**No evidence for a protective role of hearing aids for the risk of dementia in UK Biobank: a response to Jiang et al. (2023)**

**Mur, J.[[1]](#footnote-1),[[2]](#footnote-2), Klee, M****.[[3]](#footnote-3), Muniz-Terrera, G.[[4]](#footnote-4),[[5]](#footnote-5), Leist, A. K.3**

Hearing loss is considered a major risk factor for dementia1. Due to the possibility of improving hearing with the use of hearing aids, the latter have garnered considerable attention as a potentially modifiable intervention to delay the onset of dementia. The provision of hearing aids to reduce the risk of dementia may also be clinically cost-effective, which not all lifestyle interventions fulfil2. In the absence of randomised controlled trials with a sufficiently long follow-up, observational designs have been invaluable in describing the population trends of hearing loss, hearing aid use, and dementia incidence, and in assessing the direction and strength of potential effects3,4.

Jiang et al. (2023) 5 analysed the rate of dementia in users and non-users of hearing aids in the UK Biobank cohort. The study had an observational design, testing hearing loss and the use of hearing aids as exposures to modify the risk of dementia, and controlling for relevant confounders but unable to fully rule out selection effects. The authors found that those participants with hearing loss who used hearing aids at baseline did not exhibit a higher rate of dementia during follow-up compared to people without hearing loss (HR=1.04, 95% CI=0.98-1.10). Participants with hearing loss who did not use hearing aids at baseline did exhibit a higher rate of dementia when compared to people without hearing loss (HR=1.42, 95% CI=1.29-1.56). The interpretation was that among people with hearing loss, users of hearing aids were at lower risk of dementia than non-users, suggesting a potential role for hearing aid use in the prevention of dementia. The study received substantial coverage and led some to declare that the evidence for a causal role for hearing aids in the prevention of dementia was now “*as good as possible without randomised controlled trials*” 6.

In recent months, we tried and failed to reproduce the results by Jiang et al. using the same sample as the original study. First, we could not reproduce the numbers of missing observations as reported in the original paper – our numbers were considerably higher (**S1**). Second, we did not find the same proportions of dementia diagnoses in the different exposure groups as Jiang et al. (**S2-3**). Finally, we were unable to reproduce the main results – we found that before and after adjusting for confounders, hearing loss was significantly associated with the rate of dementia. Moreover, when comparing people with hearing loss using hearing aids to those without hearing loss, the rate of dementia was *greater* (HR=1.30, 95% CI=1.15-1.46) than when comparing people without hearing aids to those without hearing loss (HR=1.07, 95% CI=0.99-1.15). Our results differed from those of Jiang et al. also for Alzheimer’s disease and vascular dementia. While we could not exactly recreate all covariates using the same coding as in the original paper – the authors did not describe some crucial steps in defining or recoding of those variables (**S4**) neither in the article nor upon requests – we performed multiple sensitivity analyses to confirm the robustness of our results, including varying the method to determine dementia and the dates of right-censoring. (**S5-7**). We wrote the code for data preparation and analysis anew because we could not access the code of the original paper. Our code is available at https://github.com/JuM24/hearing\_dementia\_UKB.

Our results do not correspond well with hypotheses about the preventative potential of hearing aids. We also do not have a clear explanation for the direction of the effect. Selection bias inherent to the UK Biobank cohort7, unadjusted confounding, measurement error, suboptimal operationalisation of the exposure, timing in the provision of hearing aids, and an unsystematic attempt at confounder adjustment8 could be contributing factors.

Discrepancies such as those described in the present article could be more easily avoided if authors described in detail the definitions of new variables and transformations of existing variables, and published the code used to derive and analyse the data. We also stress that the work by Jiang et al. was not designed to assess causality. While another discovery of the co-occurrence of hearing aid use and a lower rate of dementia would be a reason for cautious optimism, claims of a definitive conclusion with hitherto published observational data are in our opinion premature. The association between hearing loss and dementia is complex and hearing aids may not be universally effective at reducing cognitive decline in all groups9. A causal role for the use of hearing aids in the risk of dementia is thus far from a settled issue.

We would welcome other teams with access to the data to check the discrepancies and Jiang et al. to respond to our observations. We encourage researchers, in the spirit of Open Science, to provide transparent accounts of their methodology by publishing their full code and any other relevant information to enhance reproducibility of research results.

**References**

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1. Centre for Clinical Brain Sciences (CCBS), University of Edinburgh, Edinburgh, UK. [↑](#footnote-ref-1)
2. Centre for Genomic & Experimental Medicine (CGEM), University of Edinburgh, Edinburgh, UK. [↑](#footnote-ref-2)
3. Department of Social Sciences, Institute for Research on Socio-Economic Inequality (IRSEI), University of Luxembourg, Esch-sur-Alzette, Luxembourg. [↑](#footnote-ref-3)
4. Centre for Dementia Prevention, University of Edinburgh, UK. [↑](#footnote-ref-4)
5. Ohio University, Athens, OH, USA. [↑](#footnote-ref-5)