

# Putting the brakes on pain

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Neuropathic pain—pain that results from a malfunction in the nervous system—is a daily reality for millions of Americans. Unlike normal pain, it doesn't go away after the stimulus that provoked it ends, and it also behaves in a variety of other unusual and disturbing ways. Someone suffering from neuropathic pain might experience intense discomfort from a light touch, for example, or feel as though he or she were freezing in response to a slight change in temperature.

A major part of the answer to the problem of neuropathic pain, scientists believe, is found in spinal [nerve cells](#) that release a signaling chemical known as GABA. These GABA neurons act as a sort of brake on [pain impulses](#); it's thought that when they die or are disabled the pain system goes out of control. If GABA neurons could be kept alive and healthy after [peripheral nerve](#) or [tissue injury](#), it's possible that neuropathic pain could be averted.

Now, University of Texas Medical Branch at Galveston researchers have found a way to, at least partially, accomplish this objective. The key, they determined, is stemming the biochemical assault by [reactive oxygen species](#) that are generated in the wake of [nerve injury](#).

"GABA neurons are particularly susceptible to oxidative stress, and we hypothesized that reactive oxygen species contribute to neuropathic sensitization by promoting the loss of GABA neurons as well as hindering GABA functions," said UTMB professor Jin Mo Chung, senior author of a paper on the research now online in the journal *Pain*.

To test this hypothesis—and determine whether GABA neurons could be saved—the researchers conducted a series of experiments in mice that had been surgically altered to simulate the conditions of neuropathic pain. In one key experiment, mice treated with an antioxidant compound for a week after surgery were compared with untreated mice. The antioxidant mice showed less pain-associated behavior and were found to have far more GABA neurons than the untreated mice.

"So by giving the antioxidant we lowered the pain behavior, and when we look at the spinal cords we see the GABA neuron population is almost the same as normal," Chung said. "That suggested we prevented those neurons from dying, which is a big thing."

One complication, Chung noted, is a "moderate quantitative mismatch" between the behavioral data and the GABA-neuron counts. While the anti-oxidant mice displayed less pain behavior, their behavioral improvement wasn't as substantial as their high number of GABA neurons would suggest. One possibility is that the surviving neurons were somehow impaired—a hypothesis supported by electrophysiological data.

Although no clinical trials are planned in the immediate future, Chung believes anti-oxidants have great potential as a treatment for neuropathic pain. "If this is true and it works in humans—well, any time you can salvage neurons, it's a good thing," he said. "Neuropathic pain is very difficult to treat, and I think this is a possibility, a good possibility."

Provided by University of Texas Medical Branch at Galveston

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