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How We Treat Acute Pain Could Be Wrong

By Emily Shiffer



June 17, 2022 -- In a surprising discovery that flies in the face of conventional medicine, McGill University researchers report that [treating pain](#) with anti-inflammatory medication, like ibuprofen or aspirin, may promote pain in the long term.

The paper, published in *Science Translational Medicine*, suggests that [inflammation](#), a normal part of injury recovery, helps resolve acute pain and prevents it from becoming chronic. Blocking that inflammation may interfere with this process, leading to harder-to-treat pain.

“What we’ve been doing for decades not only appears to be wrong, but appears to be 180 degrees wrong,” says senior study author Jeffrey Mogil, PhD, a professor in the department of psychology at McGill. “You should not be blocking [inflammation](#). You should be letting inflammation happen. That’s what stops [chronic pain](#).”

Inflammation: Nature’s Pain Reliever

Wanting to know why pain goes away for some but drags on (and on) for others, the researchers looked at pain mechanisms in both humans and mice. They found that a type of white blood cell known as a neutrophil seems to play a key role.

“In analyzing the genes of people suffering from [lower back pain](#), we observed active changes in genes over time in people whose pain went away,” says [Luda Diatchenko](#), PhD, a professor in the faculty of medicine at McGill and Canada Excellence Research Chair in Human Pain Genetics. “Changes in the blood cells and their activity seemed to be the most important factor, especially in cells called neutrophils.”

To test this link, the researchers blocked neutrophils in mice and found the pain lasted 2 to 10 times longer than normal. Anti-inflammatory drugs, despite providing short-term relief, had the same pain-prolonging effect -- though injecting neutrophils into the mice seemed to keep that from happening.

The findings are supported by a separate analysis of 500,000 people in the U.K. that showed those taking anti-inflammatory drugs to treat their pain were more likely to have pain 2 to 10 years later.

“Inflammation occurs for a reason,” says Mogil, “and it looks like it’s dangerous to interfere with it.”

Rethinking How We Treat Pain

Neutrophils arrive early during inflammation, at the onset of injury – just when many of us reach for pain medication. This research suggests it might be better *not* to block inflammation, instead letting the neutrophils “do their thing.” Taking an analgesic that alleviates pain without blocking neutrophils, like [acetaminophen](#), may be better than taking an anti-inflammatory drug or [steroid](#), says Mogil.

Still, while the findings are compelling, [clinical trials](#) are needed to directly compare anti-inflammatory drugs to other painkillers, the researchers said. This research may also lay the groundwork for new drug development for chronic pain patients, Mogil says.

“Our data strongly suggests that neutrophils act like analgesics themselves, which is potentially useful in terms of analgesic development,” Mogil says. “And of course, we need new analgesics.”

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