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Tinnitus and cardiovascular disease: the population-based Tromsø Study (2015–2016)

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ABSTRACT

Introduction People with tinnitus are likely to have other co-occurring disorders that should be considered when diagnosing and understanding tinnitus as a health problem. The association between tinnitus and cardiovascular health in the general population is, however, unclear. This study aimed to examine whether tinnitus is associated with the prevalence of hypertension, myocardial infarction or stroke, in the general adult population.

Methods We used data from the seventh survey of the Tromsø Study, a comprehensive population-based health study carried out in 2015–2016. All inhabitants aged ≥40 years in the municipality of Tromsø, Norway, (n=32 591) were invited, of which 21 083 individuals (65%), aged 40–99, participated. Poisson regression was used to analyse the relationships between tinnitus and cardiovascular disease, while adjusting for relevant covariates. We used three separate tinnitus variables as exposures in analyses: (1) 'tinnitus status', measured with the question 'During the last 12 months, have you experienced ringing in your ears lasting more than five min?'; (2) 'tinnitus symptom intensity', generated as a function of tinnitus frequentness and tinnitus bother; and (3) 'tinnitus bother', analysed only among participants with tinnitus. Hypertension (measured blood pressure and/or self-reported use of antihypertensives), and self-reported myocardial infarction and stroke were the outcomes of analyses.

Results Analyses of the complete sample (n=17 288, 51.2% women), in fully adjusted models, revealed non-significant and very weak associations between tinnitus status and all three cardiovascular outcomes (prevalence ratios (PRs): 1.04–1.11), while for tinnitus symptom intensity, we found significant positive associations between low-intensity tinnitus and hypertension (PR: 1.08, 95% CI: (1.01 to 1.16) and myocardial infarction (PR: 1.39, 95% CI: 1.07 to 1.81). Among participants with tinnitus (n=3570), there were no associations between tinnitus bother and cardiovascular outcomes.

Conclusions Results from the present study indicate that there is a weak association between tinnitus and cardiovascular disease and that tinnitus should be taken seriously even at low intensities.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Tinnitus is a commonly occurring condition that is often accompanied by comorbidities. The association between tinnitus and cardiovascular disease has been examined in previous studies, but results are inconclusive, and population-based studies examining the relationship between tinnitus and cardiovascular diseases are lacking.

WHAT THIS STUDY ADDS

⇒ In a general adult population sample, we found weak positive associations between tinnitus and the prevalence of cardiovascular disease (hypertension, myocardial infarction and stroke). Associations were strongest for tinnitus with low symptom intensity.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Our results indicate that individuals with tinnitus who seek healthcare should be examined for cardiovascular comorbidity.

INTRODUCTION

Tinnitus—the perception of sound without acoustic stimulation[1]—is a common health problem, with an international prevalence ranging between 5% and 43% in the general population.[2] Tinnitus can be accompanied by several comorbidities. Links have been drawn between tinnitus and sleep disorders,[3 4] cognitive impairment,[5 6] mental disorders such as depression and anxiety,[7] as well as chronic pain.[8] Various studies also link tinnitus to cardiovascular disease[9 10] and its risk factors, for example, high body mass index (BMI), hypertension, hypercholesterolaemia and smoking.[9–12]

There are several possible mechanisms for the association between tinnitus and cardiovascular disease. It has been suggested that circulatory factors, through impaired cochlear blood flow or ischaemia, may contribute to the development of tinnitus.[13] Further, tinnitus is associated with both stress and sleep disturbances,[3 4 14] which, if chronic, are among the common risk factors for both cardiovascular disease and other non-communicable

diseases.[15–17] Both chronic stress and sleep disturbance can affect, for example, blood pressure and lipid profile, which might promote the development of cardiovascular risk factors such as hypertension and obesity.[18 19]

Tinnitus can be compared with noise from external sources, which is defined as unwanted sound, and understood as a stressor.[20] Similarities of problems have been observed with tinnitus as what is found with exposure to external noise, such as psychological distress and sleep disturbance,[21 22] but there is a large variation in noise annoyance at a given exposure for both types of sound. Studies have shown associations between environmental noise exposure and increased risk of cardiovascular disease, with the main mechanisms suggested to be stress and sleep disturbances.[23] One may argue that the association between noise as a stressor and cardiovascular disease should exist irrespective of the source of noise, so that tinnitus, regarded as internal noise, could be expected to have the same physiological significance as an external sound source. For tinnitus, stress may also act in the reverse direction, triggering and worsening tinnitus symptoms[14] and creating a vicious circle of stress, resulting in persistently high stress levels. Given the possible mechanisms, it can thus be assumed that tinnitus may be both a consequence of, and possibly also a risk factor for, stress-related disease.

The risk of hypertension and cardiovascular disease differs with sex and age. In general, men are more at risk of hypertension and cardiovascular disease than women of similar ages.[24 25] Likewise, there are age and sex differences in both tinnitus prevalence and annoyance.[2 26] Tinnitus prevalence is typically found to increase with age, sometimes peaking in prevalence at around 70 years before declining, and prevalence is most often reported to be higher in men than women.[2] It is therefore likely that the association between tinnitus and cardiovascular disease may vary across different groups of age and sex.

Several studies have examined the associations between cardiovascular risk factors and cardiovascular disease and tinnitus, but study design and quality of studies vary, and findings are inconclusive. Some studies have reported positive associations with tinnitus for eg, hypertension, obesity, ischaemic heart disease and stroke, while others found no, or sometimes negative, associations for the same conditions.[27] Population-based studies investigating associations between cardiovascular diseases and tinnitus are lacking,[27] but based on the scarce existing evidence, more studies are warranted.[28]

Establishing the relationship between tinnitus and cardiovascular disease is important, for several reasons. Comorbid disease in general could influence the effectiveness and result of tinnitus treatment. Additionally, determining the existence of such an association might help further hypotheses generation and testing of mechanisms and causal pathways.

This study aims to explore the association between tinnitus and cardiovascular disease in a general adult population sample. We investigate if tinnitus is associated with the prevalence of hypertension, myocardial infarction or stroke—three common cardiovascular health outcomes that have been associated with environmental noise.[29] We also examine whether a possible association depends on the tinnitus symptom intensity (as a function of frequentness and bother) or the degree of bother. Lastly, we examine if associations between tinnitus symptom intensity and cardiovascular disease differ for separate groups of age and sex.

MATERIALS AND METHODS

Study sample

We used data from Tromsø7, the seventh survey of the Tromsø Study, which was carried out in 2015–2016. The Tromsø Study is a population-based study that originated in 1974 with a focus on cardiovascular disease, and has later grown into a wide-ranging health study including research areas such as diabetes, lung disease, cognitive function and pain.[30] In Tromsø7, extended questions on tinnitus were included.

Invitations to partake in Tromsø7 were sent out to all inhabitants aged 40 and above, in the municipality of Tromsø (n=32 591). Participation was 65%, totalling 21 083 people between the ages of 40 and 99. Data collection included clinical examinations, biological sampling and questionnaires. More detailed information on data collection can be found on the website of the Tromsø Study, which also includes links to questionnaires.[31] Previously published work by Jacobsen and colleagues also describes the Tromsø Study in more detail.[32]

Of the total sample, 82% (n=17 288) had no missing values for relevant variables and were used in analyses, while the quantity of missing for individual variables ranged from 0–5.7%.

Main variables

Tinnitus

Participants were asked about tinnitus: 'During the last 12 months, have you experienced ringing in your ears lasting more than five min?' (no / yes). Having experienced tinnitus for more than 5 min is the definition most frequently used in population studies,[2] and tinnitus for more than 5 min within the past year has been suggested as a standardised survey definition.[33] Participants who responded 'yes' were further asked how frequently they experienced tinnitus: 'How frequently do you have ringing in your ears?' ((1) less frequently than every week / (2) each week, but not every day / (3) each day, but not all the time / (4) most of the time), as well as how bothersome they found it: 'Please indicate how bothered you are by the ringing in your ears?' (numeric rating scale ranging from 0 (not bothered) to 10 (worst imaginable bother)).

Three separate tinnitus variables were used as exposure variables in the analyses. For the main analyses, we first used the original tinnitus question (tinnitus more than 5 min within the past 12 months) as exposure, a variable

referred to as 'tinnitus status'. Second, we created an index of tinnitus symptom intensity in the following manner: The tinnitus bother variable was categorised into four levels of bother, in accordance with previous research[8]; (1) not bothered (values 0–2), (2) a little bothered (values 3–5), (3) bothered (values 6–7), and (4) highly bothered (values 8–10). We then summed the four-category bother variable and the tinnitus frequentness variable (values 1–4), creating a tinnitus symptom intensity scale from 2 to 8. This scale was further divided into three categories by merging values 2–3, 4–6 and 7–8, creating the tinnitus symptom intensity categories 'low intensity', 'medium intensity' and 'high intensity', respectively. Participants without tinnitus were included in the category 'no tinnitus'.

The third tinnitus variable was the original tinnitus bother variable ranging from 0 to 10. When this variable was set as the exposure, the original tinnitus frequentness variable was included as a covariate in some of the analyses.

Cardiovascular disease

Blood pressure was measured three times, and the mean of the second and third measurements was used in the analyses. We defined hypertension as systolic blood pressure ≥140 mm Hg, and/or diastolic blood pressure ≥90 mm Hg, and/or current use of antihypertensive medication, according to standard criteria.[34] Antihypertensive medication use was surveyed with the question 'Do you use, or have you used blood pressure lowering drugs?' (currently/previously, not now/never used). History of myocardial infarction and stroke was recorded by participants answering the question 'Do you have, or have you had a heart attack?' (no/yes, previously) and 'Do you have, or have you had a cerebral stroke/brain haemorrhage?' (no/yes, previously).

The validity of self-reported measures of myocardial infarction and stroke has been examined elsewhere.[35] For myocardial infarction, the sensitivity was high (90.1%) and the positive predictive value was moderate (78.9%). For stroke, they were both moderate (sensitivity 81.1% and positive predictive value 64.3%).[35]

Covariates

In order to minimise bias in analyses, we used directed acyclic graphs (DAGs) as an aid in the selection of covariates. When using DAGs, the causal relationships between variables of interest are systematically considered,[36] based on existing literature. We used the software DAGitty[37] for the construction of the DAGs, and DAGitty then provided information on the minimal sufficient adjustment set, as well as which covariates could be added in the analyses without giving bias to the results. We constructed one DAG for each combination of exposure and outcome variables, and we made similar adjustment sets for each of the analyses (see online supplemental figure 1 for more information on DAGs). We adjusted for the following variables in all analyses: age, sex, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms and hearing loss.

Demographic variables

Education level was reported on a four-point scale: (1) Primary/partly secondary education: up to 10 years of schooling, (2) upper secondary education: a minimum of 3 years, (3) tertiary education, short: college/university less than 4 years and (4) tertiary education, long: college/university 4 years or more. We merged categories (3) and (4) before analyses. Marital status was reported by answering the question 'Do you live with a spouse/partner?' (no/ yes).

Alcohol and smoking

Alcohol drinking habits were measured with the question 'How often do you usually drink alcohol?' (never / monthly or less frequently / 2–4 times a month / 2–3 times a week / 4 or more times a week). This is the first question in the Alcohol Use Disorders Identification Test, which is a validated battery of questions used to screen for harmful alcohol consumption.[38] We merged some categories before analysis, generating the categories never / up to 4 times a month / 2 or more times a week. Participants were asked about their smoking status: 'Do you/did you smoke daily?' (yes, now / yes, previously / never).

Waist–height ratio

We selected waist–height ratio as the anthropometric measure used in analyses. Height was measured with light clothing and no shoes using a Jenix DS-102 height and weight scale (DongSahn Jenix, Seoul, Korea). Waist circumference was measured with a Seca measurement tape at the level of the umbilicus. We used a cut-point of 0.59 for abdominal obesity,[39] which is strongly linked to the development of cardiovascular disease.[40]

Diabetes

The diabetes status of participants was surveyed with the question 'Do you have, or have you had diabetes?' (no / yes, now / yes, previously). The categories for current and previous diabetes were merged for analyses, due to few cases in the previous diabetes category.

Dyslipidaemia

Non-fasting blood samples were taken in an antecubital vein with standard methods with the participant sitting, and analysed for serum low-density lipoprotein (LDL) cholesterol with enzymatic colorimetric methods with commercial kits on a Cobas 8000 c702 (Roche Diagnostics, Mannheim, Germany) at the Department of Laboratory Medicine at the University Hospital of Northern Norway (ISO certification NS-EN ISO 15189:2012). Participants with LDL cholesterol levels higher than 5.0 mmol/L, or those reporting to currently use lipid-lowering drugs, were considered to have dyslipidaemia, in accordance with standard criteria.[41]

Hearing loss

Hearing loss was measured through the question 'Do you have a hearing loss (one/both ears)?' (no/yes).

Mental health

Mental health symptoms were measured by the 10-item version of the Hopkins Symptom Checklist (SCL-10), which is a shortened version of the original questionnaire with 90 items. The reliability of SCL-10 is high (Cronbach's alpha 0.88).[42] SCL-10 includes questions on sudden fear, anxiousness, faintness/dizziness, tenseness, self-blame, sleep difficulties, depression, feeling of uselessness, feeling that everything is a struggle and feeling hopeless about the future, within the past week. Responses range from 1 (no problem) to 4 (very much). For participants with two missing values or less on SCL-10, we replaced missing with the sample mean value for each item, while participants with three or more missing values were removed from the sample, in accordance with previous research.[42] The SCL-10 score for each participant was calculated as the mean value over all the items.

Statistical analyses

We used Poisson regression models with robust variance to estimate prevalence ratios (PR) with tinnitus as exposure and hypertension, myocardial infarction and stroke as outcome variables, adjusting for the aforementioned covariates.

First, we used the tinnitus status variable, then the symptom intensity variable including information on tinnitus frequentness and bother, and analysed their associations with hypertension, myocardial infarction and stroke in the full sample (n=17 288). We tested interactions between tinnitus symptom intensity and age/sex and further analysed the association between tinnitus symptom intensity and cardiovascular outcomes for women <65 years, women ≥65 years, men <65 years and men ≥65 years separately. Next, to be able to examine if tinnitus frequentness or bother was most important in the potential association between tinnitus and cardiovascular disease, we performed analyses of the association between the original tinnitus bother variable (ranging from 0 to 10) and cardiovascular outcomes, both with and without tinnitus frequentness included in the adjustment set. These analyses were performed only among the participants with tinnitus (n=3570). Analyses were performed using Stata V.18.0 (StataCorp, College Station, Texas, USA), and p values<0.05 were considered statistically significant.

Patient and public involvement

Project ideas were presented to the Norwegian Association for the Hard of Hearing (HLF), which supported them when planning the application for funding. Further, the project plans were presented and discussed with a group of stakeholders (representatives from HLF and tinnitus competence/rehabilitation centre HLF Briskeby, and two patient representatives with tinnitus) at the project kickoff meeting. The research question regarding the significance of tinnitus symptom intensity and bother was informed by input from the tinnitus patients in the stakeholder group. Since the present study is a general population study using data that were already collected in the Tromsø Study, patients or the public were not involved in the design of this study. Results will be disseminated to the participants, relevant patient communities and the general public through information at the websites of HLF and the Tromsø Study.

RESULTS

According to our definition, 38.4% of participants had hypertension, while 3.1% had a previous myocardial infarction, and 2.2% had a previous stroke. Table 1 shows the prevalence of all three cardiovascular health outcomes and covariates across different levels of tinnitus symptom intensity (see online supplemental tables 1 and 2 for prevalence according to tinnitus status and tinnitus bother, respectively). Prevalence of both hypertension, and myocardial infarction and stroke was lower in the group without tinnitus than among participants with tinnitus of various degrees of symptom intensity. More men than women reported tinnitus of all intensities, and the proportion of men increased further for higher intensities of tinnitus. The categories with no tinnitus and low-intensity tinnitus had a larger proportion of individuals in the youngest age group compared with higher tinnitus intensities. Correspondingly, there was a larger proportion of older participants in categories with higher tinnitus symptom intensity, compared with no or low-intensity tinnitus. More participants with the highest tinnitus symptom intensity possessed the lowest level of education and fewer possessed the highest level of education, compared with the other groups. There was a clear increase in the prevalence of hearing loss in participants with tinnitus compared with those without, and prevalence increased steadily with increasing tinnitus symptom intensity value. The group of participants with the highest tinnitus symptom intensity also had the highest degree of mental health problems.

Results from Poisson regression analyses of associations between tinnitus and cardiovascular outcomes are shown in tables 2–4, including PR with 95% CIs. In most cases, PRs were smaller in full models than in crude models. Very weak associations were found between tinnitus status and hypertension (PR=1.04, 95% CI: 1.00 to 1.08) and stroke (PR=1.08, 95% CI: 0.85 to 1.36) (table 2). For myocardial infarction, there was a weak positive association with tinnitus (PR=1.11, 95% CI: 0.94 to 1.31) that was significant only in the crude model (PR=1.24, 95% CI: 1.04 to 1.47).

For tinnitus symptom intensity (table 3), the strongest association for all cardiovascular outcomes was found with low intensity, although only some of the associations were statistically significant. For hypertension, there was a significant weak association with low-intensity tinnitus

Table 1 Prevalence of cardiovascular health outcomes and covariates across different levels of tinnitus symptom intensity

	Tinnitus symptom intensity*				
	No tinnitus	Low intensity	Medium intensity	High intensity	Total
	n (%)	n (%)	n (%)	n (%)	n (%)
Hypertension					
No	8622 (62.9)	670 (60.5)	1124 (55.3)	239 (55.5)	10 655 (61.6)
Yes	5096 (37.2)	437 (39.5)	908 (44.7)	192 (44.6)	6633 (38.4)
Myocardial infarction					
No	13 344 (97.3)	1061 (95.8)	1941 (95.5)	407 (94.4)	16 753 (96.9)
Yes, previously	374 (2.7)	46 (4.2)	91 (4.5)	24 (5.6)	535 (3.1)
Stroke					
No	13 445 (98.0)	1078 (97.4)	1978 (97.3)	415 (96.3)	16 916 (97.9)
Yes, previously	273 (2.0)	29 (2.6)	54 (2.7)	16 (3.7)	372 (2.2)
Sex					
Women	7441 (54.2)	501 (45.3)	733 (36.1)	169 (39.2)	8844 (51.2)
Men	6277 (45.8)	606 (54.7)	1299 (63.9)	262 (60.8)	8444 (48.8)
Age (years)					
40–54	6885 (50.2)	575 (51.9)	763 (37.6)	155 (36.0)	8378 (48.5)
55–64	3631 (26.5)	311 (28.1)	599 (29.5)	135 (31.3)	4676 (27.1)
65–74	2411 (17.6)	185 (16.7)	516 (25.4)	105 (24.4)	3217 (18.6)
>75	791 (5.8)	36 (3.3)	154 (7.6)	36 (8.4)	1017 (5.9)
Education level					
Primary/partly secondary	2849 (20.8)	189 (17.1)	461 (22.7)	128 (29.7)	3627 (21.0)
Upper secondary	3752 (27.4)	332 (30.0)	579 (28.5)	131 (30.4)	4794 (27.7)
Tertiary	7117 (51.9)	586 (52.9)	992 (48.8)	172 (39.9)	8867 (51.3)
Living with spouse					
No	2991 (21.8)	251 (22.7)	485 (23.9)	97 (22.5)	3824 (22.1)
Yes	10 727 (78.2)	856 (77.3)	1547 (76.1)	334 (77.5)	13 464 (77.9)
Daily smoking					
Never	5966 (43.5)	452 (40.8)	828 (40.8)	152 (35.3)	7398 (42.8)
Yes, previously	5917 (43.1)	497 (44.9)	959 (47.2)	205 (47.6)	7578 (43.8)
Yes, now	1835 (13.4)	158 (14.3)	245 (12.1)	74 (17.2)	2312 (13.4)
Alcohol					
Never	943 (6.9)	57 (5.2)	163 (8.0)	40 (9.3)	1203 (7.0)
Up to 4 times a month	8514 (62.1)	714 (64.5)	1204 (59.3)	259 (60.1)	10 691 (61.8)
2 or more times a week	4261 (31.1)	336 (30.4)	665 (32.7)	132 (30.6)	5394 (31.2)
Diabetes					
No	13 098 (95.5)	1052 (95.0)	1921 (94.5)	401 (93.0)	16 472 (95.3)
Yes, now or previously	620 (4.5)	55 (5.0)	111 (5.5)	30 (7.0)	816 (4.7)
Dyslipidaemia†					
No	11 000 (80.2)	873 (78.9)	1551 (76.3)	307 (71.2)	13 731 (79.4)
Yes	2718 (19.8)	234 (21.1)	481 (23.7)	124 (28.8)	3557 (20.6)
Hearing loss					
No	10 603 (77.3)	665 (60.1)	755 (37.2)	93 (21.6)	12 116 (70.1)
Yes	3115 (22.7)	442 (39.9)	1277 (62.8)	338 (78.4)	5172 (29.9)
	mean (SD)	**mean (SD)**	**mean (SD)**	**mean (SD)**	**mean (SD)**
Waist–height ratio	0.554 (0.073)	0.561 (0.073)	0.561 (0.069)	0.567 (0.072)	0.556 (0.073)

Continued

Table 1 Continued

	Tinnitus symptom intensity*				
	No tinnitus	Low intensity	Medium intensity	High intensity	Total
	n (%)	n (%)	n (%)	n (%)	n (%)
Mental health score (SCL-10)	1.269 (0.360)	1.365 (0.406)	1.333 (0.410)	1.469 (0.484)	1.288 (0.375)
Total n (row %)	**13 718 (79.4)**	**1107 (6.4)**	**2032 (11.8)**	**431 (2.5)**	**17 288 (100.0)**

The Tromsø Study 2015–2016.
*Generated as a function of tinnitus frequentness (scale 1–4; (1) less frequently than every week / (2) each week, but not every day / (3) each day, but not all the time / (4) most of the time) plus tinnitus bother (scale 1–4; (1) not bothered / (2) a little bothered / (3) bothered / (4) highly bothered), further divided into four categories of tinnitus symptom intensity, ranging from 'no tinnitus' to 'high intensity'.
†Participants with serum LDL cholesterol >5.0 mmol/L or current use of lipid lowering drugs were considered to have dyslipidaemia.
LDL, low-density lipoprotein; SCL-10, 10-item version of the Hopkins Symptom Checklist.

in the crude model—the prevalence of hypertension was 9% higher than with no tinnitus. For the full model, the prevalence was slightly lower (8% increase; PR=1.08, 95% CI: 1.01 to 1.16). Additionally, there was a significant association between tinnitus symptom intensity and myocardial infarction—the prevalence of previous myocardial infarction was 39% higher when having low-intensity tinnitus (PR=1.39, 95% CI: 1.07 to 1.81) than having no tinnitus.

No interactions between tinnitus symptom intensity and age or sex were significant for any of the cardiovascular outcomes (see online supplemental table 3). Still, we chose to perform stratified analyses for separate groups of age and sex (figure 1), since it is acknowledged that the prevalence of both tinnitus and cardiovascular disease differs across age and sex.[2 24 26] Also here, the strongest associations were generally found for the group with low-intensity tinnitus. Among women below 65 years, there was a significant association between low tinnitus symptom intensity and stroke, with prevalence of stroke approximately 2.5 times higher with low tinnitus symptom intensity compared with no tinnitus (PR=2.42, 95% CI: 1.31 to 4.45). For men aged 65 and older, there were only significant associations between low-intensity tinnitus and both hypertension and myocardial infarction, where prevalences of the cardiovascular outcomes were marginally higher in both cases, compared with no tinnitus (PR=1.20, 95% CI: 1.09 to 1.31 and PR=1.59, 95% CI: 1.11 to 2.27, respectively).

Among participants with tinnitus, we analysed the associations between tinnitus bother and cardiovascular health outcomes. These prevalence ratios were close to 1 and none of the associations were significant (table 4). The inclusion of tinnitus frequentness in the adjustment set did not have a notable influence on the prevalence ratios.

DISCUSSION

In this population-based study, we found weak and mostly non-significant associations between tinnitus and the prevalence of hypertension, myocardial infarction and stroke. For crude models, there was a significant association between tinnitus status and myocardial infarction, as well as significant associations between low-intensity tinnitus and hypertension and myocardial infarction. After covariate adjustment, associations were attenuated, and only the associations between low-intensity tinnitus and the prevalence of hypertension and myocardial infarction remained significant. Stratified analyses revealed a significantly higher prevalence of stroke for women <65 years, and a marginally, but significantly higher prevalence of both hypertension and myocardial infarction for men ≥65 years, among participants with

Table 2 Prevalence ratio with 95% CI for the association between tinnitus status and hypertension, myocardial infarction and stroke

Tinnitus variable	Cardiovascular variable	Crude‡	Full§
Tinnitus status†	Hypertension	1.03 (0.99 to 1.07)	1.04 (1.00 to 1.08)
	Myocardial infarction	1.24 (1.04 to 1.47)*	1.11 (0.94 to 1.31)
	Stroke	1.15 (0.92 to 1.45)	1.08 (0.85 to 1.36)

The Tromsø Study 2015–2016. Analysed in the full sample (n=17 288).
*p<0.05.
†'During the last 12 months, have you experienced ringing in your ears lasting more than 5 min?' (no/yes).
‡Adjusted for age and sex.
§Adjusted for age, sex, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms (SCL-10) and hearing loss.
SCL-10, 10-item version of the Hopkins Symptom Checklist.

Table 3 Prevalence ratio with 95% CI for the association between tinnitus symptom intensity and hypertension, myocardial infarction and stroke

Tinnitus variable	Cardiovascular variable	Tinnitus symptom intensity	Crude‡	Full§
Tinnitus symptom intensity†	Hypertension	No tinnitus	1.00	1.00
		Low intensity	1.09 (1.02 to 1.17)*	1.08 (1.01 to 1.16)*
		Medium intensity	1.01 (0.96 to 1.06)	1.03 (0.98 to 1.09)
		High intensity	1.00 (0.90 to 1.10)	0.98 (0.88 to 1.08)
	Myocardial infarction	No tinnitus	1.00	1.00
		Low intensity	1.54 (1.15 to 2.05)**	1.39 (1.07 to 1.81)*
		Medium intensity	1.10 (0.88 to 1.36)	1.01 (0.82 to 1.24)
		High intensity	1.38 (0.94 to 2.01)	1.06 (0.74 to 1.54)
	Stroke	No tinnitus	1.00	1.00
		Low intensity	1.38 (0.94 to 2.01)	1.29 (0.89 to 1.86)
		Medium intensity	1.01 (0.76 to 1.35)	0.97 (0.72 to 1.30)
		High intensity	1.40 (0.86 to 2.29)	1.15 (0.71 to 1.89)

The Tromsø Study 2015–2016. Analysed in the full sample (n=17,288). The group without tinnitus served as the reference group for the tinnitus variable.
*p<0.05.
**p<0.01.
†Generated as a function of tinnitus frequentness (1–4) plus tinnitus bother (1–4), further divided into four categories of tinnitus symptom intensity, ranging from 'no tinnitus' to 'high intensity'.
‡Adjusted for age and sex.
§Adjusted for age, sex, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms (SCL-10) and hearing loss.
SCL-10, 10-item version of the Hopkins Symptom Checklist.

low-intensity tinnitus compared with no tinnitus in fully adjusted models.

We found no associations between tinnitus bother and the prevalence of cardiovascular conditions among participants with tinnitus, neither with nor without tinnitus frequentness included as a covariate in the model. Taken together, our results indicate a slight difference in the prevalence of some cardiovascular conditions, depending on tinnitus symptom intensity, when comparing with participants without tinnitus in the full sample, but there was no difference in the prevalence of cardiovascular disease according to tinnitus bother or tinnitus frequentness in participants with tinnitus.

Most previous studies on the association between cardiovascular disease and tinnitus have assumed cardiovascular disease to be a risk factor for tinnitus in their analyses.[9–12 27] We have found only one previous population-based study discussing possible opposite mechanisms and examining tinnitus as a risk factor for cardiovascular disease. Huang *et al* found a significantly increased risk of ischaemic stroke in young and middle-aged individuals with tinnitus (OR=1.66, 95% CI: 1.34 to

Table 4 Prevalence ratio with 95% CI for the association between tinnitus bother and hypertension, myocardial infarction and stroke

Tinnitus variable	Cardiovascular variable	Crude*	Full†	Full incl. tinnitus frequentness‡
Tinnitus bother§	Hypertension	1.00 (0.98 to 1.01)	0.99 (0.97 to 1.00)	1.02 (0.96 to 1.08)
	Myocardial infarction	1.05 (0.99 to 1.11)	1.02 (0.96 to 1.08)	0.99 (0.90 to 1.08)
	Stroke	1.03 (0.95 to 1.12)	0.99 (0.90 to 1.08)	0.99 (0.97 to 1.01)

The Tromsø Study 2015–2016. Analysed among participants with tinnitus (n=3570).
*Adjusted for age and sex.
†Adjusted for age, sex, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms (SCL-10) and hearing loss.
‡Adjusted for age, sex, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms (SCL-10), hearing loss and tinnitus frequentness.
§'Please indicate how bothered you are by the ringing in your ears?' (numeric rating scale ranging from 0 (not bothered) to 10 (worst imaginable bother)).
SCL-10, 10-item version of the Hopkins Symptom Checklist.

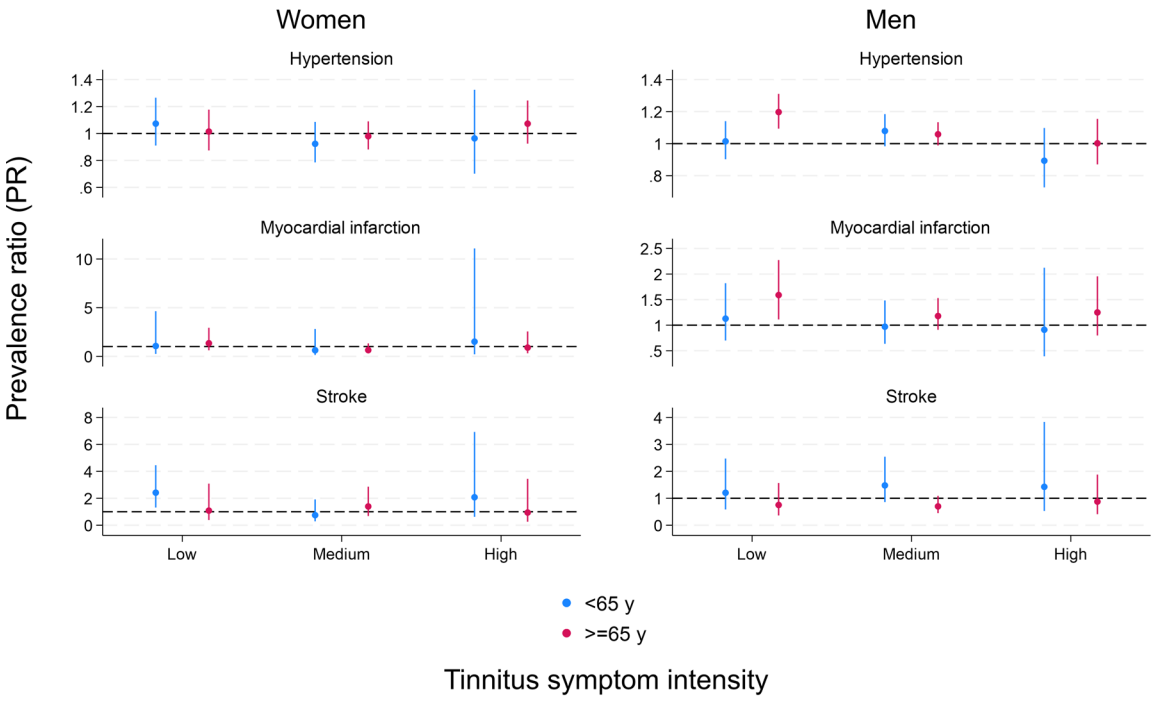

Figure 1 Stratified analyses of associations between tinnitus symptom intensity and hypertension, myocardial infarction and stroke. The Tromsø Study 2015–2016. Analysed in the full sample (n=17 288) using Poisson regression. Analyses were stratified by age (<65 years/≥65 years) and sex, generating the groups, women <65 years (n=6775), women ≥65 years (n=2069), men< 65 years (n=6279) and men ≥65 years (n=2165). 'No tinnitus' served as the reference group (PR=1), marked by dotted lines. We adjusted for age, education level, marital status, alcohol consumption, smoking, waist–height ratio, diabetes, dyslipidaemia, mental health symptoms (SCL-10) and hearing loss in the regression models. SCL-10, 10-item version of the Hopkins Symptom Checklist.

2.04) compared with a control group.[28] In addition to being based on individuals who had previously received diagnoses of tinnitus and ischaemic stroke, this study was limited to individuals aged 20–45. Hence, our results cannot be directly compared with this study. The present study thus includes individuals with a broader age span and potentially previously undiagnosed tinnitus. Studies that examined the association in the opposite direction, with cardiovascular disease as a risk factor for tinnitus, presented inconclusive results—some studies found that hypertension, ischaemic heart disease and stroke were significant risk factors for tinnitus, while others did not.[27]

Whether tinnitus was analysed as an outcome or risk factor, the previous studies identifying significant associations with cardiovascular conditions have in common that associations were mostly weak.[9 10 12 27 28] Like our study, several of the previous studies were cross-sectional.[10 12 43] Although there are possible mechanisms for tinnitus both being a risk factor and a consequence of cardiovascular disease, the choice of independent and dependent variables both in previous and our cross-sectional study must be taken as hypotheses that need to be further tested. Our study supports previous studies indicating weak associations between tinnitus and cardiovascular disease, but longitudinal studies are needed to examine the prevailing mechanisms and pathways.

We have found only one study that examined tinnitus bother in relation to cardiovascular disease.[43] This population-based study found a higher frequency of cardiovascular disease in those with bothersome compared with non-bothersome tinnitus, in a sample of individuals with self-reported tinnitus. Bothersome tinnitus was also positively associated with cardiovascular disease among women.[43] In the present study, we found no associations between tinnitus bother and the prevalence of cardiovascular conditions among participants with tinnitus.

In our study, the relatively strongest associations were generally found between low tinnitus symptom intensity and cardiovascular conditions, and this was also the only category of tinnitus symptom intensity that presented significant results. It is not clear why only low-intensity tinnitus should be related to cardiovascular health outcomes. It could be that individuals reporting the lowest level of tinnitus symptom intensity are inherently more attentive to disease, more sensitive and thus more prone to stress. This explanation is in line with findings from a Norwegian study on the association between tinnitus and mental health, where worse mental health was reported among individuals with low tinnitus symptom intensity compared with those with high symptom intensity (frequentness and duration), for some groups of age and

sex.[7] It should be noted, however, that participants in the category with low-intensity tinnitus in the present study reported to be not bothered or only a little bothered by their tinnitus. Alternatively, the association may be due to cardiovascular disease causing tinnitus through its effect on blood circulation. Pulsatile tinnitus, which is a rhythmic type of tinnitus synchronous with the heartbeat, often originates from vascular causes.[44] It may be that vascular changes related to cardiovascular disease could bring about pulsatile tinnitus occurring sporadically, for instance, in relation to physical exertion. If so, an association between only low-intensity (and thus infrequent) tinnitus and cardiovascular disease makes sense. Since we had no information on tinnitus type, we were not able to examine this any further. Importantly, since CIs were overlapping for all categories of tinnitus symptom intensity in the present study, we cannot say for certain that there were differences between categories, and the associations between low tinnitus symptom intensity and the prevalence of cardiovascular diseases need to be replicated in future studies.

The slightly higher prevalence of both hypertension and myocardial infarction among men ≥65 years with low-intensity tinnitus, compared with the same group without tinnitus, was as expected, since both tinnitus and cardiovascular disease are more common in men and their prevalences increase with age. The reason for the higher prevalence of stroke in women <65 years with low-intensity tinnitus compared with no tinnitus is, however, uncertain, although it is partially in line with results from Huang *et al*, who found significant associations between tinnitus and ischaemic stroke specifically for young individuals (<40 years).[28]

In parallel to the study of tinnitus and cardiovascular disease, results from environmental noise research also describe associations with cardiovascular disease and its risk factors.[23] Cardiovascular risk factors such as hypertension, obesity and altered blood lipid profile are likely to be influenced by external noise through mechanisms of stress and sleep disturbance.[18 19] Thus, the effects of environmental noise on the cardiovascular system are parallel to what has been reported in relation to tinnitus, including high BMI, hypertension and hypercholesterolaemia.[10 11] Additionally, stress and sleep disturbance are symptoms that have also been linked to tinnitus.[22] It might thus be that stress and sleep disturbance are factors involved in the observed association between tinnitus and cardiovascular disease. Another option is that stress or other factors are common underlying causes of both phenomena.[28 45 46] More research is warranted to explain the causality of this relationship.

A review article by Kempen *et al*[23] summarises results on the associations between the prevalence and incidence of cardiovascular disease and several types of traffic noise; air traffic, road traffic and rail traffic. For hypertension, ischaemic heart disease and stroke, results were mixed, with some indications of modest positive associations. Only some of these findings were statistically significant. Regarding the association between road traffic noise and ischaemic heart disease, however, findings were more consistent. Even though we presented results from only a single cross-sectional study on tinnitus, they align with these results on traffic noise—we found a significant positive association between tinnitus symptom intensity and prevalence of myocardial infarction in our non-stratified analyses.

There are some limitations to this study. We used cross-sectional data and did not have information on the onset of tinnitus, so we do not know which of the conditions came first. Thus, while our study contributes knowledge about the overall associations between tinnitus and the prevalence of cardiovascular disease in the general adult population, mechanisms for the associations could not be explored. Furthermore, a possible impact of tinnitus on the development of cardiovascular disease would likely be a long-term effect, possibly with a stronger association between tinnitus and cardiovascular disease for individuals with prolonged tinnitus. We could not compare prevalences according to duration of tinnitus, neither did we have information on specific types of tinnitus (like pulsatile tinnitus), which could possibly affect associations. All tinnitus and vascular condition data, except for blood pressure measurements, were self-reported. Being a general population health study covering numerous topics, it was not possible to include a thorough clinical assessment of tinnitus or any full-scale tinnitus inventory (like the Tinnitus Handicap Inventory[47]) in the Tromsø Study. Instead, and rather similar to how it has been done in other population studies,[9 10 12] tinnitus was assessed using single items measuring tinnitus status, frequentness and bother. It is a strength of the study that we used the proposed standard measure of tinnitus prevalence in population studies, 'tinnitus lasting more than five min during the past year'.[2 33] The other items were also selected to align with other population-based studies.[8]

Myocardial infarction and stroke were measured by self-report in this study. A study that has examined the validity of self-reported myocardial infarction and stroke in Sami and Norwegian populations[35] found that these measures were subject to some false-positive reporting, since small proportions of self-reported myocardial infarctions and strokes were actually other diseases. However, the study concluded that the sensitivity was high for self-reported myocardial infarction and moderate for stroke, while the positive predictive value was moderate for both measures.[35] We could not discriminate between different types of strokes in our data, but since the large majority of strokes are cerebral infarctions,[48] it is unlikely that this had a notable influence on the results of the study.

The present study also has several strengths. It used data from a large population-based study, so that the associations between tinnitus and cardiovascular conditions were examined in a general adult sample. Common to all population-based health surveys is the possibility of healthy participant bias. The participation rate in the present study was, however, high, which is a strength. A

study on the prevalence of dietary patterns found some significant differences between participants and non-participants in Tromsø7, but differences were small, and the authors concluded that their study population was fairly representative of the Norwegian population regarding sex, age and education level.[49] A recent study especially examining differences between participants and non-participants in Tromsø7 found some significant differences between participants and non-participants according to sex, age, marital status, income, ethnicity, residential ownership and socioeconomic characteristics of the living area.[50] Although associations between tinnitus and various sociodemographic factors are somewhat ambiguous in the literature,[27] there is an established association between socioeconomic factors and health.[51] It may be assumed that the inclusion of non-participants could have increased the prevalence of both tinnitus and cardiovascular disease in our data slightly, but the effect on associations would not be considered substantial. A strength of our study is further that hypertension was identified through blood pressure measurements, rather than self-report. Furthermore, we examined associations between tinnitus and specific cardiovascular diseases, as opposed to cardiovascular disease in general. Our sample had enough cases and statistical strength to detect clinically relevant associations, and we had the opportunity to adjust for potential confounders to reduce bias in analyses. We used DAGs in the selection of covariates, to minimise bias in analyses. Finally, we estimated prevalence ratios, which may be a better-suited measure of associations in cross-sectional studies than the commonly used OR.[52]

To the best of our knowledge, this is one of the first population-based studies to report associations between tinnitus and the prevalence of specific cardiovascular diseases, and to examine whether associations with cardiovascular disease vary by tinnitus symptom intensity, frequentness and bother. Although the strength of the associations can be described as only weak, it may be of importance for public health since both tinnitus and cardiovascular disease are prevalent conditions in the general population. Our results based on self-reported data suggest that tinnitus should be taken seriously even if tinnitus frequentness and bother level is low. This may be especially relevant for practitioners in primary healthcare services who receive patients with tinnitus. Longitudinal studies should be performed to determine the mechanisms and causal pathways of the relationship between tinnitus and cardiovascular disease.

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REFERENCES

1 Tonndorf J. The analogy between tinnitus and pain: a suggestion for a physiological basis of chronic tinnitus. *Hear Res* 1987;28:271–5.
2 McCormack A, Edmondson-Jones M, Somerset S, *et al*. A systematic review of the reporting of tinnitus prevalence and severity. *Hear Res* 2016;337:70–9.
3 Axelsson A, Ringdahl A. Tinnitus--a study of its prevalence and characteristics. *Br J Audiol* 1989;23:53–62.
4 Henry JA, Dennis KC, Schechter MA. General review of tinnitus: prevalence, mechanisms, effects, and management. *J Speech Lang Hear Res* 2005;48:1204–35.
5 Vanneste S, Faber M, Langguth B, *et al*. The neural correlates of cognitive dysfunction in phantom sounds. *Brain Res* 2016;1642:170–9.
6 Neff P, Simões J, Psatha S, *et al*. The impact of tinnitus distress on cognition. *Sci Rep* 2021;11:2243.
7 Krog NH, Engdahl B, Tambs K. The association between tinnitus and mental health in a general population sample: results from the HUNT study. *J Psychosom Res* 2010;69:289–98.
8 Ausland JH-L, Engdahl B, Oftedal B, *et al*. Tinnitus and associations with chronic pain: the population-based Tromsø Study (2015-2016). *PLOS ONE* 2021;16:e0247880.
9 Nondahl DM, Cruickshanks KJ, Wiley TL, *et al*. Prevalence and 5-year incidence of tinnitus among older adults: the epidemiology of hearing loss study. *J Am Acad Audiol* 2002;13:323–31.
10 Fujii K, Nagata C, Nakamura K, *et al*. Prevalence of tinnitus in community-dwelling Japanese adults. *J Epidemiol* 2011;21:299–304.
11 Martines F, Sireci F, Cannizzaro E, *et al*. Clinical observations and risk factors for tinnitus in a sicilian cohort. *Eur Arch Otorhinolaryngol* 2015;272:2719–29.
12 Shargorodsky J, Curhan GC, Farwell WR. Prevalence and characteristics of tinnitus among US adults. *Am J Med* 2010;123:711–8.

13 Mazurek B, Haupt H, Georgiewa P, *et al*. A model of peripherally developing hearing loss and tinnitus based on the role of hypoxia and ischemia. *Med Hypotheses* 2006;67:892–9.
14 Baigi A, Oden A, Almlid-Larsen V, *et al*. Tinnitus in the general population with A focus on noise and stress: a public health study. *Ear Hear* 2011;32:787–9.
15 Irwin MR. Why sleep is important for health: a psychoneuroimmunology perspective. *Annu Rev Psychol* 2015;66:143–72.
16 Agorastos A, Chrousos GP. The neuroendocrinology of stress: the stress-related continuum of chronic disease development. *Mol Psychiatry* 2022;27:502–13.
17 Lagraauw HM, Kuiper J, Bot I. Acute and chronic psychological stress as risk factors for cardiovascular disease: insights gained from epidemiological, clinical and experimental studies. *Brain Behav Immun* 2015;50:18–30.
18 Münzel T, Sørensen M, Daiber A. Transportation noise pollution and cardiovascular disease. *Nat Rev Cardiol* 2021;18:619–36.
19 Münzel T, Hahad O, Sørensen M, *et al*. Environmental risk factors and cardiovascular diseases: a comprehensive expert review. *Cardiovasc Res* 2022;118:2880–902.
20 Basner M, Babisch W, Davis A, *et al*. Auditory and non-auditory effects of noise on health. *Lancet* 2014;383:1325–32.
21 Mucci N, Traversini V, Lorini C, *et al*. Urban noise and psychological distress: a systematic review. *Int J Environ Res Public Health* 2020;17:6621.
22 Malouff JM, Schutte NS, Zucker LA. Tinnitus-related distress: a review of recent findings. *Curr Psychiatry Rep* 2011;13:31–6.
23 Kempen E van, Casas M, Pershagen G, *et al*. WHO environmental noise guidelines for the European region: a systematic review on environmental noise and cardiovascular and metabolic effects: a summary. *Int J Environ Res Public Health* 2018;15:379.
24 Reckelhoff JF. Gender differences in the regulation of blood pressure. *Hypertension* 2001;37:1199–208.
25 Pinho-Gomes AC, Peters SAE, Thomson B, *et al*. Sex differences in prevalence, treatment and control of cardiovascular risk factors in England. *Heart* 2021;107:462–7.
26 McCormack A, Edmondson-Jones M, Fortnum H, *et al*. The prevalence of tinnitus and the relationship with neuroticism in a middle-aged UK population. *J Psychosom Res* 2014;76:56–60.
27 Deklerck AN, Debacker JM, Keppler H, *et al*. Identifying non-otologic risk factors for tinnitus: a systematic review. *Clin Otolaryngol* 2020;45:775–87.
28 Huang Y-S, Koo M, Chen J-C, *et al*. The association between tinnitus and the risk of ischemic cerebrovascular disease in young and middle-aged patients: a secondary case-control analysis of A nationwide, population-based health claims database. *PLOS ONE* 2017;12:e0187474.
29 Hahad O, Kröller-Schön S, Daiber A, *et al*. The cardiovascular effects of noise. *Dtsch Arztebl Int* 2019;116:245–50.
30 The Tromsø study. Available: https://uit.no/research/tromsostudy [Accessed 03 Nov 2022].
31 The seventh survey of the Tromsø study. Available: https://uit.no/research/tromsostudy/project?pid=708909 [Accessed 03 Nov 2022].
32 Jacobsen BK, Eggen AE, Mathiesen EB, *et al*. Cohort profile: the Tromso study. *Int J Epidemiol* 2012;41:961–7.
33 Biswas R, Lugo A, Gallus S, *et al*. Standardized questions in english for estimating tinnitus prevalence and severity, hearing difficulty and usage of healthcare resources, and their translation into 11 European languages. *Hear Res* 2019;377:330–8.
34 Helsedirektoratet. Legemiddelbehandling av høyt blodtrykk. Available: https://www.helsedirektoratet.no/retningslinjer/forebygging-av-hjerte-og-karsykdom/legemidler-ved-primaerforebygging-av-hjerte-og-karsykdom/legemiddelbehandling-av-hoyt-blodtrykk [Accessed 24 Sep 2021].
35 Eliassen B-M, Melhus M, Tell GS, *et al*. Validity of self-reported myocardial infarction and stroke in regions with Sami and Norwegian populations: the SAMINOR 1 survey and the CVDNOR project. *BMJ Open* 2016;6:e012717.
36 Greenland S, Pearl J, Robins JM. Causal diagrams for epidemiologic research. *Epidemiology (Sunnyvale)* 1999;10:37–48.
37 Textor J, Hardt J, Knüppel S. DAGitty: a graphical tool for analyzing causal diagrams. *Epidemiology* 2011;22:745.
38 Babor TF, Higgins-Biddle JC, Saunders JB, *et al*. The alcohol use disorders identification test: guideline for use in primary care [World Health Organization]. 2001. Available: http://apps.who.int/iris/bitstream/handle/10665/67205/WHO_MSD_MSB_01.6a.pdf;jsessionid=9ADAA109E8BD34DC94BEA0EBA8EFFA5B?sequence=1 [Accessed 21 Feb 2022].
39 Swainson MG, Batterham AM, Tsakirides C, *et al*. Prediction of whole-body fat percentage and visceral adipose tissue mass from five anthropometric variables. *PLoS One* 2017;12:e0177175.
40 Mathieu P, Pibarot P, Larose E, *et al*. Visceral obesity and the heart. *Int J Biochem Cell Biol* 2008;40:821–36.
41 Helsedirektoratet. Bruk av statiner og andre lipidsenkende legemidler ved primærforebygging av hjerte- og karsykdom. Available: https://www.helsedirektoratet.no/retningslinjer/forebygging-av-hjerte-og-karsykdom/legemidler-ved-primaerforebygging-av-hjerte-og-karsykdom/bruk-av-statiner-og-andre-lipidsenkende-legemidler-ved-primaerforebygging-av-hjerte-og-karsykdom [Accessed 04 Oct 2021].
42 Strand BH, Dalgard OS, Tambs K, *et al*. Measuring the mental health status of the norwegian population: a comparison of the instruments SCL-25, SCL-10, SCL-5 and MHI-5 (SF-36). *Nord J Psychiatry* 2003;57:113–8.
43 Basso L, Boecking B, Brueggemann P, *et al*. Gender-specific risk factors and comorbidities of bothersome tinnitus. *Front Neurosci* 2020;14:706.
44 Madani G, Connor SEJ. Imaging in pulsatile tinnitus. *Clin Radiol* 2009;64:319–28.
45 Szczepek AJ, Mazurek B. Neurobiology of stress-induced tinnitus. In: Searchfield GD, Zhang J, eds. *The Behavioral Neuroscience of Tinnitus*. Cham: Springer International Publishing, 2021: 327–47.
46 Dar T, Radfar A, Abohashem S, *et al*. Psychosocial stress and cardiovascular disease. *Curr Treat Options Cardiovasc Med* 2019;21:23.
47 Newman CW, Jacobson GP, Spitzer JB. Development of the tinnitus handicap inventory. *Arch Otolaryngol Head Neck Surg* 1996;122:143–8.
48 Mathiesen EB, Njølstad I, Joakimsen O. Risikofaktorer for hjerneslag. *Tidsskr Nor Laegeforen* 2007;127:748–50.
49 Lundblad MW, Andersen LF, Jacobsen BK, *et al*. Energy and nutrient intakes in relation to national nutrition recommendations in a norwegian population-based sample: the Tromsø study 2015-16. *Food Nutr Res* 2019;63.
50 Vo CQ, Samuelsen PJ, Sommerseth HL, *et al*. Comparing the sociodemographic characteristics of participants and non-participants in the population-based Tromsø study. *BMC Public Health* 2023;23:994.
51 Wang T, Li Y, Zheng X. Association of socioeconomic status with cardiovascular disease and cardiovascular risk factors: a systematic review and meta-analysis. *J Public Health (Berl)* 2024;32:385–99.
52 Tamhane AR, Westfall AO, Burkholder GA, *et al*. Prevalence odds ratio versus prevalence ratio: choice comes with consequences. *Stat Med* 2016;35:5730–5.