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SENSORY AND MOTOR SYSTEMS

Molecular mechanisms of cell death in a mouse model of progressive hearing loss

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KCNQ4 is a voltage-gated K+ channel whose dysfunction in the inner ear is the main cause of the progressive hearing loss (HL) DFNA2. It develops in 2 phases: first, a mild HL (40-60 dB) and later, it progresses to a profound HL (> 90 dB). Previously, using a knock-out mouse model of the human DFNA2 (Kcnq4-/-), we reported that outer hair cell (OHC) degeneration may explain the first phase of HL and inner hair cell (IHC) and spiral ganglion neuron (SGN) degeneration occur in the second phase of HL. Now, we performed a functional hearing test, correlating these results with the molecular events leading to cell death and ultrastructural changes in the Organ of Corti's surface in both phases. We observed a profound HL starting at middle-aged (40-week-old (W)) Kcnq4-/- mice, as revealed by Preyer´s reflex test. By immunofluorescence, we found caspase 3-mediated apoptosis (Cas-3) in SGNs and OHCs of Kcnq4-/- mice at different time points: in SGNs it was found late, at 54W and 68W, which correlates with our functional studies elucidating the profound HL of the last phase. On the other hand, OHCs showed a Cas-3 positive signal in 4W and 10W Kcnq4-/- mice, which could explain the mild HL of the first phase of DFNA2. IHCs did not show Cas-3 signal but they exhibited remarkable stereocilia defects by scanning microscopy, such as fusion and giant stereocilia in old mice. Collectively, these results are useful to understand the mechanisms involved in the human DFNA2.