



Response to letter to the editor: “association between obstructive sleep apnea and hearing loss among a cohort of emergency responders”

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Dear editor,

We thank Drs. Osmo, Andersen, and co-authors for their engagement with our work. We have considered their letter carefully and responded below:

In our recent manuscript, [1] we reported an increase in hearing loss affecting both high and low frequencies with increasing Obstructive Sleep Apnea (OSA) severity. We hypothesized that OSA-induced intermittent hypoxia (IH) with reduced blood flow to the inner ear and increased oxidative stress-related inflammatory mediator release

mechanistically contributed to hearing loss among a cohort of emergency responders.

Osmo et al rightly identify the difficulty of studying the IH effect in humans and cited studies implicating both central and peripheral pathways in high-frequency hearing loss in mice exposed to 3 hours of IH. Indeed, mouse models of IH have been instrumental in clarifying mechanisms of oxidative stress and inflammation across multiple organ systems. For example, mice-IH studies also have investigated the role of oxidative stress and inflammation in promoting melanoma lung metastases [2]. These findings, while not directly related to auditory function, reinforce the plausibility of systemic inflammation and oxidative effects arising from IH that may extend to the ear structures.

As Osmo et al mentioned, there are mechanisms of oxidative stress in OSA that potentially lead to cardiovascular and neurologic adverse outcomes. In highlighting work regarding olfactory dysfunction, the authors propose an expanded list of pathophysiologic mechanisms besides IH possibly contributing to hearing loss.

One study reported an association between olfactory dysfunction and OSA severity measured by AHI [3]. Further, a population-based study provided a cross-sectional association between olfactory dysfunction and hearing loss-dysphonia among nearly 18,000 Korean adults [4].

The role of sleep architecture and duration remains unclear. Osmo et al reference a study that found a statistically significant improvement in Quick Speech-in-Noise (QuickSIN) performance with just ten additional minutes of REM sleep, however, the clinical relevance of this modest change is uncertain. They also report associations between hearing impairment and sleep duration though it remains unclear whether and how depressive symptoms may mediate

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or confound these associations. In a large cross-sectional study of 2,777 adults aged 20–69 both short and long sleep durations were associated with hearing loss, though depression was not among them [5].

We appreciate the authors' careful reading of our paper and agree that OSA should be viewed as a systemic condition. While animal models of IH provide valuable mechanistic insight, they capture only one feature of OSA. As the authors noted, the over-reliance on cross-sectional data highlights the need for longitudinal and randomized controlled trial studies, supported by collaboration across sleep medicine, hearing, and neuroscience.

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Data Availability No datasets were used for this letter. The datasets generated during and/or analyzed during the original study are available from the corresponding author on reasonable request.

Declarations

Ethical approval This study was approved by the Institutional Review Board of Albert Einstein College of Medicine. As an observational study, analyses were conducted using pre-existing data. All procedures

involving human participants adhered to the ethical principles outlined in the 1964 Declaration of Helsinki.

Consent to participate All participants consented to participate.

Competing interest The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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