MedicalXpress

Study suggests new way to treat chronic pain

March 26 2012

Nearly one in five people suffers from the insidious and often devastating problem of chronic pain.

That the problem persists, and is growing, is striking given the many breakthroughs in understanding the basic biology of pain over the past two decades. A major challenge for treating chronic pain is to understand why certain people develop pain while others, with apparently similar disorders or injuries, do not. An equally important challenge is to develop individualized therapies that will be effective in specific patient populations.

Research published online in Nature Medicine points to solutions to both challenges. A research team led by Prof. Jeffrey Mogil of McGill University in Montreal and Prof. Michael Salter of The Hospital for Sick Children (SickKids), affiliated with the University of Toronto, has identified a major gene affecting chronic pain sensitivity. The findings also suggest a new approach to individualizing treatment of chronic pain.

The gene that the researchers identified encodes the pain receptor known as P2X7. Specifically, the scientists discovered that a single amino-acid change in P2X7 controls sensitivity to the two main causes of chronic pain: inflammation and nerve damage.

The amino-acid change is known to affect only one function of P2X7 receptors – the forming of pores that permit large molecules to pass through – while leaving intact the other function, of allowing much tinier ions to flow through. Using a peptide that targets pore formation only,

the researchers found that pain behaviours were dramatically reduced.

The scientists then examined genetic differences among human patients suffering from two distinct types of persistent pain: chronic post-mastectomy pain and osteoarthritis. In both cases, they found that individuals with genetically inherited low pore formation in P2X7 receptors experienced lower pain levels.

"Our findings indicate that it may be possible to develop drugs that block pores in this crucial receptor, while leaving its other function intact – thereby killing pain while minimizing side effects," said Prof. Mogil, E.P. Taylor Professor of Pain Research in McGill's Department of Psychology.

Prof. Salter, Anne and Max Tanenbaum Chair in Molecular Medicine at SickKids, said these discoveries "point toward a new strategy for individualizing the treatment of chronic pain." Scientists from the U.S. and Israel also contributed to the study.

Provided by McGill University