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AUDITORY SYSTEM

A moving experience in the cochlea

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The ongoing debate about the mechanism by which the mammalian inner ear amplifies incoming sounds now sees the publication of new evidence in favour of a mechanism driven by an influx of calcium ions into the outer hair cells. The study uses a new type of *in vitro* cochlear preparation that allows researchers to get closer to the process than ever before.

Writing in *Nature Neuroscience*, Chan and Hudspeth describe how they removed part of the cochlea of the clawed jird (also known as the Mongolian gerbil) and mounted it in a recording chamber that had two compartments. The compartments were designed to reproduce the conditions found in the inner ear, in which the apical side of the sensory epithelium is bathed in endolymph — a fluid that is rich in potassium ions — while the basal side is bathed in sodium-rich perilymph. This set-up allowed the authors to record the responses of the cochlea to sound stimulation for up to 30 minutes.

The amplification process in the inner ear is an active, frequency-specific process that optimizes the ear's response to incoming sounds. A characteristic feature of the active process is compressive nonlinearity — a large range of stimulus intensities is compressed into a relatively narrow range of responses. The active process is also responsible for 'spontaneous otoacoustic emission' — sounds that are produced by the active movement of the inner hair cells and that can be recorded from the ear canal.

There are two theories about how the active process occurs. The first is active hair-bundle motility, and relies on a calcium current entering the cell during mechanoelectrical transduction. The calcium current is thought to

stimulate the closure of transduction channels and the activity of myosin motors, both of which could contribute to the amplification of the mechanical stimulation that is produced by sounds. The second theory is known as somatic electromotility. This is also initiated by electrical activity, but relies on conformational changes in a protein called prestin.

Chan and Hudspeth set out to test the first theory. They blocked the mechanoelectrical transduction channels with amiloride, so that calcium ions could not enter the cells, and found that this abolished the nonlinear amplification process. By contrast, when the ionic contents of the artificial endolymph were manipulated so that currents other than the calcium current were abolished, the nonlinear amplification persisted. These results provide evidence that calcium influx is both necessary and sufficient for the active process.

The debate will no doubt continue, with proponents of both theories continuing to test their ideas. It might be that both mechanisms are involved, perhaps at different frequencies. But the *in vitro* preparation used by Chan and Hudspeth should be a useful tool in the investigation.

References and links

ORIGINAL RESEARCH PAPER
Chan, D. K. & Hudspeth, A. J. Ca^{2+} current-driven nonlinear amplification by the mammalian cochlea *in vitro*. *Nature Neurosci.* **8**, 149–155 (2005) | Article | PubMed | ChemPort |
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http://www.rockefeller.edu/labheads/hudspeth/hudspeth-lab.php

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