

Chronic pain gene identified

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British researchers say they have identified the gene that controls chronic pain, opening the door to new drug therapies that block the chemical processes that cause chronic back pain, headaches or arthritis.

Chronic pain, defined as pain that is experienced most days of the week for at least three months, afflicts around 1 in 5 Australians. It commonly disrupts a sufferer's ability to lead a normal life.

Now a team led by Professor Peter McNaughton, Head of the Department of Pharmacology at the University of Cambridge, has isolated a gene called HCN2 that produces a protein that causes chronic pain.

“Individuals suffering from neuropathic pain often have little or no respite because of the lack of effective medications. Our research lays the groundwork for the development of new drugs to treat chronic pain by blocking HCN2,” said Professor McNaughton.

His team conducted a series of experiments on mice genetically engineered to be born without the HCN2 gene. The researchers observed that these mice responded normally to normal acute pain (which is produced by a sudden injury) but that removing the HCN2 gene abolished neuropathic pain.

Being able to control chronic pain without affecting normal pain responses was crucial, he said.

“Many genes play a critical role in pain sensation, but in most cases interfering with them simply abolishes all pain, or even all sensation,” Professor McNaughton said.

“This finding could be very valuable clinically because normal pain sensation is essential for avoiding accidental damage.”

Professor Richard Lewis, an expert on [chronic pain](#) from the University of Queensland’s Institute for Molecular Bioscience, said the breakthrough was significant.

“This work appears particularly exciting as it, for the first time, directly links pacemaker channel HCN2 — a membrane protein important in controlling cell excitability — to the development of several difficult-to-treat painful conditions but not normal pain sensations,” said Professor Lewis, who was not involved in the research.

Chemical disruption of HCN2 could form the basis of much needed new pain therapies, he said.

“Building on previous research in this area, the work now highlights opportunities to discover and develop safer and more effective pain therapies that can selectively target HCN2 in [pain](#) pathways.”

The UK study was published in the September 9 edition of the journal [Science](#).

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