EAAT5 mediates glutamate transport in mouse vestibular epithelium

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Synaptic transmission between hair cells and primary afferent fibres in the inner ear is mediated by glutamate. Type I vestibular hair cells are enveloped by calyx afferent terminals. The unusual geometry of the calyx, and the tonic release of glutamate by type I hair cells at this synapse means mechanisms must exist to clear glutamate from the synaptic cleft and prevent postsynaptic receptor desensitisation.

Immunofluorescence: Vestibular organs and retina (control) were obtained from mice (overdosed with Ketamine 300 mg/kg), sectioned and incubated in primary antibodies against the glial glutamate-aspartate transporter (GLAST) and EAAT5.

RT-PCR: Total RNA was extracted from retina and vestibular epithelium and the EAAT5 gene amplified using RT-PCR. Reaction products were separated on 1.5% agarose gel.

Results: Immunolabelling of GLAST was confined to supporting cells of the vestibular epithelium as shown previously. Until now, the expression of EAAT5 has only been reported in the retina. Significantly, RT-PCR and immunolabelling of EAAT5 show expression in crista and utricle. Interestingly, immunofluorescence of EAAT5 shows expression in both type I and II vestibular hair cells, as well as calyx primary afferent terminals and fibres.

Conclusions: EAAT5 is highly expressed in the mouse crista and utricle. Active uptake of glutamate by EAAT5 in both hair cells and primary afferent fibres may limit glutamate concentration within the synaptic cleft, thereby preventing glutamate receptor desensitisation. The expression of EAAT5 at tonically active glutamatergic synapses such as those in the vestibular epithelium, and retina suggests highly efficient glutamate uptake mechanisms have developed to maximize receptor sensitivity.