Fast Cochlear Amplification with Slow Outer Hair Cells

T.K. Lu, S. Zhak, P. Dallos, R. Sarpeshkar
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In mammalian cochleas, outer hair cells (OHCs) produce mechanical amplification over the entire audio-frequency range (up to 100 kHz). Under the “somatic electromotility” theory, mechano-electrical transduction modulates the OHC transmembrane potential, driving an OHC mechanical response that generates cycle-by-cycle mechanical amplification. Yet, though the OHC motor responds up to at least 70 kHz, the OHC membrane RC time constant (in vitro upper limit ~1000 Hz) reduces the potential driving the motor at high frequencies. Thus, the mechanism for high-frequency amplification with slow OHCs has been a two-decade-long mystery. Previous models that fit experimental data incorporated slow OHCs but did not explain how the OHC time constant limitation was overcome. Our key contribution is showing that negative feedback due to organ-of-Corti functional anatomy with adequate OHC gain significantly extends closed-loop system bandwidth and increases resonant gain [1]. Figure 1 shows our macromechanical model of the cochlea. The OHCs implement negative feedback by exerting a corrective force on the reticular lamina (designated “rl” in Figure 1) that opposes changes to the system output caused by changes in the input stimuli. Our model produces realistic results (Figure 2) and demonstrates that the OHC gain-bandwidth product, not just bandwidth, determines whether high-frequency amplification is possible. Due to the cochlea’s collective traveling-wave architecture, the gain of a single OHC needs not be great. The OHC piezoelectricity increases the effectiveness of negative feedback but is not essential for amplification. Thus, emergent closed-loop network dynamics differ significantly from open-loop component dynamics, a generally important principle in complex biological systems.

**REFERENCES**
