SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Addiction

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.3.10 · A-level Paper 3

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

Contents

2025 spec changes: the **theory of planned behaviour has been removed** — **Prochaska's six-stage model** is now the only behaviour-change model on the specification. Risk factors are now worded as "**social influences**" (covering stress, family and peers), and the topic refers to "**nicotine addiction**" rather than "smoking behaviour". Past-paper questions on the theory of planned behaviour are obsolete.

AQA 2025 SPECIFICATION — ADDICTION (4.3.10)

- **Describing addiction**: physical and psychological dependence, tolerance and withdrawal syndrome.
- **Risk factors**: genetic vulnerability, personality and social influences.
- **Nicotine addiction**: brain neurochemistry (dopamine); learning theory (cue reactivity).
- **Gambling addiction**: learning theory (partial and variable reinforcement); cognitive theory (cognitive bias).
- **Reducing addiction**: drug therapy; behavioural interventions (aversion therapy, covert sensitisation); CBT.
- **Behaviour change**: Prochaska's six-stage model.

1 Describing Addiction

KEY FEATURES OF ADDICTION

- **Physical dependence** — the body has adapted to a substance so that it is needed to function normally; stopping produces a physical withdrawal reaction.
- **Psychological dependence** — a compulsion to continue because of the perceived pleasure or relief from anxiety it provides.
- **Tolerance** — through neuroadaptation, more of the substance is needed to achieve the same effect.
- **Withdrawal syndrome** — the set of unpleasant physical and psychological symptoms (e.g. tremors, sweating, anxiety, nausea) experienced when a dependent person stops or reduces use.

Evaluation

A strength of these concepts is that they provide clear, measurable criteria for diagnosing addiction. Tolerance and withdrawal can be observed and even measured physiologically, which allows clinicians to identify addiction reliably and consistently. This objectivity is valuable because a shared definition underpins research and ensures that people receive an appropriate diagnosis and treatment, giving the concepts real practical utility.

However, a limitation is that the distinction between physical and psychological dependence is not clear-cut. Withdrawal from substances such as nicotine involves both bodily symptoms and powerful cravings, so the two forms of dependence overlap rather than being separate. This blurring matters because focusing only on *physical* dependence risks underestimating behavioural addictions such as gambling, which produce little physical withdrawal yet are clearly addictive — challenging a purely physiological definition.

A further point is that defining addiction is socially sensitive and value-laden. Labelling a behaviour an "addiction" frames it as a medical disorder rather than a choice, which affects whether sufferers are treated with sympathy and offered help or blamed for a lack of willpower. Because these definitions influence funding, treatment access and personal responsibility, they have real-world consequences and must be applied carefully — illustrating the **free will versus determinism** debate.

2 Risk Factors in the Development of Addiction

- **Genetic vulnerability** — addiction runs in families and shows higher concordance in MZ than DZ twins. Candidate genes affect the **dopamine reward system** (e.g. fewer D2 receptors, so a person needs more stimulation to feel reward).
- **Personality** — traits such as **impulsivity** and **sensation-seeking**, and high neuroticism, are linked to greater vulnerability; antisocial personality is also associated with addiction.
- **Social influences** — **stress**, **family** attitudes and behaviour, and **peers** (peer pressure and social norms) all raise the risk of developing addiction. *(In the 2025 spec these are unified as "social influences".)*

Evaluation

A strength of the genetic risk factor is supporting evidence from twin and family studies. Addiction consistently shows higher concordance in identical than non-identical twins, and relatives of addicts are at greater risk. Because this pattern holds across many studies, it provides solid evidence that vulnerability to addiction is partly inherited, supporting the role of genetic factors and pointing to differences in the dopamine reward system.

However, a major limitation is that no single risk factor is sufficient — the factors interact. Genetic vulnerability does not guarantee addiction: many people with a family history never develop one, and others with no genetic risk do. This is best understood as a **diathesis-stress / nature–nurture** interaction, in which an inherited predisposition is only "switched on" by environmental triggers such as peer pressure or stress, so a single-factor account is incomplete.

A counterpoint concerning the personality risk factor is the problem of cause and effect. Although impulsivity and sensation-seeking correlate with addiction, it is unclear whether these traits *cause* addiction or whether long-term substance use *changes* personality and impulse control. Because most evidence is correlational, the direction of the relationship cannot be established, weakening the claim that personality is a true cause.

On the other hand, risk-factor research has valuable real-world application. Identifying who is most vulnerable — for example, impulsive teenagers in heavy-using peer groups — allows prevention efforts and early intervention to be targeted where they are most needed. Reducing the number of people who go on to develop addictions would bring major health benefits and substantial economic savings, given the enormous cost of addiction to health services and the wider economy.

3 Explaining Nicotine Addiction

Brain Neurochemistry

Nicotine binds to **nicotinic acetylcholine receptors**, triggering the release of **dopamine** in the mesolimbic **reward pathway** (ventral tegmental area → nucleus accumbens). This rewarding "buzz" **positively reinforces** smoking. Repeated use down-regulates receptors, producing **tolerance** and a **withdrawal** state that smoking then relieves (**negative reinforcement**).

Learning Theory and Cue Reactivity

- **Operant conditioning** — smoking is reinforced both positively (the dopamine reward) and negatively (relief from withdrawal).
- **Classical conditioning / cue reactivity** — cues repeatedly paired with smoking (coffee, the pub, stress, a particular routine) become conditioned stimuli that trigger **cravings**.

Evaluation

A strength of the explanation is strong supporting evidence for both mechanisms. Brain-imaging studies confirm that nicotine causes dopamine release in the reward pathway, and **cue-reactivity** studies show that smokers experience cravings and physiological arousal when shown smoking-related cues. Because these findings come from objective, replicable methods, they give the combined neurochemical-and-learning account high validity as an explanation of how nicotine addiction develops and persists.

This explanation also has valuable real-world and economic application. The neurochemical account directly underpins **nicotine replacement therapy**, while cue reactivity supports **cue-exposure** approaches that help smokers resist triggers. Because smoking-related illness places a vast burden on the NHS, treatments derived from this explanation have major economic value, strengthening the case for the approach.

However, a limitation is that the explanation is reductionist. Reducing nicotine addiction to dopamine and conditioning ignores the cognitive and social factors — expectations about smoking, peer influence and personality — that also shape it. The fact that nicotine addiction is strongly influenced by social context suggests a purely biological/behavioural account is incomplete, and that an **interactionist** explanation is needed.

A counterpoint that further limits the explanation is that it cannot explain individual differences. Many people smoke occasionally without becoming addicted, and quit rates vary widely, yet the dopamine and conditioning mechanisms should apply to everyone equally. This implies that genetic and personality risk factors (Section 2) determine who becomes addicted, so the neurochemical/learning account is only part of the picture.

4 Explaining Gambling Addiction

Learning Theory

Gambling is positively reinforced by **wins**. Crucially, it is reinforced on a **partial, variable-ratio schedule** — wins are unpredictable, arriving after a variable number of bets — which makes the behaviour **highly resistant to extinction**. Cues such as flashing lights and sounds become conditioned through **classical conditioning**.

Cognitive Theory

Gambling is maintained by faulty thinking — **cognitive biases**:

- **Illusion of control** — overestimating one's influence over chance outcomes ("I have a system").
- **The gambler's fallacy** — believing a win is "due" after a run of losses.
- **The near-miss effect** — almost-wins are experienced as encouraging rather than as losses.
- **Biased recall** — remembering wins vividly while discounting losses.

Evaluation

A strength of the learning explanation is that the variable-ratio schedule is very well established. Research in both animals and humans shows that variable-ratio reinforcement produces high, persistent rates of behaviour that are extremely resistant to extinction. This directly mirrors the way problem gamblers continue despite repeated losses, so the explanation has strong predictive validity and accounts well for the persistence of gambling.

The cognitive explanation also has convincing supporting evidence. "Thinking-aloud" studies (e.g. **Griffiths**) found that regular gamblers make far more irrational verbalisations — such as treating the machine as having intentions — than non-gamblers, and brain-imaging shows that "near misses" activate the same reward areas as wins. This demonstrates that distorted cognition genuinely characterises problem gambling, supporting the cognitive account.

A particular strength of the cognitive approach is its real-world application. Because it locates the problem in faulty thinking, it directly underpins **CBT** for gambling, which challenges irrational beliefs such as the gambler's fallacy and is an effective treatment. The success of a therapy derived from the theory provides indirect support for it and offers economic benefits by reducing the financial and social harms of problem gambling.

However, a limitation common to both explanations is that they cannot explain individual differences. Most people who experience variable reinforcement and hold the occasional cognitive bias never become addicted gamblers. This suggests that biological vulnerability and personality factors (Section 2) determine who is affected, so neither learning nor cognitive theory is a complete account — and, since gambling involves *both* reinforcement and distorted thinking, an integrated explanation is likely to be the most valid.

5 Reducing Addiction

Drug Therapy

- **Agonists** — mimic or replace the substance to reduce withdrawal (e.g. nicotine replacement therapy; methadone for heroin).
- **Antagonists** — block the substance's rewarding effects.
- **Aversives** — make use unpleasant (e.g. disulfiram causes sickness if alcohol is taken).

Behavioural Interventions

- **Aversion therapy** — the addictive behaviour is paired with an unpleasant stimulus (e.g. an emetic drug with alcohol), so a negative association forms (classical conditioning).
- **Covert sensitisation** — the same principle carried out using aversive **imagery** (imagining the unpleasant consequences), so it is less traumatic.

Cognitive Behaviour Therapy (CBT)

CBT helps the person identify and challenge the irrational thoughts and cues that maintain the addiction, and develop coping and relapse-prevention strategies.

Evaluation

A strength of drug therapy is that it is effective and easy to use. Treatments such as NRT and methadone reliably reduce withdrawal and cravings and require little effort from the user, which improves uptake and adherence. Because they can be delivered cheaply and at scale through health services, they offer a cost-effective way to reduce the very large economic burden of addiction, giving them clear practical value.

However, a limitation of drug therapy is that it treats the symptoms rather than the underlying causes, and can have side effects. Replacing one drug with another (e.g. methadone) does not address the psychological and social factors that maintain addiction, so relapse is common once treatment stops. This suggests drug therapy is best combined with psychological approaches such as CBT rather than used alone.

A strength of CBT is that, by tackling the underlying cognitions, it can produce more durable change. Unlike drug therapy, CBT addresses the cues and irrational beliefs that drive the behaviour and equips the person with relapse-prevention skills, which should make recovery longer-lasting. The trade-off is that CBT is more **time-consuming and expensive** and demands high motivation, so it may not suit everyone.

A counterpoint, however, is that aversion therapy raises ethical and effectiveness concerns. Deliberately inducing unpleasant experiences (e.g. vomiting) is distressing and raises ethical questions about protection from harm, and the effects are often short-lived and do not generalise beyond the clinic. Covert sensitisation is less traumatic, but the modest and often temporary success of behavioural interventions means they are usually most effective as *part* of a combined treatment programme.

6 Behaviour Change: Prochaska's Six-Stage Model

> **THE TRANSTHEORETICAL (SIX-STAGE) MODEL**
>
> Prochaska's model views behaviour change as a **dynamic, cyclical** process moving through six stages, rather than an all-or-nothing event. People can **relapse** and re-enter at an earlier stage.

1. **Precontemplation** — not yet thinking about change; may deny a problem.
2. **Contemplation** — aware of the problem and weighing the pros and cons (often ambivalent).
3. **Preparation** — intends to change soon and begins to plan.
4. **Action** — actively changing the behaviour.
5. **Maintenance** — sustaining the change and avoiding relapse.
6. **Termination** — the new behaviour is established with no temptation to relapse.

Note: the **theory of planned behaviour** was removed from the 2025 spec, so Prochaska's model is now the only behaviour-change model required.

Evaluation

A strength of Prochaska's model is that it realistically treats change as a process that includes relapse. Unlike "all-or-nothing" approaches, the model recognises that people often relapse and cycle back through earlier stages before succeeding. This matches the reality of addiction recovery, in which lapses are common, and it usefully removes the sense of total failure after a relapse, encouraging people to keep trying.

A further strength is its practical, applied value for tailoring treatment. Because it identifies a person's stage, practitioners can match the intervention to readiness — for example, motivational work for someone in precontemplation versus active support for someone in action. Targeting interventions in this way should improve their effectiveness and efficiency, with clear economic benefits for health services.

However, a limitation is that the "stages" may be arbitrary and not clearly separable. Critics argue that change is really a continuous process and that the boundaries between stages are vague, so people do not move neatly from one discrete stage to the next. If the stages cannot be reliably identified, the model's central claim is weakened, and stage-matched interventions have shown mixed success in research.

A counterpoint is that the model may apply better to some addictions than others, and overstate conscious choice. It assumes deliberate, rational decision-making about change, yet much addictive behaviour is driven by automatic cravings and cue reactivity that are not under full conscious control. This illustrates the **free will versus determinism** debate and suggests the model is most useful when combined with approaches that also address the biological and conditioned aspects of addiction.

These notes were prepared for Simply Psychology and cover spec section 4.3.10 of the AQA Psychology 2025 specification (A-level only, Paper 3). The theory of planned behaviour was **removed** in 2025; Prochaska's six-stage model is the only behaviour-change model required. For deeper coverage, see simplypsychology.org/addiction.html.