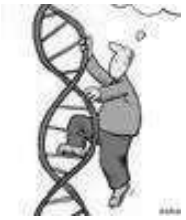
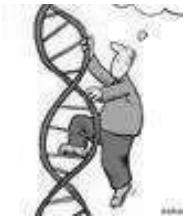
Biological explanations of schizophrenia



Genetics



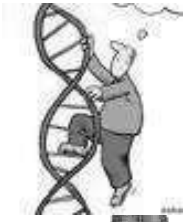
Genetics

A01

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

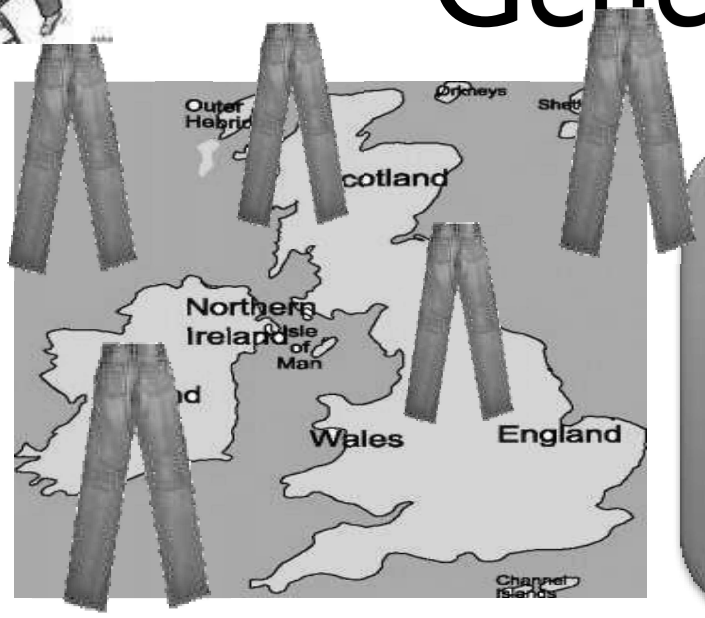
As you can see....inheriting a gene... does not mean you WILL HAVE blonde hair (or schizophrenia)....but by studying genetic links in families, it can help us to predict the likelihood of that feature occurring.

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



Genetics - evidence

A01



Much relevant evidence comes from...

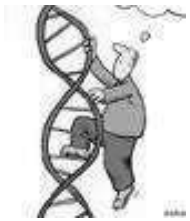
Concordance rates:
These show the percentage of family members developing schizophrenia.

Genetics – family studies

A01

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

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.

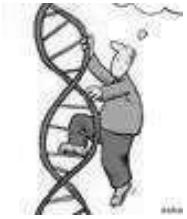
However, Gottesman (1991)

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

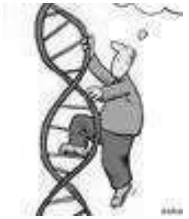
A02

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*

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:

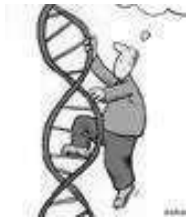
1. Family studies; 2. Twin studies;
3. Adoption studies; 4. Gene mapping.

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



Gottesman (1991) reviewed 40 twin studies and found:

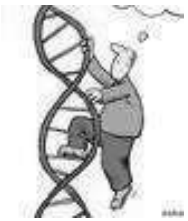
48% concordance for MZ twins

17% concordance for DZ twins

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

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



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]).

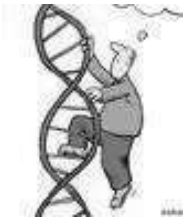
Shields (1962) can be criticised because in the studies of twins reared apart – it is unlikely that the twins spent all of their childhood apart. Some would have been raised by close relatives and may have gone to the same school – and so would have shared a very similar environment.

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

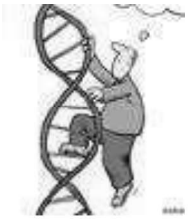
Genetics – adoption studies

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.

Kety & Ingraham (1992) found that prevalence rates of schizophrenia were 10 x higher among genetic than adoptive relatives of schizophrenics, suggesting that genetics play a greater role than environmental factors. .



Genetics – adoption studies



What does this suggest?....

Supporting evidence from Tienari (1991)

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

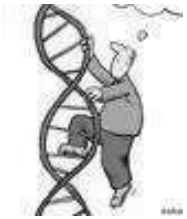
Genetics – adoption studies

Can you think of any criticisms of this evidence?

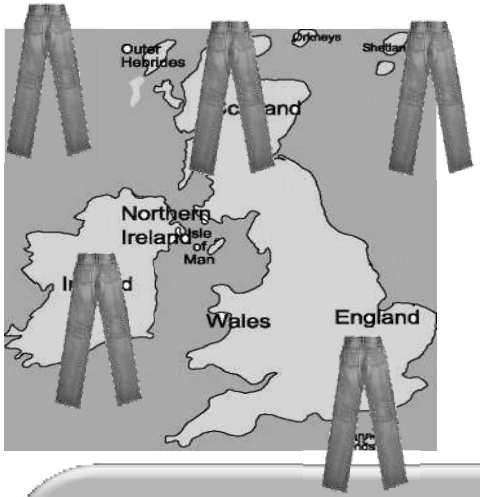
+VE: Studies such as these provide strong evidence for a genetic explanation for schizophrenia.

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.

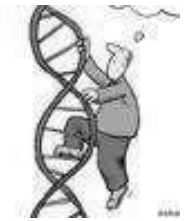


Genetics – gene mapping

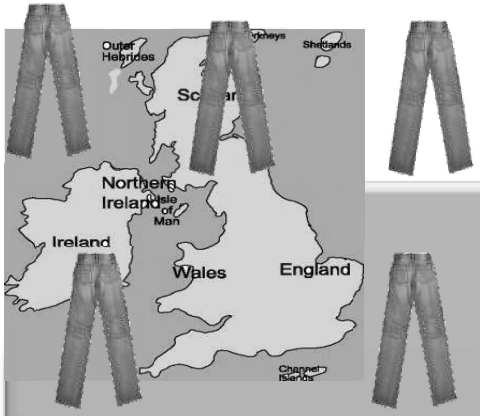


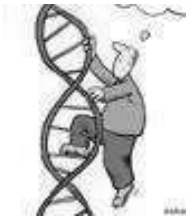
A gene located on chromosome 5 has been linked to schizophrenia in a small number of extended families where a number of family members have the disorder (**Sherrington et al 1988**)

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



Genetics – gene mapping

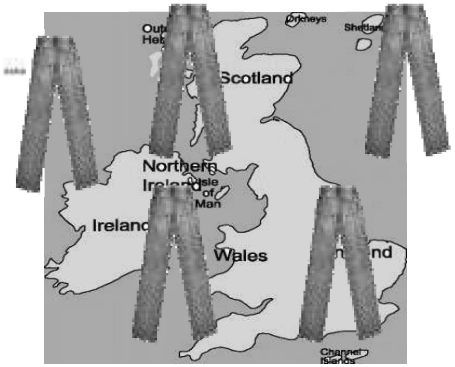




However.... it is not as straightforward as you may think..... ***(see page 5)***

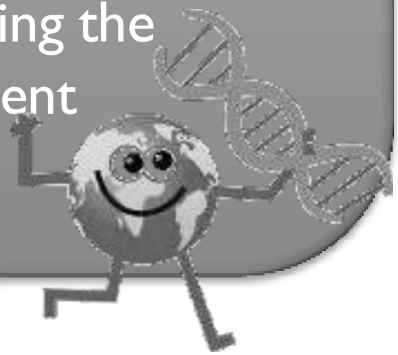
- **The Genome project has identified a much lower number of genes than first anticipated.**
- **It is now recognised that genes have multiple functions**
- **Strong evidence that schizophrenia is the result of multiple factors (genes + environment).**
- **The research on gene mapping is over-simplistic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

Genetics

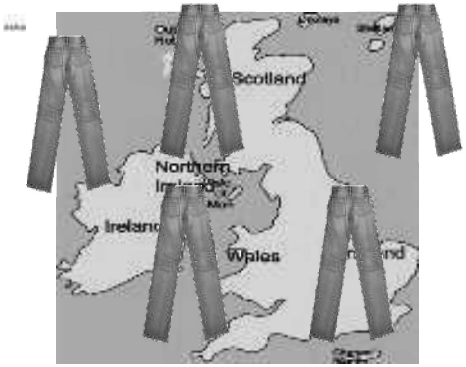


However, a theory based on "nature" alone would be reductionist and highly deterministic and with the continual development of research in this area, even biologists agree that;

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***).



Genetics



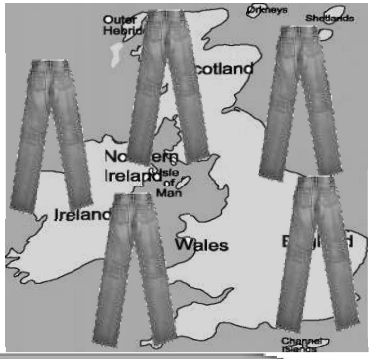
Methodological weaknesses of concordance rates

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:**

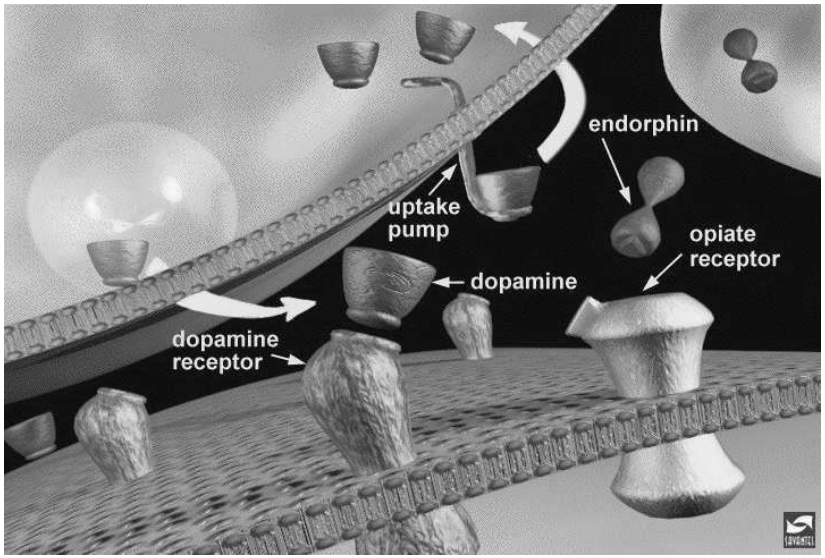
- Outline any one biological explanation of schizophrenia (9 marks)

Biological explanations of schizophrenia

Neurochemical – dopamine hypothesis

Dopamine Hypothesis

Dopamine (a neurotransmitter) is widely believed to work differently in the brain of a patient with schizophrenia and this may result in the symptoms observed in sufferers. Dopamine acts to increase the rate of firing in the synapse of neurons, which enhances the communication between neurons.



It is probable that genetic factors are linked to faulty dopaminergic systems.

Dopamine hypothesis

Hyperdopaminergic explanations (High levels)	**Hypodopaminergic explanations (low levels)**
The original version of the dopamine hypothesis focused on the possible role of **high levels/activity of dopamine in the subcortex** (central areas of the brain). For example, an excess of dopamine receptors in Broca's area (responsible for speech production) may be associated with poverty of speech and/or the experience of auditory hallucinations.	The theory was updated because high levels of dopamine are not found in all schizophrenics. More recent versions of the dopamine hypothesis have focused instead on abnormal dopamine systems in the brains **cortex**. Goldman-Rakic et al. (2004) have identified a role for **low levels of dopamine in the prefrontal cortex** (responsible for thinking and decision-making) in the negative symptoms of schizophrenia.
It may be the case that both high and low levels of dopamine in different brain regions are involved in schizophrenia.	

Biological explanations of schizophrenia

Neural correlates (areas of the brain)

Neural correlates

The idea of neural correlates is that ***abnormalities within specific brain areas may be associated with the development of schizophrenia***.

Research uses non-invasive scanning techniques, such as ***fMRI, to compare the functioning of the brains of schizophrenics with that of non-sufferers***, to identify brain areas that may be linked to the disorder.

Participants are often given tasks associated with types of functioning known to be abnormal in sufferers, for example social cognition, thought processing and working memory tasks.

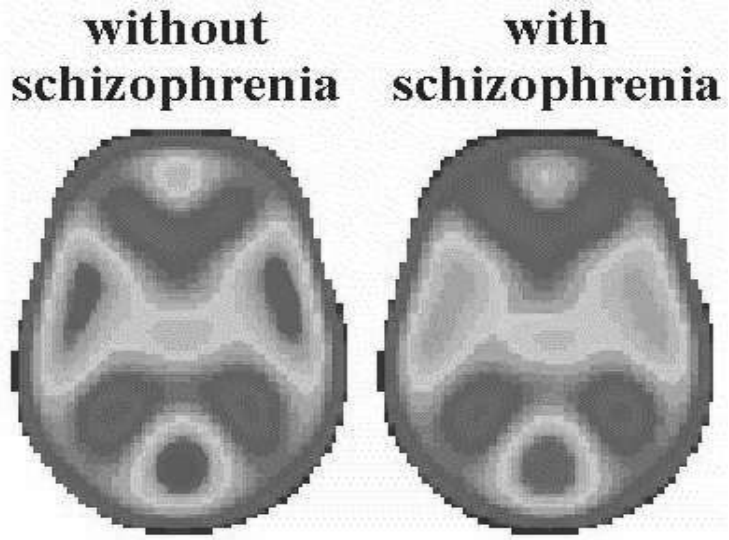
Brain Dysfunction

Negative symptoms

Early research attention (Johnstone et al., 1976) was focused on schizophrenics having enlarged ventricles (fluid-filled gaps between brain areas).

Enlarged ventricles are especially associated with damage to central brain areas and the pre-frontal cortex, which more recent scanning studies have also linked to the disorder.

Such damage has often been associated with negative symptoms but cannot explain all symptoms and incidences of schizophrenia.



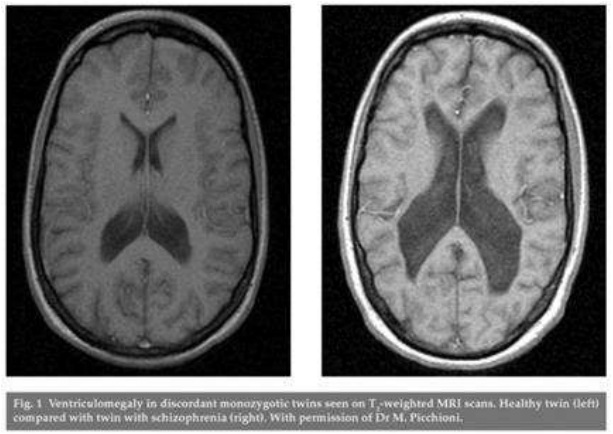


Fig. 1 Ventriculomegaly in discordant monozygotic twins seen on T_2-weighted MRI scans. Healthy twin (left) compared with twin with schizophrenia (right). With permission of Dr M. Picchioni.

Brain Dysfunction

Positive symptoms

Allen et al. (2007) found that positive symptoms also have neural correlates. They scanned the brains of patients experiencing auditory hallucinations and compared them to a control group whilst they identified pre-recorded speech as theirs or others.

Lower activation levels in the s**uperior temporal gyrus and anterior cingulate gyrus** were found in the hallucination group, who also made more errors than the control group. We can thus say that reduced activity in these two areas of the brain is a neural correlate of auditory hallucination.