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Featured Article

Peppers, Pain and Lidocaine

By Jennifer Decker Arevalo, MA, contributor.

In a recent scientific study, Massachusetts General Hospital (MGH) and Harvard Medical School (HMS) researchers found that capsaicin, the pungent ingredient in chili peppers, combined with QX-314, a normally inactive lidocaine derivative, selectively block nociceptor (pain-sensing) neurons without interfering with the excitability of all other neurons, thus reducing the common side effects of local anesthesia.

Although this technique has been tested to date only on rodents, it has the potential to revolutionize surgical and post-surgical analgesia in humans, because it specifically targets the pain system. Patients could remain awake and pain-free during surgery, and avoid unpleasant side effects, such as temporary paralysis and numbness.

"We used capsaicin to open the noxious-heat-sensitive TRPV1 ion channels or 'passageways,' which have large enough pores to allow fairly large molecules, like charged forms of membrane-impermeant lidocaine, to come into the nerve cell," said senior study author, Clifford Woolf, M.D., Ph.D., director of the neural plasticity research group in the department of anesthesia and critical care at MGH and HMS. TRPV1 is a membrane-spanning protein unique to pain-sensing neurons.

Normally, QX-314 cannot pass through the cell's lipid membrane on its own because of its positive charge, but when TRPV1 is activated by capsaicin, the TRPV1 "gate" opens. Other non-pain sensing neurons are unaffected, leaving the QX-314 to remain outside the cell. TRPV1 can be activated both by noxious heat and capsaicin, which is why chilies produce a burning sensation.

Woolf and his colleagues, Bruce Bean, Ph.D., a professor of neurobiology at HMS, and Alexander Binshtok, a postdoctoral researcher in Woolf's lab, found that once the QX-314 was inside the cell, it blocked sodium channels and inhibited excitability, shutting off the pain. This transmembrane access route for QX-314 existed only on nociceptors on which TRPV1 was expressed.

After injecting this combination into rats' hind paws, researchers noted that it produced a two-hour or more increase in the mechanical and thermal nociceptive thresholds without causing paralysis; they tested this by placing the rats on a high heat source and measuring their ability to sense pain.

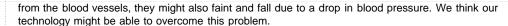
Likewise, a regional injection of QX-314 and capsaicin near the rats' sciatic nerve also reduced pain sensitivity and did not cause motor deficits that normally accompany conventional local anesthesia with lidocaine. The rats were poked with nylon probes, which they ignored. In both cases, the rats were still able to normally move about their cages and remain sensitive to touch and other non-painful stimuli.

"Since we wanted to target pain fibers specifically and knew that they selectively responded to both intense heat and capsaicin, we chose capsaicin; however, we would not envision using capsaicin in the actual medication that we hope to develop because even the slightest burning sensation upon injection may be undesirable for humans," said Woolf.

"Our plan is to take this technology further by working with a pharmaceutical company and looking for alternative ways to create the same 'warming' effect that opens the TRPV1 channels," said Woolf. "We are keen to move onto the next step and eventually develop a drug that produces a longer-lasting, pain-specific local anesthetic."

The authors believe that the earliest treatment uses of their technology may center on regional anesthesia, like epidurals for childbirth or abdominal surgery, and topical applications for patients with postherpetic neuralgia from shingles.

"Right now, most mothers are unable to stand up immediately after delivery because of the temporary paralysis caused by the epidural," said Woolf. "Additionally, since the anesthetic blocks the autonomic fibers



"We are optimistic that this method might allow regional anesthesia to expand from abdominal to thoracic surgery, which is currently not possible, as an epidural would block the nerves supplying the intercostal muscles making breathing depressed."

It is fitting that this discovery occurred at MGH, where general anesthesia using ether was first performed in October 1846, thus "transforming the practice of surgery and introducing the profession of anesthesiology," said Woolf. "We are hoping that this discovery will be the start of a new era in anesthesia, in which drugs are targeted specifically at the pain system to increase the efficacy and reduce the risks of anesthesia."

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