

PERIPHERAL FINE MODULATION OF HEARING: SLOW MOTILITY AND ELECTROMOTILITY OF OUTER HAIR CELLS

Rezső Borkó dr.

Department of Otorhinolaryngology,
Hetényi G. County Hospital, Szolnok, Hungary.

Motor activity of the outer hair cells (slow-, and fast motility or electromotility) is a significant mechanism which determines the sharp frequency selectivity and low threshold in mammals. Electromotility provides with amplification on a cycle-by-cycle basis. The exact role of slow motility in the peripheral processing, however, is much less known. The Theoris aims to examine the integration of slow and fast motility in cellular level and their influence on each other.

Slow motile length changes of isolated outer hair cells (OHCs) ($n = 36$) were induced by perfusion of saline (flow rate: $0,6 \mu\text{l}/\text{min}$) as a mechanical challenge or by perfusion of $12,5 \text{ mM}$ KCl solution for 90 s as a chemical and mechanical challenge with and without ocadaic acid (OA), a serine/threonine protein phosphatase inhibitor ($n = 9$, in each experiment). Electromotility was evoked by square pulses from $\pm 35 \text{ mV}$ to $\pm 240 \text{ mV}$ at different stages of cell shortening ($n = 36$). Stiffness of the lateral wall was measured by the micropipette aspiration technique ($n = 5$, in each experiment).

Both forms of stimulation (saline and $12,5 \text{ mM}$ KCl solution) caused a reversible shortening (KCl evoked greater). Slow shortening increased lateral wall stiffness, simultaneously, electromotility magnitude decreased. Ocadaic acid blocked slow shortening, increased lateral wall stiffness, and decreased the magnitude of electromotility. Applied stimulations of ocadaic acid treated OHCs do not further change stiffness or electromotility.

Isolated OHCs respond with slow shortening and consecutive cell stiffness increase to mechanical insult. This phenomenon seems operating with calcium-, and phosphorylation-dependent modifications of the cytoskeletal proteins. The subsequent electromotility gain decrease suggests a slow OHC shortening driven regulation of the cochlear amplifier with simultaneous safety control of the auditory periphery against overstimulation. Physiological relevance of these observations are, that a mechanically (loud sound) or chemically (K^+ intoxication, e.g. Ménière's disease) stimulated organ of Corti is capable of changing its micromechanical response properties.