Scientists find link between gene and sensitivity to emotional environment

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Researchers at the University of Essex have shown that a genetic variant could make some people more sensitive to their emotional environment - and more susceptible to anxiety disorders - than others. The study, funded by the Wellcome Trust, could have implications for predicting how well individual patients will respond to treatments for anxiety disorders.

Anxiety disorders are among the most common types of mental health problem: one in ten people in the UK experience mixed anxiety and depressive disorder, according to the Office for National Statistics. Anxiety disorders cause feelings of excessive worry that interfere with everyday activities and include phobias, post-traumatic stress disorders and obsessive-compulsive disorder.

People with anxiety disorders have a tendency to pay more attention to negative or threatening information, known as a negative attention bias. This tendency can be changed with attention bias modification (ABM) procedures, which typically involve using a computerised training programme to re-train a participant's attention bias. These are experimental techniques with the potential to be used as therapies for anxiety disorders.

It has been suggested that there is a link between a person's attention bias towards negative material and the version they have of the gene that corresponds to a protein known as the 'serotonin transporter'.

Serotonin is a neurotransmitter, a chemical released into the gap between nerve cells, which carries nerve signals from one nerve cell to the next. The serotonin transporter is a protein that plays an important part in nerve signalling. After a nerve signal has crossed to the next nerve cell, the serotonin transporter removes serotonin from the gap and transports it back into the initial nerve cell, where it can be reused for the next nerve signal.

The gene that encodes the serotonin transporter varies across the human population. Some people carry short versions of the gene, which results in them having fewer copies of the serotonin transporter and therefore higher concentrations of serotonin in the gaps between neurons. Others have long versions, which lead to more copies of the serotonin transporter and lower serotonin levels.

Previous studies have found that people with short versions of the serotonin transporter gene are more likely to show the 'toxic' negative bias that is characteristic of anxiety disorders.

Now, in a study published in the journal 'Biological Psychiatry', researchers from the Department of Psychology at the University of Essex have combined ABM training with genetic techniques for the first time to produce new evidence that could help to explain the nature of the relationship between the serotonin transporter gene and attention bias.

The researchers found that healthy people with short forms of the gene were more sensitive to negative information when doing an ABM task. In other words, these individuals developed a stronger negative bias over an hour's training session than those with the longer version of the gene.

Remarkably, people with short versions of the gene were also found to be more sensitive to positive information. They developed a stronger positive attention bias (a tendency to focus on positive information)

during training than people with the long version of the gene.

The researchers tested two groups, each containing 57 healthy people (with low levels of 'trace' attention bias normally found in healthy individuals). Both groups contained some people with short versions of the serotonin transporter gene and others with long versions. They used a computer-based task to test whether participants had an initial trace attention bias towards positive or negative images, then conducted ABM training to induce a positive bias in one group and a negative bias in the other. Participants were then re-tested to see whether their initial attention biases had changed.

Attention bias tests involved showing participants two images simultaneously on a screen: one positive (such as a smiling baby), the other negative (such as a snarling dog). Both images were removed and one was replaced with a symbol (a line of either horizontal or vertical dots), which the participant had to identify.

People with a negative attention bias were faster at finding the symbol when it appeared in the location where the negative image had been than when it appeared where the positive image had been. Conversely, those with a positive attention bias responded more quickly when the symbol appeared where the positive image had been. Each participant's response time was measured 128 times, in which there was equal probability of the symbol appearing behind either the positive or the negative image.

ABM training was conducted in the same way as attention bias testing, except that the symbol always replaced the positive image during positive attention bias training and the negative image during negative training.

Professor Elaine Fox, who led the study, explained: "Our findings suggest that people with a short serotonin transporter gene are likely to

be far more reactive to both very negative situations, such as a car crash, and very positive ones, such as a very supportive relationship. This supports the idea of short serotonin transporter genes as 'adaptability', rather than 'vulnerability', genes. They may not only increase the risk of an individual developing emotional vulnerability in a negative environment but also increase the chances of them benefiting from a supportive environment, compared to people with the long form.

"People with a long serotonin transporter gene are likely to be less influenced by their emotional environment, which may help to protect them from negative events but could also mean that they are less able to benefit from a positive environment."

These results are complemented by another recent study by researchers from Kings College London, Macquarie University in Sydney and the University of Reading, in which people with short forms of the serotonin transporter gene benefited far more from cognitive behavioural therapy (a talking therapy) than people with long versions.

Both studies support a theory proposed by Dr Catherine Harmer at the University of Oxford. Dr Harmer suggests that drugs used to treat anxiety and depression by lowering serotonin levels at the gap between nerve cells (such as selective serotonin reuptake inhibitors) might work by reducing a patient's negative attention bias, causing them to adopt a more positive outlook on life that leads to a decrease in anxiety and depression.

"This opens the door to the idea of personalized treatments for anxiety disorders. Information about the genotype and cognitive biases of a patient could be used to inform decisions about which treatments, such as ABM and cognitive behavioural therapy, are likely to be most effective," Professor Fox said.

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