

Researchers identify which sensory nerve cells contribute to chronic nerve pain

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Computer-generated artwork illustrating a migraine.
Credit: Adrian Cousins, Wellcome Images.

(Medical Xpress) -- New research from the University of Bristol has identified the subtypes of sensory nerve cells that are likely to contribute to long-term nerve pain from partial nerve injury. It is hoped this will aid in the development of more effective painkillers.

Partial [nerve injury](#) can be caused by several things, such as an accident, surgery or a disease (e.g. diabetes). Although some nerve cells will degenerate and die as a result of the injury, some will survive and continue to conduct through the damaged or inflamed nerve. This can result in serious long-term neuropathic pain.

Neuropathic pain is experienced in different ways. The most debilitating is an often relentless, spontaneous and ongoing burning or sharp shooting pain. Some people may experience extreme tenderness and pain in response to normal touch or normal movements, as well as greater pain than usual in response to normally painful events. They may also feel strange, and often unpleasant - but non-painful - sensations.

In all cases, neuropathic pain remains very hard to treat clinically, partly because we don't fully understand the types of nerve cells responsible for these different types of pain.

Sensory nerve cells are responsible for carrying

signals from the tissues, such as skin, muscle and organs, to the [central nervous system](#). The cells responsible for transmitting pain are called nociceptors. They sense and signal [tissue damage](#) or [inflammation](#), activating pathways to the brain, which results in pain.

In the study, published this week in the journal *Pain*, the researchers report profound changes in properties of different subpopulations of the sensory nerve cells that survive without damage after nerve injury. First, they observed spontaneous ongoing firing in different groups of pain nerve cells, which would cause ongoing burning and sharp shooting pain.

They also report spontaneous firing in non-pain nerve cells that could cause the abnormal non-[painful sensations](#) that are associated with neuropathic pain. In addition, they found an increased sensitivity of fast-conducting nociceptors, which could underlie the increased pain resulting from stimulation of the tissues.

Professor Sally Lawson, who led the research at the University of Bristol, said: "We hope that our findings will trigger further studies that will clarify how and why these nerve cells with uninjured fibres running through a damaged nerve change so remarkably and contribute to pathological pain, and determine how to reverse these changes."

Dr Laiche Djouhri, now at the University of Liverpool, added: "This should help the understanding of how to target pathological pain more effectively, taking into account the different neuron types involved. This may, in the longer term, help development of more effective painkillers to help sufferers of [neuropathic pain](#)."

The study was funded by the Wellcome Trust and the Medical Research Council.

More information: Djouhri L et al. Partial nerve

injury induces electrophysiological changes in conducting (uninjured) nociceptive and nonnociceptive DRG neurons: possible relationships to aspects of peripheral neuropathic pain and paresthesias. *Pain* 2012;153:1824-36.
www.ncbi.nlm.nih.gov/pubmed/22721911

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