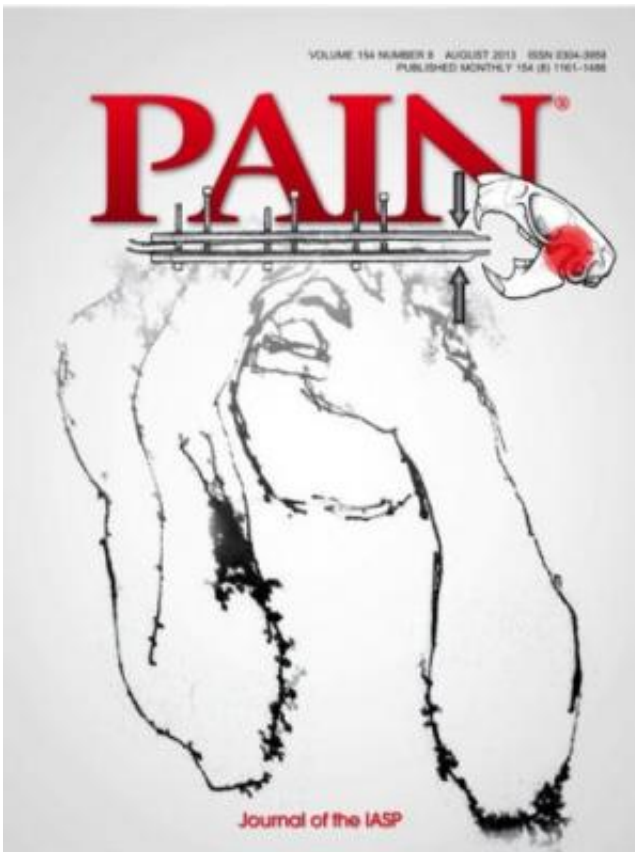


# Study reveals target for drug development for chronic jaw pain disorder

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Temporomandibular joint disorder (TMJD) is the most common form of oral or facial pain, affecting over 10 million Americans. The chronic

disorder can cause severe pain often associated with chewing or biting down, and lacks effective treatments.

In a study in mice, researchers at Duke Medicine identified a protein that is critical to TMJD pain, and could be a promising target for developing treatments for the disorder. Their findings are published in the August issue of the journal *Pain*.

Aside from cases related to trauma, little is known about the root cause of TMJD. The researchers focused on TRPV4, an ion [channel protein](#) that allows calcium to rapidly enter cells, and its role in inflammation and pain associated with TMJD.

"TRPV4 is widely expressed in [sensory neurons](#) found in the trigeminal ganglion, which is responsible for all sensations of the head, face and their associated structures, such as teeth, the tongue and temporomandibular joint," said senior study author Wolfgang Liedtke, M.D., PhD, associate professor of neurology and [neurobiology](#) at Duke. "This pattern and the fact that TRPV4 has been found to be involved in response to [mechanical stimulation](#) made it a logical target to explore."

The researchers studied both normal mice and mice genetically engineered without the *Trpv4* gene (which produces TRPV4 channel protein). They created inflammation in the temporomandibular joints of the mice, and then measured bite force exerted by the mice to assess jaw inflammation and pain, similar to how TMJD pain is gauged in human patients. Given that biting can be painful for those with TMJD, bite force lessens the more it hurts.

The mice without the *Trpv4* gene had a smaller reduction in bite force—biting with almost full force—suggesting that they had less pain. In normal mice there was more TRPV4 expressed in trigeminal sensory neurons when inflammation was induced. The increase in TRPV4

corresponded with a greater reduction in bite force.

The researchers also administered a compound to normal mice that blocked TRPV4, and found that inhibiting TRPV4 also led to smaller reductions in bite force, similar to the effects of the mice engineered without the *Trpv4* gene.

Surprisingly, the researchers found comparable bone erosion and inflammation in the jaw tissue across all mice, regardless whether the mice had TRPV4 or not.

"Remarkably, the damage is the same but not the pain," Liedtke said. "The mice that had the most TRPV4 appeared to have the most pain, but they all had similar evidence of temporomandibular joint inflammation and bone erosion in the jawbone as a consequence of the inflammation."

The results suggest that TRPV4 and its expression in trigeminal sensory neurons contribute to TMJD pain in mice. Given the lack of effective treatments for this chronic pain disorder, TRPV4 may be an attractive target for developing new therapies.

Provided by Duke University Medical Center

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