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Study solves mystery of mammalian ears

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A 30-year scientific debate over how specialized cells in the inner ear amplify sound in mammals appears to have been settled more in favor of bouncing cell bodies rather than vibrating, hair-like cilia, according to investigators at St. Jude Children's Research Hospital.

The finding could explain why dogs, cats, humans and other mammals have such sensitive hearing and the ability to discriminate among frequencies. The work also highlights the importance of basic hearing research in studies into the causes of deafness. A report on this work appears in the advanced online issue of *Proceedings of the National Academy of Science*.

"Our discovery helps explain the mechanics of hearing and what might be going wrong in some forms of deafness," said Jian Zuo, Ph.D., the paper's senior author and associate member of the St. Jude Department of Developmental Neurobiology. "There are a variety of causes for hearing loss, including side effects of chemotherapy for cancer. One strength of St. Jude is that researchers have the ability to ask some very basic questions about how the body works, and then use those answers to solve medical problems in the future."

The long-standing argument centers around outer hair cells, which are rod-shaped cells that respond to sound waves. Located in the fluid-filled part of the inner ear called the cochlea, these outer hair cells sport tufts of hair-like cilia that project into the fluid. The presence of outer hair cells makes mammalian hearing more than 100 times better than it would be if the cells were absent.

As sound waves race into the inner ear at hundreds of miles per hour, their energy—although dissipated by the cochlear fluid—generates waves in the fluid, somewhat like the tiny waves made by a pebble thrown into a pond. This energy causes the hair cell cilia in both mammals and non-mammals to swing back and forth quickly in a steady rhythm.

In mammals, the rod-shaped body of the outer hair cell contracts and then vibrates in response to the sound waves, amplifying the sound. In a previous study, Zuo and his colleagues showed that a protein called prestin is the motor in mammalian outer hair cells triggers this contraction. And that is where the debate begins.

While both mammals and non-mammals have cilia on their outer hair cells, only mammalian outer hair cells have prestin, which drives this cellular contraction, or somatic motility. The contraction pulls the tufts of cilia downward, which maximizes the force of their vibration. In mammals, both the cilia and the cell itself vibrate. Thus far the question has been whether the cilia are the main engine of sound amplification in both mammals and non-mammals.

One group of scientists believes that somatic motility in mammalian outer hair cells is simply a way to change the height of the cilia in the fluid to maximize the force with which the cilia oscillate. That, in turn, would amplify the sound. An opposing group of scientists maintains that although the vibration of the outer hair cell body itself—somatic motility—does maximize the vibration of the cilia, the cell body works independently of its cilia. That is, vibration of the mammalian cell dominates the work of amplifying sound in mammals.

“If somatic motility is the dominant force for amplifying sound in mammals, this would mean that prestin is the reason mammals amplify sound so efficiently,” Zuo said.

In the current study, Zuo and his team conducted a complex series of studies that showed in mammals that the role of somatic mobility driven by prestin is not simply to modify the response of the outer hair cells' cilia to incoming sound waves in the cochlea fluid. Instead, somatic motility itself appears to dominate the amplification process in the mammalian cochlea, while the cilia dominate amplification in non-mammals.

Zuo's team took advantage of a previously discovered mutated form of prestin that does not make the outer hair cells contract in response to incoming sound waves as normal prestin does. Instead, the mutated form of prestin makes the cell extend itself when it vibrates.

The St. Jude researchers reasoned that if altering the position of the cilia in the fluid changes the ability of the cilia to amplify sound, then hearing should be affected when the mutant prestin made the cell extend itself. Therefore, the team developed a line of genetically modified mice that carried only mutant prestin in their outer hair cells. The researchers then tested the animals' responses to sound.

Results of the studies showed no alteration in hearing, which suggested that it did not matter whether the outer hair cells contracted or extended itself, that is, raised or lowered the cilia. There was no effect on amplification. The researchers concluded that somatic motility was not simply a way to make cilia do their job better; rather, there is no connection between the hair cell contractions and how the cilia do their job. Instead, somatic motility, generated by prestin, is the key to the superior hearing of mammals.

Source: St. Jude Children's Research Hospital