Novel approach to chronic pain relief

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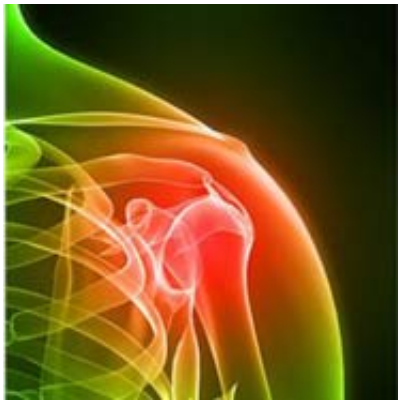

(PhysOrg.com) -- An international team of scientists have found what they believe could be a novel approach to more effective, targeted relief of chronic pain caused by nerve injuries. The research, a collaboration involving the Universities of Toronto, Seoul, Korea and Bristol, is reported in the latest edition of the journal *Science*.

Previously, scientists have been able to show that a protein molecule known as PKM zeta is required to store memories. In the case of chronic pain, there is a malfunctioning in the neural process that stores those memories, which prevents the brain from adapting the subsequent behavioral response which would ordinarily allow it to cope with the pain.

The connections between neurons through synaptic pathways in the

central nervous system are somehow flawed, causing an individual to re-experience pain as the mental record of that pain persists.

This new research has detected the cause for this malfunction and in doing so, has identified a novel target for the treatment of neuropathic pain. By inhibiting PKM zeta in a part of the brain involved in the perception of pain in a mouse model, the international team of scientists have been able to eliminate the painful memory responsible for chronic pain.

Professor Graham Collingridge, from the University of Bristol's MRC Centre for Synaptic Plasticity, the School of Physiology and Pharmacology, and part of the Bristol Neuroscience network, said: "If this translates to humans, it may be possible one day to treat some forms of chronic pain by inhibiting PKM zeta or other molecules involved in the storage of the painful memory. The challenge will be to target the drug so that it inhibits painful memories but not other forms of memory. "

By studying how brain connections operate in a part of the cortex involved in pain sensation in mice, the team found that the molecule PKM zeta actually serves to maintain pain-induced persistent changes in the brain, thereby prolonging the sensation of chronic pain.

The team combined biochemistry, electrophysiology and behavior to study the role of PKM zeta in the anterior cingulate cortex, a brain region known to be activated in humans during painful states. The identification of a molecular basis for chronic pain provides a framework for the development of more effective therapies in the future.

More information: Alleviating Neuropathic Pain Hypersensitivity by Inhibiting PKMzeta in the Anterior Cingulate Cortex, is published online

by the journal *Science*, at the *Science Express* web site on Thursday 2 December 2010.

Provided by University of Bristol