SimplyPsychology

A-LEVEL PSYCHOLOGY REVISION NOTES

Aggression

AQA Psychology 7182 (A-level only)

2025 specification · spec section 4.3.8 · A-level Paper 3

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

Contents

2025 spec: **no content was removed** from the Aggression topic in the 2025 specification — the whole topic remains examinable.

AQA 2025 SPECIFICATION — AGGRESSION (4.3.8)

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- **Ethological explanation**: innate releasing mechanisms and fixed action patterns. **Evolutionary explanations** of human aggression.
- **Social psychological explanations**: the frustration-aggression hypothesis; social learning theory; de-individuation.
- **Institutional aggression** in prisons: dispositional and situational explanations.
- **Media influences**: the effects of computer games; desensitisation, disinhibition and cognitive priming.

1 Neural and Hormonal Mechanisms

The Limbic System

The **limbic system** — especially the **amygdala** — is central to how we process and respond to threat. A reactive amygdala is associated with **reactive aggression**: studies in animals show that stimulating it produces aggression and damaging it reduces it, and **Gospic et al. (2011)** found heightened amygdala activity when participants reacted aggressively in a provocation task.

Serotonin

Normal levels of **serotonin** have a calming, inhibitory effect on the prefrontal cortex, helping to control impulses. **Low serotonin** reduces this inhibitory control, making impulsive aggression more likely.

Testosterone

Testosterone, an androgen, is linked to aggression — particularly in males, who are generally more physically aggressive. **Dabbs et al. (1987)** found higher salivary testosterone in offenders who had committed violent crimes than in those who had committed non-violent crimes.

Evaluation

A strength of the neural explanation is supporting evidence for the role of serotonin from drug studies. Research shows that drugs which increase serotonin activity tend to reduce aggressive behaviour, and that low serotonin is found in violent individuals. Because this evidence comes from experimental manipulation rather than mere correlation, it allows a cause-and-effect inference, strengthening the claim that serotonin genuinely influences aggression rather than simply co-occurring with it.

However, a key limitation is that much of the hormonal evidence is only correlational. Studies such as Dabbs et al. show that high testosterone is *associated* with violence, but they cannot establish the direction of causation: winning a confrontation can itself raise testosterone, so aggression may cause raised testosterone rather than the reverse. This means the testosterone–aggression link, although consistent, does not by itself prove that testosterone causes aggression.

A counterpoint that further limits the approach is that it is biologically reductionist. Explaining aggression purely through the amygdala, serotonin and testosterone reduces a complex social behaviour to neurochemistry and ignores well-evidenced social factors such as de-individuation, social learning and media influence (Sections 4 and 6). Aggression almost always occurs in a social context, so the fullest explanation is **interactionist**, combining biological predispositions with environmental triggers.

A further issue is the over-reliance on animal research, which raises validity and ethical concerns. Much of the evidence on the limbic system comes from animal lesion and stimulation studies. While these allow tight control, human aggression is shaped by cognition, culture and intention in ways that animal

aggression is not, so extrapolation is questionable — and such procedures raise ethical questions about harm to animals that must be weighed against the scientific benefit.

2 Genetic Factors in Aggression

Twin and Adoption Studies

Aggression shows higher concordance in **MZ than DZ twins**, and adoption studies find that adoptees' aggression correlates with their biological relatives — both pointing to a heritable component.

The MAOA Gene

> **THE MAOA GENE ("WARRIOR GENE")**
>
> The **MAOA gene** controls production of the enzyme **monoamine oxidase A**, which breaks down neurotransmitters (e.g. serotonin) after transmission. A **low-activity variant (MAOA-L)** leaves abnormal neurotransmitter levels and is associated with higher aggression.

- **Brunner et al. (1993)** — studied a Dutch family, many of whose male members were involved in violent crime and had a malfunctioning MAOA gene.
- **Caspi et al. (2002)** — MAOA-L was linked to aggression mainly in men who had also experienced **childhood maltreatment**, showing a gene–environment interaction.

Evaluation

A strength of the genetic explanation is converging evidence from family, twin and gene studies. Brunner's study of a violent family with an MAOA deficiency, combined with twin concordance data, points consistently to a heritable contribution to aggression. Because these findings come from different methods that triangulate on the same conclusion, they make a stronger case for a genetic basis than any single study could.

However, a major limitation is that genes alone cannot explain aggression — the relationship is interactionist. Caspi et al. (2002) found that the MAOA-L variant predicted aggression mainly in those who had *also* suffered childhood maltreatment, and not in those raised in supportive environments. This is powerful evidence for a **diathesis-stress / nature–nurture** account: a genetic vulnerability is only expressed when combined with adverse experience, so a purely genetic explanation is incomplete.

A further limitation is that genetic explanations of aggression are socially sensitive and raise issues of determinism. Labelling MAOA-L a "warrior gene" implies some people are biologically destined to be violent, which has already been used in court as a mitigating defence. This is problematic because it risks undermining the principle of free will and personal responsibility on which the justice system rests, and could stigmatise individuals or groups — so such research must be communicated with great care.

On the other hand, this research has potential applied value. Understanding genetic and gene–environment risk could, in principle, help identify those most vulnerable to developing aggressive behaviour and target early interventions (e.g. support for maltreated children carrying the risk variant). Reducing later violent offending in this way would carry substantial benefits for individuals and major economic savings for the criminal-justice system.

3 Ethological and Evolutionary Explanations

The Ethological Explanation

Ethology studies animal behaviour in natural settings and views aggression as **innate and adaptive** — it spaces animals out over territory and establishes dominance hierarchies.

- **Innate releasing mechanism (IRM)** — a built-in neural mechanism activated by a specific **sign stimulus** (e.g. a rival's colouring).
- **Fixed action pattern (FAP)** — the IRM triggers a stereotyped, universal, innate sequence of behaviour that, once started, runs to completion ("ballistic"). **Lorenz** noted aggression is often *ritualistic*, ending in appeasement rather than death.

Evolutionary Explanations

Human aggression is seen as **adaptive** — it was naturally selected because it helped ancestors survive and reproduce: competing for **resources and status**, and, in males, **sexual jealousy** and **mate retention** in response to paternity uncertainty (Wilson & Daly's mate-guarding behaviours).

Evaluation

A strength of the ethological approach is evidence that fixed action patterns are genuinely universal. Tinbergen showed that male sticklebacks would perform the same aggressive FAP towards any model displaying a red underside (the sign stimulus), regardless of its shape. This supports the claim that such aggression is innate and stimulus-driven rather than learned, lending the approach scientific credibility.

However, a limitation is that aggression is not as fixed or universal as the theory claims. Research shows aggressive behaviour varies considerably between cultures and even individuals — for example, the !Kung San are notably non-aggressive, whereas some cultures actively reward aggression. This variation suggests aggression is shaped by learning and environment, not just innate releasing mechanisms, which undermines the idea of a rigid, species-universal FAP.

A further limitation, particularly of the evolutionary account, is that it can be reductionist and risks justifying aggression. Explaining male violence as an evolved adaptation can be criticised as a "just-so story" that is difficult to falsify, and as **biologically deterministic** — implying aggression is natural and perhaps excusable. This is socially sensitive, especially where the theory is applied to male sexual jealousy and partner violence, so its conclusions must be treated cautiously.

A counterpoint, however, is that the evolutionary approach can explain real-world patterns that other theories struggle with. It accounts for consistent findings such as the prevalence of male-on-male violence among young men competing for status, and the link between sexual jealousy and domestic violence. The

ability to explain these robust patterns gives the approach genuine explanatory value, even if it cannot be the whole story.

4 Social Psychological Explanations

The Frustration-Aggression Hypothesis

Dollard et al. (1939) proposed that frustration (being blocked from a goal) always produces a drive towards aggression, which is often **displaced** onto a safer target. **Berkowitz** revised this: frustration creates a *readiness* for aggression that environmental **aggressive cues** (e.g. weapons — the "weapon effect") then trigger.

Social Learning Theory (Bandura)

Aggression is learned by **observing** and **imitating** models, especially when the model is rewarded (**vicarious reinforcement**). Learning is mediated by attention, retention, reproduction and motivation. The **Bobo doll** studies showed children imitated observed aggression.

De-individuation

In crowds, **anonymity** and reduced self-awareness produce a loss of personal identity and a reduced sense of **accountability**, releasing aggression that would normally be inhibited.

Evaluation

A strength of social learning theory is strong experimental support from Bandura. In the Bobo doll studies, children who watched an adult behave aggressively imitated that aggression, and (in later versions) imitated more when the model had been rewarded. Because the level of aggression was manipulated and measured under controlled conditions, this provides convincing evidence that aggression can be acquired through observation and vicarious reinforcement, exactly as SLT predicts.

However, a counterpoint is that the Bobo doll research has serious methodological weaknesses. The studies were laboratory experiments vulnerable to **demand characteristics** (children may have guessed they were meant to hit the doll), and a Bobo doll is designed to be hit and cannot retaliate as a real person would. This casts doubt on the **ecological validity** of the findings as evidence for real interpersonal aggression, weakening the support they provide.

A strength of de-individuation theory is supporting research and clear real-world application. Studies such as Diener et al.'s Halloween study found anonymous, grouped children behaved more antisocially, and analyses of crowd and football violence fit the theory. This has practical value: measures that reduce anonymity — CCTV, name badges, identifiable uniforms — should reduce aggression, offering an economically useful way to curb crowd violence and its policing costs.

A limitation common to these explanations is that they are reductionist and ignore biology. By focusing solely on situational and social factors, they neglect the neural, hormonal and genetic influences (Sections 1–2) that clearly contribute to aggression — for example, why some frustrated or anonymous individuals become

aggressive and others do not. As with most explanations of aggression, an **interactionist** account that integrates social and biological factors is the most complete.

5 Institutional Aggression in Prisons

The Dispositional Explanation — The Importation Model

Aggression in prison is "imported": inmates bring their own violent **dispositions, traits, values and histories** into the institution. People who were aggressive outside prison continue to be aggressive inside it, so violence reflects who the inmates *are*.

The Situational Explanation — The Deprivation Model

Aggression is caused by the **prison environment** itself. **Sykes** described the "pains of imprisonment" — deprivation of liberty, autonomy, goods and services, security and heterosexual relationships. Stressful, overcrowded conditions and a lack of meaningful activity generate aggression regardless of the inmates' dispositions.

Evaluation

A strength of the situational (deprivation) model is supporting evidence linking conditions to violence. Research has found that overcrowding, heat and a lack of privacy are associated with higher rates of inmate violence, and reducing these "pains of imprisonment" tends to reduce aggression. Because manipulating conditions changes the violence, this suggests the prison environment is a genuine cause, supporting the situational account and pointing to a practical, economically valuable route to safer prisons.

However, a counterpoint is that the dispositional (importation) model also has clear support. Studies show that inmates with prior gang membership or violent histories are more likely to be aggressive in prison, which the deprivation model cannot easily explain. This indicates that the characteristics inmates bring with them matter too, so neither model is sufficient on its own.

This leads to the wider point that the two explanations are best seen as complementary rather than competing. The most convincing account is **interactionist**: aggression results when individuals with violent dispositions (importation) are placed in depriving, stressful environments (deprivation). Treating the models as mutually exclusive is a false dichotomy that oversimplifies a complex phenomenon.

A further strength is the real-world and economic value of this research. If situational factors drive violence, then improving conditions, reducing overcrowding and providing meaningful activity should make prisons safer for staff and inmates. Lower levels of violence reduce the considerable costs of injuries, staff absence and damage, so the research has direct practical benefits for prison management and public spending.

6 Media Influences on Aggression

The Effects of Computer Games

Violent computer games are of particular concern because they are **interactive** and players are often **rewarded** for aggression. Evidence is mixed: experimental studies sometimes find short-term increases in aggression, while longitudinal and correlational studies produce inconsistent results.

Three Explanatory Mechanisms

Mechanism	How it increases aggression
Desensitisation	Repeated exposure to media violence reduces the emotional and physiological response to it, so aggression comes to seem normal and less wrong.
Disinhibition	When media portray violence as acceptable, justified or rewarded, the usual social constraints against aggression are weakened.
Cognitive priming	Violent media provide aggressive "scripts"/schemas that are stored in memory and later triggered by environmental cues.

Evaluation

A strength of media-effects research is supporting experimental evidence for the mechanisms. Laboratory studies have shown that participants exposed to media violence show reduced physiological arousal to later violence (desensitisation) and respond faster to aggressive words (cognitive priming). Because these mechanisms are demonstrated under controlled conditions, they provide a credible, testable account of *how* media might influence aggression, rather than merely asserting that it does.

However, a major limitation is that much of the evidence is laboratory-based and lacks ecological validity. Aggression in these studies is usually measured artificially (e.g. blasts of noise or button presses) rather than as real violence, and the effects observed are typically short-term. This means we cannot be confident that the same processes produce *real-world* aggression, so the practical significance of the findings is uncertain.

A counterpoint is that field and longitudinal research produces inconsistent and generally small effects. Long-term studies often find weak or no links between media violence and serious aggression once other factors are controlled, and meta-analyses disagree on effect sizes. This suggests media influence — if real — is modest and operates alongside more powerful factors such as upbringing and disposition, rather than being a major cause of violence.

Finally, this area is socially sensitive and prone to confounding, so conclusions must be cautious. The debate feeds directly into public policy and calls to censor games, yet the direction of causation is unclear: aggressive individuals may simply be drawn to violent media (a **"socialisation" vs "selection"** problem).

Overstating media effects risks unjustified censorship and distracts from better-evidenced causes of aggression, so claims need to rest on strong, real-world evidence.

These notes were prepared for Simply Psychology and cover spec section 4.3.8 of the AQA Psychology 2025 specification (A-level only, Paper 3). No content was removed from Aggression in the 2025 specification. For deeper coverage, see simplypsychology.org/aggression.html.