

# **AQA Psychology A-level**

## **Option 3: Addiction**

### **Summary Notes**

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## Part 1 — Describing Addiction

**Addiction** is the **physical and/or psychological dependence** on a substance or on an activity or behaviour. Examples include **nicotine, alcohol, opiates and gambling**.

Addiction can be described using four key characteristics: **physical dependence, psychological dependence, tolerance and withdrawal syndrome**.

### 1. Physical dependence

**Physical dependence** occurs when the body becomes **physiologically adapted to a substance**, meaning the person needs to continue taking it to maintain normal functioning.

- If the substance is **reduced or stopped**, the person experiences **withdrawal symptoms**.
- Taking the substance again can cause these symptoms to **reduce or disappear**.
- Therefore, the person may continue using the substance partly to **avoid withdrawal** and feel physically normal.

Physical dependence is often accompanied by **tolerance**, although the two concepts are not exactly the same.

### 2. Psychological dependence

**Psychological dependence** occurs when a substance or behaviour becomes an important part of a person's **thoughts, emotions and activities**.

- The person may experience **strong cravings or compulsions** to use the substance or engage in the behaviour.
- They may feel that they **cannot cope without it**.
- They may continue the behaviour because they want to experience its **pleasurable effects** or because it has become a **habit**.
- Psychological dependence can occur without physical dependence, particularly with **behavioural addictions such as gambling**.

### 3. Tolerance

**Tolerance** occurs when the body's response to a substance **decreases over time**. This means:

**Same dose → weaker effect → larger dose needed to produce the same effect**

As a result, the person may gradually consume **increasingly larger amounts** of the substance to achieve the desired effect.



#### 4. Withdrawal syndrome

**Withdrawal syndrome** is the **collection of unpleasant physical and psychological symptoms** experienced when a person who is dependent on a substance **stops taking it or reduces the dose**.

Symptoms can include:

- **Anxiety**
- **Insomnia**
- **Nausea**
- **Sweating**
- **Tremors**
- **Body aches**

The severity of withdrawal can vary depending on factors such as the **substance used, amount consumed and pattern of use**.

Withdrawal can encourage continued substance use because taking the substance again may **relieve the unpleasant symptoms**.

Therefore, dependence can be maintained by a cycle of **use** → **withdrawal** → **further use**.



## Part 2 — Genetic Risk Factors in the Development of Addiction

From a **biological perspective**, some individuals may inherit **genetic vulnerabilities** that increase their risk of developing an addiction. This suggests a **predisposition** rather than a deterministic effect, meaning having these genes does not guarantee that someone will become addicted.

### Dopamine receptors

One possible biological vulnerability is having **abnormally low levels of D2 dopamine receptors** in the brain.

- **Dopamine** is involved in the brain's **reward system**.
- D2 receptors are sites where dopamine can bind to **postsynaptic neurons**.
- Having fewer D2 receptors may reduce the effects of dopamine.
- This may encourage an individual to seek out **stronger or more frequent rewarding experiences**, increasing vulnerability to addictive behaviour.

### CYP2A6 and nicotine

Another example involves the **CYP2A6 gene**, which codes for an enzyme involved in **breaking down nicotine**.

**Pianezza et al. (1998)** suggested that variations in the CYP2A6 gene can affect how quickly nicotine is metabolised.

- **Higher CYP2A6 activity** → nicotine is broken down **more quickly**.
- **Lower CYP2A6 activity** → nicotine is broken down **more slowly**.
- Slow nicotine metabolism means nicotine remains in the body for **longer**, so the individual may need to smoke **less frequently** to maintain nicotine levels.
- This may reduce their risk of developing **nicotine addiction**.

Therefore, genetic differences can create **biological vulnerability or protection** against addiction rather than directly determining whether addiction will occur.

### AO3 - EVALUATION

A strength of the biological explanation of addiction is that there is **research support for a genetic predisposition**. **Kendler et al. (2012)** found that adoptees with at least one **biological parent with an addiction** had a **4.4% greater risk** of developing an addiction than adoptees without this biological risk. Because adoptees were raised apart from their biological parents, the findings reduce the influence of the **shared environment**, suggesting that genetic factors contribute to vulnerability. Therefore, the evidence supports the idea that addiction has a **heritable component**.



A further strength of the biological explanation of addiction is that it has **practical applications for prevention and treatment**. If genetic factors increase an individual's vulnerability to addiction, this could allow professionals to identify people who may be at **greater risk** and provide early support. For example, individuals with a strong family history of addiction could be encouraged to develop **healthy coping strategies** and avoid high-risk environments. Therefore, understanding the biological basis of addiction may help with **early intervention and prevention**.

A limitation is that genetic vulnerability may not come from **candidate genes alone**. Genetic factors may also influence characteristics such as **impulsivity and poor self-control**, which can increase the likelihood of developing addictive behaviour indirectly. For example, an impulsive child may experience **school difficulties, punishment and rejection**, which could lead to poorer future opportunities and increased vulnerability to addiction later in life. Therefore, addiction may result from a **complex interaction between genetic predispositions and environmental experiences**, meaning purely genetic explanations may be **too simplistic**.

A further limitation of the biological explanation of addiction is that identifying a **genetic predisposition does not necessarily provide an effective way to treat the addiction**. Unlike some medical conditions where identifying a genetic cause can lead directly to a targeted treatment, there is currently no simple way to remove or alter the genes associated with addiction. This means that even if an individual is identified as being **genetically vulnerable**, they would still require psychological or behavioural interventions to change their addictive behaviour. Therefore, the biological explanation may have **limited practical value for treating addiction**, despite helping to explain why some individuals are more vulnerable.



## Part 3 — Personality and Social Factors in the Development of Addiction

A range of **psychological and social factors** can increase or decrease an individual's **vulnerability to addiction**. These include **personality characteristics** and **social influences** such as family, peers and stress.

### Personality:

Certain personality characteristics may increase an individual's **vulnerability to addiction**. **Impulsivity** is particularly important because impulsive individuals are more likely to act **without considering the consequences** and engage in **risk-taking behaviours**. This may make them more likely to experiment with addictive substances or behaviours and continue despite negative consequences.

**Antisocial personality characteristics** have also been linked to addiction. **Ivanov et al. (2008)** found that characteristics such as **impulsivity and antisocial behaviour** were associated with an increased risk of developing addictive behaviours.

### Social influences:

Social influences can affect an individual's **exposure to addictive behaviours**, their attitudes towards them and their ability to cope with stressful situations.

| Social Influence | Role in addiction                                                                                                                                                                                                                                                                                                                                                                                                                                                                                 |
|------------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Family</b>    | <b>Parental monitoring</b> can reduce the risk of addiction by limiting access to addictive substances and behaviours. Children may also learn attitudes towards substances through <b>observational learning</b> . If parents regularly use substances, this may make their use seem <b>normal or acceptable</b> . <b>Livingston et al. (2010)</b> found that adolescents who regularly saw their parents drinking alcohol were more likely to show <b>excessive alcohol consumption later</b> . |
| <b>Peers</b>     | Peers can provide <b>access to addictive substances</b> , act as <b>role models</b> and influence what is seen as <b>normal or acceptable</b> behaviour. <b>Peer pressure</b> may also encourage individuals to experiment with addictive behaviours.                                                                                                                                                                                                                                             |
| <b>Stress</b>    | <b>Chronic stress</b> can increase vulnerability as addictive substances or behaviours may provide <b>temporary relief</b> . Repeatedly using them as a way of coping can <b>reinforce the behaviour</b> , increasing the risk of addiction. <b>Andersen and Teicher (2008)</b> suggested that chronic stress can act as a <b>diathesis</b> , increasing vulnerability when combined with other risk factors or stressful experiences.                                                            |



## Protective influences

Some social factors can **reduce**, rather than increase, vulnerability to addiction.

- **Positive family relationships** – emotional support, open communication and clear boundaries can help young people cope with difficulties without turning to addictive behaviours.
- **Parental monitoring** – supervision can reduce opportunities to access addictive substances and allow parents to recognise risky behaviour early.
- **Positive peer groups** – friends who do not engage in addictive behaviours reduce **exposure and peer pressure**, while providing social support.
- **Effective coping strategies** – being able to manage **stress and difficult emotions** in healthy ways reduces the need to use addictive substances or behaviours as a form of escape.

This is important because **risk factors do not inevitably lead to addiction**. Protective influences can **reduce an individual's vulnerability**, even when they are exposed to factors such as stress or peer pressure.

## AO3 - EVALUATION

A strength of the **risk-factor explanation of addiction** is that it recognises the importance of **multiple interacting influences**. **Mayes and Suchman (2006)** argued that no single factor is necessarily more important than another, with the combination of factors such as **stress, personality, family influences and peer influences** affecting an individual's vulnerability to addiction. For example, high stress may increase vulnerability, but supportive parenting or positive peer relationships may reduce this risk. Therefore, considering several risk factors together provides a **more comprehensive explanation** than focusing on any one influence.

A further strength is that identifying **risk and protective factors** has useful **practical applications**. **Hawkins et al. (1992)** suggested that understanding these factors can help identify individuals who may be particularly vulnerable to addiction, allowing early intervention. For example, interventions can target **stress management, family relationships, coping skills and resistance to peer pressure**. This could help prevent addictive behaviour from developing, giving research into risk factors clear **real-world value**.

A limitation of research into **risk factors for addiction** is that much of the evidence is **correlational**. For example, a relationship between **peer influence, family problems, stress or personality** and addiction does not prove that the risk factor caused the addiction. There may be a **third factor** influencing both variables, such as a biological vulnerability. The **file-drawer problem** may also mean that studies showing significant relationships are more likely to be published than studies finding no relationship. Therefore, it is difficult to establish the **cause and effect** of individual risk factors.

A further limitation is that the importance of each risk factor may **vary between individuals**. Someone may experience high stress, have a vulnerable personality and associate with substance-using peers but never develop an addiction, while another person exposed to fewer risks may become addicted. This makes it difficult to **predict who will develop an addiction** or determine which factor should be targeted in treatment. Therefore, although risk factors can help identify vulnerability, they may have **limited predictive value when considered individually**.



## Part 4 — Explanations for Nicotine Addiction: Brain Neurochemistry

### Desensitisation hypothesis

**Dani and Heinemann (1996)** proposed that changes to **nicotinic acetylcholine receptors** help explain the development and maintenance of nicotine addiction. Their explanation focuses on the interaction between **acetylcholine**, **nicotine** and **dopamine** in the brain's **reward system**, particularly in areas such as the **ventral striatum** and **nucleus accumbens**.

### Desensitisation

- **Nicotinic acetylcholine receptors** are found on the **postsynaptic membranes** of neurons, including in the **nucleus accumbens**.
- Normally, **acetylcholine** binds to these receptors and increases neural activity.
- **Nicotine** can also bind to these receptors and **mimics the effects of acetylcholine**.
- Repeated exposure to nicotine causes the receptors to become **less sensitive (desensitised)**.
- This **reduces their response to acetylcholine** and changes neurotransmission in the reward pathway.
- Nicotine also stimulates the release of **dopamine into the mesolimbic system**, producing feelings of **pleasure and euphoria**.

### Upregulation and withdrawal

When nicotine use is **reduced or stopped**, the receptors begin to **upregulate**.

- The nicotinic receptors become **more sensitive** again.
- This changes neural activity in the **nucleus accumbens**.
- The individual experiences **withdrawal symptoms**, which can include **anxiety, irritability and nausea**.
- Taking nicotine again causes the receptors to become **desensitised**, reducing the unpleasant withdrawal symptoms.

This creates a cycle:

**Nicotine → receptor desensitisation → pleasure → nicotine reduced → receptor upregulation → withdrawal → nicotine use again**

### Tolerance

Repeated nicotine use also leads to **tolerance**.

- As the receptors become increasingly **desensitised**, more nicotine is needed to produce the same pleasurable effects.
- The individual therefore increases their **nicotine consumption** to achieve the desired level of pleasure.
- This helps explain why nicotine addiction can become a **cycle of increasing consumption and withdrawal**.



### AO3 - EVALUATION

A strength of the dopamine explanation of nicotine addiction is that there is **research support** for the role of dopamine in maintaining nicotine use. **McEvoy et al. (1995)** found that patients taking a **dopamine antagonist** smoked significantly more than those who were not taking the drug. Blocking dopamine receptors reduced the effects normally associated with nicotine, so greater nicotine consumption was needed to compensate. This supports the idea that **dopamine is involved in maintaining nicotine addiction**.

A further strength is that the explanation has **practical applications for treatment**. If dopamine activity contributes to the rewarding effects of nicotine, drugs that **alter dopamine activity** could potentially be used to reduce the reinforcing effects of smoking and help individuals **reduce or stop nicotine use**. This means research into the dopamine system can contribute to the development of **biological treatments for nicotine addiction**, giving the explanation real-world value.

A limitation is that the explanation may place **too much emphasis on dopamine**. **Berrendero et al. (2010)** argued that there is no single neurotransmitter responsible for nicotine addiction, with **GABA, serotonin and endorphins** also contributing to the effects of nicotine. This suggests that dopamine is only **one part of a much more complex process**, so the explanation cannot fully account for the different effects that nicotine has on the brain and behaviour. Therefore, the dopamine explanation may provide an **incomplete account of nicotine addiction**.

A further limitation is that the dopamine explanation may be **biologically reductionist** because it reduces a complex behaviour to the activity of one neurotransmitter. **Choi et al. (2003)** suggested that biological factors may create a **diathesis**, but psychosocial factors such as **peer and family influences** can determine whether addiction actually develops. This may help explain why only around **50% of regular smokers become addicted**, suggesting that an **interactionist explanation** may provide a more complete account.



## Part 5 — Explanations for Nicotine Addiction: Learning Theory

Learning theory suggests that **nicotine addiction is a learned behaviour** that develops and is maintained through **conditioning and reinforcement**. Both **classical and operant conditioning** can contribute, with **positive and negative reinforcement** making smoking more likely to continue.

### Initiation

**Classical conditioning** suggests that smoking can initially develop through **association**.

- A young person may already associate situations such as **socialising with friends** with positive feelings such as enjoyment and relaxation.
- If they repeatedly **smoke in these situations**, smoking becomes associated with these positive feelings.
- Over time, smoking can become a **conditioned stimulus**, triggering an expectation of the pleasurable feelings associated with the situation.
- This can increase the likelihood of **smoking again**, helping to explain the **initiation of nicotine use**.

### Maintenance

Once smoking has started, **reinforcement** can make the behaviour more likely to continue.

#### Positive reinforcement – gaining pleasure

- Nicotine stimulates **dopamine release in the mesolimbic reward system**.
- This produces feelings of **pleasure and euphoria**.
- The pleasurable effects act as a **reward**, increasing the likelihood that the individual will **smoke again**.

#### Negative reinforcement – avoiding withdrawal

- As nicotine levels fall, the individual experiences **withdrawal symptoms**.
- Smoking again **restores nicotine levels** and reduces these unpleasant symptoms.
- This makes continued smoking more likely because the individual is **escaping the discomfort of withdrawal**.

Therefore, smoking is maintained through both **gaining pleasure** and **avoiding unpleasant withdrawal symptoms**

#### Cue Reactivity – maintenance and relapse

**Cue reactivity** refers to the way **environmental cues associated with smoking** can trigger cravings and increase the likelihood of smoking.



Repeated pairings between smoking and particular cues can mean that situations such as **parties, seeing other people smoke, the smell of tobacco or seeing a cigarette packet** become associated with nicotine's pleasurable effects.

- The cue can trigger a **craving to smoke** even when nicotine has not been consumed.
- This can help **maintain the addiction**.
- The response to these cues can also make **relapse more likely** after someone has attempted to quit.

### AO3 - EVALUATION

A strength of **learning theory as an explanation of nicotine addiction** is that it can account for **gender differences in smoking behaviour**. **Carpenter et al. (2014)** found that women were **less likely to give up smoking and more likely to relapse** than men. This may be because women have lower **self-efficacy**, meaning they are less confident in their ability to quit, and may also experience greater sensitivity to **social cues** associated with smoking. This supports the idea that **cue reactivity and learned associations** can influence nicotine addiction and relapse.

A further strength is that there is **biological evidence supporting the role of reinforcement in nicotine addiction**. **Levin et al. (2010)** found that rats rapidly increased their **self-administration of nicotine**, making up to **100 attempts per infusion**. This suggests that the **pleasurable effects of nicotine**, linked to increased **dopaminergic activity in the mesolimbic system**, can reinforce repeated drug-taking behaviour. Therefore, learning theory is supported by evidence that **positive reinforcement can contribute to the development of nicotine addiction**.

A further strength is that learning theory has **useful practical applications for treatment**. **Aversion therapy and covert sensitisation** are based on learning principles, aiming to associate smoking with **negative consequences** rather than pleasure. **Smith (1988)** found that aversion therapy using electric shocks produced recovery rates at least **32% higher** than simply deciding to stop smoking. This suggests that understanding the **learning processes underlying addiction** can be used to develop treatments that improve **smoking cessation**, giving the theory important real-world value.

A limitation of learning theory as an explanation of nicotine addiction is that it may be **too reductionist**. The theory focuses mainly on learned associations and reinforcement while underestimating the role of **biological factors**. Nicotine directly affects the brain's **reward system** and can lead to **tolerance and withdrawal symptoms**, which may continue to drive smoking even when there are no obvious environmental cues or rewards. Therefore, learning theory alone cannot fully explain why nicotine addiction can be difficult to overcome.

A further limitation is some of the evidence supporting learning theory comes from **animal research, limiting generalisability to humans**. For example, **Levin et al. (2010)** found that rats rapidly increased their nicotine self-administration, supporting the role of reinforcement. However, human smoking involves complex **cognitive, social and emotional factors** that are not present in the same way in lab animals. This means that findings from animal studies may not accurately explain the development and maintenance of **human nicotine addiction**, limiting the strength of the evidence for learning theory.



## Part 6 — Explanations for Gambling Addiction: Learning Theory

**Learning theory** suggests that gambling addiction is a **learned behaviour**, maintained by **reinforcement** and associations with gambling-related cues. Five forms of reinforcement are particularly relevant: **positive, negative, partial, variable and vicarious reinforcement**.

All forms of reinforcement **increase the likelihood that gambling behaviour will be repeated**.

**Types of reinforcement:**

| Type                           | How it maintains gambling                                                                                                                                                                                                                                                                                                |
|--------------------------------|--------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Positive reinforcement</b>  | Gambling produces <b>pleasant consequences</b> , such as the <b>thrill of winning, money, praise or admiration</b> . These rewards increase the likelihood of gambling again.                                                                                                                                            |
| <b>Negative reinforcement</b>  | Gambling can provide an <b>escape from unpleasant feelings or everyday problems</b> , such as stress or anxiety. The relief this provides makes the individual more likely to gamble again when similar difficulties occur.                                                                                              |
| <b>Partial reinforcement</b>   | Gambling is only <b>rewarded occasionally</b> because the gambler does not win every time. However, occasional wins can be highly rewarding, encouraging the gambler to <b>persist despite losses</b> . <b>Skinner</b> found that intermittently reinforced behaviours are particularly <b>resistant to extinction</b> . |
| <b>Variable reinforcement</b>  | The gambler knows there is a <b>chance of winning</b> , but cannot predict exactly when the reward will occur. This <b>uncertainty and anticipation</b> encourages persistent betting because the next attempt could be successful.                                                                                      |
| <b>Vicarious reinforcement</b> | Individuals may see <b>other gamblers being rewarded</b> , for example lottery winners receiving money and attention. This can motivate them to <b>imitate the behaviour</b> in the hope of receiving similar rewards.                                                                                                   |

### How the processes work together

Gambling can therefore become strongly maintained because the individual can **gain pleasure, escape unpleasant feelings**, receive rewards **occasionally and unpredictably**, and **observe other people being rewarded**. At the same time, gambling-related cues can trigger cravings and encourage **continued gambling or relapse**.



### AO3 - EVALUATION

A strength of **learning theory** is that there is research support for the role of **positive and vicarious reinforcement** in the development of gambling behaviour. **Dickerson (1979)** conducted a field study in two Birmingham betting shops and found that **high-frequency gamblers** were more likely to place their bets in the **final two minutes before a race**. This was a particularly exciting period, with increased tension, commentary and “buzz” within the betting shop. The excitement may therefore have acted as **positive reinforcement**, encouraging gamblers to repeat the behaviour. This supports the idea that reinforcement can contribute to the **development and maintenance of gambling behaviour**.

A further strength is that learning theory has **practical applications** for reducing gambling behaviour. If gambling is maintained through **reinforcement**, treatments can focus on reducing the rewards associated with gambling and replacing them with more positive behaviours. For example, **operant conditioning techniques** can be used to reinforce alternative behaviours, while exposure techniques can reduce responses to gambling-related cues. This means learning theory can be used to **inform treatment strategies**, giving it practical value as well as explanatory value.

A limitation of **learning theory** is that it may not explain **all stages of the development of gambling addiction**. **Brown (1987)** suggested that reinforcement may be particularly important in the **initial acquisition** of gambling, as winning can provide immediate pleasure or temporary relief from problems. However, **cognitive explanations** may be better at explaining the **long-term maintenance** of gambling addiction, through distortions such as the **illusion of control and gambler's fallacy**. Therefore, learning theory may be useful for explaining how gambling begins but less effective at explaining **why it develops into a persistent addiction**.

A further limitation is that **cue reactivity does not affect all individuals in the same way**. Learning theory suggests that cues associated with previous gambling experiences should continue to trigger gambling behaviour, but **Griffiths and Delfabbro (2001)** argued that this is not always the case. The effect of a cue may depend on other **psychosocial factors**, such as peer or family influences. This suggests that gambling addiction may be better explained using an **interactionist approach**, where learned cues interact with individual vulnerabilities and environmental stressors. Therefore, learning theory alone may be **too simplistic** to explain why some individuals exposed to gambling cues develop an addiction while others do not.



## Part 7 — Explanations for Gambling Addiction: Cognitive Theory

The **cognitive theory** suggests that gambling addiction develops because gamblers have **irrational expectations** about the outcomes of gambling and develop **cognitive biases** that distort the way they think about wins and losses.

**Expectancy theory** suggests that people are more likely to behave in a particular way when they expect the **benefits to outweigh the costs**.

In gambling, this expectation can become **irrational**:

- Gamblers may believe that the **potential rewards** of gambling are greater than the possible losses.
- They may focus their attention on **winning, money and other positive outcomes**, while ignoring or minimising their losses.
- This creates a distorted view of the **costs and benefits** of gambling.
- As a result, they continue gambling because they believe that the behaviour will eventually produce a **positive outcome**.

### Cognitive biases

**Cognitive biases** are systematic errors in thinking that can distort a gambler's judgement about the likelihood of winning.

**Rickwood (2010)** identified four important types:

| Cognitive bias               | What it means                                                                 | Example                                                                                                                                |
|------------------------------|-------------------------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------|
| <b>Faulty perceptions</b>    | Incorrect beliefs about <b>chance and probability</b> .                       | <b>Gambler's fallacy</b> – believing that after several losses, a win is now more likely because a losing run cannot continue forever. |
| <b>Selective recall</b>      | Remembering <b>wins and positive experiences</b> more easily than losses.     | Focusing on the time they won a large amount while forgetting how much money they have lost overall.                                   |
| <b>Skills and judgements</b> | Overestimating their own ability to <b>control random events</b> .            | <b>Illusion of control</b> – believing they can influence which numbers or machines will win.                                          |
| <b>Personal traits</b>       | Believing that personal characteristics or <b>luck</b> influence the outcome. | Having <b>superstitions</b> about particular machines, numbers or rituals.                                                             |

These biases can strengthen the gambler's belief that gambling will eventually produce a **reward**, encouraging them to continue despite repeated losses.



**Griffiths (1994)** provides evidence for the role of **cognitive biases** in gambling addiction.

- **Regular gamblers** were compared with **occasional gamblers**.
- Regular gamblers made significantly **more irrational verbalisations** than non-regular gamblers (14% vs 2.5%), despite there being **no difference in actual objective skill or money won**.
- There was **no difference in the amount of money won** between the groups.
- Examples included statements such as *"I have a knack for this machine"* and *"Everyone knows I can choose winning numbers!"*

This suggests that regular gamblers are more likely to use **irrational thoughts and beliefs**, supporting the idea that **cognitive distortions contribute to the maintenance of gambling addiction**.

The cognitive theory therefore suggests that gambling addiction is maintained because gamblers **overestimate the likelihood and value of winning**, while cognitive biases cause them to **misinterpret losses, remember wins and overestimate their control**

### AO3 - EVALUATION

A strength of the cognitive explanation of gambling addiction is that there is **research support for the role of cognitive and dispositional factors**. **Michalczuk et al. (2011)** compared **30 pathological gamblers with 30 controls** and found that the gamblers showed greater **impulsivity** and were more likely to choose **immediate rewards** rather than waiting for larger rewards. This supports the idea that gambling addiction is associated with particular **cognitive characteristics**, such as impulsivity, which may make some individuals more vulnerable to developing an addiction.

A further strength is that the cognitive explanation has **practical applications in treatment**. **Clark (2010)** suggested that understanding cognitive biases such as the **illusion of control and gambler's fallacy** can improve **CBT** for gambling addiction. Therapists can identify these irrational beliefs and use **cognitive restructuring** to challenge them and replace them with more realistic thoughts. Therefore, the theory has **practical value** because it can directly inform the treatment of gambling addiction.

A limitation of the **cognitive explanation** is that it may not explain **individual differences in gambling addiction**. **Burger and Norris (1985)** suggested that people differ in their belief about how much **control** they have over events in their lives. Some individuals may be more likely to transfer this belief onto **random events**, such as gambling, leading them to develop an illusion of control. This suggests that cognitive distortions may be particularly important for some individuals but not others, meaning the explanation cannot fully account for **why some people develop gambling addictions while others do not**.

A further limitation is that the cognitive explanation may be **too narrow** because it focuses **mainly on thoughts and beliefs**. Gambling addiction is also influenced by **behavioural and biological factors**, such as **reinforcement and impulsive reward-seeking**. This means cognitive distortions alone may not provide a complete explanation of why gambling becomes addictive. Therefore, a **multifactorial explanation** that considers cognitive, behavioural and biological factors together may provide a more comprehensive account.



## Part 8 — Reducing Addiction: Behavioural Interventions

There are two main **behavioural interventions** for addiction: **aversion therapy** and **covert sensitisation**. Both are based on **classical conditioning** and aim to weaken the association between the addictive behaviour and the **pleasure or reward** it produces.

### 1. Aversion therapy

**Aversion therapy** is a more **traditional approach** that is less commonly used today. It aims to **counter-condition** the addictive behaviour by repeatedly pairing it with an **unpleasant consequence**.

- The addictive stimulus is paired with something unpleasant, such as **nausea, vomiting or an electric shock**.
- Through **classical conditioning**, the individual learns to associate the addictive behaviour with these negative consequences.
- This aims to **break the association between addiction and pleasure**, making the behaviour less appealing.
- The process relies on the principle of **continuity**, where two stimuli repeatedly occurring together become associated.

Examples of aversion therapy:

| Addiction       | How it's used                                                                                                                                                                                                                                                                                                                                                                                                                                                       |
|-----------------|---------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>Alcohol</b>  | <ul style="list-style-type: none"> <li>• The individual consumes <b>alcohol</b> and is then given an <b>emetic</b>, causing <b>vomiting</b>.</li> <li>• Alternatively, <b>disulfiram</b> can be used to produce unpleasant <b>hangover-like effects</b> (e.g. nausea, throbbing headache, hyperventilation).</li> <li>• Repeated pairings aim to create an association between <b>alcohol and unpleasant consequences</b>, reducing the desire to drink.</li> </ul> |
| <b>Gambling</b> | <ul style="list-style-type: none"> <li>• The individual may receive a brief <b>electric shock</b> while being exposed to <b>gambling-related words or cues</b>.</li> <li>• Repeatedly pairing gambling with an unpleasant consequence aims to create a <b>negative association</b> with gambling.</li> <li>• This should make the <b>gambling behaviour less appealing</b> and reduce the likelihood of repeating it.</li> </ul>                                    |



## 2. Covert sensitisation

**Covert sensitisation** uses the same basic principles as aversion therapy but does **not** require the unpleasant consequence to be physically experienced.

- Instead, the individual is asked to **imagine** the addictive behaviour followed by a highly unpleasant consequence.
- This may involve deliberately **vivid and graphic imagery** to create a strong feeling of disgust.
- **McMurrin (1994)** suggested that personal experiences, such as an individual's existing **phobias**, can be incorporated to make the imagined consequence particularly unpleasant.
- Repeatedly pairing the addictive behaviour with an unpleasant imagined outcome aims to **weaken the original association with pleasure** and reduce the likelihood of the behaviour being repeated

### AO3 - EVALUATION

A strength of aversion therapy is that there is evidence that more modern forms, such as **covert sensitisation**, may be more effective than traditional methods. **McConaghy et al. (1983)** found that covert sensitisation produced a **60% greater recovery rate** than traditional electrical aversion therapy in the treatment of gambling addiction. This suggests that aversion-based approaches can be effective, while covert methods may offer a **more successful and practical alternative** to physically unpleasant forms of treatment.

A further strength is that the more modern use of **covert sensitisation** removes some of the **practical and ethical problems** associated with traditional aversion therapy. As the unpleasant consequences are **imagined rather than physically induced**, patients are not exposed to the same level of physical discomfort or potential harm. This may make individuals **more willing to begin and remain in treatment**, increasing the practical usefulness of aversion-based approaches.

A limitation of **aversion therapy** is that it raises significant **ethical concerns**. Traditional approaches may involve deliberately producing **pain, nausea or other unpleasant physical responses**, which could conflict with the **BPS principle of protecting participants from physical and psychological harm**. These unpleasant experiences may also reduce patients' **motivation to engage with treatment or complete it**, limiting the usefulness of aversion therapy as a long-term approach to rehabilitation.

A further limitation is that research into aversion therapy may lack **methodological validity** because **double-blind procedures are difficult to use**. **Hajek and Stead (2001)** reviewed **25 studies of aversion therapy for nicotine addiction** and highlighted the problem that researchers may know which participants received the treatment. This creates the possibility of **researcher bias**, as expectations about the effectiveness of aversion therapy could influence the interpretation of results. Therefore, conclusions about the effectiveness of aversion therapy may be **less reliable and valid**.



## Part 9 — Reducing Addiction: Cognitive Behaviour Therapy

**Cognitive Behavioural Therapy (CBT)** assumes that **faulty thinking, cognitive biases and unhelpful behaviours** help maintain addiction. CBT therefore aims to identify and change these patterns, while giving the individual **alternative ways of coping** with difficult situations.

**Functional analysis** is used to identify the factors that **trigger and maintain addictive behaviour**. The therapist and patient explore:

- **Situations and stimuli** that trigger addictive behaviour.
- The individual's **thoughts and beliefs** about the situation.
- **Emotions** experienced before, during and after the behaviour.
- The **consequences** of addictive behaviour.

For example, an individual with an **alcohol addiction** may find that being around unfamiliar people increases their **social anxiety**, leading them to drink to feel more confident. The therapist then identifies and challenges **irrational beliefs or cognitive biases** that contribute to the addiction.

Functional analysis is also an **ongoing process**. It can be revisited after **relapse** to identify what triggered the return to addictive behaviour and adapt treatment accordingly.

### Skills acquisition

CBT also teaches individuals **alternative coping skills** so that they do not rely on addictive behaviour to deal with problems.

There are three main areas:

#### 1. Cognitive restructuring

- The therapist identifies **irrational thoughts and cognitive biases**.
- These are challenged and replaced with more **rational and realistic alternatives**.
- The patient learns to recognise how their thinking may contribute to the addiction.

#### 2. Specific skills

- The therapist links the individual's **wider life problems** to their addictive behaviour.
- CBT is therefore considered a **broad-spectrum treatment**, rather than focusing only on the addiction itself.
- Skills may include **anger management** and **assertiveness training**.
- For example, **Novaco (1975)** developed anger-management techniques that help individuals recognise the **emotions and thoughts preceding aggressive behaviour**.



### 3. Social skills

- Individuals learn ways to **manage or avoid situations that trigger their addiction**.
- For example, a drug-dependent person may be encouraged to avoid **parties where drugs are commonly used**.
- They may also practise ways of **refusing drugs**, such as giving a simple reason for not taking them.
- The therapist can **model appropriate responses**, which the patient practises through **role-play** and other directive techniques.

CBT aims to help individuals understand the **triggers, thoughts and behaviours** that maintain their addiction and develop **healthier coping strategies** to replace the addictive behaviour.

### AO3 - EVALUATION

A strength of CBT for addiction is that there is research support for the importance of a **strong therapist–patient relationship**. **Petry et al. (2006)** found that chronic gamblers who received **eight CBT sessions alongside Gamblers Anonymous** showed a significant reduction in gambling one year later compared with a control group. Importantly, the improvement depended on CBT being delivered by a **therapist rather than through a self-help book**, suggesting that individual guidance and the therapeutic relationship are important to treatment effectiveness.

A limitation of CBT is that it is **cognitively demanding**, as patients must actively recognise, challenge and change the thoughts and behaviours contributing to their addiction. **Cuijpers et al. (2008)** found that **dropout rates were around five times higher** for CBT than for some alternative therapies. This suggests CBT may be less suitable for individuals who **struggle to engage with cognitively demanding treatment**, limiting its effectiveness for some addicts.

A further limitation is that CBT may not be equally effective for all types of addiction because it focuses heavily on **changing thoughts and behaviours**. Some addictions are strongly influenced by **biological and physiological factors**, such as withdrawal symptoms and changes in the brain's reward system, which cannot necessarily be changed through cognitive restructuring alone. This suggests CBT may be most effective when combined with **other treatments**, rather than being used as a single approach to addiction.

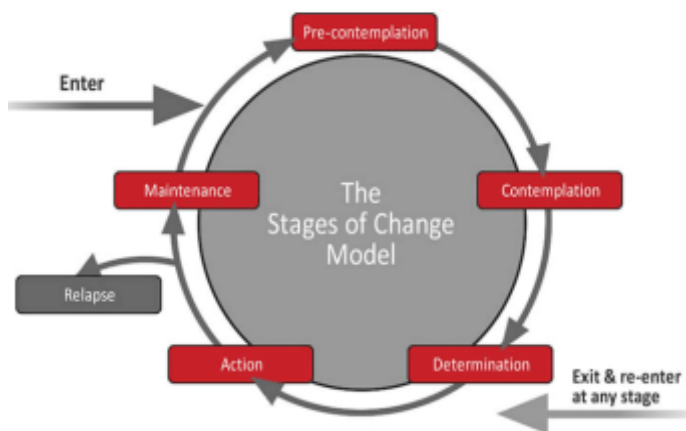
Another limitation of CBT is that access to treatment can be limited by **long waiting times for psychological therapy**, particularly within the NHS. Addiction can continue to cause significant harm while an individual waits to access CBT, meaning that the treatment may not be available when it is most needed. This may reduce the effectiveness of CBT as a treatment for addiction, particularly if the person's addiction becomes more severe during the waiting period. Therefore, the practical effectiveness of CBT may be limited by **availability and access**, rather than the treatment itself.



## Part 10 — Prochaska's Model of Behaviour Change

### Prochaska and DiClemente (1983)

proposed a **cyclical model of behaviour change** to explain how people change addictive behaviours. The model recognises that recovery is **not always a fixed, linear process**. People can move backwards between stages, and the **time spent in each stage varies between individuals** depending on their readiness to change.



The **six stages** are:

| Stage                       | What Happens                                                                                                                                                                                                                                                                                                         |
|-----------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| <b>1. Pre-contemplation</b> | The individual is <b>not ready to change</b> and may not recognise that they have a problem. This can be due to <b>denial</b> or a lack of motivation following previous unsuccessful attempts to stop.                                                                                                              |
| <b>2. Contemplation</b>     | The individual begins to consider <b>changing their behaviour</b> and weighs up the <b>costs and benefits</b> of stopping. Some people remain in this stage for a long time because they are unsure whether the benefits of change outweigh the costs.                                                               |
| <b>3. Preparation</b>       | The individual has <b>decided to change</b> and begins making plans for how to do this. They may seek help, such as <b>contacting a GP or support service</b> , and prepare themselves for difficulties such as withdrawal or lifestyle changes.                                                                     |
| <b>4. Action</b>            | The individual <b>actively changes their behaviour</b> . For example, they may throw away drugs or remove alcohol from their home. <b>Behavioural interventions</b> are likely to be most effective when the individual is actively ready to make changes.                                                           |
| <b>5. Maintenance</b>       | The individual works to <b>maintain the change and prevent relapse</b> . They develop alternative ways of coping with triggers such as <b>stress, loneliness and withdrawal symptoms</b> , rather than returning to the addictive behaviour.                                                                         |
| <b>6. Termination</b>       | The individual no longer feels <b>dependent on the substance or behaviour</b> and has little or no desire to return to it. Not everyone reaches this stage, particularly those with <b>long-term or severe addictions</b> , so treatment may instead focus on maintaining progress and reducing the risk of relapse. |



## The Cyclical nature of change

A key feature of the model is that **relapse does not necessarily mean failure**. If an individual returns to the addictive behaviour, they can **move back to an earlier stage**, learn from the experience and begin the process again. This makes the model more realistic than approaches that assume everyone moves through recovery in exactly the same order.

## AO3 - EVALUATION

A strength of Prochaska and DiClemente's six-stage model is that it recognises **recovery from addiction as a dynamic and non-linear process**. Unlike approaches which suggest that individuals move through a fixed sequence, the model allows people to **recycle through stages, move backwards or even skip stages**. This reflects the fact that recovery is often influenced by changing circumstances, motivation and individual differences, rather than following one predictable pathway. This makes the model a **more realistic account of addictive behaviour**, as it can explain why two people attempting to overcome the same addiction may progress in very different ways. It also avoids suggesting that a person has permanently failed simply because they have moved backwards.

A further strength is that the model has **important practical applications for the treatment of addiction**. The model treats **relapse as a normal part of the recovery process**, rather than viewing it as evidence that treatment has failed. This is important because people who relapse may otherwise experience **guilt, hopelessness or reduced self-efficacy**, increasing the likelihood that they will abandon attempts to stop the addictive behaviour. By recognising relapse as an opportunity to identify difficulties and return to an earlier stage, the model can help individuals **maintain motivation and continue treatment**. It can also help therapists **tailor interventions to the individual's current stage of readiness**, making treatment more appropriate to their needs. Therefore, the model has value beyond explaining addiction because it can **guide how recovery is managed in practice**.

A limitation of the model is that there is evidence to suggest that using the stages does **not necessarily make treatment more effective**. Taylor et al. (2006) and Cahill et al. (2010) reviewed evidence considered by NICE and found that alternative approaches to treating **nicotine addiction were equally effective** as stage-based approaches. This suggests that although identifying an individual's stage may help therapists understand their readiness to change, it may not actually result in **better treatment outcomes**. If stage-based treatment is no more successful than alternative approaches, this reduces the model's **practical usefulness** and suggests that it may be more valuable as a **theoretical framework** than as a basis for deciding which treatment should be used.

A further limitation is that the model may **oversimplify the process of addiction and recovery**. Although the model allows individuals to move backwards and forwards, it still divides recovery into **six broad stages**, which may not capture the complexity of addictive behaviour. Addiction can be influenced by **biological factors, psychological processes, social influences and environmental circumstances**, all of which can change over time. Someone may also show characteristics of more than one stage at the same time, making it difficult to place them accurately into a single category. This means the model may give an **overly simplified picture of recovery**, potentially ignoring important individual differences in why people become addicted, why they relapse and what helps them to recover.

