A further limitation is that the published evidence may overstate effectiveness due to **publication bias**. Pharmaceutical-company funded trials are more likely to report positive results, and trials with null or negative results are less likely to be published (the file-drawer problem). Goldacre (2012) argued that this systematically inflates apparent drug effectiveness. This is important because clinical decisions are based on the published evidence, which may be a biased subset of all evidence. This limits confidence in published effectiveness figures.

Limitation — ethical concerns about over-prescription. A further limitation is the ethical concern of over-prescription. Drug therapy is cheap and easy to prescribe, which can lead to GPs offering drugs as a default rather than referring patients for psychological therapies. This is important because it can mean patients receive symptom suppression when CBT might address the underlying issue. This is an ethical limitation about clinical decision-making rather than the drugs themselves.

Application — economic value. Despite these limitations, drug therapy has substantial economic value. SSRIs reduce healthcare contact, lost work time and the secondary costs of untreated OCD (loss of relationships, employment difficulties, mental-health complications). This is important because applied economic benefits demonstrate the treatment has measurable real-world value, supporting its continued use as part of a stepped-care model alongside psychological interventions.

Conclusion. Drug therapy — particularly SSRIs — is an effective, well-evidenced and cost-effective first-line treatment for OCD. Its main limitations (side effects, symptom rather than cause focus, high relapse on discontinuation) are best addressed by combining drug therapy with CBT in a comprehensive treatment plan —

the approach now recommended by NICE guidelines.

These revision notes were prepared for Simply Psychology and cover spec section 4.1.4 of the AQA Psychology 2025 specification (A-level only, Paper 1 — formerly titled *Psychopathology*). Definitions in the field of mental health and the AQA terminology for phobias, depression, OCD, the two-process model, CBT and drug therapy follow AQA's official 2025 *Subject specific vocabulary*. For deeper coverage of any topic, see simplypsychology.org/abnormal-psychology.html.