***Genetic Factors in Aggression***

There are several ways in which genetic factors in aggression have been investigated. In the 1960s and 70s a popular theory was that males who have inherited an extra Y chromosome are more 'male' and therefore, more aggressive; however, this hypothesis as been shown to be incorrect. Nevertheless, animal breeding studies consistently show that it is possible to select for aggressiveness and twin and adoption studies in humans suggest that genes influence aggressive behaviour; more recently, psychologists and biologists have developed a better understanding of how genes can influence behaviour by using new technology to investigate the genetic mechanisms that lead to increased aggressiveness.

**XYY Karyotype**

During the 1960s William Court-Brown and his colleagues carried out chromosome studies on 315 patients in a maximum security hospital for patients with dangerous, violent, or criminal propensities in Carstairs, Scotland. Out of the 315 patients, 15 were found to have chromosomal abnormalities, including 9 who had an extra Y chromosome. Since the Y chromosome is the male sex chromosome and the incidence of XYY in the general population is 1 in 1000, this suggests that having an extra Y chromosome may have predisposed these men to increased aggressive behaviour. Nevertheless, six other patients in the sample had chromosomal abnormalities, including one with an XXY karyotype and one with a condition known as mosaicism, where one individual has more than one set of genes.

Further research into the XYY karyotype has revealed that although XYY participants are on average taller than the general population, there is no evidence of increased aggression (Milunsky, 2004) or higher than average testosterone levels (Ratcliffe et al., 1994). This suggests that the XYY karyotype is not related to aggression at all.

**Animal Breeding Studies**

In animal breeding studies, animals are selectively bred for certain traits. If it is possible to breed for aggression, this would suggest that genes contribute to aggressiveness.

One of the earliest animal breeding studies to investigate aggression was carried out by Russian scientist Dmitri Belyaev in the 1950s. Belyaev's aim was to reduce the aggressiveness of Silver Foxes to make them easier to breed for their much sought after silver fur. He found that after 18 generations the animals were tame: they approached humans, barked like dogs and even rolled over to have their tummies tickled. Unfortunately for Belyaev, the animals even looked like dogs, with floppy ears and a piebald coat – not the silver coat that the fur industry required. The silver fox breeding programme was also able to breed extremely viscious silver foxes, which supports the hypothesis that genes are involved in aggression.

Other studies have shown that it is possible to breed highly aggressive male and female mice (Cairnes, 1983). However, Cairnes found that the aggressiveness was linked to a particular developmental stage: the animals were only aggressive during mid life, but not during early or late life. Nevertheless, the study does show that genes can affect aggressiveness.

A more direct approach has shown that mice bred to lack a receptor for the neurotransmitter serotonin attack with twice the intensity of those with the gene. This fits with research in humans that aggression increases when serotonin levels are low.

A problem with research on animals is that it is difficult to generalise the results to humans, nevertheless, mice are often used in genetic studies because their genes are homologous to those of humans, this means they produce the same proteins, which are used in a similar way.

**Heritability Studies**

Research on humans has also supported the notion that genes are an important factor in aggressiveness. Rutter et al. (1990) carried out a meta-analysis of twin studies on criminality and found that Dizygotic Twins have concordance rates between 13 & 22%, while Monozygotic Twins have concordance rates between 26 & 51%. Since both types of twins grow up together in the same environment, the differences between the two are likely to be due to genetics. However, it is important to consider that as monozygotic twins do not show a 100% concordance rate there must be an environmental contribution to aggressiveness.

An adoption study by Mednick, Gabrielli & Hutchings (1987) shows an interesting interaction between genes and the environment. The researchers studied the criminal records of all Danish children adopted outside their biological family between 1924 and 1947. They reasoned that if the criminal records of adopted children were more similar to those of their biological parents than their adopted parents then this would suggest a genetic component in aggression. The results did show that having a criminal biological father increased the risk of criminality, but the highest risk was for those with a criminal biological father and a criminal adoptive father.

The heritability studies above, although providing support for a genetic contribution to aggressiveness are limited in that they studied criminality, rather than aggressiveness and not all crime is violent. In fact, Mednick and his colleagues reported that the largest effect in their study was for non-violent crime.

**Molecular Genetics**

New technology has enabled researchers to examine DNA at the molecular level. Researchers are now able to investigate the proteins a particular sequence of DNA codes for, which cells in the body express those proteins and the role of that protein in the body. Molecular geneticists are then able to collaborate with psychologists to examine any effects of genetic differences on behaviour.

This type of research has produced a number of breakthroughs in the effect of genes on aggressive behaviour:

**MAOA Promotor**

The promotor region of a gene is a sequence of DNA that promotes gene transcripton, which is the first step in the process of gene expression; gene expression is the process by which a gene creates a protein. Differences in the promotor region of the MAO-A gene have been associated with increased aggressiveness. The MAO-A gene produces Monoamine Oxidase, a chemical that is involved in the breakdown of the neurotransmitters serotonin, dopamine and norepinephrine in synapses.

There are a number of versions (alleles) of this gene, each with a different number of repeats of a particular sequence of DNA in the promotor region. The number of repeats of this sequence determines the amount of MAO produced. The shorter version (2 repeats), which produces less MAO, is associated with high aggression and has been called the “warrior gene” by some researchers.

It is thought that having too little MAO leads to the brain is flooded with too much serotonin, norepinephrine and dopamine, which eventually leads to a lowered sensitivity to these neurotransmitters. Other studies have shown low serotonin activity to be associated with aggression and this fits with the data, as low sensitivity is equivalent to low activity.

Moffat et al. (2002) conducted a longitudinal study of 422 males in New Zealand. He studied their history of abuse and criminal convictions, their penchant for violence and any symptoms of antisocial personality disorder. His findings showed that the 2 repeat MAOA allele did increase the risk of being convicted of a violent crime; however, this was only when participants also suffered abuse as children. This is a clear example of a gene-environment interaction, the gene only exerts its effect in particular environmental conditions.

[Video Clip: Jim Fallon on the "Warrior Gene"](http://alevelpsychology.co.uk/component/option%2Ccom_video/Itemid%2C2889/cat%2C12/feature%2Cyoutube_gdata/s%2C1/vid%2Cu2V0vOFexY4/)

More recent research by Guang Guo and his colleagues (2008) has revealed further insights into the effect of genes on aggression. Saliva samples were taken from 1,100 males in grades 7 through 12 whose DNA and social-control measures were available through the National Longitudinal Study of Adolescent Health. Guo and his colleagues found similar results to Moffat et al. with the two-repeat MAOA allele being associated with violent delinquency, but only when participants experienced failure at school and had to repeat a year.

Guo et al. also found that that an allele of a DRD2 gene (178/304) was associated with violent delinquency. The DRD2 is the gene that manufactures the D2 dopamine receptor. A gene environment interaction was also found with the DRD2 gene: there was only an association with delinquency when parents did not have regular meals with the adolescents. Having only one biological parent at home also dramatically increased the rate of delinquency in those with the 178/304 allele. This suggests that parental involvement in the child's development is a critical factor in those with this particular genetic risk factor for aggression.

Another gene identified by the Guo et al study was the DAT1 gene, which codes for a dopamine transporter. Transporters remove neurotransmitters from synapses and stop them exerting their action. The research showed that inheriting a 9 repeat allele of DAT1 from both parents lowered the incidence of aggressive and risky behaviour.

**Conclusion**

There is a great deal of evidence that genes do play a significant role in aggressiveness. Animal breeding studies have shown that it is possible to select for aggressive behavioural traits and family studies in humans have shown that aggressiveness is highly heritable. Some of the actual genetic mechanisms responsible for aggression have been revealed by molecular genetics; however, the importance of environmental factors has also been highlighted by researchers such as Guang Guo. It appears that genes do increase the risk of aggression, but only when combined with enviromental risk factors, such as abuse and a neglectful family environment.