Slutrapport til Arbejdsmiljøforskningsfonden:

Støjbelastning i arbejdsmiljøet og risiko for tinnitus og nedsat høreevne. En forløbsundersøgelse af støjkarakteristika, tærskelværdier, forvarsler, individuel følsomhed og forebyggelse: STØJRISK projektet

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Aarhus og København 2017

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Projektet er støttet af Arbejdsmiljøforskningsfonden (projektnummer: 20100020116/3)

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Forfattere: Henrik Kolstad, Thomas Winther Frederiksen, Matias Brødsgaard Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper Medom Vestergaard, Jens Peter Bonde, Zara Ann Stokholm
Institution: Arbejdsmedicinsk Klinik, Dansk Ramazzini Center, Aarhus Universitetshospital
Udgiver: Arbejdsmedicinsk Klinik, Dansk Ramazzini Center, Aarhus Universitetshospital
Finansiel støtte: Projektet er støttet af Arbejdsmiljøforskningsfonden (Projektnummer: 20100020116/3)

Arbejdsmedicinsk Klinik, Dansk Ramazzini Center
Aarhus Universitetshospital
Nørrebrogade 44, bygning 2C
8000 Aarhus C
Tlf.: 7846 4290
Fax: 7846 4260
E-post: auharb@rm.dk
http://www.auh.dk/om+auh/afdelinger/arbejdsmedicinsk+klinik
http://ramazzini.dk/index.php/da/

Indhold

Forord

Arbejdsmiljøforskningsfonden støttede gennemførelsen af STØJRISK projektet, som har løbet fra den 1.1 2010 til 31.12. 2016. Denne rapport giver et overblik over de resultater, som er opnået indenfor bevillingsperioden.

Projektgruppen har bestået af Thomas Winther Frederiksen, Aarhus Universitetshospital (AUH) og Regionshospitalet Holstebro, Zara Ann Stokholm (AUH), Åse Marie Hansen, Københavns Universitet (KU), Søren Peter Lund, Det Nationale Forskningscenter for Arbejdsmiljø (NFA), Jesper Kristiansen (NFA), Jesper Medom Vestergaard, AUH, Jens Peter Bonde (Bispebjerg Universitetshospital) og Henrik Kolstad (AUH).

Matias Brødsgaard Grynderup, KU, har bistået ved de statistiske analyser og Mai Arlien-Søborg (AUH) og Astrid Schmedes (AUH) har stået for analyser og afrapportering af undersøgelsen af støj og serum lipider under vejledning af Zara Ann Stokholm..

Mogen Erlansen, AU, Vivi Schünssen, AU, Ioannis Basinas, AU og Institute of Occupational Medicine, Edinburgh, og Jens Brandt, CRECEA A/S, har bistået med udvikling af støj job eksponeringsmatricen (Støj-JEM).

Thomas Winther Frederiksen har været PhD studerende på projektet på halv tid kombineret med hans kliniske virke som øre-næse-hals læge under vejledning af Cecilia Høst Ramlau-Hansen, AU, og Henrik Kolstad, AUH.

Vi retter en varm tak til alle deltagende virksomheder og alle ansatte, som har deltaget i denne undersøgelse of Arbejdsmiljøforskningsfonden, som gjorde undersøgelsen mulig.

Århus og København 27. marts 2017

Thomas Winther Frederiksen, Zara Ann Stokholm, Åse Marie Hansen, Søren Peter Lund,
Jesper Kristiansen, Jesper Medom Vestergaard, Jens Peter Bonde og Henrik Kolstad

Resumé

En stor del af arbejdsstyrken er generet af støj og støjskader er blandt de hyppigste arbejdsskader. Selvom den høreskadende effekt er velkendt, og vi kender forebyggelsesmetoder, må vi forvente at støj vedbliver med at være et væsentligt arbejdsmiljøproblem i en lang årrække, og der er behov for ny viden om de helbredsmæssige konsekvenser og effektiv forebyggelse.

Dette projekt havde to overordnede formål: For det første, at fremskaffe konkret viden om forekomst og forebyggelse af støjskader indenfor støjbelastede brancher i Danmark: Industri, bygge- og anlæg og daginstitutioner. For det andet, at fremskaffe ny videnskabelig viden om årsagssammenhænge mellem lavdosis støj omkring 80 dB(A), impulsstøj, tinnitus og hørenedsættelse, samt samspilseffekter mellem støj, psykosociale belastninger og individuel følsomhed.

Projektet følger op på vores tidligere undersøgelse fra 2001-2002, som viste at støjniveauer over 85 dB(A) og støjskader stadig var hyppigt forekommende på danske arbejdspladser. Undersøgelsen viste også at støjudsatte personer, som anvender høreværn, bevarer en normal hørelse.

Vi fandt i STØJRISK projektet at det gennemsnitlige støjniveau målt gennem en hel arbejdsdag i 10 særligt støjbelastede industribrancher (fremstilling af næringsmidler og drikkevarer, træindustri, møbelindustri, grafisk industri, sten, ler og glasindustri, jern og metalvareindustri, fremstilling af metal, maskinindustri, fremstilling af biler mv.), bygge og anlægsvirksomhed og børnehaver i gennemsnit faldt med 1,1 dB(A) fra 83,9 dB(A) i 2001-3 til 82,8 dB(A) i 2009-10. Andelen af deltagere eksponeret for støj over 85 dB(A) (grænsen for hvornår Arbejdstilsynet kræver at der anvendes høreværn), som anvendte høreværn, steg fra 70 til 78 procent i den samme periode.

Vi fandt ikke at høretærskelen i de kritisk støjfølsomme hørefrekvenser steg (faldende hørelse) med stigende støjeksponering mellem 2001 og 2010. Os bekendt er vi de første til at dokumentere faldende støjniveauer over tid med direkte sammenlignelige data. Dette har været vist for en række andre erhvervseksponeringer og vores fund kom derfor ikke som en overraskelse. Ved et gennemsnitligt støjniveau under 83 dB(A) er den kendte effekt på hørelsen begrænset og sammen med den udbredte brug af høreværn tyder dette på at de fleste medarbejdere i de undersøgte industrier er godt beskyttet mod støjskader.

Årsagsmekanismerne der knytter støjeksponering og høreskade sammen, er kun delvist kortlagte. Som en af flere mekanismer har man foreslået at støj ændrer koncentrationen af kolesterol og andre lipider i blodet, forøger forekomsten af atherosklerose (åreforkalkning) og nedsætter blod flowet i de fine arterier, som forsyner høresneglen og det Cortiske organ. Vi fandt at et højt niveau af triglycerider og et lavt niveau af

HDL kolesterol samt høj BMI og rygning var associeret med reduceret hørelse. Men vi fandt ingen sammenhæng mellem støjniveau og disse lipider, når vi tog højde for høreværn og kendte risikofaktorer for dyslipidæmi. Disse analyser peger således mere i retning af at kost og livsstil end støj kan medføre høretab via atherosklerose.

Vi fandt ingen sammenhæng mellem generende tinnitus og psykosocialt arbejdsmiljø beskrevet som graden af krav og kontrol i arbejdet, eller fysiologisk stress målt som niveauet af kortisol i spyt. Det er velkendt at høretab forårsaget af støj ofte er ledsaget af tinnitus. Men vi fandt ikke holdepunkter for at risikoen for tinnitus var associeret med støjniveauet i denne population. Det kan hænge sammen med at støjniveauerne var for lave til at kunne forårsage høretab.

Hovedbudskabet fra denne undersøgelse er at vi mellem 2001 og 2010 ikke kunne finde sammenhæng mellem støjudsættelse på arbejdet og høretab indenfor de mest støjudsatte brancher i Danmark. Vi fandt at støjniveauet i disse brancher er lavt og brugen af høreværn er høj, og dette er nærliggende forklaringer på dette resultat.

Et hovedformål for arbejdsmiljøforskningen er at forebygge arbejdsbetingede lidelser. Ny viden om risikofaktorer i arbejdsmiljøet er helt centralt for at kunne nå dette mål. Men ny viden får kun effekt, hvis den udmøntes i grænseværdier eller anden regulering, som efterleves ude på arbejdspladserne. Dette projekt tyder på at dette har været tilfældet for støjbetinget høretab: Forebyggelse nytter. Det er også arbejdsmiljø-forskningens opgave at vurdere om mistænkte risikofaktorer også er reelle risikofaktorer. Denne undersøgelse tyder ikke på at generende tinnitus primært er en følge af arbejdsmiljøeksponeringer.

Abstract

Many workers are annoyed by noise exposure at work and hearing loss is among the most frequent work injuries. Even thought the effect of noise on hearing is well documented and preventive measures are well known, we expect noise exposure to be problem in many jobs also in the future. Thus there is need for new insights into the health effects of noise and evidence based prevention.

The STØJRISK project had two main objectives: First, to obtain new knowledge about the occurrence and prevention of noise induced hearing loss within industries with the highest levels of occupational noise in Denmark. Second, to generate new scientific evidence for the relation between low level noise exposure about 80 dB(A), tinnitus and hearing loss as well as the joint effects of noise, psychosocial factors, and individual susceptibility.

This is a follow up on our earlier studies from 2001-2003 that showed noise levels above 85 dB(A) and noise induced hearing loss to be prevalent at many Danish work sites. That study also showed that noise exposed workers using hearing protective devices (HPD) maintained a normal hearing.

In the STØJRISK project, we observed that the average full shift noise level declined by 1.1 dB(A) from 83,9 dB(A) in 2001-3 to 82,8 dB(A) in 2009-10. The proportion of workers exposed to noise levels above 85 dB(A) that used HPD increased from 70 % to 78 % during this period. We observed no association between noise exposure levels 2001-2010 and haring threshold shift during this period. As far as we are aware, we are the first to document declining industrial noise exposure levels. Previously, this has been shown for several other occupational exposures and our findings did thus not come as a surprise.

The causal mechanisms linking noise exposure and hearing loss is only partly known. According to one theory, noise exposure may increase serum lipid levels and atherosclerosis and reduce blood flow to cochlea and the organ of Corti. On the one hand, we observed that a high level of triglyceride and a low level of HDL cholesterol as well as a high BMI and smoking were associated with hearing loss. On the other hand, we observed no association between noise exposure levels and serum lipids when we accounted for HPD and well established risk factors of lipid levels. These analyses suggest that diet and lifestyle factors and not noise may cause hearing loss through altering lipid levels.

We observed no association between tinnitus and psychosocial work factors, or physiologic stress as measured by cortisol level. It is well known that noise induced hearing loss frequently is followed by tinnitus. However, we observed no increased risk of tinnitus by increasing noise levels in this population. This may be because noise levels were to low to cause hearing loss.

The main message from this study is that we were unable to document an association between hearing loss and the occupational noise exposure levels encountered between 2001 and 2010 within industries with the expected highest noise levels in Denmark. The low noise levels and high prevalence of HPD use are likely explanations.

Prevention of occupational injuries is a main objective of occupational health research. Detection of new risk factors within the work environment is essential. But even so is the implementation of occupational exposure limits and other regulations at the work sites. This project indicates that this has been the case for noise and hearing loss: prevention makes a difference. Another objective of occupational health research is to assess whether suspected risk factors are real risk factors. This study did not indicate that occupational factors increase the risk of tinnitus.

Formål

En stor del af arbejdsstyrken er generet af støj og støjskader er blandt de hyppigste arbejdsskader. Selvom den høreskadende effekt er velkendt, og vi kender forebyggelsesmetoder, må vi forvente at støj vedbliver med at være et væsentligt arbejdsmiljøproblem i en lang årrække, og der er behov for ny viden om de helbredsmæssige konsekvenser og effektiv forebyggelse.

Dette projekt havde to overordnede formål: For det første, at fremskaffe konkret viden om forekomst og forebyggelse af støjskader indenfor støjbelastede brancher i Danmark: Industri, bygge- og anlæg og daginstitutioner. For det andet, at fremskaffe ny videnskabelig viden om årsagssammenhænge mellem lavdosis støj omkring 80 dB(A), impulsstøj, tinnitus og hørenedsættelse, samt samspilseffekter mellem støj, psykosociale belastninger og individuel følsomhed.

Projektet fulgte op på en undersøgelse fra 2001-2003, som viste at støjniveauer over 85 dB(A) og støjskader stadig var hyppigt forekommende på danske arbejdspladser. Undersøgelsen viste også at støjudsatte personer, som anvender høreværn, bevarer en normal hørelse.

I dette projekt var formålene at analysere ændringer i høretærskel og nytilkomne tilfælde af tinnitus i 10-års perioden 2001-2010 som funktion af støjbelastning. Vi ville se om deltagere med tinnitus i 2001-2002 har særlig risiko for at udvikle hørenedsættelse i 2009-2010 og om tinnitus er forvarsel om støjskade. Vi ville undersøge om risikoen for høreskade er den samme i 2009-2010 som i 2001-2003, som udtryk for at støjforebyggelsen er på rette vej, og om deltagere fra 2001-2003, som anvendte høreværn stadig har normal hørelse. Endelig ville vi inddrage individuelle faktorer, blandt andet genetiske analyser, som kan anvendes til at fastsætte støjniveauer, som er også er sikre for personer med større følsomhed.

Metoder og udførelse

Population

Undersøgelsen er baseret på 85 virksomheder, som i 2001-2003 deltog i vores tidligere undersøgelser af støj og auditive effekter [1-3]. Virksomhederne var alle rekrutteret blandt de 10 brancher, som i følge Arbejdstilsynet havde de hyppigste anmeldelser af høreskader som følge af støj. Det drejede sig om følgende brancher: Fremstilling af næringsmidler og drikkevarer, træindustri, møbelindustri, grafisk industri, sten, ler og glasindustri, jern og metalvareindustri, fremstilling af metal, maskinindustri, fremstilling af biler mv. og bygge og anlægsvirksomhed. Herudover inkluderede vi børnehaver, hvor der havde været stor offentlig opmærksomhed om støjniveauerne, samt finanssektoren, som en referencegruppe med forventede lave støjniveauer.

I 2009-2010 indvilligede 42 af de 85 virksomheder, som deltog i 2001-2003, i at deltage i anden runde, samt 34 nye virksomheder rekrutteret efter lignende fremgangsmåde som i 2001-2003. Fra disse virksomheder deltog 516 personer (129 fra undersøgelsen i 2001-03 og 387 nye deltagere). Herudover blev alle medarbejdere fra de 43 virksomheder fra 2001-2003, som ikke deltog i anden runde, inviteret til det lokale sygehus for at deltage igen. I alt 149 personer accepterede dette. Disse 149 personer repræsenterede en gruppe arbejdere (i. industriarbejdere inden for de 10 brancher; ii. industriarbejdere udenfor de 10 brancher, iii. Medarbejdere inden for finanssektoren; iv. service medarbejdere; v. fridag, orlov eller barsel) eller pensionister. I alt deltog 665 personer gennem 2 på hinanden følgende dage. To bioanalytikere instruerede, udleverede måleudstyr og indsamlede prøver fra deltagerne på virksomhederne og de lokale sygehuse.

Støjmålinger

Gennem 24 timer målte vi støjniveauet hvert 5. sekund med bærbare dosimetre (Brüel & Kjær type 4443 og 4445). Vi beregnede LAEq værdien for arbejdstiden ud fra synkronisering med dagbøger og vurderede den kumulerede støjeksponering baseret på historiske arbejdsoplysninger siden 1980 og i opfølgningsperioden 2001-2010. For en del af deltagerne estimerede vi LAEq-værdien for øret under høreværn i den periode de anvendte høreværn.

Høretærskelbestemmelse

Ved begge runder fik alle deltagere bestemt høretærskler for hvert øre ved 0.25, 0.5, 1, 2, 3, 4, 6 og 8 kHz med rentone audiometri. Undersøgelserne blev udført ude på arbejdspladserne med et Voyager 522 audiometer udstyret med TDH-39 høretelefoner (Madsen Electronics, Taastrup, Danmark). Undersøgelserne blev udført i lyddæmpet boks (model AB-4240, Eckel Noise Control Technologies, Bagshot, UK) monteret i en varevogn af trænet personale og efter en standard protokol. Alle deltagere blev bedt om at anvende høreværn fra arbejdsdagens start indtil undersøgelsen blev udført.

Spørgeskemaoplysninger

Samtlige deltagere fik ved begge runder udleveret og returnerede et spørgeskema om erhvervsstatus, arbejdstid, høreværn, tinnitus og andre helbredsforhold, søvn, tobaksforbrug, og arbejdsmiljø. Deltagere i 2009-2010 rapporterede også deres psykosociale arbejdsmiljø angivet som krav og kontrol i overensstemmelse med Karaseks job strain model. Vi klassificerede de enkelte deltagere efter det gennemsnitlige niveau af krav og kontrol blandt alle deltagere i hver virksomhed i et forsøg på at reducere informations bias.

Spytkortisol og blodprøver

Deltagerne i 2009-2010 leverede hver 3 spytprøver, som blev analyseret for kortisol koncentration på NFA. Deltagerne i 2009-2010 afleverede en veneblodprøve, som blev analyseret for total kolesterol, HDL kolesterol, LDL kolesterol, triglycerider og HBA1c. Desuden blev der opsamlet blodprøver til nedfrysning, som ligger i en biobank med henblik på senere undersøgelser.

Blodtryksmåling

Deltagerne i 2009-2010 fik over 24 timer målt ambulant blodtryk hvert 20. minut klokken 7-23 og hvert 30. minut klokken 23-07 ved hjælp af fuldautomatiserede blodtryksmålere (Spacelaps mode 90217). Vi beregnede middelværdier for blodtrykket på arbejdet, i fritid og gennem nattetimerne.

Statistiske analyser

Vi analyserede sammenhængene mellem serum-lipider og andre atherogene risikofaktorer, støj og høretærskel med multivariat lineær regression. Sammenhængene mellem støj, psykosociale faktorer, kortisol og tinnitus analyserede vi med multivariat logistisk regression. I alle analyser inkluderede vi potentielle confoundere baseret på en gennemgang af den eksisterende litteratur.

Resultater: Om projektets formål og hensigt er blevet opnået

Vi undersøgte det gennemsnitlige støjniveau gennem en hel arbejdsdag (full-shift) blandt 627 deltagere 2001-2003 og 467 deltagere 2009-2010. Vi fandt at støjniveauet i de 10 industribrancher (fremstilling af næringsmidler og drikkevarer, træindustri, møbelindustri, grafisk industri, sten, ler og glasindustri, jern og metalvareindustri, fremstilling af metal, maskinindustri, fremstilling af biler mv.), bygge og anlægsvirksomhed og børnehaver faldt med 1,1 dB(A) fra 83,9 dB(A) i 2001-3 til 82,8 dB(A) i 2009-10. Andelen af deltagere eksponeret for støj over 85 dB(A), som anvendte høreværn, steg fra 70 til 78 procent i den samme periode.

Vi analyserede sammenhængen mellem full-shift støjniveau mellem 2001-2003 og 2009-2010 og ændring i høretærskel i den samme periode blandt 271 deltagere, som havde komplette data ved begge runder. Vi fandt ingen sammenhæng mellem støjniveauerne og ændring i høretærskel.

Vi analyserede sammenhængen mellem total kolesterol, HDL kolesterol, LDL kolesterol, triglycerider, HBA1c og høretærskler blandt 576 deltagere fra 2009-2010, som havde komplette data. Vi fandt at højt niveau af triglycerider, lavt niveau af HDL kolesterol, højt BMI og rygning var associeret med nedsat hørelse.

Vi analyserede sammenhængen mellem full-shift støjniveau og total kolesterol, HDL kolesterol, LDL kolesterol, triglycerider blandt 508 deltagere fra 2009-2010. I ujusterede analyser var der stærk sammenhæng mellem støj og disse serum lipider. Men når vi tog højde for brug af høreværn og kontrollerede for BMI, tobaksrygning og andre kendte risikofaktorer forsvandt disse sammenhænge.

Vi analyserede psykosociale faktorer, nuværende og kumuleret støjniveau, kortisol niveau og risiko for tinnitus blandt 534 deltagere fra 2009-2010. Vi fandt ingen sammenhæng mellem tinnitus og støjeeksponering på arbejdet, psykosocialt arbejdsmiljø angivet som graden af krav og kontrol i arbejdet eller fysiologisk stress målt som niveauet af kortisol i spyt.

Baseret på 1357 full-shift støjmålinger fra 2001-2010 og ekspertvurderinger af støjniveauerne har vi udviklet en kvantitativ støj job eksponerings matrice (støj-JEM) for 373 fagkoder (DISCO-88) som tager højde for ændringer i støjniveauer over tid. Vi analyserede data med mixed lineær regression efter en metode som nyligt vist at Susan Peters[4]. Vi vil afrapportere denne i et videnskabeligt tidskrift for eksponeringsvurdering (exposure assessment). Den vil danne grundlag for fremtidige analyser af hjertekarsygdomme og andre helbredseffekter, som er under mistanke for at være forårsaget af støj.

Det var også vores hensigt at analysere om særlige genetisk varianter var forbundet med særlig sårbarhed for nedsat høretærskel ved støjeksponering. Men da vi ikke kunne påvise en sammenhæng mellem støj og høretab afstod fra disse analyser.

Erfaringer og konklusioner

Hovedbudskabet fra denne undersøgelse er at vi ikke mellem 2001 og 2010 kunne finde sammenhæng mellem støjudsættelse på arbejdet og høretab indenfor de mest støjudsatte brancher i Danmark. Vi fandt at støjniveauerne i disse brancher er faldet og brugen af høreværn er steget i løbet af opfølgningsperioden og dette er nærliggende forklaringer på dette resultat. Man må dog formode at overordnede samfundsmæssige

forhold også har spillet ind på denne udvikling, fx den generelle tekniske udvikling og outsourcing af de mest støjende arbejdsopgaver. Vi kunne ikke underbygge at støj, psykosociale arbejdsforhold eller kortisol (som mål for fysiologisk stress) er associeret med kronisk tinnitus.

Perspektiver: Hvordan projektets resultater på kort og langt sigt kan bidrage til at forbedre arbejdsmiljøet

Et hovedformål for arbejdsmiljøforskningen er at forebygge arbejdsbetingede lidelser. Ny viden om risikofaktorer i arbejdsmiljøet er helt centralt for at kunne nå dette mål. Men denne viden får kun effekt, hvis den udmøntes i grænseværdier eller anden regulering, som efterleves ude på arbejdspladserne. Dette projekt tyder på at dette har været tilfældet for støjbetinget høretab: Forebyggelse nytter. Det er også arbejdsmiljøforskningens opgave at vurdere om mistænkte risikofaktorer også er reelle risikofaktorer. Denne undersøgelse tyder ikke på at generende tinnitus er forårsaget af arbejdsmiljøeksponeringer.

Litteratur

1. Kock S, Andersen T, Kolstad HA, Kofoed-Nielsen B, Wiesler F, Bonde JP. Surveillance of noise exposure in the danish workplace: A baseline survey. *Occup Environ Med*. 2004;61(10):838-843.

2. Rubak T, Kock SA, Koefoed-Nielsen B, Bonde JP, Kolstad HA. The risk of noise-induced hearing loss in the danish workforce. *Noise Health*. 2006;8(31):80-87.

3. Rubak T, Kock S, Koefoed-Nielsen B, Lund SP, Bonde JP, Kolstad HA. The risk of tinnitus following occupational noise exposure in workers with hearing loss or normal hearing. *Int J Audiol*. 2008;47(3):109-114.

4. Peters S, Vermeulen R, Olsson A, et al. Development of an exposure measurement database on five lung carcinogens (ExpoSYN) for quantitative retrospective occupational exposure assessment. *Ann Occup Hyg*. 2012;56(1):70-79.

Publikationer og produkter fra projektet

Skriftlig videnskabelig formidling med fagfælle bedømmelse

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad Atherogenic Risk Factors and Hearing Thresholds. Audiology and Neurotology 2014;19:310–318).

Arlien-Søborg MC[1], Schmedes AS[2], Stokholm ZA[3], Grynderup MB[4], Bonde JP[5], Jensen CS[3], Hansen ÅM[4,6], Frederiksen TW[3], Kristiansen J[6], Christensen KL[6,7], Vestergaard JM[3], Lund SP[6], Kolstad HA[3]. Ambient and at-the-ear occupational noise exposure and serum lipid levels. Int Arch Occup Environ Health. 2016 Oct;89(7):1087-93.

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Occupational Noise Exposure, Psychosocial Working Conditions and the Risk of Tinnitus. International Archives of Occupational and Environmental Health, December 2016

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara Ann Stokholm, Matias B. Grynderup, Åse Marie Hansen PhD, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Noise Induced Hearing Loss – a preventable disease? Results of a 10-year longitudinal study of occupationally noise exposed workers. Noise and Health, in press

Øvrig skriftlig videnskabelig formidling

Thomas W. Frederiksen. Occupational Noise Exposure and Individual Risk Factors for Hearing Loss and Tinnitus. PhD dissertation, Health, Aarhus University, March 2016

Manuskripter indsendt til videnskabelig tidsskrift eller under udarbejdelse

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Salivary Cortisol and Tinnitus. Manuscript.

STOKHOLM Z.A., ERLANDSEN M., SCHLÜNSSEN V., BASINAS I., BONDE J.P, BRANDT J., VESTERGAARD J.M., KOLSTAD H.A. A general population job exposure matrix for occupational noise.

Skriftlig populærformidling

Kolstad, HA, Stokholm, ZA, Vestergaard, JM, Erichsen, TR, Frederiksen, TW, Bonde, JP. Noise and health: Danish studies on noise at work. Miljø og sundhed, nr. 3, december 2012.

Mundtlig populær formidling

Zara Ann Stokholm. Støjbelastning i arbejdsmiljøet og risiko for tinnitus og nedsat høreevne. Arbejdsmiljøforskningsfondens Årskonference, 11. januar 2017

Mundtlig videnskabelig formidling

Kolstad, HA, Stokholm, ZA, Vestergaard, JM, Erichsen, TR, Frederiksen, TW, Bonde, JP. Noise and health: Danish studies on noise at work. Noise and Health. Sundhedsstyrelsens Rådgivende Videnskabelige Udvalg om Miljø og Sundhed, oktober 2012.

Schmedes, A, Arlien-Søborg, MC, Stokholm, ZA, Hansen, AM, Bonde, JP, Christensen, KL, Frederiksen, TW, Kristiansen, J, Lund, SP, Vestergaard, JM, Wetke, R, Kolstad, HA. Occupational noise exposure and serum lipids: the impact of noise exposure level and hearing protection. Mundtligt præsenteret ved Epidemiology in Occupational Health Conference, Utrecht, juni 2013.

T. W. Frederiksen, C.H. Ramlau-Hansen, Stokholm ZA,Vestergaard JM, H.A. Kolstad. Occupational Noise Exposure, Psychosocial Working Conditions and the Risk of Tinnitus. The 25th International Epidemiology in Occupational Health (EPICOH) Conference, September 2016, Barcelona

Zara Ann Stokholm, Mogen Erlansen, Vivi Schünssen, Ioannis Basinas, Jens Peter Bonde, Jens Brandt, Jesper Medom Vestergaard, Henrik Kolstad. A general population job exposure matrix for occupational noise. The 25th International Epidemiology in Occupational Health (EPICOH) Conference, September 2016, Barcelona

Øvrige artikler, nyheder og hjemmesider

Høretab koster milliarder. Interview med Henrik Kolstad i Magasinet Penge, DR1 7. oktober 2015.

Støj for nogle er musik for andre. Interview med Zara Stokholm i TV2 Østjylland den 7. december 2016.

Forsker: Indsatsen mod støj er lykkedes. Interview med Henrik Kolstad. Videncenter for Arbejdsmiljø, 7. september 2015.

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Atherogenic Risk Factors and Hearing Thresholds

Thomas Winther Frederiksen[a, b] Cecilia Høst Ramlau-Hansen[c]
Zara Ann Stokholm[a] Matias Brødsgaard Grynderup[d] Åse Marie Hansen[d, e]
Søren Peter Lund[e] Jesper Medom Vestergaard[a] Jesper Kristiansen[e]
Jens Peter Bonde[g] Henrik Albert Kolstad[a]

[a]Department of Occupational Medicine, Danish Ramazzini Centre, Aarhus University Hospital, Aarhus, [b]Department of Otolaryngology/Head and Neck Surgery, Regionshospitalet Holstebro, Holstebro, [c]Department of Public Health, Section for Epidemiology, Aarhus University, Aarhus, [d]Department of Public Health, University of Copenhagen, Copenhagen, [e]National Research Centre for the Working Environment, Copenhagen, [f]Department of Public Health, University of Copenhagen, Copenhagen, [g]Department of Occupational and Environmental Medicine, Bispebjerg University Hospital, Copenhagen, Denmark

Key Words
Sensorineural hearing loss · Blood lipids · Glycosylated hemoglobin · Smoking habits · Body mass index · Ambulatory blood pressure

Abstract
The objective of this study was to evaluate the influence of atherogenic risk factors on hearing thresholds. In a cross-sectional study we analyzed data from a Danish survey in 2009–2010 on physical and psychological working conditions. The study included 576 white- and blue-collar workers from children's day care units, financial services and 10 manufacturing trades. Associations between atherogenic risk factors (blood lipids, glycosylated hemoglobin, smoking habits, body mass index (BMI), and ambulatory blood pressure) and hearing thresholds were analyzed using multiple linear regression models. Adjusted results suggested associations between smoking, high BMI and triglyceride level and low high-density lipoprotein level and increased low-frequency hearing thresholds (average of pure-tone hearing thresholds at 0.25, 0.5 and 1 kHz). Furthermore, an increasing load of atherogenic risk factors seemed associated with increased low-frequency hearing thresholds, but only at a borderline level of statistical significance. Associations were generally strongest with hearing levels of the worst hearing ear. We found no statistically significant associations between atherogenic risk factors and high-frequency hearing thresholds (average of pure-tone hearing thresholds at 4, 6 and 8 kHz).

Introduction

Sensorineural hearing thresholds generally increase with age due to a gradual degeneration of the cochlea and its central neural pathways [Schuknecht, 1964; Schuknecht and Gacek, 1993]. A complex interplay of environmental and genetic factors is thought to be the reason for this [Van Eyken et al., 2007].

Twin studies suggest that around half of the variance in sensorineural hearing thresholds in the middle-aged and older age groups is derived from genetic factors and the other half from environmental factors [Karlsson et al.,

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E-Mail karger@karger.com
www.karger.com/aud

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Thomas Winther Frederiksen
Department of Occupational Medicine
Danish Ramazzini Centre, Aarhus University Hospital
Nørrebrogade 44 building 2C, DK–8000 Aarhus C (Denmark)
E-Mail thofre@rm.dk

1997]. This allows for marked variation in median hearing thresholds within age groups [International Organization for Standardization, 2000] and the potential for the prevention of hearing loss if we learn more about the underlying nongenetic risk factors.

Occupational and leisure-time noise exposure [Daniel, 2007], ototoxic medication [Schacht et al., 2012] and industrial chemicals such as styrene and toluene [Hoet and Lison, 2008; Sliwinska-Kowalska, 2008] are among the already known risk factors for sensorineural hearing loss. In industrialized countries, this knowledge has led to legislation and new procedures intending to reduce the impact of these factors [Osguthorpe and Klein, 1991; Rybak and Whitworth, 2005]. This makes it relevant to look for other risk factors for sensorineural hearing loss as the composition of environmental exposures may have changed.

Smoking [Katsiki et al., 2013], hypertension [Chobanian, 1988], impaired blood sugar regulation [Selvin et al., 2006], high body mass index (BMI) [Van Gaal et al., 2006] and dyslipidemia [Koba and Hirano, 2011; Talayero and Sacks, 2011] are known to cause atherosclerotic vascular disease, leading to narrowing of arteries and decreased blood flow. As the cochlea is metabolically a very active organ depending on a steady supply of nutrients and oxygen from its vasculature to maintain homeostasis, atherosclerosis may be involved in the pathogenesis of sensorineural hearing loss.

In the Framingham cohort, cardiovascular disease events were associated with low-frequency hearing loss [Gates et al., 1993] and a more recent study has supported this finding [Friedland et al., 2009]. As atherosclerosis is intimately related to cardiovascular disease events, this may represent the common cause of both cardiovascular disease events and hearing loss, explaining the association found in the Framingham study. However, studies exploring the direct association between atherogenic risk factors and hearing loss show inconsistent findings: dyslipidemia in terms of elevated levels of total cholesterol, low-density lipoprotein (LDL), triglycerides (TG) and low levels of high-density lipoprotein (HDL) have shown mainly adverse effects on hearing ability [Gates et al., 1993; Suzuki et al., 2000; Shargorodsky et al., 2010] but a gainful effect of high total cholesterol level has also been reported [Jones and Davis, 2000]. A relation between hearing ability and diabetes-related measures has been reported in several studies [Austin et al., 2009; Jang et al., 2011; Akinpelu et al., 2014], which is also the case for smoking [Fransen et al., 2008; Shargorodsky et al., 2010] and high BMI [Fransen et al., 2008; Lalwani et al., 2013]. Other studies, however, have shown conflicting results for these factors [Gates et al., 1993; Shargorodsky et al., 2010]. The possible effect of hypertension has been evaluated both independently and in combination with noise exposure, showing both increased risk of hearing loss [Gates et al., 1993; Toppila et al., 2000] and no association [Shargorodsky et al., 2010]. These conflicting results could indicate weak associations that may have to act in combination to significantly affect hearing.

The aim of this study was to evaluate the association between well-established risk factors for atherosclerosis (high levels of LDL, TG and total cholesterol, low levels of HDL, elevated systolic and diastolic ambulatory blood pressure, smoking habits, high levels of glycosylated hemoglobin and high BMI) and hearing thresholds.

Materials and Methods

Participants

This cross-sectional study takes advantage of a survey of 819 workers conducted between 2001 and 2002 in Aarhus, Denmark, with the purpose of monitoring occupational noise exposure and hearing levels among blue- and white-collar workers. The cohort was recruited from children's day care units, financial services and 10 manufacturing trades. In 2009–2010, the same companies and workers were asked to participate again. This time the purpose was extended to also include psychosocial work factors, stress-related disorders, medical risk factors and parameters concerning auditory function. A total of 271 workers agreed to participate again and a further 394 workers were recruited de novo, making a total of 665 participants in 2009–2010. At the company level all participants were as far as possible selected at random. However, to avoid disruption of workflow, selection in some cases had to be done in accordance with the local manager.

In the present study, we excluded 88 participants with possible conductive hearing loss due to questionnaire-reported middle ear disease. Furthermore, we excluded 1 participant reporting Ménière's disease. In total, 576 workers were included in the present study. The age range was 20–73 years (mean 44.1). Workers only participating in 2001–2002 were not included in the present study due to lack of information on atherogenic risk factors as these were only measured in 2009–2010. Eight participants reported to be on antidiabetic medication, 22 participants were on high cholesterol medication and 53 participants took antihypertensives. The local scientific ethics committee approved the study (M.20080239). All participants gave written, informed consent to participate.

Variables

Audiometric Measures

Air conduction thresholds were determined for each ear at 0.25, 0.5, 1, 2, 3, 4, 6 and 8 kHz by pure-tone audiometry at the workplaces, using a Voyager 522 audiometer equipped with TDH-39 headphones (Madsen Electronics, Taastrup, Denmark). The audiometer was installed in a mobile examination unit equipped with a sound-proof booth (model AB-4240; Eckel Noise Control Technologies, Bagshot, UK). Audiometry was performed by trained examiners using a standardized protocol.

To avoid temporary threshold shifts from possible noise sources, all participants were asked to wear hearing protection from the beginning of the day until the audiometry was done. Otoscopy was performed initially to verify that ears were free of wax and the tympanic membrane was visible. The audiometer was calibrated every 6 months according to the standards of the International Organization for Standardization. Based on air conduction thresholds we calculated low- and high-frequency hearing thresholds for each ear. Low-frequency hearing thresholds were calculated as the average of pure-tone hearing thresholds at 0.25, 0.5 and 1 kHz and high-frequency hearing thresholds were defined as the average of pure-tone hearing thresholds at 4, 6 and 8 kHz. As analyses were performed on both the better and the worse hearing ear, we defined 4 hearing thresholds: low-frequency hearing threshold better ear (LFHT-better), low-frequency hearing threshold worse ear (LFHT-worse), high-frequency hearing threshold better ear (HFHT-better) and high-frequency hearing threshold worse ear (HFHT-worse). The better and worse hearing ear were defined as the ear with the lowest and highest average thresholds in the given spectrum, respectively. If hearing levels were equal in both ears, the same threshold value was used for statistical analysis of the better and the worse ear. Correspondingly, low- and high-frequency hearing loss for the better and the worse ear (LFHL-better, LFHL-worse, HFHL-better and HFHL-worse) were defined if LFHT-better, LFHT-worse, HFHT-better or HFHT-worse were above 25 dB hearing level, respectively.

Occupational Noise Exposure Assessment

Individual dosimeters (model 4443; Bruel & Kjær, Nærum, Denmark) measuring A-weighted equivalent sound levels (L_{Aeq}) in 5-second intervals were handed out to the participants. Microphones were fitted at the right-side collar if right-handed and vice versa if left-handed. The measuring range was set to 70–120 dB(A). Individual A-weighted equivalent noise levels were computed for the full work shift ($L_{Aeq, work}$).

Based on 1,268 noise exposure recordings from the 2001–2002 study and the 2009–2010 study, we predicted noise exposure levels for each combination of trade, occupation (blue- vs. white-collar worker) and calendar year (1980–2010) by mixed regression analyses, including these as fixed effects and the participants as random effect. The predicted noise exposure levels were linked with the employment histories of the participants by trade, occupation and calendar year. Information on employment histories (1980–2010) were retrieved from the Danish Supplementary Pension Fund. Using information from the resulting noise exposure matrix we calculated cumulative occupational noise exposure levels for each participant as the product of estimated noise exposure level [L_{Aeq} in dB(A)] and duration of employment (T) using the formula: $10 \times \log [\Sigma(10^{dB(A)/10} \times T)]$, resulting in 'dB(A)-year' on a logarithmic scale.

Questionnaire Information

A questionnaire was handed out to the participants at the time of the audiometric examination to provide information on medical and professional history. For the purpose of this study, information on socioeconomic status (personal income and educational level), middle ear disease (perforated ear drum, recurrent aural discharge and chronic otitis), family history of hearing handicap before the age of 70 years, military service, leisure-time noise exposure (hunting, use of fire arms, heavy use of portable music player, motor sport, playing electrically amplified musical instruments), smoking habits (ever-, never- or current smoker and smoking intensity) and medication (lipid-lowering medication, antidiabetics and antihypertensives) was used. The number of pack-years was calculated as the number of cigarettes smoked per day divided by 20 and multiplied by the number of years smoking.

Biochemical Data, Biometry and Ambulatory Blood Pressure Monitoring

For each worker, height and weight were measured and non-fasting venous blood was sampled by a medical laboratory technologist. Equipment for 24-hour ambulatory blood pressure monitoring (Space Labs 90217) was fitted together with the noise dosimeter and worn by the participant until the next day. Blood pressure was measured every 20 min during daytime (7 a.m. to 11 p.m.) and every 30 min during nighttime (11 p.m. to 7 a.m.) and average 24-hour systolic and diastolic blood pressure values were calculated. As some participants removed the equipment during nighttime, only measurements containing at least 4 nighttime observations were accepted as '24-hour ambulatory blood pressure'.

BMI was calculated as weight in kilograms divided by height in meters squared. Venous blood was refrigerated immediately after extraction, separated and frozen after being returned to the hospital. Biochemical analyses were done at the Department of Biochemistry, Aarhus University Hospital, Denmark, after all samples were collected at the work site. LDL levels were estimated using the Friedewald equation: estimated LDL = total cholesterol – HDL – (TG/5). A total of 15 participants (2.6%) had TG levels over 4.5 mmol/l, making the calculation unreliable. These values were excluded in the analysis of associations between LDL and hearing levels.

Statistics

We tabulated possible confounders according to LFHL-better, LFHL-worse, HFHL-better and HFHL-worse status.

We computed percentage differences in low- and high-frequency hearing thresholds for both the better and the worse ear by atherogenic risk factors using linear regression analysis. For these analyses, hearing threshold values were log transformed to normalize distribution of residuals. As LFHT-better for 82 workers (14.2%), LFHT-worse for 23 workers (4.0%), HFHT-better for 21 workers (3.6%) and HFHT-worse for 4 workers (0.7%) were zero or negative (minimum −5 dB hearing level) these values were replaced with a value of 1 dB hearing level before log transformation.

We adjusted for age, sex, educational level (none, short courses, skilled worker, short-range training, middle-range training, long-range training), personal income (DDK ≤299.999, DDK 300.000–499.999, DDK ≥500.000), family history of hearing loss (yes/no), ear disease (yes/no), military service (yes/no), noisy leisure-time activities (yes/no), hunting and shooting (yes/no). Crude results and results adjusted only for age and sex were also calculated, but were presented only in the text.

As atherosclerosis may result from the combined effect of several risk factors, we calculated an atherogenic risk factor score to examine the combined effect on hearing levels. For this purpose, the highest tertile of total cholesterol, TG, LDL, glycosylated hemoglobin, cumulative smoking, BMI, and 24-hour systolic and diastolic blood pressures was given a score of 1; otherwise a score of 0 was given. For HDL we reversed the scoring. The total atherogenic risk factor score was then calculated as the sum of the individual scores, ranging from 0 to 9 (i.e. a higher score indicated a higher risk of atherosclerosis). All statistical analyses were performed using Stata version 13.

We performed subanalyses in which participants reporting lipid-lowering medications, antidiabetics and antihypertensives were excluded.

Results

The characteristics of participants according to low- and high-frequency hearing loss in the better and the worse ear are presented in table 1. Participants with hearing loss in both low and high frequencies were generally around 10 years older than participants without hearing loss. Among participants with low-frequency hearing loss in the better and the worse ear, the prevalence was higher of males and participants reporting a family history of early hearing loss and former military service than among those with no low-frequency hearing loss. Among participants with high-frequency hearing loss in the better and the worse ear, the prevalence was higher of males, participants with a family history of early hearing loss, former military service, leisure-time hunting or shooting activities and blue-collar work compared with those with no high-frequency hearing loss. Cumulative occupational noise exposure was slightly higher for participants with high- and low-frequency hearing loss for the better as well as the worse ear.

Table 2 shows adjusted percentage differences in low-frequency hearing thresholds in the better and the worse ear by atherogenic risk factors. Results for the better ear showed statistically significant associations between TG ($p = 0.02$), status as former smoker ($p = 0.01$) and low-frequency hearing threshold. Also, there seemed to be a strong association between BMI and LFHT-better, but only at a borderline level of statistical significance ($p = 0.08$). Results for the worse ear showed statistically significant associations between HDL ($p = 0.03$; inverse association), TG ($p = 0.01$), status as former smoker ($p = 0.03$), BMI ($p = 0.03$) and low-frequency hearing threshold. Associations between average 24-hour diastolic blood pressure ($p = 0.07$), the atherogenic risk factor score ($p = 0.07$) and LFHT-worse also appeared strong, albeit only at a borderline level of statistical significance. For the remaining atherogenic risk factors, we generally observed weak positive associations with both better and worse ear low-frequency hearing thresholds. In general, the atherogenic risk factors showed stronger associations for the worse ear than the better ear at low frequencies.

Table 3 gives results for high-frequency hearing thresholds for the worse and the better ear as those presented for low frequencies in table 2. Adjusted results showed no statistically significant results for any of the atherogenic risk factors. As with results for low frequencies, we did, however, observe a general trend of weak positive associations (except for HDL) and the associations were in general stronger with worse ear than better ear thresholds.

To test if the association between BMI and low-frequency hearing threshold in the worse ear was mediated through high TG and low HDL, we performed a multivariable analysis that included BMI, TG and HDL in addition to the other confounders and LFHT-worse. The association between BMI and LFHT-worse decreased substantially. Thus, the mean percentage difference in LFHT-worse was 1.1% (95% CI: –0.6 to 2.8, $p = 0.22$) per unit of BMI, when TG and HDL were included in the model and 1.8% (95% CI: 0.2–3.4, $p = 0.03$) when not included.

Excluding participants taking lipid-lowering medication ($n = 22$, 3.8%), antidiabetics ($n = 8$, 1.4%) and antihypertensives ($n = 53$, 9.2%), respectively, from the statistical analyses testing for associations between blood lipids, glycosylated hemoglobin and blood pressures and hearing thresholds did not alter results noticeably. For example, the adjusted percentage difference in worse ear low-frequency hearing threshold changed from 8.6 (95% CI: 2.4–15.2) per mmol/l of TG to 8.9 (95% CI: 2.6–15.6) when excluding participants on lipid-lowering medications. Comparable differences were observed when testing associations between glycosylated hemoglobin and ambulatory blood pressures and hearing thresholds, excluding participants on antidiabetics and antihypertensives, respectively.

We also analyzed associations between atherogenic risk factors and hearing levels adjusted only for sex and age. This was done to enable a comparison of results to most previous studies that only adjusted for these factors. As expected, adjusting for only sex and age resulted generally in moderately stronger associations, but not to such an extent that the overall results were changed.

Discussion

The main findings of our analyses suggest an association between high BMI, high TG level, low HDL level and smoking and increased worse ear low-frequency hearing threshold. Comparable associations were observed for LFHT-better but these were generally weaker and only at a statistically significant level for TG and status as former smoker. Associations between atherogenic risk factors and high-frequency hearing thresholds for the better and the worse ear were, for the most part, weakly positive, but

Table 1. Characteristics of 576 industrial, financial and day care workers with or without low- and high-frequency hearing loss in the better and the worse ear, Aarhus, Denmark, 2009–2010

Characteristic	Low-frequency hearing loss				High-frequency hearing loss			
	better ear		worse ear		better ear		worse ear	
	yes (n = 9)	no (n = 567)	yes (n = 28)	no (n = 548)	yes (n = 153)	no (n = 423)	yes (n = 233)	no (n = 343)
Sex								
Female	1 (11.1)	151 (26.6)	5 (17.8)	147 (26.8)	18 (11.7)	134 (31.7)	25 (10.7)	127 (37.0)
Male	8 (88.9)	416 (73.4)	23 (82.1)	401 (73.2)	135 (88.2)	289 (68.3)	208 (89.3)	216 (63.0)
Missing	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Age, years	52.1±10.5	44.0±10.7	53.4±9.6	43.6±10.6	52.8±8.6	41.0±9.6	50.5±9.2	39.8±9.5
Missing	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Education								
None	3 (33.3)	66 (11.6)	6 (21.4)	63 (11.5)	20 (13.1)	49 (11.6)	29 (12.5)	40 (11.7)
Short courses	1 (11.1)	59 (10.4)	4 (14.3)	56 (10.2)	20 (13.1)	40 (9.5)	33 (14.2)	27 (7.9)
Skilled worker	4 (44.4)	308 (54.3)	15 (53.6)	297 (54.2)	89 (58.2)	223 (52.7)	132 (56.6)	180 (52.5)
Short-range training	0 (0.0)	35 (6.2)	0 (0.0)	35 (6.4)	6 (3.9)	29 (6.9)	11 (4.7)	24 (7.0)
Middle-range training	1 (11.1)	85 (15.0)	2 (7.1)	84 (15.3)	16 (10.5)	70 (16.6)	24 (10.3)	62 (18.1)
Long-range training	0 (0)	14 (0)	1 (3.6)	13 (2.4)	2 (1.3)	12 (2.8)	4 (1.7)	10 (2.9)
Missing	0 (0)	0 (0)	0 (0.0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Personal annual income, DDK								
<299,999	2 (22.2)	247 (43.6)	10 (35.7)	239 (43.6)	53 (34.6)	196 (46.3)	89 (38.2)	160 (46.7)
300,000–499,999	5 (55.6)	282 (49.7)	13 (46.4)	274 (50.0)	90 (58.8)	197 (46.6)	129 (55.4)	158 (46.1)
>500,000	1 (11.1)	32 (5.64)	4 (14.3)	29 (5.3)	8 (5.2)	25 (5.9)	12 (5.2)	21 (6.1)
Missing	1 (11.1)	6 (1.1)	1 (3.6)	6 (1.1)	2 (1.3)	5 (1.2)	3 (1.3)	4 (1.2)
Family history of early hearing loss								
No	1 (11.1)	264 (46.6)	6 (21.4)	259 (47.3)	52 (34.0)	213 (50.4)	86 (36.9)	179 (52.2)
Yes	5 (55.6)	161 (28.4)	10 (35.7)	156 (28.5)	52 (34.0)	114 (27.0)	75 (32.2)	91 (26.5)
Do not know	3 (33.3)	135 (23.8)	12 (42.9)	126 (23.0)	45 (29.4)	93 (22.0)	67 (28.8)	71 (20.1)
Missing	0 (0)	7 (1.2)	0 (0)	7 (1.3)	4 (2.6)	3 (0.7)	5 (2.2)	2 (0.6)
Military service								
Yes	5 (55.6)	163 (28.8)	13 (46.4)	155 (28.3)	66 (43.1)	102 (24.1)	95 (40.8)	73 (21.3)
No	3 (33.3)	391 (69.0)	14 (50.0)	380 (69.3)	83 (54.3)	311 (73.5)	133 (57.1)	261 (76.1)
Missing	1 (11.1)	13 (2.3)	1 (3.6)	13 (2.4)	4 (2.6)	10 (2.4)	5 (2.2)	9 (2.6)
Leisure-time hunting or shooting								
Yes	1 (11.1)	83 (14.6)	5 (17.9)	79 (14.4)	29 (19.0)	55 (13.0)	44 (18.9)	40 (11.7)
No	8 (88.9)	484 (85.4)	23 (82.1)	469 (85.6)	124 (81.1)	368 (87.0)	189 (81.1)	303 (88.3)
Missing	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Leisure-time noisy activities								
Yes	2 (22.2)	141 (24.9)	4 (14.3)	139 (25.4)	32 (20.9)	111 (26.2)	57 (54.5)	86 (25.1)
No	7 (77.8)	426 (75.1)	24 (85.7)	409 (74.6)	121 (79.1)	312 (73.8)	176 (75.5)	257 (74.9)
Missing	0 (0.0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Trade								
Manufacture	8 (88.9)	453 (79.9)	25 (89.3)	436 (79.6)	135 (88.2)	326 (77.1)	207 (88.8)	254 (74.1)
Day-care	0 (0)	49 (8.64)	1 (3.6)	48 (8.8)	3 (2.0)	46 (10.9)	7 (3.0)	42 (12.2)
Finance and other services	1 (11.1)	65 (11.5)	2 (7.1)	64 (11.7)	15 (9.8)	51 (12.1)	19 (8.2)	47 (13.7)
Missing	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Occupation								
White-collar worker	1 (11.1)	126 (22.2)	3 (10.7)	124 (22.6)	20 (13.1)	107 (25.3)	30 (12.9)	97 (28.3)
Blue-collar worker	8 (88.9)	441 (77.8)	25 (89.3)	424 (77.4)	133 (86.9)	316 (74.7)	203 (87.1)	246 (71.7)
Missing	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)
Cumulative occupational noise exposure, dB-years	96.6 [90.2–99.8]	96.1 [87.8–99.1]	97.3 [91.8–99.8]	96.0 [87.6–99.0]	97.6 [91.8–99.5]	95.5 [87.2–98.3]	97.4 [91.8–99.5]	95.0 [86.6–98.1]
Missing	0	0	0	0	0	0	0	0

Values are presented as numbers (with percentages), means ± SD or medians (with percentiles p10–p90 in square brackets), where appropriate. Low-frequency hearing loss was defined if the average of pure-tone hearing thresholds at 0.25, 0.5 and 1 kHz were above 25 dB. High-frequency hearing loss was defined if the average of pure-tone hearing thresholds at 4, 6 and 8 kHz were above 25 dB.

Table 2. Adjusted percentage differences in the better and worse ear low-frequency hearing threshold according to atherogenic risk factors

Exposure	Number	Better ear		Worse ear	
		adjusted percentage difference	p value	adjusted percentage difference	p value
Total cholesterol (per 1 mmol/l)	555	–1.4 (–8.8 to 6.4)	0.71	–1.2 (–7.8 to 5.7)	0.72
HDL (per 1 mmol/l)	555	–8.5 (–25.5 to 12.5)	0.40	–18.4 (–32.2 to –1.9)	0.03
TG (per 1 mmol/l)	555	8.1 (1.3 to 15.4)	0.02	8.6 (2.4 to 15.2)	<0.01
LDL (per 1 mmol/l)	540	–5.2 (–13.2 to 3.4)	0.23	–4.1 (–11.4 to 3.8)	0.30
Glycosylated hemoglobin (per 1%)	554	3.7 (–9.7 to 19.0)	0.61	7.3 (–5.2 to 21.4)	0.26
Smoking status					
Never	253	Ref.		Ref.	
Current	169	11.7 (–6.8 to 33.9)	0.23	13.6 (–3.4 to 33.6)	0.12
Former	152	26.8 (5.3 to 52.7)	0.01	21.6 (2.9 to 43.6)	0.02
Cumulative smoking (per 10 pack-years)	564	3.4 (–2.2 to 9.1)	0.23	3.7 (–1.4 to 8.7)	0.15
BMI (per 1 kg/m^2)	576	1.6 (–0.2 to 3.4)	0.08	1.8 (0.2 to 3.4)	0.03
Average 24-hour ambulatory systolic BP (per 10 mm Hg)	565	3.6 (–2.9 to 10.2)	0.28	3.9 (–2.0 to 9.8)	0.20
Average 24-hour ambulatory diastolic BP (per 10 mm Hg)	565	5.0 (–4.7 to 14.8)	0.32	8.1 (–0.7 to 16.9)	0.07
Atherogenic risk factor score (per 1 point)	576	2.4 (–1.6 to 6.5)	0.25	3.4 (–0.2 to 7.1)	0.07

Values in parentheses are 95% CI. Adjusted percentage differences: adjusted for age, sex, education, income, family history of hearing loss before age 70, military service, cumulative occupational noise exposure, leisure-time noisy activities (heavy use of portable music player, playing electrically amplified instrument, doing motor sports, shooting and hunting). Atherogenic risk factor score (0–9 points): 1 point for each atherogenic risk factor belonging in the high tertile (lowest tertile for HDL, as this factor is assumed to protect against atherosclerosis).

Table 3. Adjusted percentage differences in the better and worse ear high-frequency hearing threshold according to atherogenic risk factors

Exposure	Number	Better ear		Worse ear	
		adjusted percentage difference	p value	adjusted percentage difference	p value
Total cholesterol (per 1 mmol/l)	555	0.9 (–5.3 to 7.5)	0.782	2.3 (–2.7 to 7.7)	0.371
HDL (per 1 mmol/l)	555	1.5 (–14.6 to 20.5)	0.867	–2.9 (–15.4 to 11.4)	0.676
TG (per 1 mmol/l)	555	1.4 (–4.0 to 7.1)	0.608	3.5 (–0.9 to 8.1)	0.123
LDL (per 1 mmol/l)	540	0.3 (–6.9 to 8.0)	0.944	1.2 (–4.6 to 7.3)	0.687
Glycosylated hemoglobin (per 1%)	554	–1.9 (–12.6 to 10.1)	0.747	0.4 (–8.4 to 10.1)	0.928
Smoking status					
Never	253	Ref.		Ref.	
Current	169	5.4 (–9.4 to 22.7)	0.494	8.9 (–3.5 to 22.9)	0.166
Former	152	5.5 (–9.7 to 23.4)	0.494	4.5 (–7.7 to 18.3)	0.487
Cumulative smoking (per 10 pack-years)	564	1.7 (–2.9 to 6.4)	0.465	2.4 (–1.3 to 6.1)	0.203
BMI (per 1 kg/m^2)	576	0.4 (–1.0 to 1.9)	0.579	0.9 (–0.3 to 2.1)	0.137
Average 24-hour ambulatory systolic BP (per 10 mm Hg)	565	0.0 (–5.5 to 5.5)	0.995	–0.3 (–4.7 to 4.1)	0.894
Average 24-hour ambulatory diastolic BP (per 10 mm Hg)	565	1.0 (–9.1 to 7.2)	0.815	1.2 (–5.3 to 7.8)	0.718
Atherogenic risk factor score (per 1 point)	576	0.1 (–3.2 to 3.5)	0.958	1.8 (–0.8 to 4.5)	0.175

Values in parentheses are 95% CI. Adjusted percentage differences: adjusted for age, sex, education, income, family history of hearing loss before age 70, military service, cumulative occupational noise exposure, leisure time noisy activities (heavy use of portable music player, playing electrically amplified instrument, doing motor sports, shooting and hunting). Atherogenic risk factor score (0–9 points): 1 point for each atherogenic risk factor belonging in the high tertile (lowest tertile for HDL, as this factor is assumed to protect against atherosclerosis).

none were at a statistically significant level. Generally, associations between atherogenic risk factors and hearing thresholds were strongest at low-frequency hearing levels in the worse ear.

In a large European multicenter study on risk factors for age-related hearing impairment, an association between high BMI and hearing thresholds was also observed [Fransen et al., 2008]. The effect was equally distributed over all frequencies and not predominantly restricted to specific frequencies as observed in our study. Regrettably, for the comparison with this study, lipids were not accounted for. In the same study, a dose-dependent association between smoking and high-frequency hearing thresholds was observed. In our study, we were only able to demonstrate weak positive associations between cumulative smoking and hearing thresholds. In contrast, we demonstrated statistically significant associations between status as former smoker and low-frequency hearing thresholds. We would have expected significant results for cumulative smoking as well. A suggested explanation for our finding may be that participants had quit smoking due to adverse health effects, including cardiovascular disease, which we expected to be associated with hearing loss.

One of the rare prospective studies in this research field, including 26,917 participants, demonstrated a higher risk of hearing loss in participants with hypercholesterolemia and a past history of smoking [Shargorodsky et al., 2010]. BMI ≥30, a history of hypertension or diabetes were not associated with hearing loss in that study. All exposures and outcomes were, however, self-reported and frequency-specific analyses were thus not conducted.

Apart from cardiovascular disease events, cardiovascular risk factors in relation to hearing were also analyzed in the Framingham study [Gates et al., 1993]. In brief, the authors observed associations between blood pressure, blood glucose level and HDL and hearing levels, whereas no association with smoking, relative weight, serum cholesterol or TG were observed. Some of the associations were restricted to women and most associations were strongest for worse ear low-frequency thresholds, as also demonstrated in our study.

This seemingly 'low frequency- and worse ear-specific effect' of cardiovascular risk factors (in our study synonymous with atherogenic risk factors) also observed in our study is interesting. From studies investigating the relative contribution of genetic and nongenetic factors to hearing thresholds, we know that that the proportion of variance in hearing levels accounted for by nongenetic factors are higher in the worse hearing ear, particularly at low frequencies [Gates et al., 1999; Viljanen et al., 2007]. However, this still offers no explanation of the possible causal pathway. A hypothetical causal pathway, also suggested by others [Gates et al., 1993; Friedland et al., 2009], is that atherosclerosis causes microvascular disturbances in the mainly terminal vessels of the cochlea. This, subsequently, results in the ischemic degeneration of inner ear structures responsible for the detection and propagation of auditory signals. As apical parts of the cochlea (where blood supply is most distal and low-frequency sound is transmitted) are, theoretically, the most vulnerable to ischemia, this would explain the higher effect of atherogenic risk factors on low-frequency thresholds.

According to Schuknecht et al., who correlated audiometric patterns with cochlear histopathology [Schuknecht and Ishii, 1966; Schuknecht and Gacek, 1993], the hallmark of strial presbycusis (characterized by the degeneration of the stria vascularis) compared to the more common sensory presbycusis (characterized by loss of hair cells in the base of the cochlea) is a flattened audiogram, showing a relatively higher impact on low frequencies compared to the more common high-frequency sloping audiogram of, for example, the sensory presbycusis. If, hypothetically, the stria vascularis due to its highly vascular structure is susceptible to atherosclerotic vascular changes, this could also explain why atherogenic risk factors affect low frequencies most in the present and corresponding studies.

Finally, we cannot exclude that the apparent lack of impact on high-frequency thresholds is due to masking from additional risk factors for high-frequency hearing loss that were not taken into account in the present study. The background prevalence of high-frequency hearing loss in our study is about 10 times the background prevalence of low-frequency hearing loss (table 1), indicating that frequent risk factors are involved. We carefully adjusted for age, various sources of leisure-time noise and occupational noise, but additional noise exposure and other unknown factors may still have influenced our results.

Concerning blood lipids, we observed the strongest associations with BMI, TG and HDL and no association with LDL. This is interesting from a clinical point of view because recent studies found high BMI strongly associated with high TG and low HDL but not with LDL [Nicholls et al., 2006; Shamai et al., 2011]. This could indicate a causal pathway from high BMI through elevated TG and lowered HDL to hearing loss. We tested this, and results showed a substantial attenuation in the effect of BMI when adjusting for TG and HDL, supporting a link between obesity and low-frequency hearing levels that is partially mediated by high TG and low HDL.

Atherosclerotic vascular disease is often the result of a joint effect of multiple risk factors. We, therefore, assessed whether an increasing load of the risk factors included in this study affected hearing thresholds by an atherogenic risk factor score. This score showed a borderline statistically significant association with low-frequency hearing threshold in the worse ear and weaker positive associations with low-frequency hearing threshold in the better ear and high-frequency hearing threshold in the worse ear, indicating that the effects of the individual risk factors sum up.

Our study has a number of strengths. Firstly, we have analyzed hearing thresholds on a continuous scale, allowing us to keep as detailed information on individual hearing levels in the analyses as possible. Further, we analyzed different frequency hearing levels, as we assumed from previous studies [Gates et al., 1993; Friedland et al., 2009] that atherogenic risk factors would have frequency-specific effects.

Audiometric data were complete for all participants, and missing data on explanatory variables were limited. Furthermore, we had objective measures of most variables, leaving little room for differential misclassification. As hazardous noise levels are frequent in manufacturing industries, from which many of our participants were recruited, this was a potential source of bias to our study. To address this problem we carefully evaluated cumulative noise exposure for each participant back to 1980 and adjusted for this in our analyses.

If the effect of our exposure variables is mediated through atherosclerotic vascular changes, we suppose that the effect will be on the cochlea and thus affect sensorineural hearing thresholds. Testing bone conduction thresholds would have made evaluation of sensorineural thresholds more precise. However, due to time constraints in this epidemiological field study, we refrained from this. Instead we excluded participants with questionnaire information indicating conductive hearing loss due to middle ear disease.

The high number of tests for possible associations between exposures and outcomes is a possible limitation of our study as it increases the risk of obtaining significant results just by chance. However, we find our results consistent and in line with prior studies and suggested mechanisms, speaking against the risk of observing spurious significant associations.

A substantial part of our study population consisted of blue-collar workers from manufacturing industries, the rest being day care workers and employees from the financial sector. The general population is more heterogeneous, but this should not have conflicted with the external validity of our study because the effect of atherogenic risk factors is not expected to depend on population characteristics.

Another possible limitation of our study is the potential risk of a healthy worker survivor effect as this was a cross-sectional study that consisted not only of newly recruited participants but, supposedly, also included the healthiest earlier hired workers (little hearing loss and healthy lifestyle causing few atherogenic risk factors). This would be a possible source of selection bias in our study. The result of this would, however, be an underestimation of associations. Furthermore, it is unlikely that, for example, the level of TG, which is unknown to most subjects, predicts employment status conditional on hearing threshold.

Individual sensorineural hearing level varies due to a complex interplay of environmental exposures over time and genes determining individual susceptibility to these exposures. This study has been an attempt to uncover the possible contribution from atherogenic risk factors which, in contrast to the irreversibility of sensorineural hearing loss, are potentially reversible if treated through either modification of lifestyle or pharmacological intervention. According to our findings, lifestyle intervention will not only have preventive effects on cardiovascular disease but also on low-frequency sensorineural hearing loss.

Conclusion

We observed that low HDL, high TG, high BMI and history of former smoking were associated with increased low-frequency hearing thresholds, particularly in the worst hearing ear. Moreover, we found that as the number of risk factors for atherosclerosis increased, so did hearing levels. Generally, associations were strongest for low-frequency hearing thresholds.

In this study, we found no statistically significant associations between atherogenic risk factors and high-frequency hearing thresholds.

According to these results, atherogenic risk factors represent a potential risk for increased low-frequency hearing thresholds and lifestyle intervention is therefore a relevant target for hearing protection.

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References

Akinpelu OV, Mujica-Mota M, Daniel SJ: Is type 2 diabetes mellitus associated with alterations in hearing? A systematic review and meta-analysis. Laryngoscope 2014;124:767–776.

Austin DF, Konrad-Martin D, Griest S, McMillan GP, McDermott D, Fausti S: Diabetes-related changes in hearing. Laryngoscope 2009;119:1788–1796.

Chobanian A: Overview: hypertension and atherosclerosis. Am Heart J 1988;116:319–322.

Daniel E: Noise and hearing loss: a review. J Sch Health 2007;77:225–231.

Fransen E, Topsakal V, Hendrickx JJ, et al: Occupational noise, smoking, and a high BMI are risk factors for age-related hearing impairment and moderate alcohol consumption is protective: a European population-based multicenter study. J Assoc Res Otolaryngol 2008;9:264–276, discussion 261–263.

Friedland DR, Cederberg C, Tarima S: Audiometric pattern as a predictor of cardiovascular status: development of a model for assessment of risk. Laryngoscope 2009;119:473–486.

Gates GA, Cobb JL, D'Agostino RB, Wolf PA: The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. Arch Otolaryngol Head Neck Surg 1993;119:156–161.

Gates GA, Couropmitree NN, Myers RH: Genetic associations in age-related hearing thresholds. Arch Otolaryngol Head Neck Surg 1999;125:654–659.

Hoet P, Lison D: Ototoxicity of toluene and styrene: state of current knowledge. Crit Rev Toxicol 2008;38:127–170.

International Organization for Standardization (ISO): Acoustics – Statistical Distribution of Hearing Thresholds as a Function of Age (ISO 7029:2000). Geneva, ISO, 2000.

Jang TW, Kim BG, Kwon YJ, Im HJ: The association between impaired fasting glucose and noise-induced hearing loss. J Occup Health 2011;53:274–279.

Jones NS, Davis A: A retrospective case-controlled study of 1,490 consecutive patients presenting to a neuro-otology clinic to examine the relationship between blood lipid levels and sensorineural hearing loss. Clin Otolaryngol Allied Sci 2000;25:511–517.

Karlsson KK, Harris JR, Svartengren M: Description and primary results from an audiometric study of male twins. Ear Hear 1997;18:114–120.

Katsiki N, Papadopoulou SK, Fachantidou AI, Mikhailidis DP: Smoking and vascular risk: are all forms of smoking harmful to all types of vascular disease? Public Health 2013;127:435–441.

Koba S, Hirano T: Dyslipidemia and atherosclerosis. Nihon Rinsho 2011;69:138–143.

Lalwani AK, Katz K, Liu YH, Kim S, Weitzman M: Obesity is associated with sensorineural hearing loss in adolescents. Laryngoscope 2013;123:3178–3184.

Nicholls SJ, Tuzcu EM, Sipahi I, Schoenhagen P, Hazen SL, Ntanios F, Wun CC, Nissen SE: Effects of obesity on lipid-lowering, anti-inflammatory, and antiatherosclerotic benefits of atorvastatin or pravastatin in patients with coronary artery disease (from the REVERSAL study). Am J Cardiol 2006;97:1553–1557.

Osguthorpe JD, Klein AJ: Occupational hearing conservation. Otolaryngol Clin North Am 1991;24:403–414.

Rybak LP, Whitworth CA: Ototoxicity: therapeutic opportunities. Drug Discov Today 2005;10:1313–1321.

Schacht J, Talaska AE, Rybak LP: Cisplatin and aminoglycoside antibiotics: hearing loss and its prevention. Anat Rec (Hoboken) 2012;295:1837–1850.

Schuknecht HF: Further observations on the pathology of presbycusis. Arch Otolaryngol 1964;80:369–382.

Schuknecht HF, Gacek MR: Cochlear pathology in presbycusis. Ann Otol Rhinol Laryngol 1993;102:1–16.

Schuknecht HF, Ishii T: Hearing loss caused by atrophy of the stria vascularis. Nihon Jibiinkoka Gakkai Kaiho 1966;69:1825–1833.

Selvin E, Wattanakit K, Steffes MW, Coresh J, Sharrett AR: HbA_{1c} and peripheral arterial disease in diabetes: the Atherosclerosis Risk in Communities study. Diabetes Care 2006;29:877–882.

Shamai L, Lurix E, Shen M, Novaro GM, Szomstein S, Rosenthal R, Hernandez AV, Asher CR: Association of body mass index and lipid profiles: evaluation of a broad spectrum of body mass index patients including the morbidly obese. Obes Surg 2011;21:42–47.

Shargorodsky J, Curhan SG, Eavey R, Curhan GC: A prospective study of cardiovascular risk factors and incident hearing loss in men. Laryngoscope 2010;120:1887–1891.

Sliwinska-Kowalska M: Organic solvent exposure and hearing loss. Occup Environ Med 2008;65:222–223.

Suzuki K, Kaneko M, Murai K: Influence of serum lipids on auditory function. Laryngoscope 2000;110:1736–1738.

Talayero BG, Sacks FM: The role of triglycerides in atherosclerosis. Curr Cardiol Rep 2011;13:544–552.

Toppila E, Pyykko II, Starck J, Kaksonen R, Ishizaki H: Individual risk factors in the development of noise-induced hearing loss. Noise Health 2000;2:59–70.

Van Eyken E, Van Camp G, Van Laer L: The complexity of age-related hearing impairment: contributing environmental and genetic factors. Audiol Neurootol 2007;12:345–358.

Van Gaal LF, Mertens IL, De Block CE: Mechanisms linking obesity with cardiovascular disease. Nature 2006;444:875–880.

Viljanen A, Kaprio J, Pyykko I, Sorri M, Kauppinen M, Koskenvuo M, Rantanen T: Genetic and environmental influences on hearing at different frequencies separately for the better and worse hearing ear in older women. Int J Audiol 2007;46:772–779.

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ORIGINAL ARTICLE

Ambient and at-the-ear occupational noise exposure and serum lipid levels

Mai C. Arlien-Søborg[1] · Astrid S. Schmedes[1] · Z. A. Stokholm[1] · M. B. Grynderup[2] · J. P. Bonde[3] · C. S. Jensen[1] · Å. M. Hansen[2,4] · T. W. Frederiksen[1] · J. Kristiansen[4] · K. L. Christensen[4,5] · J. M. Vestergaard[1] · S. P. Lund[4] · H. A. Kolstad[1]

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Abstract
Objectives Occupational and residential noise exposure has been related to increased risk of cardiovascular disease. Alteration of serum lipid levels has been proposed as a possible causal pathway. The objective of this study was to investigate the relation between ambient and at-the-ear occupational noise exposure and serum levels of total cholesterol, low-density lipoprotein–cholesterol, high-density lipoprotein–cholesterol, and triglycerides when accounting for well-established predictors of lipid levels.
Methods This cross-sectional study included 424 industrial workers and 84 financial workers to obtain contrast in noise exposure levels. They provided a serum sample and wore portable dosimeters that every 5-s recorded ambient noise exposure levels during a 24-h period. We extracted measurements obtained during work and calculated the full-shift mean ambient noise level. For 331 workers who kept a diary on the use of a hearing protection device (HPD), we subtracted 10 dB from every noise recording obtained during HPD use and estimated the mean full-shift noise exposure level at the ear.
Results Mean ambient noise level was 79.9 dB (A) [range 55.0–98.9] and the mean estimated level at the ear 77.8 dB (A) [range 55.0–94.2]. Ambient and at-the-ear noise levels were strongly associated with increasing levels of triglycerides, cholesterol–HDL ratio, and decreasing levels of HDL–cholesterol, but only in unadjusted analyses that did not account for HPD use and other risk factors.
Conclusion No associations between ambient or at-the-ear occupational noise exposure and serum lipid levels were observed. This indicates that a causal pathway between occupational and residential noise exposure and cardiovascular disease does not include alteration of lipid levels.

Keywords Manufacturing industries · Cardiovascular disease · Causal pathways · Hearing protective devices

✉ Mai C. Arlien-Søborg
maiarlie@rm.dk

✉ Astrid S. Schmedes
astrid_ss@hotmail.com

[1] Department of Occupational Medicine, Danish Ramazzini Centre, Aarhus University Hospital, Nørrebrogade 44 Building 2C, 8000 Aarhus C, Denmark

[2] Department of Public Health, University of Copenhagen, Copenhagen, Denmark

[3] Department of Occupational and Environmental Medicine, Bispebjerg University Hospital, Copenhagen, Denmark

[4] National Research Centre for the Working Environment, Copenhagen, Denmark

[5] Department of Cardiology B, Aarhus University Hospital, Aarhus, Denmark

Introduction

Ample empirical evidence suggests a causal association between occupational and residential noise exposure and cardiovascular diseases such as stroke, hypertension, and acute myocardial infarction (Chang et al. 2003; Davies et al. 2005; Gan et al. 2011; Hansell et al. 2013; Melamed et al. 1999; Virkkunen et al. 2005; Virtanen and Notkola 2002). Noise exposure is believed to evoke a stress response activating the sympathetic branch of the autonomous nervous system and the endocrine system, respectively (Andren 1982; Anticaglia and Cohen 1970; Cavatorta et al. 1987; Lehmann and Tamm 1956; Ortiz et al. 1974). Elevated levels of stress hormones are associated with changes in physiological functions and the metabolism of

the organism including blood lipids (cholesterol, triglycerides, and free fatty acids) (Atkinson and Milsum 1983; Lundberg 1999; Selye 1955).

Serum lipids play a major role in the causation of cardiovascular disease and could represent a causal pathway between noise exposure and cardiovascular disease (Nabel 2003). Increased cholesterol and triglyceride levels have been observed in workers exposed to noise above 80 dB (A) (Melamed et al. 1997; Ortiz et al. 1974; Rai et al. 1981; Vangelova and Deyanov 2007), but this effect has not been observed by others (Chang et al. 2003; Virkkunen et al. 2005, 2006). Serum lipid levels are influenced by sex, age, body mass index (BMI), waist circumference, treatment with statins, beta blockers and other medicines, diabetes, smoking, alcohol, physical activity, and social status (American heart association 2002; Carroll et al. 2005; Gossett et al. 2009; Hu et al. 2000; Kasiske et al. 1995; Primatesta and Poulter 2006; Stone 1994; Virtanen and Notkola 2002); however, this has only rarely been accounted for (Melamed et al. 1997).

Hearing protective devices (HPD) are increasingly used by workers occupationally exposed to noise and are in Denmark recommended by the Danish Working Environment Authority with noise exposure levels above 80 dB (A) (Danish Working Environment Authority 2014). HPD may significantly reduce noise exposure levels at the ear (Neitzel et al. 2006).

This study analysed whether increasing levels of ambient and at-the-ear occupational noise exposure are associated with increasing levels of total cholesterol, low-density lipoprotein (LDL)–cholesterol, and triglycerides and a decreasing level of high-density lipoprotein (HDL)–cholesterol when accounting for well-documented risk factors.

Materials and methods

Subjects

In 2009–2010, we recruited workers from 76 companies within manufacturing industries (manufactures of food, wood products, non-metallic mineral products, basic metals, fabricated metal, machinery, motor vehicles, furniture, publishing and printing, and construction) with high reporting of noise-induced hearing loss according to the Danish Working Environment Authority (2014), children day care units and workers from financial and others services to obtain contrast in exposure levels. A total of 665 workers agreed to participate for two consecutive days according to a protocol that included individual noise recording and blood sampling. We excluded 61 children day care workers because of noise recordings of questionable validity, and 43 participants invited to the local hospital that were not exposed to work-related noise on the measurement day (33 unemployed or retired, 10 on sick leave, maternity leave or off duty). All participants filled in a questionnaire, 526 participants provided a noise measurement during work on the first day, and of these, 508 provided a blood sample that was analysed for serum lipid levels (424 blue-collar industrial workers and 84 white-collar financial and other service workers). A total of 331 participants completed a HPD diary concurrent with the noise recording. All subjects gave informed consent. The study protocol was approved by the local ethics committee (M-200880239) and the Danish Data Protection Agency (2009-41-3072).

Noise exposure assessment

Ambient noise exposure levels were measured by portable dosimeters (*Bruel and Kjær 4443*). The dosimeters were calibrated, handed over, worn in a pouch attached to a belt at the participant's waist, and collected at the workplaces. Microphones were placed on the right shoulder if right-handed and left shoulder if left-handed. The A-weighted equivalent sound level (L_{Aeq}) was recorded every 5 s for 24 h. The dosimeters were set to a range of 70–120 dB. The display of the dosimeters was dimmed during measurements to minimize noise-dependent influence on the participant's behaviour. During noise measurement, participants registered the beginning and ending of working hours and leisure time, and we estimated the ambient L_{Aeq} values for these periods. The 331 workers who kept a HPD diary registered the time of beginning and ending of every period of HPD use. This information was synchronized with the noise recordings, and we subtracted 10 dB from every 5-s noise recording obtained during HPD use to estimate the L_{AEq} value at the ear (Giardino and Durkt 1996; Neitzel et al. 2006).

Personal data

At the day of examination, height, weight, and waist circumference were measured by biomedical laboratory technologists. BMI was calculated based on height measured without shoes and weight measured with clothes. Levels of total cholesterol, HDL–cholesterol, and triglycerides were analysed by a chromogenic catalytic method. We estimated LDL–cholesterol from the formula of Friedwald: LDL–cholesterol = total cholesterol − (HDL–cholesterol + 0.45 × triglycerides) when triglycerides <4.5 mmol/l. The participants completed a questionnaire about lifestyle, medications, education, and income.

Established risk factors for dyslipidemia

Based on a review of the literature, we identified the following factors as potential confounders to be included in

our statistical models: treatment with statin (Stone 1994), levothyroxine, antihypertensive, corticoid, immunosuppressive, and oestrogen medications (Kasiske et al. 1995; Stone 1994), smoking (Gossett et al. 2009), alcohol consumption (Stone 1994), physical activity (Stone 1994), BMI, waist circumference (Hu et al. 2000), income, and educational level (American heart association 2002). In addition, we also included noise exposure outside work, which has been associated with the occurrence of cardiovascular disease (Babisch et al. 2005; Babisch 2011; Passchier-Vermeer and Passchier 2000).

Statistical analysis

From the individual noise recordings, participants were classified into four full-shift occupational noise exposure categories: *low* [<75 dB (A)], *medium* [75–79 dB (A)], *high* [80–84 dB (A)], and *very high* [≥85 dB (A)]. Lipid levels were normally distributed; means, standard deviations, and 95 % confidence intervals were tabulated by noise exposure levels. We analysed serum lipids as a function of occupational noise exposure level by linear regression. The models were adjusted for the following a priori selected potential confounders: sex, age, BMI (kg/m^2), medication affecting serum lipid levels (statins, beta blockers, oestrogens, retinoids, diuretics, levothyroxine, or glucocorticoids; yes/no), diabetes (yes/no), current smoking (yes/no), alcohol consumption (>84 g/week for women and >168 g/week for men; yes/no which are the maximum weekly low-risk intakes recommended by the Danish Health and Medicines Authority), low physical activity (<4 h of low-intensity physical activity per week or <2–4 h of moderate-intensity physical activity per week), low educational level (yes/no, low level defined as no education at all or skilled worker), and personal annual income (<52.849 US$). Furthermore, we included the leisure-time noise exposure levels that were extracted from the individual noise recordings. As a sensitivity check, we excluded finance and other services workers, because they may differ from the industrial workers with respect to extraneous predictors of lipid levels.

Data processing and analysis were performed with Stata version 12 (StataCorp, College Station, TX).

Results

The median full-shift ambient noise exposure level was 79.9 dB (A) [range 55.0–98.9 dB (A)]. In all, 112 workers were exposed at a low [<75 dB (A)], 112 at a median [75–79 dB (A)], 154 at a high [80–85 dB (A)], and 130 at a very high noise level [>85 dB (A)]. Only 37 (7 %) workers were exposed above 90 dB (A). The mean estimated level at the ear was 77.8 dB (A) (range 55.0–94.2).

Table 1 shows significantly increasing proportions of men, current smokers, and participants with low level of education, low personal income, increasing mean waist circumference or BMI, and increasing leisure-time noise exposure level with increasing full-shift ambient occupational noise exposure level. An inverse association was observed for use of medications that may affect serum lipid levels.

Table 2 shows mean serum lipid levels with 95 % confidence intervals (95 % CI) by ambient occupational noise exposure level. Triglyceride level and cholesterol–HDL ratio increased, whereas HDL–cholesterol level decreased by increasing ambient occupational noise exposure (p values <0.05). Cholesterol level was almost unchanged with increasing ambient noise level.

Table 1 Characteristics of the study population of 503 Danish workers according to ambient occupational noise exposure level, 2009–2010

Worker characteristics	Ambient occupational noise exposure level				
	<75 dB (A)	75–79 dB (A)	80–84 dB (A)	≥85 dB (A)	p value
	$N = 112$	$N = 112$	$N = 154$	$N = 130$	
Sex, men, no. (%)	62 (55)	92 (82)	135 (88)	115 (88)	<0.05
Age, mean (SD)	45 (10)	44 (10)	43 (10)	42 (10)	0.03
BMI, kg/m^2 mean (SD)	25 (4)	27 (4)	27 (4)	27 (4)	0.006
Waist circumference, cm (SD)	90 (13)	95 (13)	96 (13)	96 (15)	0.002
Heavy alcohol consumption, no. (%)	18 (16)	16 (14)	27 (18)	26 (20)	0.7
Current smoking, no. (%)	18 (16)	39 (35)	49 (32)	47 (36)	<0.05
Low physical activity, no. (%)	61 (54)	60 (54)	97 (63)	80 (62)	0.3
Use of medications affecting serum lipid levels, no. (%)	21 (19)	17 (15)	17 (11)	11 (8)	0.04
Low educational, no. (%)	63 (56)	96 (86)	135 (88)	118 (91)	<0.05
Personal income, <52.849 US$ no. (%)	30 (27)	42 (38)	44 (68)	57 (44)	0.004
Leisure-time noise, dB (A), mean (SD)	70 (6)	72 (7)	73 (6)	74 (7)	<0.05

Table 2 Serum lipid levels according to ambient and at-the-ear occupational noise exposure levels among Danish workers, 2009–2010

Serum lipid parameter	Mean (95 %CI)							Test for trend[a]
	Noise exposure level							*p* value
	N	<75 dB (A)	*N*	75–79 dB (A)		80–84 dB (A)	*N*	≥85 dB (A)	
Ambient occupational noise exposure level									
Cholesterol (mmol/l)	112	5.2 (5.0–5.4)	112	5.4 (4.9–5.9)	154	5.3 (4.8–5.7)	129	5.3 (4.8–5.8)	0.338
Triglycerides (mmol/l)	112	1.5 (1.3–1.8)	112	1.7 (1.1–1.3)	154	1.9 (1.4–2.5)	129	1.8 (1.3–2.4)	0.039
HDL (mmol/l)	112	1.5 (1.5–1.6)	112	1.3 (1.2–1.4)	154	1.3 (1.2–1.4)	129	1.3 (1.2–1.5)	0.001
LDL (mmol/l)	111	3.0 (2.8–3.2)	111	3.3 (2.8–3.7)	146	3.1 (2.7–3.6)	122	3.2 (2.8–3.6)	0.099
Cholesterol–HDL ratio	112	3.7 (3.4–4.0)	112	4.2 (3.6–4.9)	154	4.4 (3.7–5.0)	129	4.3 (3.6–4.9)	0.006
At-the-ear occupational noise exposure level									
Cholesterol (mmol/l)	81	5.2 (5.0–5.4)	61	5.4 (4.9–6.0)	99	5.2 (4.7–5.7)	89	5.3 (4.8–5.8)	0.294
Triglycerides (mmol/l)	81	1.3 (1.1–1.5)	61	1.6 (0.9–2.1)	99	1.7 (1.2–2.2)	89	1.8 (1.3–2.3)	0.002
HDL (mmol/l)	81	1.6 (1.5–1.7)	61	1.4 (1.2–1.6)	99	1.3 (1.1–1.5)	89	1.3 (1.1–1.5)	0.000
LDL (mmol/l)	81	3.0 (2.8–3.2)	61	3.3 (2.8–3.8)	95	3.1 2.6–3.5)	85	3.3 (2.8–3.7)	0.052
Cholesterol–HDL ratio	81	3.5 (3.2–3.8)	61	4.1 (3.3–3.9)	99	4.2 (3.5–4.9)	89	4.3 (3.6–5.0)	0.000

CI confidence interval, *HDL* high-density lipid, *LDL* low-density lipid

[a] *p* value of trend tests that included noise exposure level as a continuous variable

Table 3 Lipid level differences by increasing ambient and at-the-ear occupational noise exposure levels

Serum lipid parameter	Adjusted difference (95 %CI)[a]			Test for trend[b]
	Noise exposure level			Adjusted *p* value
	75–79 dB (A)	80–84 dB (A)	≥85 dB (A)	
Ambient occupational noise exposure level				
Cholesterol (mmol/l)	0.2 (−0.1–0.4)	0.0 (−0.2–0.3)	0.1 (−0.2–0.4)	0.512
Triglycerides (mmol/l)	−0.2 (−0.5–0.2)	−0.1 (−0.4–0.3)	−0.1 (−0.5–0.2)	0.591
HDL (mmol/l)	−0.0 (−0.1–0.1)	−0.0 (−0.1–0.1)	−0.0 (−0.1–0.1)	0.994
LDL (mmol/l)	0.2 (−0.0–0.5)	0.1 (−0.1–0.3)	0.2 (−0.7–0.4)	0.221
Cholesterol−HDL ratio	0.1 (−0.3–0.4)	0.2 (−0.2–0.5)	0.1 (−0.3–0.4)	0.927
Estimated occupational noise exposure level at the ear				
Cholesterol (mmol/l)	0.2 (−0.2–0.5)	−0.1 (−0.4–0.3)	0.1 (−0.3–0.5)	0.785
Triglycerides (mmol/l)	0.0 (−0.3–0.4)	0.1 (−0.3–0.4)	0.1 (−0.3–0.5)	0.619
HDL (mmol/l)	0.0 (−0.1–0.1)	−0.1 (−0.2–0.1)	−0.1 (−0.2–0.1)	0.462
LDL (mmol/l)	0.2 (−0.1–0.5)	−0.0 (−0.3–0.3)	0.1 (−0.1–0.5)	0.356
Cholesterol–HDL ratio	0.1 (−0.4–0.5)	0.2 (−0.2–0.7)	0.3 (−0.2–0.8)	0.399

Results are relative to a reference category <75 dB (A) and adjusted for recognized predictors of lipid levels, Danish workers 2009–2010

[a] Difference between means of the exposure, adjusted for sex, current smoking, heavy drinking, age, BMI, waist width, medicines, physical activity, leisure-time noise, education, and income

[b] Trends on noise exposure [continuous data per 1 dB (A)]

Table 3 presents the adjusted lipid level differences with 95 % CI intervals for each of the three exposure categories ≥75 dB (A) relative to the reference category <75 dB (A). Results of tests for linear trend are also provided. Contrary to the unadjusted analyses presented in Table 2, we observed no indications of trends by ambient occupational noise exposure level.

In analyses comprising only the 331 participants with an estimated noise exposure level at the ear (Tables 2, 3), we found crude trends by noise exposure level for all serum lipid measures ($p < 0.05$), but cholesterol. However, as for the ambient noise exposure levels, no trends were apparent in the adjusted analyses.

Excluding finance and other services workers did not change findings substantially.

Discussion

We observed that occupational ambient and at-the-ear noise exposure levels were strongly associated with increasing level of triglycerides, cholesterol–HDL ratio, and decreasing level of HDL–cholesterol. However, we also observed corresponding associations with male sex, smoking, low-level education, low personal income, waist circumference, and BMI. We observed no association between occupational noise exposure levels and serum lipid levels when account was taken for these established predictors of lipid levels.

Occupational noise exposure has been associated with cardiovascular disease in recent longitudinal studies of high exposed worker populations (Chang et al. 2013; Sbihi et al. 2008). A large register-linked study recently showed no increasing risk of hypertension at noise exposure levels within the lower half of the 80–90 dB (A) range and likewise no increased risk of stroke by increasing levels of occupational noise exposure (Stokholm et al. 2013b, a). Van Kempen and colleagues concluded in a meta-analysis that the relation between noise exposure and ischaemic heart disease is inconclusive primarily due to limitations in exposure characterization and adjustment for important confounders (van Kempen et al. 2002)

Alteration of serum lipid levels has been proposed as a possible causal pathway between noise exposure and cardiovascular disease. We could, however, not confirm earlier suggested findings of such an association. This discrepancy may be due to insufficient control for documented risk factors in earlier studies. Six studies have investigated the association between occupational noise exposure and lipid levels (Chang et al. 2003; Melamed et al. 1997; Ortiz et al. 1974; Rai et al. 1981; Vangelova and Deyanov 2007; Virkkunen et al. 2005, 2006). Only Melamed et al. adjusted for a limited set of extraneous risk factors, and they only observed an association for a subpopulation of young men (Melamed et al. 1997). No former study has accounted for the use of hearing protective devices. Four studies reported increasing lipid levels by increasing noise exposure level, but findings were not consistent across studies with respect to which lipids were affected (Melamed et al. 1997; Ortiz et al. 1974; Rai et al. 1981; Vangelova and Deyanov 2007). Our findings are in line with those of Virkunen et al. and Chang et al. that observed no association between noise exposure level and serum lipid levels (Chang et al. 2003; Virkkunen et al. 2005, 2006).

This study has several strengths. First, we accounted for well-established extraneous risk factors of serum lipid levels. Interestingly, the crude association between occupational noise exposure and dyslipidemia vanished when account was taken for them. To our knowledge, this study is the first with an extensive adjustment for potential confounders.

Second, we measured full-shift ambient noise exposure level with personal dosimeters ensuring a more valid individual exposure assessment compared with stationary measurements often used (Melamed et al. 1997; Ortiz et al. 1974; Rai et al. 1981; Vangelova and Deyanov 2007; Virkkunen et al. 2006). Third, the 24-h noise measurements made us able to account for noise exposure outside work which may be relevant since noise exposure at the residence may also affect cardiovascular health (Babisch et al. 2005; Babisch 2011; Passchier-Vermeer and Passchier 2000). It is noteworthy that occupational noise exposure levels were associated with noise exposure levels during leisure time in accordance with findings by Agrawal et al. (2010). Fourth, we were able to take the use of hearing protectors into account and assessed noise exposure at the ear for a large proportion of the workers since we synchronized the individual noise recording with information of HPD use from the HPD diaries. These analyses neither suggested an effect of noise exposure on lipid levels when adjustment was made for well-established predictors of lipid levels.

The study has also limitations. Because this was a cross-sectional study, selection into and out of the population may have affected findings. However, this is perhaps not plausible since lipid levels are not likely to affect such movements in the work force, at least if workers are unaware of their lipid status. For similar reasons, differential recall is also unlikely.

We only collected one serum sample per worker and serum lipid levels may vary between days (Roades and Bell 2009). Inclusion of more serum samples per worker would have reduced misclassification of lipid parameters. The high number of study participants in our study had a similar effect.

Our noise exposure assessment was based on one-day monitoring that did not account for expected day-to-day variation. Thus, within-worker variability may be larger than between-worker variability, which may have attenuated the exposure response relations.

The study population did only include few workers (7 %) exposed above 90 dB (A), and we were thus not able to assess effects of high occupational noise exposure levels that previously have been associated with ischaemic heart disease (Chang et al. 2013; Sbihi et al. 2008). On the other hand, we obtained a more than eightfold exposure contrast partly due to the inclusion of low exposed finance and other services workers. Because we were able to adjust for all major predictors of lipid levels, we think this should have accounted for incomparability with the industrial workers.

Different types of HPD (earmuffs, earplugs etc.) show different attenuation of noise between 5 and 20 dB (A) (Giardino and Durkt 1996; Park and Casali 1991; Toivonen et al. 2002). We were not able to include such information when we assessed noise level at the ear.

To conclude, we observed no association between ambient or at-the-ear occupational noise exposure level and serum lipid levels when adjusted for well-established predictors of lipid levels. We could thus not support earlier findings suggesting that altered serum lipid levels may be part of a causal pathway between occupational noise exposure and cardiovascular disease.

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Author's contribution Arlien-Søborg MC and Schmedes AS contributed equally to this work as first authors. Stokholm ZA contributed with good advice and suggestions for improvement during the process. Kolstad HA acted as main supervisor. Grynderup MB contributed with statistical support when needed. Jensen CS was responsible for collecting data and contributed with practical help. Bonde JP, Hansen AM, Frederiksen TW, Kristiansen J, Christensen KL, Vestergaard JM, Lund SP all contributed with their expertise in the field and participated in meetings regarding the study.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interests.

References

Agrawal Y, Niparko JK, Dobie RA (2010) Estimating the effect of occupational noise exposure on hearing thresholds: the importance of adjusting for confounding variables. Ear Hear 31:234–237

American heart association (2002) Third Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel) final report. Circulation 106:3143–3421

Andren L (1982) Cardiovascular effects of noise. Acta Med Scand Suppl 657:1–45

Anticaglia JR, Cohen A (1970) Extra-auditory effects of noise as a health hazard. Am Ind Hyg Assoc J 31:277–281

Atkinson C, Milsum JH (1983) A system model of the metabolic response to stress. Behav Sci 28:268–273

Babisch W (2011) Cardiovascular effects of noise. Noise. Health 13:201–204

Babisch W, Beule B, Schust M, Kersten N, Ising H (2005) Traffic noise and risk of myocardial infarction. Epidemiology 16:33–40

Carroll MD, Lacher DA, Sorlie PD, Cleeman JI, Gordon DJ, Wolz M, Grundy SM, Johnson CL (2005) Trends in serum lipids and lipoproteins of adults, 1960–2002. JAMA 294:1773–1781

Cavatorta A, Falzoi M, Romanelli A, Cigala F, Ricco M, Bruschi G, Franchini I, Borghetti A (1987) Adrenal response in the pathogenesis of arterial hypertension in workers exposed to high noise levels. J Hypertens Suppl 5(5):S463–S466

Chang TY, Jain RM, Wang CS, Chan CC (2003) Effects of occupational noise exposure on blood pressure. J Occup Environ Med 45:1289–1296

Chang TY, Hwang BF, Liu CS, Chen RY, Wang VS, Bao BY, Lai JS (2013) Occupational noise exposure and incident hypertension in men: a prospective cohort study. Am J Epidemiol 177:818–825

Danish Working Environment Authority (2014) http://arbejdstilsynet.dk/en/engelsk.aspx

Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA (2005) Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology 16:25–32

Gan WQ, Davies HW, Demers PA (2011) Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999–2004. Occup Environ Med 68:183–190

Giardino DA, Durkt G Jr (1996) Evaluation of muff-type hearing protectors as used in a working environment. Am Ind Hyg Assoc J 57:264–271

Gossett LK, Johnson HM, Piper ME, Fiore MC, Baker TB, Stein JH (2009) Smoking intensity and lipoprotein abnormalities in active smokers. J Clin Lipidol 3:372–378

Hansell AL, Blangiardo M, Fortunato L, Floud S, de HK, Fecht D, Ghosh RE, Laszlo HE, Pearson C, Beale L, Beevers S, Gulliver J, Best N, Richardson S, Elliott P (2013) Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. BMJ 347:f5432

Hu D, Hannah J, Gray RS, Jablonski KA, Henderson JA, Robbins DC, Lee ET, Welty TK, Howard BV (2000) Effects of obesity and body fat distribution on lipids and lipoproteins in nondiabetic American Indians: the strong heart study. Obes Res 8:411–421

Kasiske BL, Ma JZ, Kalil RS, Louis TA (1995) Effects of antihypertensive therapy on serum lipids. Ann Intern Med 122:133–141

Lehmann G, Tamm J (1956) Changes of circulatory dynamics of resting men under the effect of noise. Int Z Angew Physiol 16:217–227

Lundberg U (1999) Coping with stress: neuroendocrine reactions and implications for health. Noise Health 1:67–74

Melamed S, Froom P, Kristal-Boneh E, Gofer D, Ribak J (1997) Industrial noise exposure, noise annoyance, and serum lipid levels in blue-collar workers—the CORDIS Study. Arch Environ Health 52:292–298

Melamed S, Kristal-Boneh E, Froom P (1999) Industrial noise exposure and risk factors for cardiovascular disease: findings from the CORDIS study. Noise Health 1:49–56

Nabel EG (2003) Cardiovascular disease. N Engl J Med 349:60–72

Neitzel R, Somers S, Seixas N (2006) Variability of real-world hearing protector attenuation measurements. Ann Occup Hyg 50:679–691

Ortiz GA, Arguelles AE, Crespin HA, Villafane CT (1974) Modifications of epinephrine, norepinephrine, blood lipid fractions and the cardiovascular system produced by noise in an industrial medium. Horm Res 5:57–64

Park MY, Casali JG (1991) A controlled investigation of in-field attenuation performance of selected insert, earmuff, and canal cap hearing protectors. Hum Factors 33:693–714

Passchier-Vermeer W, Passchier WF (2000) Noise exposure and public health. Environ Health Perspect 108(Suppl 1):123–131

Primatesta P, Poulter NR (2006) Levels of dyslipidemia and improvement in its management in England: results from the Health Survey for England 2003. Clin Endocrinol (Oxf) 64:292–298

Rai RM, Singh AP, Upadhyay TN, Patil SK, Nayar HS (1981) Biochemical effects of chronic exposure to noise in man. Int Arch Occup Environ Health 48:331–337

Roades RA, Bell DR (2009) Medical physiology, principle for Clinical medicine, 3rd edn. Lippincott Williams & Wilkins

Sbihi H, Davies HW, Demers PA (2008) Hypertension in noise-exposed sawmill workers: a cohort study. Occup Environ Med 65:643–646

Selye H (1955) Stress and disease. Science 122(3171):625–631

Stokholm ZA, Bonde JP, Christensen KL, Hansen AM, Kolstad HA (2013a) Occupational noise exposure and the risk of hypertension. Epidemiology 24:135–142

Stokholm ZA, Bonde JP, Christensen KL, Hansen AM, Kolstad HA (2013b) Occupational noise exposure and the risk of stroke. Stroke 44:3214–3216

Stone NJ (1994) Secondary causes of hyperlipidemia. Med Clin North Am 78:117–141

Toivonen M, Pääkkönen R, Savolainen S, Lehtomäki K (2002) Noise attenuation and proper insertion of earplugs into ear canals. Ann Occup Hyg 46:527–530

Van Kempen EE, Kruize H, Boshuizen HC, Ameling CB, Staatsen BA, de Hollander AE (2002) The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspect 110:307–317

Vangelova KK, Deyanov CE (2007) Blood pressure and serum lipids in industrial workers under intense noise and a hot environment. Rev Environ Health 22:303–311

Virkkunen H, Kauppinen T, Tenkanen L (2005) Long-term effect of occupational noise on the risk of coronary heart disease. Scand J Work Environ Health 31:291–299

Virkkunen H, Harma M, Kauppinen T, Tenkanen L (2006) The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. Occup Environ Med 63:378–386

Virtanen SV, Notkola V (2002) Socioeconomic inequalities in cardiovascular mortality and the role of work: a register study of Finnish men. Int J Epidemiol 31:614–621

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ORIGINAL ARTICLE

Occupational noise exposure, psychosocial working conditions and the risk of tinnitus

Thomas Winther Frederiksen[1,2] · Cecilia Høst Ramlau-Hansen[3] · Zara Ann Stokholm[1] · Matias Brødsgaard Grynderup[5] · Åse Marie Hansen[4,5] · Søren Peter Lund[4] · Jesper Kristiansen[4] · Jesper Medom Vestergaard[1] · Jens Peter Bonde[6] · Henrik Albert Kolstad[1]

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Abstract
Purpose The purpose of this study was to evaluate the influence of occupational noise (current and cumulative doses) and psychosocial work factors (psychological demands and decision latitude) on tinnitus occurrence among workers, using objective and non-self-reported exposure measures to prevent reporting bias.
Methods In a cross-sectional study, we analyzed data from a Danish survey from 2009 to 2010 that included 534 workers from children day care units and 10 manufacturing trades. Associations between risk factors (current noise exposure, cumulative noise exposure and psychosocial working conditions) and tinnitus were analyzed with logistic regression.
Results We found no statistically significant associations between either current [OR 0.95 (95% CI 0.89; 1.01)] or cumulative [OR 0.93 (95% CI 0.81; 1.06)] occupational noise exposure and tinnitus. Likewise, results for psychosocial working conditions showed no statistically significant association between work place decision latitude [OR 1.06 (95% CI 0.94; 1.13)] or psychological demands [OR 1.07 (95% CI 0.90; 1.26)] and tinnitus.
Conclusions Our results suggest that current Danish occupational noise levels (in combination with relevant noise protection) are not associated with tinnitus. Also, results indicated that the psychosocial working conditions we observed in this cohort of mainly industrial workers were not associated with tinnitus. Therefore, psychosocial working conditions comparable to those observed in this study are probably not relevant to take into account in the evaluation of workers presenting with tinnitus.

Keywords Tinnitus · Noise · Psychological demands · Decision latitude · Psychosocial work factors

Introduction

Tinnitus is the perception of sound in the absence of an external sound. It represents a frequent disorder with a prevalence of around 10–15% depending on study population and criteria applied (Henry et al. 2005). Though tinnitus is a frequent complaint, only around 0.5% of the population have debilitating symptoms such as severe annoyance, concentration difficulty and insomnia (Baguley et al. 2013).

The etiology of tinnitus is heterogeneous and includes both somatic and psychological factors. Among somatic factors, hearing loss is probably the most important risk factor for tinnitus (Henry et al. 2005; Axelsson and Prasher 2000). Factors contributing to hearing loss therefore also represent potential risk factors for tinnitus including age, acute and long-term (occupational) noise exposure, middle- and inner ear diseases, and ototoxic medications (Baguley

✉ Thomas Winther Frederiksen
thofre@rm.dk

1 Department of Occupational Medicine, Danish Ramazzini Centre, Aarhus University Hospital, Nørrebrogade 44 Building 2C, 8000 Åarhus C, Denmark

2 Department of Otolaryngology/Head and Neck Surgery, Regionshospitalet Holstebro, 7500 Holstebro, Denmark

3 Department of Public Health, Section for Epidemiology, Aarhus University, 8000 Åarhus C, Denmark

4 National Research Centre for the Working Environment, 2100 Copenhagen Ø, Denmark

5 Department of Public Health, Section of Social Medicine, University of Copenhagen, 1014 Copenhagen K, Denmark

6 Department of Occupational and Environmental Medicine, Bispebjerg University Hospital, 2400 Copenhagen NV, Denmark

et al. 2013). However, many people without hearing loss also experience tinnitus and often no obvious cause is found in the individual patient (Meikle and Griest 1989).

In spite of stricter occupational noise legislation, millions of workers worldwide are still exposed to occupational noise levels that increase the risk of hearing loss and tinnitus (Axelsson and Prasher 2000; Verbeek et al. 2012). Prevention programs in developed countries that include noise assessment, noise controls, audiometric monitoring of workers' hearing, worker education and appropriate use of hearing protection have, however, resulted in a decreasing incidence of hearing loss in this part of the World (Nelson et al. 2005).

Psychological factors such as mental stress, psychosocial strain, anxiety and depression have been suggested to either cause tinnitus or exacerbate tinnitus symptoms (Oishi et al. 2011; Holgers et al. 2005; Salviati et al. 2014; Evered and Lawrenson 1981). As high job strain is associated with increased mental stress (Nieuwenhuijsen et al. 2010; de Jonge et al. 2000), the risk of tinnitus may be affected by psychosocial working conditions.

A Taiwanese study from 2008 (Lin et al. 2009) found a statistically significant association between feeling stressed at work and tinnitus. Correspondingly, a Swedish study from 2011 revealed a relationship between work-related stressors and hearing problems (tinnitus and hearing complaints) (Hasson et al. 2011), but otherwise epidemiologic evidence of a possible association between job-related stress factors and tinnitus is scarce.

Retrospective evaluation of noise exposure and psychosocial factors often causes problems in epidemiological studies, especially if evaluation relies on self-reported data. People suffering from tinnitus may search their memory more thoroughly for explanatory factors than unaffected individuals, potentially leading to differential misclassification of exposure levels and inflated results. This problem is potentially circumvented by using work unit-aggregated levels of psychosocial exposures (Kolstad et al. 2011), objective noise measurements and construction of noise exposure matrices (Seixas and Checkoway 1995; Davies et al. 2009).

The two main objectives of this study were: (1) to evaluate the association between objective measures of occupational noise (based on noise dosimetries) and tinnitus and (2) to evaluate the association between work unit-aggregated measures of psychosocial work factors (psychological demands and decision latitude) and tinnitus.

Materials and methods

Participants

This cross-sectional study takes advantage of an initial survey of 819 workers conducted between 2001 and 2002 in Aarhus, Denmark, with the purpose of monitoring occupational noise exposure and auditory function among noise-exposed workers. Participants were recruited from randomly selected companies within 12 trades: children day care, financial services and the 10 manufacturing trades in Denmark with the highest reporting of noise-induced hearing loss according to the Danish Working Environment Authority. Financial workers were selected as a reference group. In 2009–2010, the same companies and workers were asked to participate again. This time the purpose was extended to also include psychosocial work factors and medical risk factors. A total of 271 workers (33.1%) agreed to participate again, and further 394 workers were recruited de novo, making a total of 665 participants in 2009–2010. At the company level, all participants were as far as possible selected at random. However, to avoid disruption of workflow, selection in some cases had to be done in accordance with the local manager. Only participants from the 2009–2010 study were included in the present study, as psychosocial work factors were not accounted for in the 2001–2002 survey. To restrict the analyses to potentially noise-exposed workers, 67 financial workers and 64 workers from the original 2001 cohort now either unemployed ($n = 44$) or no longer working in noise-exposed industries ($n = 20$) were excluded, leaving 534 participants eligible for this study. The local ethical scientific committee (Central Region Denmark) approved the study (M.20080239), and informed consent was obtained from all individual participants included.

Occupational noise exposure

Individual dosimeters (Bruel & Kjær, model 4443, Nærum, Denmark) measuring A-weighted equivalent sound levels (L_{Aeq}) in 5-s intervals were handed out to the participants. Microphones were fitted at the right side collar if right handed and vice versa if left handed. Measuring range was set to 70–120 dB(A). Individual A-weighted equivalent noise levels were computed for the full work shift ($L_{Aeq,\,work}$).

Based on 1268 noise exposure recordings from the 2001–2002 study and the 2009–2010 study, we predicted noise exposure levels for each combination of trade, occupation and calendar year (1980–2010) by mixed regression analyses including these as fixed effects and the participants as random effect resulting in a noise exposure matrix. Based on information on historical employment status (1980–2010) retrieved from the Danish Supplementary Pension Fund and the noise exposure matrix, we calculated cumulative occupational noise exposure levels for each participant as the product of estimated noise exposure level [L_{Aeq} in dB(A)] and duration of employment (T) using the formula: $10 \times \log [\Sigma(10^{dB(A)/10} \times T]$, resulting in "dB(A)-year" on a logarithmic scale.

Audiometric measures

Air conduction thresholds were determined for each ear at 0.25, 0.5, 1, 2, 3, 4, 6 and 8 kHz by pure tone audiometry at the workplaces, using a Voyager 522 audiometer equipped with TDH-39 headphones (Madsen Electronics, Taastrup, Denmark). The audiometer was installed in a mobile examination unit equipped with a soundproof booth (model AB-4240, Eckel Noise Control Technologies, Bagshot, UK). Audiometry was performed by trained examiners using a standardized protocol.

To avoid temporary threshold shifts from possible noise sources, all participants were asked to wear hearing protection from the beginning of the day until the audiometry was done. Otoscopy was performed initially to verify that ears were free of wax and the tympanic membrane was visible. The audiometer was calibrated every 6 months according to ISO standards. We defined two hearing measures: 0.5–4.0 kHz hearing threshold (0.5–4.0 kHz HT) was computed as the average of pure tone hearing thresholds at 0.5, 1, 2, 3 and 4 kHz in the worse ear. 0.5–4.0 kHz hearing handicap was defined if 0.5–4.0 kHz HT >25 dBHL (according to WHO hearing impairment definition). Worse ear hearing ability was chosen over better ear, as we assumed that hearing levels at the worse ears were the most predictive of tinnitus status.

Questionnaire information

A questionnaire was handed out to the participants at the time of the audiometric examination to provide information on tinnitus and its related symptoms, psychosocial work factors, mental symptoms, use of hearing protection device, income and education (as described below).

Tinnitus

Tinnitus was defined in the questionnaire as "ringing or buzzing in one or both ears." Related questions included frequency of tinnitus [(1) almost never experiencing tinnitus, (2) experiencing periods of tinnitus at least monthly, (3) experiencing periods of tinnitus at least weekly or (4) experiencing tinnitus daily], frequency of annoyance when experiencing tinnitus [(1) almost never or never, (2) rarely, (3) sometimes, (4) often or (5) always] and frequency of insomnia due to tinnitus [(1) almost never or never, (2) rarely, (3) sometimes, (4) often or (5) always]. A person was classified as having tinnitus if experiencing tinnitus daily accompanied by either annoyance (sometimes, often or always) or insomnia (sometimes, often or always).

Measures of psychosocial working conditions

Psychosocial working conditions were measured according to Karasek and Theorell's demand-control model (Karasek 1990) with scales from the Copenhagen Psychosocial Questionnaire (Kristensen et al. 2005). Psychological demands, decision authority and skill discretion were each measured by four items on a scale from "always" (1) to "never" (5). For each scale, a mean value of the four items was calculated. Decision latitude was computed as the mean value of decision authority and skill discretion.

Furthermore, we calculated mean values of decision latitude and psychological demands for each work unit after exclusion of participants with tinnitus. Participants with tinnitus were excluded from the calculation of the mean scores as tinnitus distress could influence their assessment of the psychosocial work environment, thus introducing reporting bias. The mean values were then assigned to all employees at the particular work place. This method was recently used in a study of depression (Grynderup et al. 2012).

Measures of mental symptoms

Symptoms of depression, anxiety and somatoform disorder (illness worries) were assessed using the Common Mental Disorders Questionnaire (CMDQ) (Christensen et al. 2005). The CMDQ is a brief case finding instrument designed to screen for mental symptoms in general practice. All questions referred to the last 4 weeks and were measured on a 5-point response scale from "not at all" (0 points) to "extremely" (4 points). We used the six-question subscale for depression, the four-question subscale for anxiety and the seven-question subscale for somatoform disorder. Participants were classified as depressive if scoring $\geq$3 on $\geq$3 of the 6 depressive symptom questions. Anxiety was classified if the score was $\geq$3 on $\geq$3 of the 4 anxiety symptom questions, and somatoform disorder was classified if the score was $\geq$3 on $\geq$3 of the 7 somatoform disorder symptom questions. These selection criteria were chosen to obtain optimal validity (Christensen et al. 2005).

Use of hearing protection devices

Of the 534 workers, 333 reported to use HPD. Among HPD users, 140 participants completed a detailed log-book specifying when they used HPD during the day of noise measurements.

Income and education

Participants were asked about gross household income (<299,999 DDK, 300,000–499,999 DDK, >500,000 DDK) and educational level (none, short courses, skilled worker, short-range training, middle-range training, long-range training).

Statistics

We tabulated possible confounders and information on occupational background according to presence of tinnitus. Odds ratios of tinnitus according to noise exposures were analyzed by logistic regression and performed using both continuous-scale exposure information (if available) and exposure divided into relevant groups or tertiles. These analyses were adjusted for age and sex.

Associations between psychosocial working conditions and tinnitus were analyzed by logistic regression with robust clusters based on the work unit of the participants and adjusted for: (1) age and sex, and (2) age, sex, depression, anxiety, somatization disorder, income and education. These potential confounders were decided upon a priori. Analyses were performed using both continuous-scale exposure information and exposure divided into tertiles. We analyzed for interaction between psychological demands and decision latitude. The interaction term was calculated based on both continuous and trichotomized data.

To test whether associations were independent of auditory function, we performed additional analyses adjusting for mean hearing levels at worse hearing ear (mean of 0.5, 1, 2, 3 and 4 kHz HL). Another subanalysis was conducted to investigate whether the use of hearing protection devices (HPD) influenced the observed associations between current noise exposure and tinnitus. In this analysis, we subtracted 10 dB(A) from each 5-s noise recording obtained at work while using HPD. This analysis was restricted to the 342 workers with valid information on HPD use (140 workers returning the HPD log-book and 202 workers reporting not to use HPD at work).

All analyses were conducted using Stata 13 statistical software (StataCorp LP, College Station, TX, USA).

Results

A total of 41 (8%) participants were classified as suffering from tinnitus according to our criteria. Characteristics of participants according to tinnitus status are presented in Table 1. Of the 534 participants, 126 were women (23.6%). Age range was 20–64 years (mean 43.0 years). Among participants with tinnitus, we observed a tendency toward higher prevalence of males, workers above 45 years of age, workers with anxiety and somatization disorder and workers with hearing impairment compared with participants without tinnitus. Median speech frequency hearing thresholds (0.5–4 kHz) were on average 7.5 dB higher in the tinnitus group. The highest number of tinnitus cases was found among workers manufacturing fabricated metals.

For each 10 dB(A) increase in current occupational noise exposure level, we observed an age-and-gender-adjusted OR_{adj1} of 0.95 (95% CI 0.89; 1.01) for tinnitus, and the association seemed to decrease with higher noise levels (Table 2). Further adjustment for mental disorders, education and income did not change this result markedly. Results for cumulative occupational noise exposure showed no statistically significant association with tinnitus [OR_{adj1} 0.94 (95% CI 0.82; 1.07 for each dB(A)-year)]. Again, further adjustment for mental disorders, education and income did not change this result.

For psychosocial working conditions, we observed no statistically significant associations between either low decision latitude [OR_{adj1} 1.09 (95% CI 1.02; 1.16) for one unit increase on a 32-level scale] or psychological demands and tinnitus [OR_{adj1} of 1.04 (95% CI 0.91; 1.91) for one unit increase on a 16-level scale]. Results for decision latitude and psychological demands did not change noticeably when further adjusting for mental disorders, education and income.

We observed no interaction between psychological demands and decision latitude (all p values >0.05 for both continuous and trichotomized exposure variables).

Accounting for the use of HPD by subtracting 10 dB(A) from every 5-s noise recording obtained at work for the subgroup with valid HPD information did not change the OR for the association between current occupational noise and tinnitus [OR_{Adj2} after 10 dB(A) subtraction: 0.97 (95% CI 0.91; 1.05)].

Testing whether associations were independent of participant's hearing levels by further adjusting analyses for hearing levels at worse hearing ear, resulted in minimal changes in the association between current occupational noise exposure and tinnitus. Thus, the OR_{Adj2} changed from 0.95 (95% CI 0.89; 1.01) to 0.96 (95% CI 0.89; 1.05) for the association between continuous current noise exposure and tinnitus. Associations between cumulative noise exposure and psychosocial working conditions and tinnitus were practically unchanged.

Discussion

The objectively measured current and cumulative occupational noise levels observed in this study were not statistically significantly associated with tinnitus. Moreover, for psychosocial working conditions, we found no association with tinnitus.

In previous epidemiological studies on risk factors for tinnitus such as "The Blue Mountain Hearing Study" and "The Beaver Dam Offspring Study," rather strong associations between both cumulative and current occupational noise and tinnitus have been reported (Nondahl et al. 2011; Sindhusake et al. 2003). Both current and historical exposure assessment, however, relied on self-reported noise

Table 1 Characteristics of 534 noise-exposed workers aged 20–64 years, Åarhus, Denmark, 2009–2010

Characteristic	Tinnitus ($n = 41$)				No tinnitus ($n = 493$)			
	n	%	Median	p10; p90	*n*	%	Median	p10; p90
Sex, no (%)								
Female	6	(14.6)			120	(24.3)		
Male	35	(85.4)			373	(75.7)		
Age, no (%)								
<35 years	5	(12.2)			99	(20.1)		
35–44 years	9	(22.0)			174	(35.3)		
45–54 years	17	(41.5)			158	(32.1)		
≥55 years	10	(24.4)			62	(12.6)		
Education								
None	3	(7.3)			62	(12.6)		
Short courses	7	(17.1)			61	(12.4)		
Skilled worker	26	(63.4)			273	(55.4)		
Short-range training	1	(2.4)			22	(4.5)		
Middle-range training	3	(7.3)			72	(14.6)		
Long-range training	1	(2.4)			3	(0.6)		
Annual income								
0–299,999 DDK	18	(43.9)			232	(47.3)		
300,000–499,999 DDK	23	(56.1)			242	(49.4)		
>500,000 DDK	0	(0)			16	(3.3)		
Hearing thresholds at 0.5–4 kHz (dB HL),			20.0	6.3; 33.8			12.5	3.8; 28.8
Hearing impairment[a]								
No	24	(58.5)			424	(86.0)		
Yes	17	(41.5)			69	(14.0)		
Depression								
No	35	(85.4)			416	(84.4)		
Yes	6	(14.6)			77	(15.6)		
Anxiety								
No	35	(85.4)			445	(90.3)		
Yes	6	(14.6)			48	(9.7)		
Somatoform disorder								
No	33	(80.5)			416	(84.4)		
Yes	8	(19.5)			77	(15.6)		
Industry								
Manufacture of food	5	(12.2)			76	(15.4)		
Manufacture of wood products	4	(9.8)			41	(8.3)		
Publishing and printing	5	(12.2)			61	(12.4)		
Manufacture of non-metallic mineral products	2	(4.9)			35	(7.1)		
Manufacture of basic metals	3	(7.3)			37	(7.5)		
Manufacture of fabricated metals	7	(17.1)			58	(11.8)		
Manufacture of machinery	5	(12.2)			58	(11.8)		
Manufacture of motor vehicles	3	(7.3)			42	(8.5)		
Manufacture of furniture	1	(2.4)			5	(1.0)		
Construction	2	(4.9)			24	(4.9)		
Day care	4	(9.8)			56	(11.4)		

[a] WHO definition. See "Audiometric measures" section

Tabel 2 Odds ratios (OR) of having tinnitus according to occupational noise exposure and psychosocial working conditions

Exposure	Tinnitus ($n = 41$)	No tinnitus ($n = 493$)	OR_{Crude}	95% CI	OR^{a}_{Adj1}	95% CI	OR^{b}_{Adj2}	95% CI
Occupational noise exposure								
Current occupational noise ($L_{Aeq, work}$), dB(A)								
<80	17	142	1		1		1	
80–84	15	192	0.65	0.31; 1.35	0.67	0.44;1.33	0.67	0.32; 1.42
85–90	7	107	0.55	0.22; 1.36	0.52	0.53; 2.92	0.52	0.20; 1.33
>90	2	40	0.42	0.51; 2.71	0.46	0.10; 2.15	0.51	0.11; 2.41
Missing	0	12						
Continuous pr. 10 dB(A)			0.95	0.89; 1.01	0.95	0.89;1.01	0.95	0.89; 1.01
Cumulative occupational noise (dB(A)-years)								
Low (79.6–94.9)	11	167	1		1		1	
Medium (95.0–97.4)	12	166	1.10	0.47; 2.56	0.67	0.27;1.68	0.63	0.24; 1.61
High (97.5–101.1)	18	160	1.71	0.78; 3.73	0.63	0.23; 1.76	0.58	0.24; 1.67
Missing	0	0						
Continuous			1.08	0.96; 1.19	0.94	0.82; 1.07	0.93	0.81; 1.06
Psychosocial working conditions (exposure range[c])								
Psychological demands								
Low (0–3)	12	157	1		1		1	
Medium (4–6)	12	158	0.99	0.47; 2.07	1.08	0.50; 2.36	0.98	0.44; 2.20
High (7–16)	15	176	1.12	0.54; 2.07	1.18	0.57; 2.45	1.09	0.53; 2.22
Missing	2	2						
Continuous (0–16)			1.06	0.88; 1.28	1.09	0.90; 1.30	1.07	0.90; 1.26
Decision latitude								
High (20–32)	11	166	1		1		1	
Medium (15–19)	13	165	1.18	0.53; 2.64	1.13	0.51; 2.52	1.07	0.51; 2.21
Low (0–14)	15	160	1.41	0.65; 3.06	1.46	0.67; 3.16	1.37	0.67; 2.78
Missing	2	2						
Continuous (32-0)			1.05	0.97; 1.15	1.05	0.96; 1.15	1.06	0.94; 1.13

[a] Adjusted for age and gender

[b] Adjusted for age, gender, depression, anxiety, somatization disorder, education and income

[c] According to method described in "Measures of psychosocial working conditions" section

levels, and results could therefore potentially be biased. Moreover, these studies were conducted a decade or two before this study, and participants were generally older, meaning that both historical and current occupational noise exposure levels for participants in these studies were probably higher.

We analyzed objective measures of occupational noise exposure in relation to tinnitus and were not able to reproduce comparable risk estimates. As long-term exposure to high noise levels [>85 dB(A)] is generally accepted to cause hearing loss (International Organization for Standardization (ISO) 1990) which is a well-established risk factor for tinnitus, we found our negative results surprising. As described in "Occupational noise exposure" section, we calculated individual "dB(A)-years" as a sum-measure of the average daily occupational noise exposure through each year of employment back to 1980. Tabulating the number of years exposed to average daily occupational noise levels >85 dB(A) for each participant revealed that, with regard to the risk of inner ear damage, the retrospective noise exposure for our participants was generally low. Thus, 62.9% of the population had never been exposed to more than one year with average daily occupational noise exposure above 85 dB(A) and only 21.7% had been exposed for more than 5 years above this level. No participants had been exposed to a full year of average daily occupational noise exposure above 90 dB(A). If the causal pathway from noise to tinnitus is through hearing loss, the low historical noise exposure levels in this cohort may therefore partly explain our finding.

Potential selection bias from a healthy worker effect is another possible explanation for our results: As tinnitus is often accompanied by hearing loss and hypersensitivity to noise (Gilles et al. 2014; Nelson and Chen 2004b), this

Tabel 3 Reported use of hearing protection device according to current occupational noise exposure levels

	Using hearing protection device at work			
	No ($n = 202$)		Yes ($n = 332$)	
	n	%	*n*	%
Current occupational noise($L_{Aeq, work}$), dB(A)				
<85	166	45.4	200	54.6
85–90	27	23.7	87	76.3
>90	5	11.9	37	88.1
Missing	4	33.3	8	66.7

may exclude workers with low thresholds for developing tinnitus and hearing loss from noise-exposed employment. If this argument holds true, our noise-exposed population may represent a selection of workers with a high resistance to noise in terms of developing tinnitus and hearing loss. Indeed, this cross-sectional study may have been particularly vulnerable to this type of bias as it consisted not only of newly recruited participants but also of "survivors" from the original study group from 2001.

Non-differential misclassification of historical noise levels which is an inherent limitation of exposure matrices is another possible source of bias affecting our results for cumulative occupational noise exposure.

Concerning current noise exposure, only 114 (21%) and 42 (8%) of workers were exposed to current average occupational noise levels >85 and >90 dB(A), respectively (Table 2). Table 3 shows that in these two groups there were many HPD users (76 and 88%, respectively). Again, if noise-related tinnitus is the result of either temporary or permanent threshold shifts [neither of which should occur at noise levels <85 dB(A)], we would not expect to observe strong associations between the observed current noise levels in this study and tinnitus, especially if HPD use was as adequate, as indicated in Table 3.

Furthermore, we performed additional regression analyses to see whether current or cumulative noise levels were associated with participant's hearing levels. Indeed, no significant association was observed, which again supports that the cumulative and current noise exposure levels we observed were not large enough to cause tinnitus, through a pathway including hearing loss.

The causal pathway from noise exposure to tinnitus could also, potentially, be mediated through mental stress resulting from noise exposure as suggested in some studies (Ising and Kruppa 2004; van Dijk et al. 1987). In this case, noise should only cause annoyance and would not have to be at deleterious levels to also cause tinnitus. Our results, however, do not support this hypothesis either, at least at the given noise levels.

Based on noise recordings and questionnaire data from 752 workers from the original 2001–2002 cohort (see "Participants" section), Rubak et al. conducted a study published in 2008, analyzing the association between occupational noise exposure and tinnitus with and without concomitant hearing handicap (Rubak et al. 2008). In Rubak's study, current occupational noise levels were higher than we measured in 2009–2010, and cumulative occupational noise was calculated from partly self-reported levels. The authors found no association between occupational noise exposure and tinnitus without concomitant hearing handicap, but interestingly an increased risk of tinnitus was observed if hearing handicap was also present.

According to Karasek & Theorell's job strain model, mental strain is the result of the interaction of high psychological demands and low decision latitude (Karasek 1990). Traditionally, the combined effect of the two factors has therefore been analyzed as a quadrant term with median splits of psychological demands and decision latitude. In this study, we found no statistically significant interaction effects between psychological demands and decision latitude and therefore decided to report associations separately, as this method, in our opinion, would give us more detailed information on the effect of each component (Mikkelsen et al. 2011).

Due to distressing tinnitus symptoms possibly affecting the individual's perception and reporting of the work environment, the association between self-reported psychological working conditions and tinnitus may be affected by reporting bias. This is potentially circumvented using work unit-aggregated measures as we did in the present study. These measures are independent of a specific worker's appraisal of his or her working conditions and thus provide a more objective description of the working environment (Kolstad et al. 2011; Kasl 1998).

Making use of the above-mentioned method in the analysis of our data for psychological working conditions, we found no statistically significant associations with tinnitus of either psychological demands or decision latitude. Prior studies have studied the association between self-reported (not job-related) mental stress and tinnitus; the majority finding positive associations (Canlon et al. 2013) (Heinecke et al. 2008; Horner 2003). We were able to find two studies evaluating the effect of job-related stress factors on tinnitus (Lin et al. 2009; Hasson et al. 2011) (both reporting positive associations with self-reported occupational stress factors), but no studies analyzing the association between non-self-reported psychosocial working conditions and tinnitus as performed in the present study. This study therefore offers a new perspective on this issue and indicates that current psychosocial working conditions in Danish industrial trades are not associated with tinnitus.

In our main analyses, we did not adjust for hearing level as it was our assumption that it was in the causal pathway from noise to tinnitus. Hearing disabilities can, however, cause mental distress and may also cause participants to avoid noise exposure (Nelson and Chen 2004a). In that regard, hearing ability could be a potential confounder. We therefore performed a sensitivity check by further adjusting for hearing levels. This resulted in practically unchanged results.

We also subtracted 10 dB(A) from every 5-s noise recording obtained during work while using HPD for those providing a log-book (Park and Casali 1991). This was done to investigate the potential effect of noise attenuation from HPD's on our results for current noise exposure. This did not alter results substantially.

This study has a number of strengths. Firstly, we used objective and non-self-reported exposure measures with little missing information, leaving little room for reporting biased results. Also, we had detailed information on potential confounders enabling us to perform analyses adjusted for other potential risk factors.

Among limitations is the cross-sectional nature of this study, preventing us from drawing strong conclusions regarding causality. Also, our definition of tinnitus may have led to misclassification of tinnitus status. Using a standardized tinnitus questionnaire as the Tinnitus Handicap Inventory (Zeman et al. 2012) possibly could have refined our tinnitus classification, but due to pressure of space in the questionnaire, we refrained from this. Regarding noise exposure, higher exposure levels (current and cumulative) and contrast would have enabled us to also explore the association between noise and tinnitus status at higher levels than given in this study. As occupational noise levels in other countries worldwide are possibly higher than what we measured, this limits the external validity of our results.

Conclusion

Overall, our results suggest that occupational noise exposure at the levels given in this study is not associated with tinnitus. However, our results do not rule out a possible increased risk of tinnitus from occupational noise levels exceeding the levels measured in this population.

Likewise, we found no indication of an association between psychosocial working conditions (in terms of high psychological demands and low decision latitude) and tinnitus, suggesting that psychosocial working factors comparable to those observed in this study are probably not relevant in the evaluation of a worker presenting with tinnitus.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflicts of interest.

References

Axelsson A, Prasher D (2000) Tinnitus induced by occupational and leisure noise. Noise Health 2:47–54

Baguley D, McFerran D, Hall D (2013) Tinnitus. Lancet. doi:10.1016/S0140-6736(13)60142-7

Canlon B, Theorell T, Hasson D (2013) Associations between stress and hearing problems in humans. Hear Res 295:9–15. doi:10.1016/j.heares.2012.08.015

Christensen KS, Fink P, Toft T, Frostholm L, Ornbol E, Olesen F (2005) A brief case-finding questionnaire for common mental disorders: the CMDQ. Fam Pract 22:448–457. doi:10.1093/fampra/cmi025

Davies HW, Teschke K, Kennedy SM, Hodgson MR, Demers PA (2009) A retrospective assessment of occupational noise exposures for a longitudinal epidemiological study. Occup Environ Med 66:388–394. doi:10.1136/oem.2008.040881

de Jonge J, Bosma H, Peter R, Siegrist J (2000) Job strain, effort-reward imbalance and employee well-being: a large-scale cross-sectional study. Soc Sci Med 50:1317–1327. doi:10.1016/S0277-9536(99)00388-3

Evered D, Lawrenson G (eds) (1981) Tinnitus. Ciba foundation symposium 85. Pitman, London, pp 193–198

Gilles A, Goelen S, Van de Heyning P (2014) Tinnitus: a cross-sectional study on the audiologic characteristics. Otol Neurotol 35:401–406. doi:10.1097/MAO.0000000000000248

Grynderup MB, Mors O, Hansen AM, Andersen JH, Bonde JP, Kaergaard A, Kaerlev L, Mikkelsen S, Rugulies R, Thomsen JF, Kolstad HA (2012) A two-year follow-up study of risk of depression according to work-unit measures of psychological demands and decision latitude. Scand J Work Environ Health 38:527–536. doi:10.5271/sjweh.3316

Hasson D, Theorell T, Wallen MB, Leineweber C, Canlon B (2011) Stress and prevalence of hearing problems in the Swedish working population. BMC Public Health 11:130. doi:10.1186/1471-2458-11-130

Heinecke K, Weise C, Schwarz K, Rief W (2008) Physiological and psychological stress reactivity in chronic tinnitus. J Behav Med 31:179–188. doi:10.1007/s10865-007-9145-0

Henry JA, Dennis KC, Schechter MA (2005) General review of tinnitus: prevalence, mechanisms, effects, and management. J Speech Lang Hear Res 48:1204–1235. doi:10.1044/1092-4388(2005/084)

Holgers KM, Zoger S, Svedlund K (2005) Predictive factors for development of severe tinnitus suffering-further characterisation. Int J Audiol 44:584–592

Horner KC (2003) The emotional ear in stress. Neurosci Biobehav Rev 27:437–446

International Organization for Standardization (ISO) (1990) ISO 1999–1990

Ising H, Kruppa B (2004) Health effects caused by noise: evidence in the literature from the past 25 years. Noise Health 6:5–13

Karasek RT, Theorell T (1990) Healthy work: stress, productivity and the reconstruction of working life. Basic Books, New York

Kasl SV (1998) Measuring job stressors and studying the health impact of the work environment: an epidemiologic commentary. J Occup Health Psychol 3:390–401

Kolstad HA, Hansen AM, Kaergaard A, Thomsen JF, Kaerlev L, Mikkelsen S, Grynderup MB, Mors O, Rugulies R, Kristensen AS, Andersen JH, Bonde JP (2011) Job strain and the risk of depression: Is reporting biased? Am J Epidemiol 173:94–102. doi:10.1093/aje/kwq318

Kristensen TS, Hannerz H, Hogh A, Borg V (2005) The Copenhagen Psychosocial Questionnaire—a tool for the assessment and improvement of the psychosocial work environment. Scand J Work Environ Health 31:438–449

Lin YH, Chen CY, Lu SY (2009) Physical discomfort and psychosocial job stress among male and female operators at telecommunication call centers in Taiwan. Appl Ergon 40:561–568. doi:10.1016/j.apergo.2008.02.024

Meikle M, Griest S (1989) Gender-based differences in characteristics of tinnitus. Hear J 42:68–76

Mikkelsen S, Bonde JP, Andersen JH (2011) Analysis of job strain effects. Occup Environ Med 68:786. doi:10.1136/oemed-2011-100203

Nelson JJ, Chen K (2004) The relationship of tinnitus, hyperacusis, and hearing loss. Ear Nose Throat J 83:472–476

Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M (2005) The global burden of occupational noise-induced hearing loss. Am J Ind Med 48:446–458. doi:10.1002/ajim.20223

Nieuwenhuijsen K, Bruinvels D, Frings-Dresen M (2010) Psychosocial work environment and stress-related disorders, a systematic review. Occup Med (Lond) 60:277–286. doi:10.1093/occmed/kqq081

Nondahl DM, Cruickshanks KJ, Huang GH, Klein BE, Klein R, Javier Nieto F, Tweed TS (2011) Tinnitus and its risk factors in the Beaver Dam offspring study. Int J Audiol 50:313–320. doi:10.3109/14992027.2010.551220

Oishi N, Shinden S, Kanzaki S, Saito H, Inoue Y, Ogawa K (2011) Influence of depressive symptoms, state anxiety, and pure-tone thresholds on the tinnitus handicap inventory in Japan. Int J Audiol 50:491–495. doi:10.3109/14992027.2011.560904

Park MY, Casali JG (1991) A controlled investigation of in-field attenuation performance of selected insert, earmuff, and canal cap hearing protectors. Hum Factors 33:693–714

Rubak T, Kock S, Koefoed-Nielsen B, Lund SP, Bonde JP, Kolstad HA (2008) The risk of tinnitus following occupational noise exposure in workers with hearing loss or normal hearing. Int J Audiol 47:109–114. doi:10.1080/14992020701581430

Salviati M, Bersani FS, Terlizzi S, Melcore C, Panico R, Romano GF, Valeriani G, Macri F, Altissimi G, Mazzei F, Testugini V, Latini L, Delle Chiaie R, Biondi M, Cianfrone G (2014) Tinnitus: clinical experience of the psychosomatic connection. Neuropsychiatr Dis Treat 10:267–275. doi:10.2147/NDT.S49425

Seixas NS, Checkoway H (1995) Exposure assessment in industry specific retrospective occupational epidemiology studies. Occup Environ Med 52:625–633

Sindhusake D, Golding M, Newall P, Rubin G, Jakobsen K, Mitchell P (2003) Risk factors for tinnitus in a population of older adults: the blue mountains hearing study. Ear Hear 24:501–507. doi:10.1097/01.AUD.0000100204.08771.3D

van Dijk FJ, Souman AM, de Vries FF (1987) Non-auditory effects of noise in industry. VI. A final field study in industry. Int Arch Occup Environ Health 59:133–145

Verbeek JH, Kateman E, Morata TC, Dreschler WA, Mischke C (2012) Interventions to prevent occupational noise-induced hearing loss. Cochrane Database Syst Rev 10:CD006396. doi:10.1002/14651858.CD006396.pub3

Zeman F, Koller M, Schecklmann M, Langguth B, Landgrebe M, TRI database study group (2012) Tinnitus assessment by means of standardized self-report questionnaires: psychometric properties of the Tinnitus Questionnaire (TQ), the Tinnitus Handicap Inventory (THI), and their short versions in an international and multi-lingual sample. Health Qual Life Outcomes 10:128. doi:10.1186/1477-7525-10-128

Original Article

Noise-Induced Hearing Loss – A Preventable Disease? Results of a 10-Year Longitudinal Study of Workers Exposed to Occupational Noise

Thomas W. Frederiksen[1,2], Cecilia H. Ramlau-Hansen[3], Zara A. Stokholm[1], Matias B. Grynderup[1], Åse M. Hansen[4,5], Jesper Kristiansen[4], Jesper M. Vestergaard[1], Jens P. Bonde[6], Henrik A. Kolstad[1]

[1]Department of Occupational Medicine, Danish Ramazzini Centre, Aarhus University Hospital, Aarhus C, [2]Department of Otolaryngology/Head and Neck Surgery, Regionshospitalet Holstebro, Holstebro, [3]Department of Public Health, Section for Epidemiology, Aarhus University, Aarhus C, [4]National Research Centre for the Working Environment, Copenhagen Ø, [5]Department of Public Health, University of Copenhagen, Copenhagen K, [6]Department of Occupational and Environmental Medicine, Bispebjerg University Hospital, Copenhagen NV, Denmark

Abstract

Aims: To survey current, Danish industrial noise levels and the use of hearing protection devices (HPD) over a 10-year period and to characterise the association between occupational noise and hearing threshold shift in the same period. Furthermore, the risk of hearing loss among the baseline and the follow-up populations according to first year at occupational noise exposure is evaluated. **Materials and Methods:** In 2001–2003, we conducted a baseline survey of noise- and hearing-related disorders in 11 industries with suspected high noise levels. In 2009–2010, we were able to follow up on 271 out of the 554 baseline workers (49%). Mean noise levels per industry and self-reported HPD use are described at baseline and follow-up. The association between cumulative occupational noise exposure and hearing threshold shift over the 10-year period was assessed using linear regression, and the risk of hearing loss according to year of first occupational noise exposure was evaluated with logistic regression. **Results:** Over the 10-year period, mean noise levels declined from 83.9 dB(A) to 82.8 dB(A), and for workers exposed >85 dB(A), the use of HPD increased from 70.1 to 76.1%. We found a weak, statistically insignificant, inverse association between higher ambient cumulative noise exposure and poorer hearing (−0.10 dB hearing threshold shift per dB-year (95% confidence interval (CI): −0.36; 0.16)). The risk of hearing loss seemed to increase with earlier first year of noise exposure, but odds ratios were only statistically significant among baseline participants with first exposure before the 1980s (odds ratio: 1.90, 95% CI: 1.11; 3.22). **Conclusions:** We observed declining industrial noise levels, increased use of HPD and no significant impact on hearing thresholds from current ambient industrial noise levels, which indicated a successful implementation of Danish hearing conservation programs.

Keywords: Hearing conservation, noise exposure assessment, noise-induced hearing loss, noise surveillance, occupational noise exposure

Introduction

Occupational noise exposure is recognised as a substantial risk factor for hearing loss, and worldwide, it remains the most frequent cause of preventable sensorineural hearing loss.[1,2] This has led to an extensive research into the auditive effects of occupational noise, and in consequence, preventive measures have been implemented. These include engineering solutions minimising noise emission and reflection, and legislations limiting the time of work-related noise exposure and obliging the use of hearing protection devices (HPD).[3-5] This means that industrial noise levels and individual occupational noise exposure have potentially changed over the last few decades, at least in the developed countries. There are, therefore, good reasons to continue assessing the burden of auditive disease from occupational noise at national or sub-national levels to follow up on the

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Address for correspondence: Dr. Thomas Winther Frederiksen, MD, Department of Occupational Medicine, Aarhus University Hospital, Nørrebrogade 44, Building 2C, 8000 Aarhus C, Denmark. E-mail: thofre@rm.dk

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possible effect of preventive initiatives. A recent systematic review on occupational noise exposure and hearing concluded that hearing loss due to workplace noise was a significant problem in the 1960s and 1970s in industrialised countries, but the impact seemed to have decreased since that period.[6] This was suggested to be due to preventive measures, improved regulation or decreased noise exposure. The evidence, however, was still limited mainly due to blunt or incomplete exposure data. Hearing data were concluded to be generally good. Results from other recent studies, also seem to differ between industries, and these studies are often based on one specific profession, limiting generalisation of results.[7-9]

On the basis of the cross-sectional data collected in 2001–2003, we found a three-fold increased risk of hearing handicap among the workers with first exposure to occupational noise before the 1980s.[10] However, the workers starting their work in a noisy environment during later years showed no increased risk. We interpreted these findings as the result of successful preventive programmes enforced during 1980–1990. To follow up on these results, we conducted an equivalent survey in 2009–2010.

The main objectives of this study were to describe the trends in industrial noise exposure levels and use of HPD over a 10-year period. Furthermore, we aimed to evaluate the association between current, Danish industrial noise levels and hearing threshold shift in the same period and analyse whether year of first occupational noise exposure was associated with hearing loss.

Materials and Methods

Participants

This study has taken advantage of an initial survey of 819 workers conducted between 2001 and 2003 in Aarhus, Denmark, with the purpose of monitoring occupational noise exposure, auditory function and preventive measures (use of hearing HPD) among noise-exposed workers. Participants were recruited from randomly selected companies within 12 trades: children day care (due to reports indicating high noise levels in these units), financial services (expected to have low-level noise exposure) and the 10 manufacturing trades in Denmark with the highest reporting of noise-induced hearing loss according to the Danish Working Environment Authority. In 2009–2010, the same companies and workers were asked to participate again. We were able to re-identify 756 participants. Owing to time and economic restraints, 202 participants (27%) were not contacted (at random) leaving 554 eligible for follow-up. A total of 271 workers (49%) responded and agreed to participate again. At follow-up, 394 workers within the 12 trades were recruited *de novo* to include new workers first to have been noise-exposed during later years, making a total of 665 participants in the follow-up cohort.

For cross-sectional analysis of the baseline population, we excluded 76 workers with incomplete questionnaire exposure information or no noise dosimetry, 16 workers with incomplete audiometry, 109 white-collar workers (typically managers and office workers considered to differ considerably from the remaining population with respect to extraneous predictors of hearing loss), 65 workers reporting current or prior chronic middle-ear infection or tympanic membrane perforation (possible conductive hearing loss) and finally 14 workers with asymmetrical hearing loss (possible hearing loss from other causes than noise, as defined in section 'Audiometric measures'), resulting in 539 eligible workers for baseline cross-sectional analysis.

Correspondingly, for cross-sectional analyses on the follow-up population, we excluded 38 workers with incomplete questionnaire exposure information or no noise-dosimetry, 98 white-collar workers, 75 workers reporting current or prior chronic middle-ear infection or tympanic membrane perforation and 30 workers with asymmetrical hearing loss, resulting in 424 eligible workers.

For the longitudinal analyses, we focused on the 271 workers participating in both surveys. Of these, 262 had complete audiometries from both surveys. We excluded two workers with incomplete questionnaire exposure information, 48 white-collar workers and the workers reporting either chronic middle ear infection ($n=2$), tympanic membrane perforation ($n=2$), scull fracture ($n=0$) concussion ($n=1$), meningitis ($n=0$) or Meniere's disease ($n=0$) in the follow-up period, resulting in a final study population of 207 persons.

The local ethical scientific committee approved the study (M.20080239). All the participants gave written, informed consent to participate.

Audiometric measures

Air-conduction thresholds were determined for each ear at 0.25, 0.5, 1, 2, 3, 4, 6 and 8 kHz by pure-tone audiometry at the workplaces, using a Voyager 522 audiometer equipped with TDH-39 headphones (Madsen Electronics, Taastrup, Denmark). The audiometer was installed in a mobile examination unit equipped with a soundproof booth (model AB-4240, Eckel Noise Control Technologies, Bagshot, UK). Audiometry was performed by trained examiners using a standardised protocol (according to ISO 8253-1:2010).

To avoid the temporary threshold shifts (TTS) from possible noise sources, all participants were asked to wear HPD from the beginning of the workday until the audiometry was performed. The majority of the workers were daytime workers (around 90% in both surveys), and we expected a limited noise exposure prior to starting the work (mostly night time noise at home) and thus limited risk of TTS. Otoscopy verified that ears were free of wax, and the tympanic membrane was visible. The audiometer was calibrated every six months according to ISO

389-1:1998. On the basis of pure-tone air-conduction thresholds, we calculated an average binaural hearing threshold level for the critically noise-sensitive frequencies at baseline and follow-up (3–6 kHz-HTL-BL or 3–6 kHz-HTL-FU). Correspondingly, a baseline hearing loss variable and a follow-up hearing loss variable (3–6 kHz-HL-BL and 3–6 kHz-HL-FU) were defined, if 3–6 kHz-HTL-BL or 3–6 kHz-HTL-FU was above 20 dB. Threshold shift from baseline to follow-up (Δ3–6 kHz-HTL) was calculated by subtracting the baseline hearing thresholds (3–6 kHz-HTL-BL) from the follow-up hearing thresholds (3–6 kHz-HTL-FU). Thus, the worsened hearing was reflected by a positive threshold shift. We regarded an inter-aural difference of 20 dBHL or more in two consecutive frequencies from 3 to 6 kHz as an asymmetrical hearing loss.

Questionnaire information

All participants filled in a questionnaire providing information on medical and professional history. For the purpose of this study, information on age, sex, professional history (current and prior employment, duration, industry, occupation (blue vs. white collar)), use of HPD and the workers judgement (whether noise levels in prior jobs were higher, comparable or lower) was retrieved.

Occupational noise exposure assessment

At baseline and follow-up, individual dosimeters (Bruel & Kjr, model 4443, Naerum, Denmark) measuring A-weighted equivalent sound levels (L_{Aeq}) in 5-second intervals during the full work shift were handed out to the participants. Microphones were fitted at the right side collar if they were right-handers and vice versa if left-handers. Measuring range was set at 70–120 dB(A). Individual A-weighted equivalent noise levels were computed for the full work shift ($L_{Aeq,work}$).

Subsequently, workplace and trade-specific mean noise levels were calculated based on the individual dosimetries. As noise levels were expected to vary more from day to day for the individual worker than between the different workers,[11,12] we estimated the most efficient grouping strategy based on the highest contrast in mean exposure level between the groups. This was accomplished by modelling the noise exposure with two mixed effect models including either worker and the industry or worker and company as random effects. The highest contrast was found using company-means, and thus worker's noise exposure was classified by the mean L_{Aeq}-value calculated for their workplace and not by her/his individual measurement.

The estimation of cumulative occupational noise exposure in the follow-up period was based on (1) the questionnaire information on current and previous employment details including company, period, and the workers' subjective judgement of whether any previous jobs involved comparable or higher noise exposure levels than their current job, and (2) workplace average L_{Aeq} levels at baseline and follow-up. Each individual employment year was given a noise exposure level based on the following criteria: (1) if the year was within an employment period in a company included in the study, the average workplace level was applied (2) for employment periods in companies not included in the study, the noise exposure was classified from the participants' judgement of the noise levels,that is, (a) if the worker reported that the noise levels in a prior job were comparable to or higher than the level of the current job (were noise measurements was performed), these years were given the same level as in the current workplace or (b) if the noise level was judged to be substantially lower than that of the exposure at the current company, then this employment period would be classified as non-exposed.

Finally, we calculated cumulative occupational noise exposure levels for each participant in the follow-up period as the product of estimated noise exposure level (L_{Aeq} in dB(A)) and the duration of employment (T) using the formula: $10 \times \log [(10^{dB(A)/10} \times T]$, resulting in 'dB(A)-year' on a logarithmic scale.

The same model was used to estimate the first year of occupational noise exposure >80 dB(A) and the number of years exposed to the mean occupational noise levels >80 dB (A) and >85 dB(A), respectively.

Statistics

We tabulated sex, age and industry across decade of first year of an occupational noise exposure above 80 dB(A) for the baseline and follow-up populations [Table 1]. For the workers who participated in both surveys, we tabulated sex, age, 3–6 kHz-HL-BL, occupational noise exposure before baseline and HPD use across three categories of cumulative occupational noise in the follow-up period [Table 2].

Logistic regression was used to estimate the association between first year of occupational noise exposure >80 dB (A) and hearing loss in the critically noise-sensitive frequencies for the baseline and the follow-up populations, adjusting for age and sex [Table 3].

Among the workers participating in both the surveys, the crude and the adjusted associations between noise exposure variables and hearing threshold shift in the follow-up period were examined, using the linear regression with the lowest exposure group as a reference [Table 4]. Outcome variables as well as residuals were assessed and found normally distributed. Stratified analyses were performed to evaluate the possible effect modification from a prior occupational noise exposure and baseline hearing loss on the association between cumulative noise exposure and hearing threshold shift in the follow-up period. A Wald

Table 1: Characteristics of 539 workers from the baseline population and 424 workers from the follow-up population by year of first occupational noise exposure >80 dB(A), Aarhus, Denmark

	Baseline population						Follow-up population							
	Year of first noise exposure						Year of first noise exposure							
	1990–1999		1980–1989		<1980		2000–2010		1990–1999		1980–1989		<1980	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Sex, no. (%)														
Women	52	19.5	22	15.1	14	11.1	29	29.9	38	25.7	20	19.2	10	13.3
Men	215	80.5	124	84.9	112	88.9	68	70.1	110	74.3	84	80.8	65	86.7
Age (years), mean (SD)	267	38.1 (9.4)	146	36.9 (7.0)	126	47.1 (7.0)	97	34.6 (10.0)	148	42.6 (9.2)	104	45.0 (6.1)	75	54.8 (5.5)
Industry (NACE-codes)														
Manufacture of food (15)	33	12.4	19	13.1	18	14.3	20	20.6	16	10.8	14	13.5	5	6.7
Manufacture of wood products (20)	38	14.2	13	9.0	16	12.7	9	9.3	12	8.1	8	7.8	6	8.0
Publishing and printing (22)	33	12.4	25	17.2	16	12.7	8	8.3	17	11.5	9	8.7	7	9.3
Manufacture of non-metallic mineral prod. (26)	25	9.4	12	8.3	15	11.9	7	7.2	7	4.7	5	4.8	7	9.3
Manufacture of basic metals (27)	16	6.0	8	5.5	8	6.4	4	4.1	12	8.1	12	11.5	6	8.0
Manufacture of fabricated metals (28)	34	12.7	21	14.5	17	13.5	11	11.3	24	16.2	13	12.5	8	10.7
Manufacture of machinery (29)	25	9.4	16	11.0	10	7.9	11	11.3	11	7.4	12	11.5	11	14.7
Manufacture of motor vehicles (34)	25	9.4	16	11.0	13	10.3	8	8.3	12	8.1	8	7.7	6	8.0
Manufacture of furniture (36)	7	2.6	6	4.1	4	3.2	1	1.0	2	1.4	2	1.9	0	0.0
Construction (45)	14	5.2	7	4.8	5	4.0	2	2.1	7	4.7	5	4.8	2	2.7
Day care (85)	17	6.4	2	1.4	4	3.2	16	16.5	16	10.8	8	7.7	4	5.3
Other industries	–	–	–	–	–	–	0	0.0	5	3.4	6	5.8	4	5.3
Retired or unemployed	–	–	–	–	–	–	0	0.0	7	4.7	2	1.9	9	12.0

Table 2: Characteristics of the 207 workers participating at both baseline and follow-up by tertiles of cumulative occupational noise exposure (dB(A)-years) in the follow-up period, Aarhus, Denmark, 2009

	Cumulative occupational noise exposure (dB(A)-years)					
	67.7–91.8		91.9– 94.6		94.7–107.0	
	n	%	*n*	%	*n*	%
Sex, no. (%)						
Women	21	45.7	11	13.9	13	15.9
Men	25	54.4	68	86.1	69	84.2
3–6 kHz-HL-BL*						
No	33	71.7	51	64.6	56	68.3
Yes	13	28.3	28	35.4	26	31.7
Duration of daily occupational noise exposure > 80 dB(A) before baseline						
<10 years	24	52.2	28	35.4	44	53.7
≥10 years	22	47.8	51	64.6	38	46.4
Reporting daily use of HPD at baseline						
Yes	21	47.7	46	60.5	56	71.8
No	23	52.3	30	39.5	22	28.2
Reporting daily use of HPD at follow-up						
Yes	22	47.8	47	59.5	55	67.1
No	24	52.2	32	40.5	27	32.9
Age in 2009 (years), mean (SD)	46	50.9 (8.2)	79	48.6 (8.7)	82	46.0 (8.4)

*Defined as an average binaural hearing threshold > 20 dB in the noise sensitive frequencies (3, 4 and 6 kHz).

Table 3: Age and sex adjusted odds ratios (OR) of hearing handicap in the critically noise sensitive frequencies* according to year of first noise exposure among the baseline and follow-up populations

Year of first noise exposure > 80 dB	Subjects	Cases	OR	95% CI
Baseline population				
1990–1999	265	70	Reference	
1980–1989	148	32	1.02	0.59; 1.77
<1980	126	79	1.90	1.11; 3.22
Continuous pr. year	539	181	1.02	1.00; 1.04
Follow-up population				
2000–2010	97	30	Reference	
1990–1999	147	69	1.04	0.55; 1.95
1980–1989	105	62	1.30	0.66; 2.57
<1980	75	61	1.48	0.58; 3.77
Continuous pr. year	424	222	1.00	0.98; 1.04

*Defined as an average binaural hearing threshold > 20 dB in the noise sensitive frequencies (3, 4 and 6 kHZ)

test was performed to test the hypothesis of no effect modification.

HPD use at baseline and follow-up was cross-tabulated with age and gender to identify possible changes in use over the follow-up period [Table 5]. To look for changes in noise emission from the industries included in this study, we calculated mean industry noise levels based on all individual blue-collar noise recordings at baseline and follow-up [Table 6].

In a sub-analysis, we subtracted 10 dB(A) from company noise levels if workers reported to use HPD, and we repeated the analyses between the cumulative noise exposure variable and hearing threshold shift in the follow-up period as described above.

The STATA statistical package (version 13, StataCorp, College Station, TX, USA) was used for all analyses.

Results

As shown in Table 1, the women-to-man ratio was lower with earlier first noise exposure in baseline and follow-up populations. In addition, mean age was higher with earlier first noise exposure in both the populations.

Among the 207 workers participating in both surveys, we observed a tendency towards a higher prevalence of males among the workers exposed to higher cumulative noise levels and more frequent use of HPD, but no difference in the prevalence between baseline and follow-up [Table 2]. Conversely, the mean age seemed

Table 4: Crude and adjusted associations between noise exposure variables and bilateral hearing threshold shift in the critically noise sensitive frequencies (3–6 kHz) among 207 workers followed from baseline to follow-up

	n	Crude	Adjusted[1]	Adjusted[2]
		Δ3–6 kHz-HTL-BI	Δ3–6 kHz-HTL-BI	Δ3–6 kHz-HTL-BI
Cumulative occupational noise exposure, dB (A)-years				
Low (76.6–91.3)	46	Reference	Reference	Reference
Medium (91.4–94.8)	79	−1.14 (−3.79; 1.52)	−1.34 (−4.04; 1.35)	−1.44 (−4.15; 1.27)
High (94.9–107.0)	82	−0.88 (−3.51; 1.76)	−0.51 (−3.29; 2.20)	−0.70 (−4.15; 2.01)
Continuous		−0.13 (−0.39; 0.13)	−0.09 (−0.35; 0.17)	−0.10 (−0.36; 0.16)
Baseline occupational noise exposure (L_{Aeq})				
80–85 dB (A)	99	Reference	Reference	Reference
>85 dB (A)	106	1.08 (−0.92; 3.08)	0.77 (−1.20; 2.74)	0.56 (−1.41; 2.54)
Continuous (80.2–92,8)		0.01 (−0.32; 0.33)	0.00 (−0.32; 0.32)	−0.01 (−0.33; 0.31)
Years exposed >80 dB (A) from baseline to follow-up				
0–5	43	Reference	Reference	Reference
6–10	166	−0.42 (−2.76; 1.91)	−0.14 (−2.53; 2.26)	−0.24 (−2.64; 2.15)
Continuous (0–10)		−0.25 (−0.68; 0.17)	−0.06 (−0.50; 0.37)	−0.09 (−0.52; 0.34)
Years exposed >85 dB (A) from baseline to follow-up				
0–5	133	Reference	Reference	Reference
6–10	76	0.75 (−139; 2.89)	0.64 (−1.41; 2.68)	0.65 (−1.41; 2.70)
Continuous (0–10)		0.07 (−0.21; 0.35)	0.08 (−0.20; 0.36)	0.06 (−0.22; 0.34)

[1]Adjusted for sex and age. [2]Adjusted for sex, age, baseline hearing threshold and prior noise exposure >10 years

Table 5: HPD use at baseline and follow-up according to occupational noise level, sex and age group

	HPD use among baseline participants (*n*=539)								HPD use among follow-up participants (*n*=424)							
	<85 dB (A)				≥85 dB (A)				<85 dB (A)				≥85 dB (A)			
	Yes		No		Yes		No		Yes		No		Yes		No	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%	*n*	%
Sex, no (%)																
Female	22	37.3	37	62.7	12	44.4	15	55.6	15	20.0	60	80.0	9	56.3	7	43.8
Male	98	50.3	97	49.7	171	73.1	63	26.9	119	71.7	47	28.3	109	78.4	30	21.6
Age, no (%)																
<40	66	52.8	59	47.2	101	71.6	40	28.4	46	56.8	35	43.2	48	71.6	19	28.4
40–50	43	47.8	47	52.2	45	63.4	26	36.6	53	55.8	42	44.2	33	80.5	8	19.5
>50	11	28.2	28	71.8	37	75.5	12	24.5	35	53.9	30	46.2	37	78.7	10	21.3
All	120	47.2	134	52.8	183	70.1	78	29.9	134	55.6	107	44.4	118	76.1	37	23.9

Table 6: Mean noise levels per industry at baseline and follow-up, Aarhus, Demnark

	No. of noise measurements	Mean noise level at baseline ($L_{Aeq,work}$), min, max (dB(A))	No. of noise measurements	Mean noise level at follow-up ($L_{Aeq,work}$), min, max (dB(A))	Difference (dB(A))
Industry (NACE code)					
Manufacture of food (15)	79	84.7 (74.0–99.1)	58	84.5 (76.6–91.6)	−0.2
Manufacture of wood products (20)	72	85.3 (76.5–96.3)	40	84.9 (72.8–96.2)	−0.4
Publishing and printing (22)	87	81.9 (64.7–90.7)	53	81.7 (67.8–89.4)	−0.2
Manufacture of non-metallic prod. (26)	64	85.2 (74.8–97.2)	40	84.0 (75.4–106.0)	−1.2
Manufacture of basic metals (27)	44	85.6 (75.4–100.0)	24	83.0 (74.9–93.0)	−2.6
Manufacture of fabricated metals (28)	84	85.4 (73.7–97.4)	58	83.2 (71.7–94.9)	−2.2
Manufacture of machinery (29)	55	81.3 (73.3–90.7)	65	81.8 (67.5–91.3)	+0.5
Manufacture of motor vehicles (34)	65	83.8 (70.2–96.2)	44	82.6 (72.3–100.0)	−1.2
Manufacture of furniture (36)	18	81.0 (73.4–88.0)	7	80.6 (73.7–85.7)	−0.4
Construction (45)	27	84.6 (73.7–91.3)	22	80.1 (70.9–88.3)	−4.5
Day care (85)	32	82.2 (68.4–92.5)	56	81.9 (76.0–103.0)	−0.3
All noisy trades	627	83.9 (64.7–100.0)	467	82.8 (67.5–106.0)	−1.1

to be lower with the higher cumulative noise exposure [Table 2].

Table 3 shows adjusted odds ratios (ORs) of hearing loss in the critically noise-sensitive frequencies (as defined in section 'Audiometric measures') by year of first occupational noise exposure for the baseline and follow-up populations. For the baseline population, we observed no increased risk of hearing loss among those with first exposure after the 1980s compared to that of the reference group (adjusted OR: 1.02, 95% confidence interval (CI) 0.59; 1.77). For the baseline workers with the first exposure before the 1980s, we found a statistically significantly increased risk of hearing loss (adjusted OR: 1.90, 95% CI 1.11; 3.22) compared to that of the reference group. For each extra year since the first exposure, we found an OR of 1.02 for the hearing loss (95% CI 1.00; 1.04) among the baseline workers.

For the follow-up population, we also observed a tendency towards an increased risk of hearing loss with a longer time since the first exposure, but results were statistically insignificant. Thus, the adjusted OR for hearing loss for the group with the earliest exposure (before the 1980s) was 1.48 (95% CI 0.58; 3.77).

In the longitudinal analyses of the 207 workers, participating in both the surveys, we initially performed analyses on the association between the cumulative noise exposure and the hearing threshold shift in the follow-up period stratified by baseline hearing status and prior noise exposure, to account for a possible effect modification from these factors [Table 4]. Results showed only marginal differences between the strata, and Wald tests indicated no effect modification by these variables.

Therefore, we proceeded with the main longitudinal analyses without stratification for baseline hearing status and prior noise exposure. Adjusted results showed a weak, statistically insignificant, inverse association between higher cumulative noise exposure and the hearing threshold shift during the 10-year period. Thus, an average hearing threshold shift in the period was −0.09 dB for each extra noise-year (95% CI −0.35; 0.17) (adjusted for age and sex). A vague inverse association was also found between higher number of years exposed >80 dB (−0.06 dB threshold shift per extra year exposed >80 dB(A) (95% CI −0.57; 0.29) (adjusted for age and sex), but this association turned weakly positive when analysing number of years exposed >85 instead (0.08 dB threshold shift per extra year exposed >85 dB(A)) (adjusted for age and sex). No association was found between occupational noise level measured at baseline and hearing threshold shifts.

Accounting for the use of HPD by adjusting analyses for HPD use or subtracting 10 dB(A) from company noise levels for the sub-group reporting daily use of HPD did not noticeably change the association between the cumulative occupational noise and hearing threshold shift in the follow-up period (association when adjusting for HPD: −0.11 dB per noise-year (95% CI −0.38; 0.16), and association when subtracting 10 dB if HPD was used: −0.09 (95% CI −0.26; 0.10)).

According to Table 5, 70% of the baseline population exposed to noise levels >85 dB(A) used HPD, raising to 76% among the follow-up population. Around 75% of men and 50% of women used HPD when exposed >85 dB(A) at both surveys. No distinctive differences in HPD use between the age groups were observed at either the baseline or follow-up populations.

Table 6 shows a general decline in noise levels from baseline to follow-up across the noisy industries included in this study. Only 'manufacture of machinery' showed an increasing noise level from 81.3 dB(A) at baseline to 81.8 dB(A) at follow-up. The most prominent fall in noise level over the follow-up period was seen in 'construction' (−4.5 dB(A)). Average decline for all the included industries from baseline to follow-up was 1.1 dB(A).

Discussion

Main results from this study indicate that worker's cumulative occupational noise exposure during the follow-up period from 2000 to 2010 was not associated with statistically significant changes in hearing in the critically noise-sensitive frequencies. By categorising the baseline and the follow-up workers by their year of first noise exposure >80 dB(A), we found the highest risk of hearing loss among workers with first exposure before the 1980s in the baseline as well as the follow-up populations.

The prevalence of HPD use among workers exposed to average occupational noise levels >85 dB(A) increased from 70.1% in 2001–2003 to 76.1% in 2009–2010, whereas mean noise levels in the included industries decreased with 1.1 dB(A).

An average decline in noise level of 1.1 dB(A) over 10 years may appear small, but remembering that 1 dB represents a power ratio of approximately 1.26 (the decibel is a logarithmic unit), the effect on hearing preservation may be significant. In addition, some of the largest declines in mean noise levels are found among the industries with the highest baseline levels, meaning that no mean industry levels exceeded 85 dB(A) in 2009–2010. However, mean company noise levels used to classify worker's noise exposure still exceed 85 dB(A) for a substantial part of workers, and in this case, around three-quarters of workers reported to use HPD. Accordingly, the finding of no association between recent occupational noise levels and hearing threshold shift among our participants was not unexpected.

In a longitudinal cohort study from 2006,[13] an inverse association between 10-year binaural hearing loss rates in the noise-sensitive frequencies (3, 4 and 6 kHz) and higher occupational noise exposure was found among 6217 noise-exposed employees. The authors found no indication of a

healthy worker bias in their analyses and, therefore, speculated if the result could be related to differential use of HPD as they found the majority of large threshold shifts among workers exposed to average noise levels <85 dB, where HPDs may not be used as consistently. Unfortunately, data on HPD use were not available in that study. We asked workers whether they used HPD in their current job and found that among workers exposed to average noise levels <85 dB(A), the use of HPD was in fact substantially lower than that at higher levels [Table 5]. Misclassification of actual noise at the ear from differential use of HPD could, therefore, also have introduced a similar bias in our study explaining the null findings.

Another 10-year longitudinal study recently conducted on construction workers in the USA[14] demonstrated that noise levels in this particular industry still constitute a risk for hearing loss in the noise-sensitive frequencies (3, 4 and 6 kHz), even though the average estimated noise exposure *L*(EQ) for the workers was only 2 dB(A) above 85 dB(A). The study population included only newly hired construction apprentices (mean age 27.6 years) assumed to have a limited prior noise exposure and good hearing at inception. Interestingly, they found a poor compliance of HPD use among the workers. Thus, only 50% of the construction workers reported to use HPD, and when observed, the fraction of exposure time, in which HPDs were used, was only 17–24%.[15] Including newly hired apprentices is an advantage to the study, because an effect modification otherwise may occur from prior noise exposure and poor baseline hearing.[16] We also included workers with prior noise exposure and workers from a broader age spectrum (mean age at baseline: 39.9 years) and, therefore, also performed stratified analyses.

A review from 2015 on occupational noise exposure and hearing concluded that the industrial noise levels in general had been reduced over the last few decades, and that this led to an improved hearing in noise-exposed groups in recent years.[6] Only among construction workers, results showed that noise was still a substantial problem with regard to hearing. Our population was too small to allow for trade-specific sub-analyses, but in general, the conclusions of the review were in line with our findings and, interestingly, we observed the largest fall in noise exposure level from baseline to follow-up among construction workers (4.4 dB(A)).

Among the strengths of our study is the longitudinal design. Much of prior literature in this field is derived from cross-sectional studies lacking temporal specificity.[17-19] Furthermore, our exposure quantification derived from individual dosimetries gives objective measures instead of subjective questionnaire information as often used to classify noise level. We did not have the capacity to measure bone conduction thresholds, which would have been a better measure of sensorineural hearing threshold. Instead, we excluded participants with possible conductive hearing loss and asymmetric hearing loss from analyses to avoid misclassification. As the white-collar workers were considered to differ considerably from the remaining population with respect to covariates (e.g. leisure time noise) that we were not able to adjust for, we decided to restrict the population to occupationally noise-exposed workers. Exposure contrast in this group was considered sufficient, with individual exposure levels ranging from 67.5 dB(A) to 106.0 dB(A).

A lower loss to follow-up than 51% in our study would have been desirable, but in our selected industries with expected low job tenancy, we find a follow-up of 49% reasonable.

Among the workers participating in both the surveys, we identified 12 workers (4.4%), who moved from high to low exposure jobs. If this shift was made because of a higher susceptibility to noise exposure among the 12 workers, it could potentially introduce a 'healthy worker bias' by attenuating the exposure response relationship. By regression analysis, we, therefore, analysed if there was an association between a change from high-to-low exposure job during the 10-year period and baseline hearing levels. We found no significant association, indicating that this was not an issue of concern.

Another possibility of bias in our study is the misclassification of noise exposure due to HPD use. Information on HPD use was retrieved from the questionnaire and was not controlled by observation of actual behaviour. As mentioned above, prior studies have revealed a large discrepancy between self-reported use and actual behaviour[15] which could also be the case in our study. To analyse whether (self-reported) HPD use had any impact on our results, we performed the sub-analyses subtracting 10 dB from the company noise exposure levels for workers reporting the HPD use and also tried to adjust the regression analyses for the use of HPD. Both sub-analyses revealed only slight changes of the main results. However, as mentioned above, a differential misclassification of actual 'noise at the ear' by a more consistent use of HPD at noise levels above 85 dB(A) is still a possibility and could have biased our results by attenuating the exposure response relationship.

To avoid TTS, we instructed participants to wear HPD from the beginning of the working day until an audiometry was performed. Participants' hearing could, however, still be affected by, for example, prior traffic or leisure time noise exposure. As most participants (around 90% in both surveys) worked only daytime (approximately 7 A.M. to 4 P.M.), we expected their prior noise exposure (mostly night time noise at home) to be low and should, therefore, not cause significant TTS. Using average company noise levels to classify worker's exposure could furthermore add to noise misclassification. We expected the sound levels to vary more from one day to another day for the individual workers than that between the different workers and chose it over industry means, because analyses of variance

showed most exposure contrast using company levels. Misclassification is, however, still a possibility but should be non-differential across noise exposure levels and would, therefore, bias results towards the null.

Conclusion

This study demonstrates a fall in recent industrial noise levels, increasing use of HPD and no association between the current occupational noise levels and hearing threshold shift.

We interpret these findings as an indication of a successful implementation of preventive measures enforced in Denmark during the last few decades to prevent noise-induced hearing loss.

Acknowledgements

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Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Stucken EZ, Hong RS. Noise-induced hearing loss: An occupational medicine perspective. Curr Opin Otolaryngol Head Neck Surg 2014;22:388-93.
2. Tak S, Calvert GM. Hearing difficulty attributable to employment by industry and occupation: An analysis of the National Health Interview Survey – United States, 1997 to 2003. J Occup Environ Med 2008;50:46-56.
3. Verbeek JH, Kateman E, Morata TC, Dreschler WA, Mischke C. Interventions to prevent occupational noise-induced hearing loss: A Cochrane systematic review. Int J Audiol 2014;53:S84-96.
4. Arenas JP, Suter AH. Comparison of occupational noise legislation in the Americas: An overview and analysis. Noise Health 2014;16:306-19.
5. El Dib RP, Mathew JL, Martins RH. Interventions to promote the wearing of hearing protection. Cochrane Database Syst Rev 2012; CD005234. doi: 10.1002/14651858.CD005234.pub5.
6. Lie A, Skogstad M, Johannessen HA, Tynes T, Mehlum IS, Nordby K, *et al.* Occupational noise exposure and hearing: A systematic review. Int Arch Occup Environ Health 2016;89:351-72.
7. Lee-Feldstein A. Five-year follow-up study of hearing loss at several locations within a large automobile company. Am J Ind Med 1993;24:41-54.
8. Bergström B, Nyström B. Development of hearing loss during long-term exposure to occupational noise: A 20-year follow-up study. Scand Audiol 1986;15:227-34.
9. Kamal AA, Mikael RA, Faris R. Follow-up of hearing thresholds among forge hammering workers. Am J Ind Med 1989;16:645-58.
10. Rubak T, Kock SA, Koefoed-Nielsen B, Bonde JP, Kolstad HA. The risk of noise-induced hearing loss in the Danish workforce. Noise Health 2006;8:80-7.
11. Malchaire J, Piette A. A comprehensive strategy for the assessment of noise exposure and risk of hearing impairment. Ann Occup Hyg 1997;41:467-84.
12. Neitzel R, Seixas NS, Camp J, Yost M. An assessment of occupational noise exposures in four construction trades. Am Ind Hyg Assoc J 1999;60:807-17.
13. Rabinowitz PM, Galusha D, Dixon-Ernst C, Slade MD, Cullen MR. Do ambient noise exposure levels predict hearing loss in a modern industrial cohort? Occup Environ Med 2007;64:53-9.
14. Seixas NS, Neitzel R, Stover B, Sheppard L, Feeney P, Mills D, *et al.* 10-Year prospective study of noise exposure and hearing damage among construction workers. Occup Environ Med 2012;69:643-50.
15. Neitzel R, Seixas N. The effectiveness of hearing protection among construction workers. J Occup Environ Hyg 2005;2:227-38.
16. ANSI S3.44-1996. Determination of Occupational Noise Exposure and Estimation of Noise-induced Hearing Impairment.
17. Ivarsson A, Bennrup S, Toremalm NG. Models for studying the progression of hearing loss caused by noise. Scand Audiol 1992;21:79-86.
18. Martin RH, Gibson ES, Lockington JN. Occupational hearing loss between 85 and 90 dBA. J Occup Med 1975;17:13-8.
19. Somma G, Pietroiusti A, Magrini A, Coppeta L, Ancona C, Gardi S, *et al.* Extended high-frequency audiometry and noise induced hearing loss in cement workers. Am J Ind Med 2008;51:452-62.

Bilag 2. Afslutningsskema til Arbejdsmiljøforskningsfonden

Afslutning på projekt

Indsendes til:
forskningsfonden@at.dk

Generelle oplysninger om projektet

1	**Projektets titel**
	Støjbelastning i arbejdsmiljøet og risiko for tinnitus og nedsat høreevne. En forløbsundersøgelse af støjkarakteristika, tærskelværdier, forvarsler, individuel følsomhed og forebyggelse: STØJRISKprojektet

2	**Ansøger**	
	CVR-nummer:	29762929
	Institutionens navn:	Arbejdsmedicinsk Klinik, Århus Universitetshospital,
	Arbejdsadresse:	Nørrebrogagde 44, 8000 Århus C
	Tlf.nr.:	89494290
	e-mail:	henkol@rm.dk

3	**Ansvarlig projektleder/kontaktperson**	
	Navn:	Henrik Kolstad
	Arbejdssted:	Arbejdsmedicinsk Klinik, Århus Sygehus
	Arbejdsadresse:	Nørrebrogagde 44, 8000 Århus C
	Tlf.nr.:	89494290
	e-mail:	henkol@rm.dk

4	**Projektet formål og hovedhypoteser** (Kortfattet beskrivelse af projektets formål og hovedhypoteser herunder begrundelse for evt. ændringer i formål og hovedhypoteser):

En stor del af arbejdsstyrken er generet af støj og støjskader er blandt de hyppigste arbejdsskader. Selvom den høreskadende effekt er velkendt, og vi kender forebyggelsesmetoder, må vi forvente at støj vedbliver med at være et væsentligt arbejdsmiljøproblem i en lang årrække, og der er behov for ny viden om de helbredsmæssige konsekvenser og effektiv forebyggelse.

Dette projekt havde to overordnede formål: For det første, at fremskaffe konkret viden om forekomst og forebyggelse af støjskader indenfor støjbelastede brancher i Danmark: Industri, bygge- og anlæg og daginstitutioner. For det andet, at fremskaffe ny videnskabelig viden om årsagssammenhænge mellem lavdosis støj omkring 80 dB(A), impulsstøj, tinnitus og hørenedsættelse, samt samspilseffekter mellem støj, psykosociale belastninger og individuel følsomhed.

5	**Resultater og videnskabelig nyhedsværdi** (Redegør for de videnskabelige resultater af projektet og deres nyhedsværdi. Redegør desuden for hvilke resultater der var forventede henholdsvis uventede. Hvordan forholder resultaterne sig til øvrige forskningsresultater?):
	Vi fandt i STØJRISK projektet at det gennemsnitlige støjniveau målt gennem en hel arbejdsdag i 10 særligt støjbelastede industribrancher (fremstilling af næringsmidler og drikkevarer, træindustri, møbelindustri, grafisk industri, sten, ler og glasindustri, jern og metalvareindustri, fremstilling af metal, maskinindustri, fremstilling af biler mv.), bygge og anlægsvirksomhed og børnehaver i gennemsnit faldt med 1,1 dB(A) fra 83,9 dB(A) i 2001-3 til 82,8 dB(A) i 2009-10. Andelen af deltagere eksponeret for støj over 85 dB(A) (grænsen for hvornår Arbejdstilsynet kræver at der anvendes høreværn), som anvendte høreværn, steg fra 70 til 78 procent i den samme periode. Vi fandt ikke at høretærskelen i de kritisk støjfølsomme hørefrekvenser steg (faldende hørelse) med stigende støjeksponering mellem 2001 og 2010. Os bekendt er vi de første til at dokumentere faldende støjniveauer over tid med direkte sammenlignelige data. Dette har været vist for en række andre erhvervseksponeringer og vores fund kom derfor ikke som en overraskelse. Ved et gennemsnitligt støjniveau under 83 dB(A) er den kendte effekt på hørelsen begrænset og sammen med den udbredte brug af høreværn giver det god mening at de fleste medarbejdere i de undersøgte industrier ser ud til at være godt beskyttet mod støjskader. Årsagsmekanismerne der knytter støjeksponering og høreskade sammen, er kun delvist kortlagt. Som en af flere mekanismer har man foreslået at støj ændrer koncentrationen af kolesterol og andre lipider i blodet, forøger forekomsten af atherosklerose (åreforkalkning) og nedsætter blod flowet i de fine arterier, som forsyner høresneglen og det Cortiske organ. Vi fandt at et højt niveau af triglycerider og et lavt niveau af HDL kolesterol samt høj BMI og rygning var associeret med reduceret hørelse. Men vi fandt ingen sammenhæng mellem støjniveau og disse lipider, når vi tog højde for høreværn og kendte risikofaktorer for dyslipidæmi. Disse analyser peger således primært i retning af at kost og livsstil men ikke støj kan medføre høretab via påvirkning af lipid niveauer. Vi fandt ingen sammenhæng mellem generende tinnitus og psykosocialt arbejdsmiljø angivet som graden af krav og kontrol i arbejdet, eller fysiologisk stress målt som niveauet af kortisol i spyt. Det er velkendt at høretab forårsaget af støj ofte er ledsaget af tinnitus. Men vi fandt ikke holdepunkter for at risikoen for tinnitus var associeret med støjniveauet i denne population. Det kan hænge sammen med at støjniveauerne var for lave til at forårsage høretab.

6	**Arbejdsmiljøperspektiver (**Hvilken relevant viden er der skabt i projektet? Hvilken betydning har det for arbejdsmiljøet og arbejdsmiljøarbejdet? Kan projektets resultater omsættes til praktisk anvendelse for målgrupperne? Hvordan kan resultaterne overføres til andre målgrupper?)
	Hovedbudskabet fra denne undersøgelse er at vi mellem 2001 og 2010 ikke kunne finde sammenhæng mellem støjudsættelse på arbejdet og høretab indenfor de mest støjudsatte brancher i Danmark. Vi fandt at støjniveauerne i disse brancher er faldet og brugen af høreværn er steget i løbet af opfølgningsperioden og dette er nærliggende forklaringer på dette resultat. Man må dog formode at overordnede samfundsmæssige forhold også har spillet ind på denne udvikling, fx den generelle tekniske udvikling og outsourcing af de mest støjende arbejdsopgaver. Vi kunne ikke underbygge at støj, psykosociale arbejdsforhold eller kortisol (som mål for fysiologisk stress) er associeret med kronisk tinnitus. Et hovedformål for arbejdsmiljøforskningen er at forebygge arbejdsbetingede lidelser. Ny viden om risikofaktorer i arbejdsmiljøet er helt centralt for at kunne nå dette mål. Men denne viden får kun effekt, hvis den udmøntes i grænseværdier eller anden regulering, som efterleves ude på arbejdspladserne. Dette projekt tyder på at dette har været tilfældet for støjbetinget høretab: Forebyggelse nytter. Det er også arbejdsmiljøforskningens opgave at vurdere om mistænkte risikofaktorer også er reelle risikofaktorer. Denne undersøgelse tyder ikke på at generende tinnitus er forårsaget af arbejdsmiljøeksponeringer.

7	**Medarbejdere** (angiv navne på videnskabelige medarbejdere i projektet)
	1. Thomas Winther Frederiksen 2. Zara Ann Stokholm 3. Cecilia Høst Ramlau-Hansen 4. Matias Brødsgaard Grynderup 5. Åse Marie Hansen 6. Søren Peter Lund 7. Jesper Kristiansen 8. Jesper Medom Vestergaard 9. Jens Peter Bonde. 10. Mai Arlien-Søborg 11. Astrid Schmedes

8	**Øvrige institutioner** (angiv øvrige institutioner som har deltaget i projektet)
	1. Det Nationale Forskningscenter for Arbejdsmiljø (NFA) 2. Københavns Universitet 3. Aarhus Universitet 4. Arbejds- og miljømedicinsk afdeling, Bispebjerg Hospital 5. Regionshospitalet Holstebro, Øre-næse-hals afdelingen 6. Institute of Occupational Medicine, Edinburgh 7. CRECEA A/S

9	**Interessentgruppe** (Angive navne på medlemmer af evt. interessentgruppe)

10		
	Startdato for projektet:	1.7.2010
	Planlagt slutdato:	31.6.2013
	Faktisk slutdato:	31.12.2016

Formidling (Ved fortrolighed skal denne aftales med Arbejdsmiljøforskningsfondens sekretariat inden indsendelse af slutrapporten.)

11	**Har Videncenter for Arbejdsmiljø været inddraget i formidling undervejs i projektforløbet?**	**Nej** ☐ **Ja** x☐

12	**Populærformidling (mundtlig)**	
	Konferencer, seminarer m.v.:	Zara Ann Stokholm. Støjbelastning i arbejdsmiljøet og risiko for tinnitus og nedsat høreevne. Arbejdsmiljøforskningsfondens Årskonference, 11. januar 2017
	Målgruppe for de væsentligste aktiviteter (max 5):	1. Industriarbejdere og andre udsat for høj støj på arbejdet 2. Arbejdstilsynet 3. Arbejdsmiljøkonsulenter 4. Arbejdsmarkedets erhvervssikring 5. Forskermiljøer beskæftiget med auditive effekter af støj

11	**Populærformidling (skriftlig)**	
	Projektets populær-videnskabelige artikel:	
	Øvrige artikler, nyheder m.v.: (Angiv titel og medie)	Høretab koster milliarder. Interview med Henrik Kolstad i Magasinet Penge, DR1 7. oktober 2015.
	Hjemmeside/sociale medier:	Støj for nogle er musik for andre. Interview med Zara Stokholm i TV2 Østjylland den 7. december 2016. Forsker: Indsatsen mod støj er lykkedes. Interview med Henrik Kolstad. Videncenter for Arbejdsmiljø, 7. september 2015.

12	**Videnskabelig formidling (mundtlig)**

Oplæg på konference, seminarer m.v. (angiv konference og titel på oplæg):

Kolstad, HA, Stokholm, ZA, Vestergaard, JM, Erichsen, TR, Frederiksen, TW, Bonde, JP. Noise and health: Danish studies on noise at work. Noise and Health. Sundhedsstyrelsens Rådgivende Videnskabelige Udvalg om Miljø og Sundhed, oktober 2012.

Schmedes, A, Arlien-Søborg, MC, Stokholm, ZA, Hansen, AM, Bonde, JP, Christensen, KL, Frederiksen, TW, Kristiansen, J, Lund, SP, Vestergaard, JM, Wetke, R, Kolstad, HA. Occupational noise exposure and serum lipids: the impact of noise exposure level and hearing protection. Mundtligt præsenteret ved Epidemiology in Occupational Health Conference, Utrecht, juni 2013.

T. W. Frederiksen, C.H. Ramlau-Hansen, Stokholm ZA,Vestergaard JM, H.A. Kolstad. Occupational Noise Exposure, Psychosocial Working Conditions and the Risk of Tinnitus. The 25th International Epidemiology in Occupational Health (EPICOH) Conference, September 2016, Barcelona

Zara Ann Stokholm, Mogen Erlansen, Vivi Schünssen, Ioannis Basinas, Jens Peter Bonde, Jens Brandt, Jesper Medom Vestergaard, Henrik Kolstad. A general population job exposure matrix for occupational noise. The 25th International Epidemiology in Occupational Health (EPICOH) Conference, September 2016, Barcelona

13	**Videnskabelig formidling (skriftlig)**

Skriftlig videnskabelig formidling med fagfælle bedømmelse

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad Atherogenic Risk Factors and Hearing Thresholds. Audiology and Neurotology 2014;19:310–318).

Arlien-Søborg MC, Schmedes AS, Stokholm ZA, Grynderup MB, Bonde JP, Jensen CS, Hansen ÅM[6], Frederiksen TW, Kristiansen J, Christensen KL, Vestergaard JM, Lund SP, Kolstad HA. Ambient and at-the-ear occupational noise exposure and serum lipid levels. Int Arch Occup Environ Health. 2016 Oct;89(7):1087-93.

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Occupational Noise Exposure, Psychosocial Working Conditions and the Risk of Tinnitus. International Archives of Occupational and Environmental Health, December 2016

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara Ann Stokholm, Matias B. Grynderup, Åse Marie Hansen PhD, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Noise Induced Hearing Loss – a preventable disease? Results of a 10-year longitudinal study of occupationally noise exposed workers. Noise and Health, in press

Øvrig skriftlig videnskabelig formidling

Thomas W. Frederiksen. Occupational Noise Exposure and Individual Risk Factors for Hearing Loss and Tinnitus. PhD dissertation, Health, Aarhus University, March 2016

Manuskripter indsendt til videnskabelig tidsskrift eller under udarbejdelse

Thomas W. Frederiksen, Cecilia H. Ramlau-Hansen, Zara A. Stokholm, Matias B. Grynderup, Åse Marie Hansen, Søren Peter Lund, Jesper Kristiansen, Jesper M. Vestergaard, Jens P. Bonde, Henrik A. Kolstad. Salivary Cortisol and Tinnitus. Manuscript.

STOKHOLM Z.A., ERLANDSEN M., SCHLÜNSSEN V., BASINAS I., BONDE J.P, BRANDT J., VESTERGAARD J.M., KOLSTAD H.A. A general population job exposure matrix for occupational noise. Manuscript.

14	**Projektansvarliges underskrift og dato** Undertegnede erklærer, at ovenstående oplysninger og oplysningerne i bilag er rigtige. Hvis der er afgivet urigtige eller vildledende oplysninger i ansøgningen, eller hvis oplysninger, som kan have betydning for afgørelse om tilsagn, er tilbageholdt, kan et tilsagn annulleres, og evt. udbetalinger kræves tilbagebetalt.
	Århus 27/3/2017