Bristol scientists pave the way to tackling anxiety disorders

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Anxiety disorders are severely debilitating, the commonest cause of disability in the US workplace1, and a source of great anguish to individuals and their families.

Although fear and anxiety are part of our natural response to stress, the causes of chronic and inappropriate levels of anxiety are complex and treatments unsatisfactory.

A study by Bristol researchers, published this week in the prestigious [*Journal of Neuroscience*](http://www.jneurosci.org/content/31/18/6721.full.pdf+html?sid=5742438d-72aa-44b9-b9cc-d1e947a7e509) 2, has identified a specific protein that appears to be critically important in the manifestation of anxiety-like symptoms.

Professor David Lodge, Dr Laura Ceolin, and collaborators in the MRC Centre for Synaptic Plasticity, based in the School of Physiology and Pharmacology, have identified a specific protein that appears to be critically important in the manifestation of anxiety-like symptoms.

This could pave the way to finding new treatments for anxiety disorders.

The protein’s normal function is to detect and respond to the neurotransmitter L-glutamate, one of the most important mammalian neurotransmitters - the chemicals that mediate communication between nerve cells in the brain and nervous system.

There are a number of subtypes of glutamate receptor proteins.  Researchers, who worked largely in Dr Zuner Bortolotto’s laboratory, discovered a strain of rat that lacked one particular subtype, the mGlu2 receptor, and that these rats displayed anxiety-like behaviours echoing symptoms of human anxiety disorders.

Drugs that affect several types of mGlu receptors have demonstrated some success in clinical trials of treating anxiety.

These new findings are important as they allow future drug development to selectively target the mGlu2 receptor subtype, potentially increasing treatment efficacy and limiting unwanted side effects.

Moreover, mGlu receptors are also implicated in other brain diseases.

Being now able to study rats specifically lacking the mGlu2 receptor thus opens up a valuable test-bed for treatments for disorders including schizophrenia, stress, epilepsy and neuropathic pain.

David Lodge and Laura Ceolin are now collaborating with colleagues in [Bristol Neuroscience](http://www.bristol.ac.uk/neuroscience/) to further understand the molecular biology underlying this lack of mGlu2 receptors and investigate the behavioural and therapeutic implications.