HL IB Psychology

Genetic Similarity

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Genetic Similarity as an Explanation for Behaviour

What is genetic similarity?

- Human beings are 99.9% **genetically** similar to each other i.e. you are almost 100% identical to every other person on the planet
- However, as we know from experience, there is huge variety amongst human beings in terms of physical appearance, skills, abilities, personalities etc.
- The only truly **DNA**-identical people in the world are **monozygotic (MZ)** twins whose birth is the result of one egg splitting post-**fertilisation** (from one **sperm**)
- MZ twins inhabit two, separate identical embryos that share 100% of their DNA
- **Dizygotic (DZ)** twins are the result of two separate eggs being fertilised by two separate sperm and thus share 50% of their DNA

Why are twins so useful for genetic similarity research?

- MZ twins - because they have 100% shared DNA, they act as each other's **control group** in research which is looking for a **biological basis to behaviour**
- Twin studies begin by identifying a **proband** and then using the second twin as a comparison
- Studies which use **concordance rates** use MZ and DZ twins to look for evidence of specific behaviours being inherited e.g. rates of depression in MZ twins are compared to rates of depression in DZ twins
- Genetic similarities are useful for determining whether there is a biological explanation for a behaviour or whether the environment is also a key contributor to the behaviour being investigated

Which other types of genetic similarity have been investigated?

- **Adoption studies** look for concordance rates between adopted children and their biological parents compared to their adoptive parents
- Adopted children share no genes with their adoptive parents therefore any similarities between them are thought to be caused by the environment
- **Family studies** look at inherited traits within the same family i.e. people can inherit conditions which predispose them to particular behaviours or conditions

Which studies investigate genetic similarity?

- **McGuffin et al. (1996)** – a twin study using concordance rates for depression in MZ and DZ twins
- **Brunner et al. (1993)** – a family study in which a dysfunctional MAOA gene was linked to anti-social behaviour seen in the affected males of a large family in the Netherlands

The studies by McGuffin et al. (1996) and Brunner et al. (1993) can be found in 'Two Key Studies of Genetic Similarity' on this site: just navigate the Genetics & Behaviour topic to find it

Worked Example

ERQ (Extended Response Question) – 22 marks

'To what extent could genetic similarity be said to affect behaviour?' [22]

The following paragraph addresses the command term 'To what extent' by assessing how successful the research is in explaining behaviour:

Twin studies such as Mc Guffin et al. (1996) use concordance rates to measure the key variable of interest, in this case depression, as experienced by MZ and DZ twins. The findings of this study could provide some evidence for the idea that depression has a biological basis i.e. that the outcome of one MZ twin can be determined by the outcome of the other MZ twin due to their identical DNA. What this study does not do is to provide clear biological evidence alone in support of nature over nurture. The fact that a higher concordance rate for depression in MZ twins was found may instead be based on the fact that MZ twins are more likely to spend greater amounts of time together than DZ twins (who may be of the opposite sex).

Two Key Studies of Genetic Similarity: McGuffin (1996) & Brunner et al. (1993)

Key Study: McGuffin (1996)

Aim: To investigate **concordance rates** for depression in **MZ** and DZ twins.

Participants: 177 twin **probands** and who were registered between 1948 and 1986 with the Maudsley Hospital in London as suffering from depression. Their same-sex twin siblings were used as comparison participants.

Procedure: The twins were assessed by **clinicians** via a series of tests and **interviews** by **blind** researchers i.e. the researchers were unaware as to whether the twin was MZ or DZ and whether they had depression or not. The **data** also consisted of the twins' medical records.

Results: MZ twins showed a 46% concordance rate for depression compared to 20% in DZ twins. Shorter periods of depression in one MZ twin was matched by depression in their twin sibling. MZ twins aged 65 showed heightened levels of depression compared to the general population.

Conclusion: Depression may be highly **heritable** rather than a product of environmental factors. Short-term depression in one MZ twin appears to increase the probability of their MZ twin sibling also developing depression, which reinforces the idea that depression is genetic.

Evaluation of McGuffin et al. (1996)

Strengths

- This was a **longitudinal** study, conducted over decades which means that it is high in validity as the participants could be tracked over time to look for real differences in behaviour
- The use of a blind interviewer means that the study was free from bias which increases the validity of the findings

Limitations

- The concordance rate for MZ twins was 46% - if depression is entirely genetic then it should show a 100% concordance rate
- This research was conducted prior to **DNA** testing was available so it is possible that some of the MZ twins may actually have been DZ i.e. they may have looked identical but in fact have been biologically non-identical

Key terms:

- **MZ**
- **DZ**
- **Proband**

Key Study: Brunner et al. (1993)

Aim: To investigate the violent, anti-social behaviour of specific male members of a large family in the Netherlands. The behaviour exhibited by the males in the family was borderline **mental retardation** (their average **IQ** was around 85), and violent behaviour.

Participants: 5 males from a family in the Netherlands, all of whom had the same **genetic** condition, transmitted via the **X chromosome** on the **MAOA gene**. The family lived in a remote rural region of the Netherlands. Two **carrier** females and one **non-carrier** female were used as a control and compared with 3 clinically affected males.

*(Carrier means that some of the females carried the faulty gene in their **genotype** but it was not expressed in the **phenotype** i.e. their behaviour)*

All of the affected males acted aggressively when angry, fearful, or frustrated. Examples of their violent, anti-social behaviour included attempted rape of one of the female members of the family, arson, attacking a mental institute warden with a pitchfork, voyeurism (spying on the females in the family at night), exhibitionism (appearing naked in public). Only one of the males in the family with the faulty gene finished primary education.

Procedure: A **case study** (close study of a small group of individuals from one family) and quasi-experiment. A **quasi-experiment**is one in which the IV is naturally occurring i.e. it can't be manipulated by the researcher – in this case the individuals involved either had the faulty gene or they didn't have the faulty gene. Brunner conducted DNA analysis, obtained via urine samples. Observations of the males and interviews with the family provided qualitative data.

Results: None of the affected males had **dysmorphic** signs of the genetic mutation i.e. they didn't 'look abnormal' or different physically to the unaffected males. Unaffected males in this family attended normal schools, and most had steady jobs. All the females (including several carriers) also functioned normally.

A **base change** in the DNA structure was identified in all 5 affected males. This in turn resulted in flawed **monoamine metabolism**, which is linked with a deficit of the **enzyme monoamine oxidase A (MAOA)** – an enzyme which (among other functions) regulates the supply of **serotonin** levels to the brain. The reason only males are affected is because it is specifically the single X chromosome which is responsible for the production of MAOA.

Conclusion: The dysfunctional MAOA gene may be linked to irregular serotonin **metabolism** which could in turn be responsible for the mental retardation and aggressive behaviour of the affected males. MAOA deficiency may account for an individual's inability to regulate their aggression. This MAOA deficiency is now known as **'Brunner syndrome'**

Evaluation of Brunner et al. (1993)

Strengths

- This research was highly influential and resulted in the faulty MAOA gene being known as 'Brunner Syndrome'
- By using one extended family the researchers were able to directly test their theory by using family members as **control** samples rather than an unrelated general population, thus validating the idea that the males' behaviour was **genetic** rather than as a result of their **environment**

Limitations

- There are some **ethical concerns** arising from this study e.g. could the affected males give fully informed consent considering their low IQ?
- The affected males may have encountered more adverse reactions from others e.g. hostility, aggression, confrontations due to their reduced IQ and lack of impulse-control which could have exacerbated their anti-social tendencies i.e. **nurture** may have influenced their behaviour as well as **nature**

Key terms:

- **X Chromosome**
- **MAOA gene**
- **Serotonin**