

Scientists reveal new avenue for drug treatment in neuropathic pain

November 24 2017



Credit: CC0 Public Domain

New research from King's College London has revealed a previously undiscovered mechanism of cellular communication, between neurons and immune cells, in neuropathic pain.

The authors, who published their findings today in *Nature Communications*, identified a new method of treating [neuropathic pain](#) in mice, which could be more safe and effective than current treatments comprising of opioids and [antiepileptic drugs](#).

Neuropathic pain is a type of chronic pain that is usually caused by an injury to nerves, but the pain persists long after the injury has healed. Neuropathic pain may occur after surgery or a car accident, or in some cases when a limb has been amputated.

Currently the only available drugs for neuropathic pain are either opioids or antiepileptic medication. Opioids, like morphine and tramadol, are highly addictive and the NHS have recently raised concerns about prescription of these drugs, due to opioid overdoses more than doubling in the last decade. In the US an opioid 'epidemic' has recently been declared due to the rising number of deaths linked to these drugs. In contrast, antiepileptic medication is not addictive but is often accompanied by a whole host of unpleasant side effects such as dizziness, fatigue, nausea and weight gain.

However, people with neuropathic pain have very little choice when it comes to other treatment options because the cause of neuropathic pain is so poorly understood.

Using cellular and mouse models of neuropathic pain the authors studied a cluster of neurons in the dorsal root ganglion (DRG), which are part of the [sensory neurons](#) that play an important role in communicating [pain information](#) to the brain. They found that after nerve injury, pain neurons in this area released very small [biological particles](#) containing microRNA-21. These particles were then taken up by surrounding [immune cells](#), ultimately leading to local inflammation and neuropathic pain.

The authors showed that when they blocked DRG pain neurons from releasing microRNA-21 in particles, this had an anti-inflammatory effect at a cellular level, which prevented neuropathic pain from occurring in mice. The advantage of this method is that these particles, containing agents that block microRNA-21, do not infiltrate the brain and lead to side effects.

In humans, a similar method could be applied to block pain neurons from releasing microRNA-21 in particles, which would prevent neuropathic pain from occurring. If successful, this would be the first drug to target neuropathic pain in specific areas without side effects, which is in stark contrast to the non-specific painkillers currently available.

Fortunately, similar treatments are already being trialled in cancer patients receiving immunotherapy, making the application to other conditions like neuropathic pain highly feasible.

Professor Marzia Malcangio, senior author from the Wolfson Center for Age-Related Diseases at the Institute of Psychiatry, Psychology & Neuroscience (IoPPN), King's College London, said "If new treatments based on the findings of this study, targeting microRNA-21, could be designed for patients with neuropathic [pain](#) this could provide a brand new avenue for drug treatment. Our next steps are to explore whether the same mechanism applies to other [chronic pain](#) conditions."

More information: Raffaele Simeoli et al. Exosomal cargo including microRNA regulates sensory neuron to macrophage communication after nerve trauma, *Nature Communications* (2017). [DOI: 10.1038/s41467-017-01841-5](https://doi.org/10.1038/s41467-017-01841-5)

Provided by King's College London

Citation: Scientists reveal new avenue for drug treatment in neuropathic pain (2017, November 24) retrieved 20 March 2024 from <https://medicalxpress.com/news/2017-11-scientists-reveal-avenue-drug-treatment.html>

This document is subject to copyright. Apart from any fair dealing for the purpose of private study or research, no part may be reproduced without the written permission. The content is provided for information purposes only.