New evidence touch-sensing nerve cells may fuel 'ringing in the ears'

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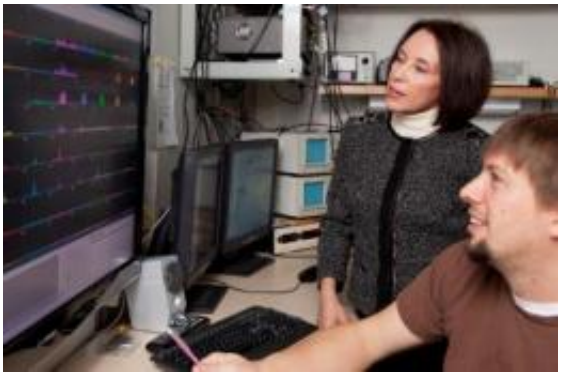

U-M researcher Susan Shore and graduate student Seth Koehler discuss hearing data. Credit: University of Michigan Health System

We all know that it can take a little while for our hearing to bounce back after listening to our iPods too loud or attending a raucous concert. But new research at the University of Michigan Health System suggests over-exposure to noise can actually cause more lasting changes to our auditory circuitry – changes that may lead to tinnitus, commonly known as ringing in the ears.

U-M researchers previously demonstrated that after hearing damage, touch-sensing "somatosensory" nerves in the face and neck can become overactive, seeming to overcompensate for the loss of auditory input in a way the brain interprets – or "hears" – as noise that isn't really there.

The new study, which appears in the Feb. 1 issue of The *Journal of Neuroscience*, found that somatosensory neurons maintain a high level of activity following exposure to loud noise, even after hearing itself returns to normal.

The findings were made in guinea pigs, but mark an important step toward potential relief for people plagued by tinnitus, says lead investigator Susan E. Shore, Ph.D., of U-M's Kresge Hearing Research Institute and a professor of otolaryngology and molecular and integrative physiology at the U-M Medical School.

"The animals that developed tinnitus after a temporary loss in their hearing after loud noise exposure were the ones who had sustained increases in activity in these neural pathways," Shore says. "In the future it may be possible to treat tinnitus patients by dampening the hyperactivity by reprogramming these auditory-touch circuits in the brain."

In normal hearing, a part of the brain called the dorsal cochlear nucleus is the first stop for signals arriving from the ear via the auditory nerve. But it's also a hub where "multitasking" neurons process other sensory signals, such as touch, together with hearing information.

During hearing loss, the other sensory signals entering the dorsal cochlear nucleus are amplified, Shore's earlier research found. This overcompensation by the somatosensory neurons, which carry information about touch, vibration, skin temperature and pain, is believed to fuel tinnitus in many cases.

Tinnitus affects up to 50 million people in the United States and millions more worldwide, according to the American Tinnitus Association. It can range from intermittent and mildly annoying to chronic, severe and debilitating. There is no cure.

It especially affects baby boomers, who, as they reach an age at which hearing tends to diminish, increasingly find that tinnitus moves in. The condition most commonly occurs with hearing loss, but can also follow head and neck trauma, such as after an auto accident, or dental work. Tinnitus is the number one disability afflicting members of the armed forces.

The involvement of touch sensing (or "somatosensory") nerves in the head and neck explains why many tinnitus sufferers can change the volume and pitch of the sound by clenching their jaw, or moving their head and neck, Shore explains.

While the new study builds on previous discoveries by Shore and her team, many aspects are new.

"This is the first research to show that, in the animals that developed tinnitus after hearing returned to normal, increased excitation from the somatosensory nerves in the head and neck continued. This dovetails with our previous research, which suggests this somatosensory excitation is a major component of tinnitus," says Shore, who serves on the scientific advisory committee of the American Tinnitus Association.

"The better we understand the underlying causes of tinnitus, the better we'll be able to develop new treatments," she adds.

More information: "Noise over-exposure alters long-term somatosensory-auditory processing in the dorsal cochlear nucleus – possible basis for tinnitus-related hyperactivity?" *Journal of Neuroscience,* Feb. 1, 2012.

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