

Physiologists discover molecular mechanism for stabilizing inner ear cells, with implications for hearing loss

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Mechanosensory hair cells in the inner ear pick up the softest sounds, such as whispers and distant noises.

Unlike other cells in the <u>human body</u>, these <u>sensory cells</u> are fragile and finite. At birth, the <u>human ear</u> contains approximately 15,000 of these cells. They do not regenerate or divide and, therefore are susceptible to permanent damage from exposure to loud sounds. Scientists believe understanding the molecular mechanisms that maintain the structure of these cells throughout the lifespan can provide insight into the fundamental causes of hearing loss and deafness.

University of Kentucky physiologists Catalina Vélez-Ortega, Gregory Frolenkov and their collaborators in the UK College of Medicine have discovered a <u>molecular mechanism</u> necessary for stabilizing stereocilia, the "hairs" of the sensory cells of the inner ear. Stereocilia are nanoscale structures grouped together in staircase-like rows and interconnected by extracellular filaments. Hearing happens when sound vibrations deflect stereocilia, tension the extracellular filaments and open mechanotransduction ion channels allowing calcium and other ions to enter the hair cell.

Frolenkov's team discovered that blockage of these channels cause the stereocilia to retract and that this retraction depends on the <u>calcium</u> <u>influx</u> through these channels. UK researchers uncovered the mechanism



maintaining the remarkable staircase-like architecture of the stereocilia. This contribution provides new insight into the molecular mechanisms that facilitate the detection of sound within the inner ear, with opportunities to explore molecular therapies to maintain the stereocilia structure.

This research will appear in an upcoming issue of *eLife*.

More information: A Catalina Vélez-Ortega et al. Mechanotransduction current is essential for stability of the transducing stereocilia in mammalian auditory hair cells, *eLife* (2017). <u>DOI:</u> <u>10.7554/eLife.24661</u>

Provided by University of Kentucky

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