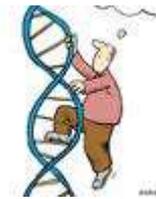
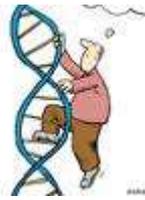


# Biological explanations of schizophrenia



Genetics



# Genetics

A01

The fact that schizophrenia tends to run in families led to the conclusion that it has a genetic basis.

According to the genetic hypothesis, the more closely related the family member to the schizophrenic, the greater their chance of developing the disorder.

As you can see...inheriting a gene... does not mean you **WILL HAVE** blonde hair (or schizophrenia)...but by studying genetic links in families, it can help us to predict the likelihood of that feature occurring.



# Genetics - evidence

A01



Much relevant evidence comes from...

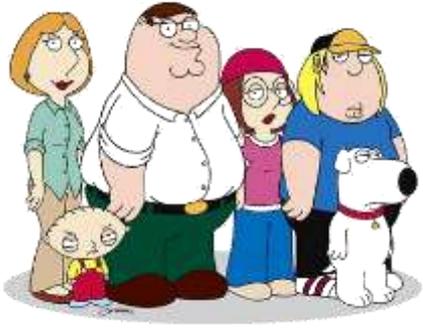


Concordance rates:  
These show the percentage of family members developing schizophrenia.



# Genetics – family studies

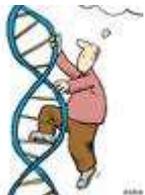
A01



**Family studies:** Show that the risk of developing schizophrenia is greater for those more closely related to the schizophrenic.

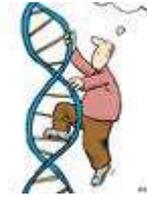
**AO1:**

**Kendler et al (1985)** found that the first-degree relatives of those with schizophrenia were 18 times more at risk of developing the disorder.



**X 18**

# Genetics – family studies



A01/2



If there was **no genetic element** then there should be no difference in the level of risk between first degree relatives and a random member of the public.

**AO2:**

**However, Gottesman (1991)**

provided **supporting** evidence for the genetic explanation using family studies.

- 1) Both parents = **46%** risk
- 2) One schizophrenic parent = **16%** risk
- 3) If a sibling has schizophrenia = **8%** risk
- 4) If a grandparent has schizophrenia = **5%** risk
- 5) If the identical twin of one parent has schizophrenia = **17%** risk.

***The comparison:***

there is a **1%** probability of schizophrenia in someone selected at random.

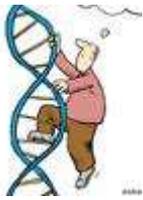
**1%**

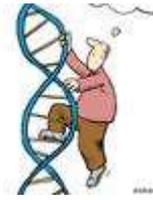
# Genetics – family studies

A02

Using family studies can you think of any evidence against the genetic explanation?

- 1) The fact that concordance rates increase with genetic relatedness may be explained by the fact that this is because people in families are also likely to spend more time together, which means that environmental factors may be influential.
- 2) Less than 50% of children where both parents have schizophrenia develop the disorder, which can be used as evidence against a direct genetic link.
- 3) Psychological explanations can be used against the Biological explanations (for example????)





# Genetics – family studies *recap*

A01

## 1. Define Concordance rate

### Concordance rate:

This is the extent to which twins/ family members are similar and they show the likelihood of family members developing a particular disorder.

## 2. Identify the 4 types of evidence used to study genetic explanations:

1. Family studies; 2. Twin studies;  
3. Adoption studies; 4. Gene mapping.

## 3. What did Kendler et al (1985) find?

**Kendler et al (1985)** found that the first-degree relatives of those with schizophrenia were 18 times more at risk of developing the disorder.

**X 18**

# Genetics – twin studies

A01



**Gottesman (1991)** reviewed 40 twin studies and found:

**48%** concordance for MZ twins

**17%** concordance for DZ twins

**Shields (1962)** found that **Concordance rates** for MZ twins brought up apart are similar to those brought up together.

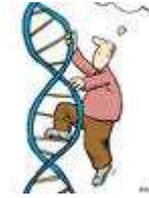
Elaboration

AO2

***This suggests*** a genetic link for schizophrenia because the likelihood of developing schizophrenia appears to be correlated with genetic relatedness. If there was no genetic factor then there should be no difference between MZ and DZ twins. The fact that there is a difference suggests a strong genetic link for schizophrenia.



# Genetics – twin studies



A02

## Can you think of any criticisms of this piece of evidence?

The higher concordance rate in MZ twins may be due to the fact that MZ twins tend to be treated more similarly than DZ twins and so **NURTURE** may explain the concordance rates rather than **NATURE** (Loehlin and Nichols [1976]).

Shields (1962) can be criticised because in the studies of twins reared apart – it is unlikely that the twins spent all of their childhood apart. Some would have been raised by close relatives and may have gone to the same school – and so would have shared a very similar environment.

The fact that the **concordance rates are not 100%** means that schizophrenia cannot be a wholly genetic disorder.

# Genetics – adoption studies

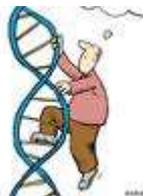
A01



**Adoption studies:** Allow researchers to look at people who were born to schizophrenic mothers but brought up by adoptive parents with no history of the disorder.

**AOI:**

**Kety (1994)** found high rates of schizophrenia in people whose biological parents had the disorder but who had been adopted by psychologically healthy parents.

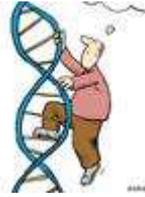


A02

**What does this suggest?**

# Genetics – adoption studies

A01



What does this suggest?....

**AO2: Supporting evidence from Tienari (1991)**

<b>Adopted children Group</b>	<b>Biological mother/ biological family</b>	<b>% of children developing schizophrenia</b>
1	Schizophrenic	10%
2	No family history of schizophrenia	1%

# Genetics – adoption studies

A01

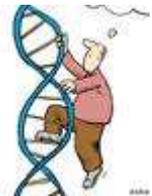


**Can you think of any criticisms of this evidence?**

**+VE:** Studies such as these provide strong evidence for a genetic explanation for schizophrenia.

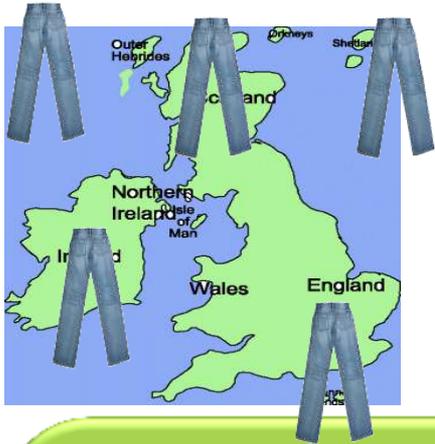
**BUT**

**-VE:** Again, it is unlikely that the twins spent all of their childhood apart. Some would have been raised by close relatives would have gone to the same school – and so would have shared a very similar environment.



# Genetics – gene mapping

A01



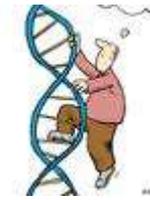
## AOI:

A gene located on chromosome 5 has been linked to schizophrenia in a small number of extended families where a number of family members have the disorder (**Sherrington et al 1988**)

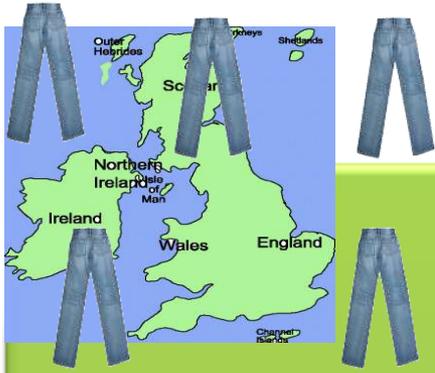
**What does this suggest?  
...Your thoughts?.....**



# Genetics – gene mapping



A02

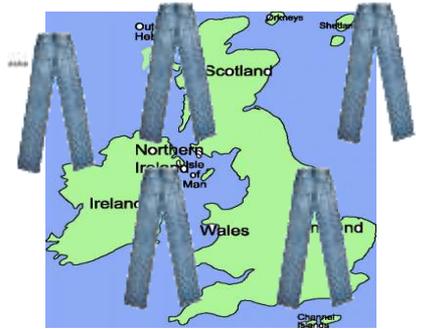
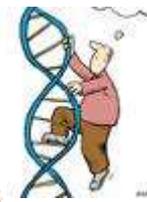


***However... it is not as straightforward as you may think..... (see page 5)***

- The Genome project has identified a much lower number of genes than first anticipated.
- It is now recognised that genes have multiple functions
- Strong evidence that schizophrenia is the result of multiple factors (genes + environment).
- The research on gene mapping is over-simplistic as schizophrenia is not due to a single gene

A02

# Genetics



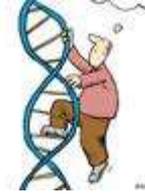
However, a theory based on “nature” alone would be **reductionist** and highly **deterministic** and with the continual development of research in this area, even biologists agree that;

Although twin studies appear to provide strong genetic evidence, there is **not 100% concordance**, therefore schizophrenia can't be a wholly genetic disorder. It is likely that genes alone do not **cause schizophrenia** instead they **predispose** an individual to the disorder i.e., they place the individual at a greater risk for developing the disorder if the necessary “trigger” is also available in the environment (*see Diathesis-stress model on page 6 in booklet*).



A03

# Genetics



## Methodological weaknesses of concordance rates

Family, twin and adoption studies must be considered cautiously because they are **retrospective** and may be biased by knowledge that another family member has been diagnosed.

## Sample bias

The **sample size** of twin, family and adoption studies tends to be **small** and so **population validity may be low**. This casts doubt on the generalisability of the findings. The gene mapping study was conducted on a restricted number of families and has failed to be replicated; which shows the problems of sample bias.

# Genetics



## Independent written activities:

1. Complete the **BUZZ WORDS** on page 10
2. Complete questions 1 to 4 on page 11 in booklet 2.
3. Draw a mind map or create a set of cue cards for the genetic explanation of schizophrenia.
4. Prepare to complete a timed summary:
  - Outline any one biological explanation of schizophrenia (9 marks)



# Biological explanations of schizophrenia

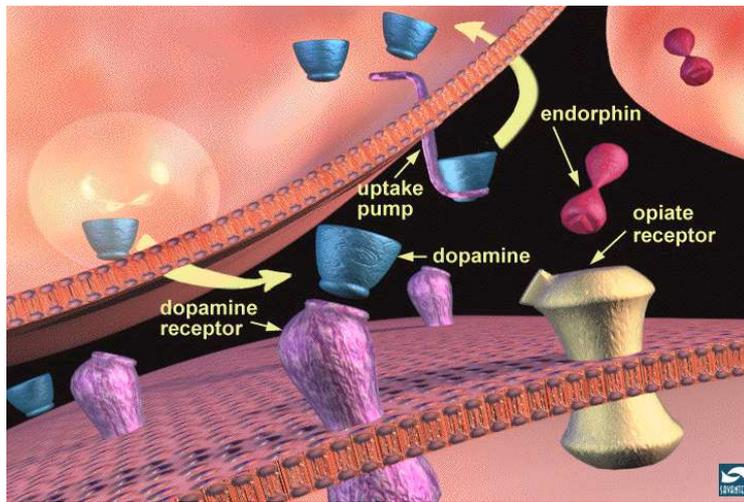
Neurochemical – dopamine hypothesis

# Dopamine Hypothesis

A01

Genetic factors may lead to imbalance in neurotransmitters

According to the *dopamine hypothesis* excess levels of the neurotransmitter dopamine is the cause of the disorder



# Dopamine Hypothesis

A01 /  
A02

## Biological Evidence

If inhibiting dopamine reduces schizophrenic symptoms what does that tell us about the cause of the disorder?

Phenothiazines  
(*anti-psychotics*) act to inhibit dopamine and reduce the symptoms of the disorder

L-Dopa increases dopamine levels and can produce many of the symptoms of schizophrenia

L-Dopa is given to sufferers of Parkinson's disease  
(*which is caused by low levels of dopamine*)

# Dopamine Hypothesis

A01 /  
A02

## Biological Evidence

If drugs that increase dopamine produce schizophrenic symptoms what does this tell you about the cause of schizophrenia?

Amphetamines (*drugs that increase dopamine*) produce symptoms which are similar to those experienced by someone with schizophrenia

# Dopamine Hypothesis

A01 /  
A02

## Randrup et al (1966)

Behaviour similar to that found with schizophrenia was induced in rats by administering amphetamines and the effect was reversed with anti-psychotic drugs

Randrup's research provides evidence for the link between high levels of dopamine and schizophrenia

# Dopamine Hypothesis

Additional  
A02

## Post-mortem examinations

have shown an increase of dopamine in parts of the brain.

**Seeman (1987)** found increases in dopamine receptor density of between 60-110% compared to controls

## PET scans

have allowed the investigation of a live brain.

**Wong et al (1986)** found a 2-fold increase in density of dopamine receptor sites in schizophrenic patients who hadn't been treated with anti-psychotics

# Dopamine Hypothesis

## Approaches:

Biological – objective and measurable

## Issues:

Reductionist

## Debates:

Determinism Vs. Free will  
Nomothetic Vs. Idiographic

## Ethics:

Long term effects of intentionally increasing dopamine level in humans

## Animal Ethics:

Randrup – increasing dopamine levels in rats

Scientific measurements  
Laboratory experiments



# Biological explanations of schizophrenia

Brain Structure – brain dysfunction

# Brain Dysfunction

A01

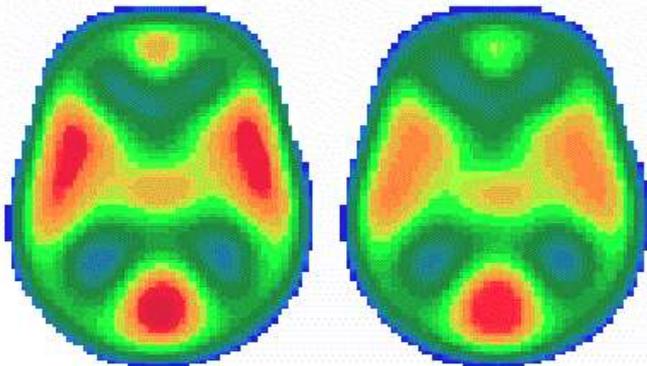
## Enlarged Ventricles

It has been found that the ventricles (cavities that supply nutrients and remove waste) are 15% larger in schizophrenics

Recent advances in technology have enabled the medical profession to examine the life brains of people with schizophrenia

Enlarged ventricles tend to be associated with negative rather than positive symptoms, are linked with greater cognitive disturbances and poor responsiveness to drug therapy

**without schizophrenia**      **with schizophrenia**



# Brain Dysfunction

A02

## MRI evidence

**Young et al (1990)** found a number of structural differences using MRI scans when comparing the brains of schizophrenic and non-schizophrenic individuals.

*This suggests that brain differences are strongly linked with the development of schizophrenia*

## Schizophrenia and brain damage

**Warner (1994)** suggested that structural abnormalities in the brains of some schizophrenics indicates some earlier brain trauma – drugs taken whilst the mother was pregnant, viral infection during pregnancy, complicated delivery.

*This suggest that brain damage is a predisposing factor in the development of schizophrenia*

# Brain Dysfunction

A02

## Inconclusive

There is great difficulty in establishing cause and effect – whether the brain damage is the **cause** of the schizophrenia or has developed as a **result** of the schizophrenia

## Enlarged ventricles in non-schizophrenics

Enlarged ventricles have been found in people not suffering from schizophrenia – this contradicts enlarged ventricles as a cause

## Negative Symptoms

Brain dysfunction is linked to the negative symptoms of schizophrenia only – can the Brain Dysfunction theory be generalised?