Department für Neurowissenschaften

INSTITUT FÜR BIOLOGIE
UND UMWELTWISSENSCHAFTEN

Einladung zum Vortrag
im Rahmen des gemeinsamen Kolloquiums des Instituts für Biologie und Umweltwissenschaften und des Departments für Neurowissenschaften

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A connexin30 mutation rescues hearing and reveals roles for gap junctions in cochlear amplification and micromechanics

The CD-1mouse strain (CD-1) suffers early-onset age-related hearing-loss (ARHL), which commences at maturity. Like the human condition, ARHL becomes progressively worse with age until only low frequency hearing remains. High frequency hearing is restored completely through a mutation (p.Ala88Val, A88V) mutation in a gap junction protein (connexin 30, Cx30), although accompanied by a small decline in low frequency hearing.

Connexins 26 and 30 combine to form gap junctions between supporting cells in the cochlea, especially in the sensory organ of Corti and the stria vascularis, which generates the endocochlea potential (EP), and the high K+ concentration of the endolymph that bathes the apical surface of the hair cells. The EP and endolymph are essential for cochlea sensory transduction. Loss or genetic mutation of Cx26 and Cx30 is responsible for almost all inherited hearing loss, including ARHL. It is surprising, therefore, that a Cx30 mutation can rescue ARHL, especially since the EP was found to be greatly reduced. In all other known circumstances reduced EP is associated with hearing loss. In mice with the Cx30 mutation, the auditory responses are as sharply tuned and sensitive as those of a wild type mouse. Outcomes from this study throw light on the cochlear amplifier and how a voltage-dependent process, which should be frequency limited by the electrical low-pass filtering of cell membranes, can meet the demands of the mouse's ultrasonic hearing.

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Gastgeber: Prof. Dr. Christine Köppl, Prof. Dr. Georg Klump, Prof. Dr. Hans Gerd Nothwang

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