Environment International 161 (2022) 107104

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

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Letter to the editor regarding, “The effect of occupational exposure to noise on ischaemic heart disease, stroke and hypertension: A systematic review and *meta*-analysis from the WHO/ILO joint estimates of the work-related burden of disease and injury”

Teixeira et al. (2021) recently published a systematic review on the association between occupational noise exposure and the risk of developing ischemic heart disease, stroke, and hypertension. The authors concluded that there is limited evidence of a harmful association between occupational noise exposure and ischemic heart disease and inadequate evidence of a harmful association between occupational noise exposure or stroke and hypertension.

We also recently published a systematic review on the association between occupational noise exposure and hypertension based on six cohort studies, one case-control study, and 17 cross-sectional studies (Bolm-Audorff et al. 2020). We included only studies with measured noise exposures of > 80 dB(A) and a comparison group with occupational noise exposure of ≤ 80 dB(A). Our *meta*-analysis found the risk for developing hypertension (≥ 140/90 mmHg) was increased by a factor of 1.81 (95% CI 1.51–2.18) for workers with occupational noise exposure of > 80 dB(A). We also detected a positive dose–response relationship. The hypertension risk in two studies looking at noise exposures of < 80 dB(A) compared to even lower noise exposures (<75 dB(A) or < 50 dB (A)) was 1.21 (95% CI 0.78–1.87; 2 studies). Risk of hypertension increased to 1.77 (95% CI 1.36–2.29; 5 studies) at > 80 to ≤ 85 dB(A) and increased further to 3.50 (95% CI 1.56–7.86; 3 studies) at > 85 to ≤ 90 dB(A). According to GRADE (Morgan et al. 2019; Schünemann et al. 2017), we rated the confidence of evidence for the association between occupational noise exposure and hypertension as high (Bolm-Audorff et al., 2020).

Given the substantial public health relevance of a causal relationship between occupational noise exposure and hypertension, it is crucial to address our conflicting results. The methodological differences summarized in Table 1 help explain the divergent results. However, after careful evaluation of the systematic review by Teixeira and colleagues (2021), we have the following criticisms regarding their analysis of the association between occupational noise and hypertension:

1. Teixeira et al. (2021) excluded cross-sectional studies from their review. Indeed, cross-sectional studies tend to underestimate the association between occupational exposures and disease because of the healthy worker effect. However, we believe that the influence of the healthy worker effect in the association between occupational noise exposure and hypertension is not severe (particularly in studies that screened for hypertension) because hypertension is largely asymptomatic in the early stages. The exclusion of cross-sectional studies disregarded much of the evidence regarding occupational noise exposure and the risk of hypertension. While our inclusion of cross-sectional studies might have resulted in an overestimation of risk, even when we exclude cross-sectional studies from our *meta*-analysis we find a statistically significant relative increase in hypertension risk of 1.27 (95% CI 1.02–1.57) (Fig. 1). In comparison, Teixeira et al. (2021) report a pooled risk ratio of 1.07 (95% CI 0.90–1.28) for hypertension. This remaining difference is due in part to the noise exposure levels considered by both systematic reviews.
2. In our opinion, it is inappropriate to define workers with noise exposure of ≥ 85 dB(A) as exposed and workers exposed to noise < 85 dB(A) as unexposed. In our systematic review, workers with occupational exposures between > 80 and ≤ 85 dB(A) had a statistically significant increased risk of developing hypertension (≥ 140/90 mmHg) of 1.77 (95% CI 1.36–2.29). Therefore, we hypothesize that the choice of a reference group with occupational noise exposure as high as < 85 dB(A) in the systematic review by Teixeira et al. (2021) resulted in a substantial underestimation of risk.
3. Teixeira et al. (2021) also defined workers exposed to noise > 85 dB (A) for less than three years as unexposed. Our results found that the risk of hypertension doubles after five years of exposure to 90 dB(A). While this indistinct reference group definition does not seem to have impacted the hypertension results, it may have caused an underestimation of the risks for ischemic heart disease and stroke.
4. Although including only prospective studies, Teixeira et al. (2021) did not consider the association between occupational exposure duration and the risk of hypertension. We concluded that the risk of developing hypertension is statistically significantly increased by a factor of 1.88 (95% CI 1.12–3.15) for every 10 dB(A) increase in noise exposure experienced over a 40-year working life. Furthermore, we concluded that the duration of noise exposure associated with a doubling of the risk of hypertension was inversely related to the level of noise exposure. At a noise exposure level of 81 dB(A), the required exposure duration for a doubling of the hypertension risk is 40 years, and the risk of hypertension doubles after five years at 90 dB(A).
5. Teixeira et al. (2021) included three studies in their *meta*-analysis of the association between occupational noise exposure and hypertension incidence: Chang et al. (2013), Stokholm et al. (2013), and Tessier-Sherman et al 2017. The *meta*-analysis of these three studies resulted in a statistically non-significant increased risk of hypertension of 1.07 (95% CI 0.90–1.28). However, the following should be noted regarding these three studies:
   a. Stokholm et al. (2013) conducted a longitudinal data-linkage study of 108,402 male and 36,788 female workers in Denmark. This study found a statistically significant association between occupational noise exposure and hypertension only in women. In this study, the relative risks for hypertension compared industrial workers to unexposed financial-sector employees or were based

https://doi.org/10.1016/j.envint.2022.107104
Received 16 July 2021; Received in revised form 7 December 2021; Accepted 18 January 2022
Available online 18 February 2022

Table 1
Summary of differences in the systematic reviews (regarding hypertension risk).

	Teixeira et al. 2021	Bolm-Audorff et al. 2020
Study design	Longitudinal intervention and observational studies: - randomized controlled trials (including parallel-group, cluster, cross-over, and factorial trials): - cohort studies - case-control studies - other non-randomized intervention studies (including quasi randomized controlled trials, controlled before-after studies, and interrupted time series studies)	All observational studies (cross-sectional or longitudinal): - cohort studies, - case-control studies - cross-sectional studies (if > 10% of the target population participated) - case-cohort studies - nested-case-control studies
Exposition	occupational noise ≥ 85 dB(A) objective or subjective measurements - sound level meter - expert assessment - job exposure matrix - self-reported exposure (subjective)	occupational noise > 80 dB(A) quantified objective noise measurements - sound level meter - expert assessment - job exposure matrix
Comparator	occupational noise < 85 dB(A) *"In some studies, the comparator was exposure to ≥ 85 dB(A) for < 3 years…"*	occupational noise ≤ 80 dB(A)
Outcome	Hypertensive heart disease (ICD 10 = I10–I11, I13–I15) Prevalence, incidence, or mortality based on: - physician diagnosis - hospital discharge data - administrative data (e.g. registered sickness) - registry data of treatments - certified cause of death - self-reported (subjective)	Primary arterial hypertension (ICD 10 = I10) Prevalence or incidence defined using one of the following criteria: - SBP ≥ 130 mmHg – 24-hour SBP ≥ 130 mmHg - DBP ≥ 80 mmHg – 24-hour DBP ≥ 80 mmHg - physician diagnosis - drug treatment
Confounders	age, sex, and socioeconomic position	age, sex (minimum) additionally considered: pre-existing hypertension, use of hearing protection, stress levels, amount of physical work, sound quality
Longitudinal studies included in the *meta*-analysis		
Studies included in both	Chang et al. (2013); Stokholm et al. (2013) *- adjusted risk ratio for 3–9 years vs < 3 years of exposure to > 85db(A)*	Chang et al. (2013); Stokholm et al. (2013) *- adjusted risk ratio for cumulative noise exposure to 95–99 dB(A)-year vs < 70 dB(A)-year*
Studies included only by Teixeira et al. (2021)	Tessier-Sherman et al. (2017)	Tessier-Sherman et al. (2017) *- excluded because the comparison group was exposed to > 80 dB(A)*
Studies included only in Bolm-Audorff et al. (2020)	Fokin et al. (2018) Hwang et al. (2007) Lui et al. (2016) Melamed et al. (2001) *- studies excluded because noise exposed group included exposures in the range 80–85 dB (A)* Siagian et al. (2009) *- not included (reason not reported)*	Fokin et al. (2018) Hwang et al. (2007) Lui et al. (2016) Melamed et al. (2001) Siagian et al. (2009)

SBP systolic blood pressure; DBP diastolic blood pressure.

on cumulative noise exposures, and were adjusted for age, socioeconomic status, and possibly exposure duration as employment duration. The study found a positive dose–response relationship in men as a function of cumulative noise exposure with a relative risk of 4.66 (95% CI 3.63–5.97) at > 100 [dB(A) × years]. This association disappeared after adjustment: relative risk = 0.99 (95% CI 0.75–1.31). In women, a positive dose–response relationship was also found. Risk of hypertension was statistically significantly increased for women by a factor of 2.40 (95% CI 1.99–2.89) in the highest noise category of 95 – 99 [dB(A) × years]. After adjustment, the risk of hypertension was attenuated but still statistically significantly increased by a factor

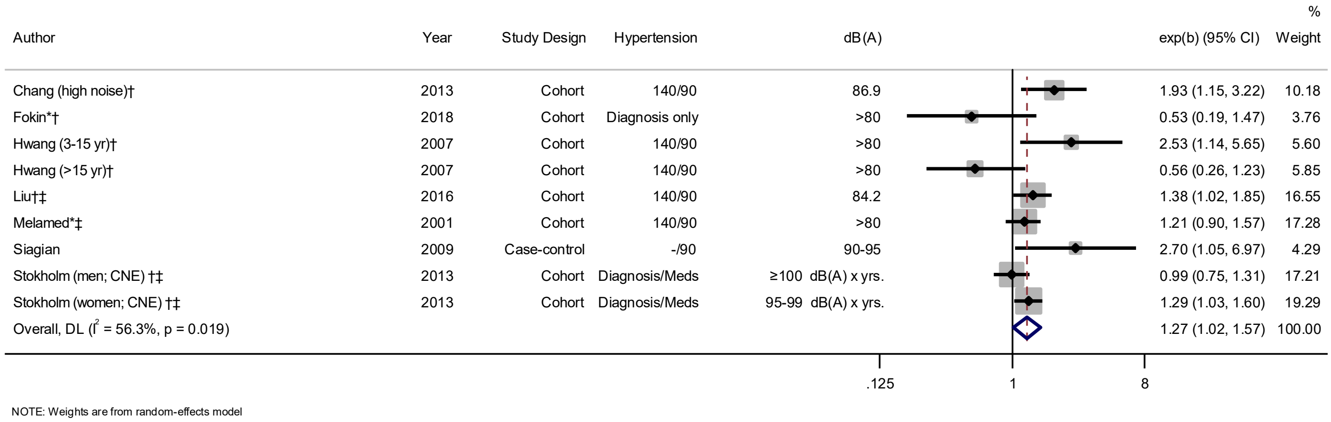

Fig. 1. Forest plot of longitudinal studies examining hypertension risk associated with occupational noise exposure > 80 dB(A) versus ≤ 80 dB(A). *The * indicates studies that we calculated the effect size (*exp*[b]) from the reported prevalences. † indicates that a physician diagnoses of hypertension was included in hypertension definition, and ‡ indicates that anti-hypertensive use was included in the hypertension definition.* CNE Cumulative Noise Exposure.

of 1.29 (95% CI 1.03–1.60) in the highest category. It could not be determined if the fully adjusted models adjusted for exposure duration (as duration of employment). If so, this may be an over-adjustment that is obscuring the positive dose–response relationship. Furthermore, individual noise exposure is known only among 710 of the 145,190 cohort members (0.5 %), so there may be non-differential misclassification regarding occupational noise exposure. In addition, the study can only correctly estimate risk if the employees exposed to noise visit a doctor and are treated for hypertension with the same frequency as the comparison group of financial sector employees. This was not investigated in the study. Due to their higher social status, financial sector employees might visit a doctor and receive treatment for hypertension more frequently than noise-exposed industrial workers (Fouriaud et al. 1984). Although Stokholm et al. (2013) adjusted their analysis for social status, it is questionable whether this reliably eliminated the potential detection bias which could result from a higher treatment prevalence for hypertension among financial sector employees compared to noise-exposed workers. Also, the risk ratio Teixeira et al. (2021) included in their *meta*-analysis represents the risk associated with a duration of exposure to > 85 dB(A) for 3 to 9 years versus < 3 years and does not correspond to the risk estimated for cumulative noise exposures.

b. Chang et al. (2013) conducted a retrospective cohort study of 152 highly exposed aircraft manufacturing workers in Taiwan with noise exposure of ≥ 85 dB(A), 221 workers with noise exposure of 80 – 85 dB(A), and 205 workers with noise exposure of < 80 dB (A). This found a statistically significant increased risk of hypertension (≥ 140/90 mmHG) of 1.93 (95% CI 1.15–3.22) in workers exposed to ≥ 85 dB(A) compared to workers exposed to < 80 dB (A). Teixeira et al. (2021) rated this as one of the best studies. However, the adjustment for exposure (employment) duration could also result in an underestimation of risk.

c. We excluded the study by Tessier-Sherman et al. (2017) from our systematic review because the study did not include a control group exposed to < 80 dB(A). However, the final risk estimates of this study were also overadjusted because the final regression model included the annual loss of hearing threshold, which (at least in part) is a direct result of occupational noise exposure. However, the risk ratio attributed to Tessier-Sherman et al. (2017) in the *meta*-analysis by Teixeira et al. (2021) does not appear in the original publication.

6. In addition, Teixeira et al. (2021) included the following studies in their review but excluded them from the *meta*-analysis:

a. We excluded the cohort study by Huo Yung Kai et al. (2018) from our review because the data on noise exposure were based on interviews of study participants rather than objective measurements.

b. We excluded the case-control study by Tong et al. (2017) from our systematic review because the unexposed workers had noise exposures up to < 85 dB(A).

7. In contrast to the systematic review by Teixeira et al. (2021), we included the cohort studies by Fokin et al. (2018), Hwang et al. (2012), Liu et al. (2016), and Melamed et al. (2001) in our review. These studies considered occupational noise exposures between ≥ 80 – <85 dB(A).

8. Teixeira et al. (2021) did not include the nested case-control study by Siagian (2012) in their systematic review. This study found a statistically significantly increased odds ratio for hypertension of 2.70 (95% CI 1.05–6.97) in aircraft pilots with occupational noise exposure of 90 – 95 dB(A). Teixeira et al. (2021) only provide exclusion reasons for the first 30 of 172 excluded studies in their supplementary data, so we could not determine the reason for exclusion.

Thus, despite the use of an extensive study protocol (Teixeira et al. 2019), the evidence examined by Teixeira et al. (2021) may have been incomplete. Also this review overlooked potential problems with the primary studies (e.g., overadjustment) and the presentation of the review results lacked transparency (i.e., complete list of reasons for study exclusion). Moreover, the overall assessment of the evidence for acquired hypertension was, in our opinion, overly critical. Teixeira et al. (2021) downgraded the confidence of evidence two levels for indirectness of evidence because few studies included women and most did not include national populations. While we agree more research on women is needed, there is direct evidence for men, so downgrading would not make sense here. Possibly, a gender-specific statement on the confidence of evidence should also be considered here. It is also unclear why studies of working populations in certain industries or occupations lack directness. According to their own protocol (Teixeira et al. 2019) a downgrading for directness of evidence should be considered a) when the populations studied differ (biologically) from the population of interest, b) when the exposure (intervention) differs from the exposure of interest, or c) when the outcome differs from the primary outcome. We argue that studies examining working populations in selected industries or occupations do provide direct evidence, and it is not always necessary (or fitting) to study the consequences of occupational exposures in the general population.

In summary, we believe our methods provide valid results. We find the methods applied by Teixeira et al. (2021) tend to lead to a (possibly strong) underestimation of the association between occupational noise and hypertension and to an underestimation of the "Confidence of Evidence."

Declaration of 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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Handling Editor: Adrian Covaci