

Antioxidants and hearing loss

February 8 2017, by Ziba Kashef



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Severe hearing loss is the third most prevalent chronic condition in older Americans and more than 15% of people in their 30s are also affected. The condition leads to communication problems, social isolation, depression, and cognitive decline. To study the cause, Yale researchers led by research scientist Alla Ivanova and professor of surgery Joseph Santos-Sacchi developed a novel mouse model of hearing loss.

Through experiments, the researchers found that the loss of a small mitochondrial protein, Fus1, in mice results in progressive [hearing loss](#) similar to human disease, but occurring at a young adult age.

Mitochondria are cellular structures that produce energy and maintain the balance of critical functions within cells.

The researchers found that hearing loss in their [mouse model](#) develops due to changes in mitochondria of the cochlea, a structure in the inner ear that transforms the sound to neural message sent to brain. They found that reactive oxygen species—potentially harmful molecules formed during mitochondrial respiration and produced in abundance by diseased mitochondria—are what trigger events leading to inability of cochlear tissues to process sound. Supplementation of young but not old mice with a common anti-oxidant, NAC (N-acetyl-cysteine), that neutralizes harmful reactive oxygen species prevented hearing loss.

This study emphasizes the role of healthy mitochondria in normal hearing and suggests that treatment with certain antioxidants or other mitochondria-improving drugs before development of irreversible damaging changes could be beneficial for hearing.

More information: Winston Tan et al. Novel role of the mitochondrial protein Fus1 in protection from premature hearing loss via regulation of oxidative stress and nutrient and energy sensing pathways in the inner ear, *Antioxidants & Redox Signaling* (2017). [DOI: 10.1089/ars.2016.6851](https://doi.org/10.1089/ars.2016.6851)

Provided by Yale University

Citation: Antioxidants and hearing loss (2017, February 8) retrieved 25 December 2022 from <https://medicalxpress.com/news/2017-02-antioxidants-loss.html>

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