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Erratum: Age-related Decline in Kv3.1b Expression in the Mouse Auditory Brainstem Correlates with Functional Deficits in the Medial Olivocochlear Efferent System

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In the original submitted manuscript of this article [J. Assoc. Res. Otolaryngol. 8(2):280–293, 2007, DOI 10.1007/s10162-007-0075-x], our analysis showed a weak, but statistically insignificant, age-related decline in somatic Kv3.1b expression in the medial nucleus of the trapezoid body (MNTB). At the request of an anonymous reviewer, we re-analyzed the Kv3.1b immunoreactivity in MNTB to see whether age had a differential effect on expression of the channel protein along the tonotopic axis of this nucleus. After subdividing the nucleus to analyze immunoreactivity in high, mid, and low frequency regions of MNTB separately, we indeed found a significant age-related decline in somatic Kv3.1b immunostaining. We corrected the section of the Results and the graphs in Figure 4G accordingly, but neglected to change the text elsewhere in the article to reflect this new finding.

The accompanying figure showing age-related decline in somatic MNTB staining is meant to replace Figure 7 of the published article.

The following text corrects the concluding paragraph of the published article:

Kv3.1b channel protein declined in neuronal cell bodies of the MOC efferent system (SPN, VNTB, and LNTB) and the MNTB in the CBA mouse by middle age (15 months), as summarized in Figure 7. Previous work by our group has shown decline in MOC function in CBA mice occurring by middle age, and Kv3.1b knockout mice showed poor MOC function as compared to +/+ and +/− genotypes. In addition, expression also declined at about the same rate in the neuropil of AVCNa, MNTB, and LSO. This decline is likely traced to distal axon segments and terminals of neurons projecting to these nuclei, and the most likely sources of these projections are the MOC neurons that likewise showed declines in somatic expression. Our data suggest that Kv3.1b channel protein may be important for normal MOC efferent function, and that declines in expression with age may result in functional deficits that precede outer hair cell loss, and some of the eventual cochlear pathologies that characterize age-related hearing loss—presbycusis—in mammals, including humans.

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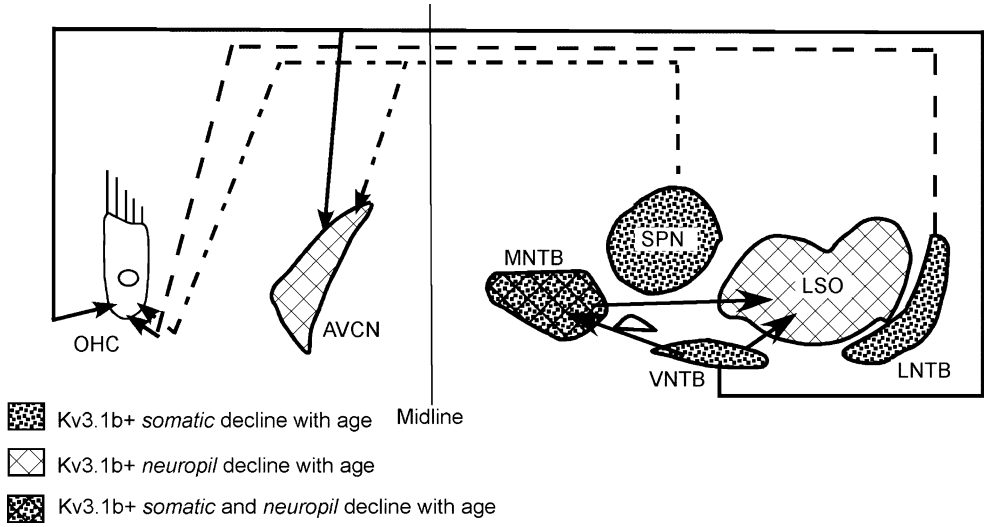

FIG. 7. Illustration showing connections between the MOC nuclei where age-related declines in somatic Kv3.1b expression (stippled) were found, and nuclei where Kv3.1+ neuropil declined (hatched). The figure does not show all connections, but is meant to demonstrate how decline in neuropil expression in the LSO, MNTB, and AVCNa may at least partially result from declines in expression within MOC neurons projecting to them