**Genetic Explanations**

Genetic Factors in Aggression, including the MAOA Gene

**AO1- Description: How are Genetic Factors Studied?**

(1) Twin Studies:

Several studies have suggested that heritability accounts for about 50% of the variance in aggressive behaviour. Monozygotic (identical) twins share 100% of their genes, while dizygotic (non-identical) twins share only about 50%, as a result we would expect to find greater similarities in aggressive behaviour between MZ twins if aggression is mostly influenced by genetic factors. Examples of concordance rates for different types of aggression are below.

 Physical aggression MZ=50% and DZ=19%, Verbal aggression MZ=28% and DZ=7%.

(2) Adoption studies:

Adoption studies can also help to separate out the effects of genes and environment. If a positive correlation is found between aggressive behaviour in adopted children and their biological parents, then this is suggestive of genes because the environmental factor has been removed.

 Meta-analysis was carried out looking at adoption and its relationship to direct aggression and antisocial behaviour. They found that genetic influences accounted for 41% of the variance in aggression.

**MAOA Gene (AO1 – Description)**

What is MAOA?

 A gene (monoamine oxidase A), that has been implicated in aggression, mice that possessed the gene were extremely aggressive. The MAOA gene is thought to affect neurotransmitters.

**How does the MAOA gene link to aggressive behaviour?**

• The gene determines the production of the enzyme MAOA

• MAOA metabolises (‘mops up’) neurotransmitters (e.g. serotonin, dopamine and noradrenaline) in the brain after a nerve impulse, it breaks them down to be recycled or excreted.

• A dysfunction in the operation of this gene may lead to abnormal activity of the MAOA enzyme, which in turn affects levels of neurotransmitters in the brain.

• People with the low-activity form (MAOA-L) produce less of the enzyme and this is correlated with aggression. The high-activity form (MAOA-H) is not correlated with aggression.

**Research showing a link between aggression and the MAOA gene (AO3 – Evaluation):**

Han Brunner et al (1993) studied 28 males from a large Dutch family who were repeatedly involved in impulsively aggressive violent criminal behaviour. It was found that these men had abnormally low levels of MAOA in their brains and the low-activity version of the MAOA gene. This research supports the role of MAOA in aggression as when this gene is not functioning correctly, individuals display aggressive behaviour.

**MAOA Gene – AO3 – Evaluation:**

Caspi - Aims

To test the hypothesis that the presence of the low-activity MAOA genotype was associated with an increased response to abuse exposure.

Method

Longitudinal study on over 1000 boys over 26 years and measured, MAOA variants, antisocial behaviour to age 30; and exposure to childhood sexual and physical abuse.

Results

Regression models were fitted to five antisocial behaviour outcomes (self-reported property offending; self-reported violent offending; convictions for property/violent offending; conduct problems; hostility) observed from age 16 to 30, using measures of childhood exposure to sexual and physical abuse. The analyses revealed consistent evidence of G × E interactions, with those having the low-activity MAOA variant and who were exposed to abuse in childhood being significantly more likely to report later offending, conduct problems and hostility.

Conclusions - The present findings add to the evidence suggesting that there is a stable G × E interaction involving MAOA, abuse exposure and antisocial behaviour across the life course. Interaction of MAOA problem AND abusive childhood led to aggression. If boys with the MAOa – L gene suffered abuse in childhood, they were 3 times more likely to be aggressive when they reached adulthood.

**AO3: IDA / Level 4 evaluation**

A weakness of attempting to explain aggressive behaviour in terms of genetic factors is that these explanations are highly deterministic. If an individual inherits a particular gene that predisposes them to act aggressively the assumption is that they will inevitably be more likely to be more aggressive or violent as a result, ignoring the role of free will. Even explanations that consider the role of environmental triggers where there is a genetic predisposition are limited in this way. In the case of genetic low MAOA combined with childhood mistreatment for example, the assumption is that aggression is inevitable when both the genetic and environmental conditions are set in place for aggression. As both the genetic and environmental factors have exerted their influence well before adulthood, there appears to be little an individual can do to avoid being aggressive where they have been dealt this hand.

The determinism also seems to suggest that violence is a behaviour that cannot be helped and perhaps in some way excuses or even “medicalises” aggressive behaviour that is the result genetic factors. For example, lawyers have used the presence of low MAOA and childhood mistreatment as a defence for murder in a few cases. As the validity and reliability of the evidence to support this idea is flawed this is a potentially dangerous practice.

AO3 Point: There are potential issues in how aggressive behaviour is measured

Evidence: For example, aggression is often measured through questionnaires or responses to a hypothetical scenario, and therefore predictive validity is poor.

Elaboration: This is a weakness because such measures of aggression are subjective (often influenced by individual judgement) and therefore it is difficult to draw conclusions about aggression from research that has used such measures.

Self report methods tend to show genetic links whereas observations tend to suggest environmental factors causing aggression. Are we accurately measuring aggression?