Caspi et al (2003) carried out a study to the role that gene mutation and epigenetics may play in major depressive disorder. You can use this study for the following content in the biological approach:

Research methods used in the biological approach.

To what extent does genetic inheritance influence behaviour?

In addition, you may use it in the abnormal option.

To what extent do biological factors influence abnormal behaviour?

The aetiology of one disorder.

The original study is available here.

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**Procedure**

**Diathesis-stress theories** of depression predict that an individual's reaction to stressful events depends on their genetic make-up. If an individual has a specific genotype, then interaction with the environment may cause these genes to be *expressed*. The aim of this study was to determine whether there is evidence for a gene-environment interaction (G x E) for a mutation of the serotonin transporter gene - 5-HTT. The serotonin transporter is involved in the reuptake of serotonin in brain synapses.

Caspi and his team looked at a sample of 847 New Zealand 26-year-olds. All were members of a cohort that had been assessed for mental health on an every-other-year basis until they were 21. They were divided into three groups based on their 5-HTT alleles: Group 1 had two short alleles; Group 2 had one short and one long allele; Group 3 had two long alleles. The mutation of the 5-HTT gene has the shorter alleles. Roughly 43% of people have the shorter alleles.

The participants were asked to fill in a "Stressful life events" questionnaire which asked them about the frequency of 14 different events - including financial, employment, health and relationship stressors - between the ages of 21 and 26. They were also assessed for depression.

**Results**

People who had inherited one or more short versions of the allele demonstrated more symptoms of depression and suicidal ideation in response to stressful life events. The effect was strongest for those with three or more stressful life events. Simply inheriting the gene was not enough to lead to depression, but the genes’ interaction with stressful life events increased one’s likelihood of developing depression.

In a later study by Wilhelm et al (2006), the researchers looked at DNA samples from 127 people who are part of a longitudinal prospective study looking at mental health. The sample had been monitored for over 25 years. At five-year intervals, scientists recorded any major life events and signs of depression. They found that 80 percent of those with two short 5-HTT
genes became depressed after three or more negative life events in a year, whereas those with two long genes appeared resilient - only 30 percent developed the illness in similar situations. They also found that childhood maltreatment predicted adult depression only among individuals carrying a short allele and not among those carrying the longer allele.

However, much more research is needed before a clear relationship between a gene and a depression can be established.

The researchers have also found evidence to support this in cases of bullying.

Evaluation

- The study is correlational, so no cause and effect relationship can be determined.
- The study makes an assumption that serotonin causes depression. This may not actually be the case. In fact, a meta-analysis of attempts to replicate Capsi's findings has shown that they have not been successfully replicated.
- Information about life-events was self-reported. It may be the salience of the negative life events which plays a role in depression - that is, those that recalled them more easily may have a tendency toward depression. Those who are more resilient, may not recall negative life events as easily.
- The theory acknowledges the interaction between both biological and environmental factors in depression. This is a more holistic approach, not reductionist.
- There were some participants who did not carry the gene mutation who became depressed; therefore, we cannot say that gene expression alone can cause depression.