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Long-term treatment with aldosterone slows the progression of age-related hearing loss.

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Abstract

Age-related hearing loss (ARHL), clinically referred to as presbycusis, is one of the three most prevalent chronic medical conditions of our elderly, with the majority of persons over the age of 60 suffering from some degree of ARHL. The progressive loss of auditory sensitivity and perceptual capability results in significant declines in workplace productivity, quality of life, cognition and abilities to communicate effectively. Aldosterone is a mineralocorticoid hormone produced in the adrenal glands and plays a role in the maintenance of key ion pumps, including the Na-K(+)-Cl co-transporter 1 or NKCC1, which is involved in homeostatic maintenance of the endocochlear potential. Previously we reported that aldosterone (1 μM) increases NKCC1 protein expression in vitro and that this up-regulation of NKCC1 was not dose-dependent (dosing range from 1 nM to 100 μM). In the current study we measured behavioral and electrophysiological hearing function in middle-aged mice following long-term systemic treatment with aldosterone. We also confirmed that blood pressure remained stable during treatment and that NKCC1 protein expression was upregulated. Pre-pulse inhibition of the acoustic startle response was used as a functional measure of hearing, and the auditory brainstem response was used as an objective measure of peripheral sensitivity. Long-term treatment with aldosterone improved both behavioral and physiological measures of hearing (ABR thresholds). These results are the first to demonstrate a protective effect of aldosterone on age-related hearing loss and pave the way for translational drug development, using aldosterone as a key component to prevent or slow down the progression of ARHL.

KEYWORDS: Aging; Aldosterone; Drug development; Hearing loss; Startle reflex

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