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ORIGINAL ARTICLE

Determinants of early-stage hearing loss among a cohort of young workers with 16-year follow-up

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ABSTRACT

Objectives The authors had a unique opportunity to study the early impacts of occupational and recreational exposures on the development of noise-induced hearing loss (NIHL) in a cohort of 392 young workers. The objectives of this study were to estimate strength of associations between occupational and recreational exposures and occurrence of early-stage NIHL and to determine the extent to which relationships between specific noise exposures and early-stage NIHL were mitigated through the use of hearing protection.

Methods Participants were young adults who agreed to participate in a follow-up of a randomised controlled trial. While the follow-up study was designed to observe long-term effects (up to 16 years) of a hearing conservation intervention for high school students, it also provided opportunity to study the potential aetiology of NIHL in this worker cohort. Study data were collected via exposure history questionnaires and clinical audiometric examinations.

Results Over the 16-year study period, the authors documented changes to hearing acuity that exceeded 15 dB at high frequencies in 42.8% of men and 27.7% of women. Analyses of risk factors for NIHL were limited to men, who comprised 68% of the cohort, and showed that risks increased in association with higher levels of the most common recreational and occupational noise sources, as well as chemical exposures with ototoxic potential. Use of hearing protection and other safety measures, although not universal and sometimes modest, appeared to offer some protection.

Conclusions Early-stage NIHL can be detected in young workers by measuring high-frequency changes in hearing acuity. Hearing conservation programmes should focus on a broader range of exposures, whether in occupational or non-occupational settings. Priority exposures include gunshots, chainsaws, power tools, smoking and potentially some chemical exposures.

What this paper adds

- Most hearing conservation programmes have been developed for specific occupational situations and do not necessarily consider recreational noise exposures and lifestyle factors that extend beyond the formal worksite.
- Few studies of hearing conservation programmes have focused on detecting early-stage NIHL in young workers.
- The study examined a diversity of occupational and recreational exposures as potential risk factors for NIHL in a young worker cohort.
- Findings suggest that early-stage NIHL can be detected in young workers by measuring high-frequency changes on audiograms.
- Hearing conservation programmes must recognise the important and potentially complicating impacts of non-occupational exposures, including gunshots, chainsaws, power tools, smoking and some chemical exposures.
- Hearing conservation policies and practices should focus on early detection of NIHL in young workers and address a broader range of exposures, both occupational and non-occupational.

INTRODUCTION

Early-stage noise-induced hearing loss (NIHL) is a topic of contemporary importance to the health of young people.[1–3] Detection of NIHL at an early stage is of paramount importance, as these losses are non-reversible[4] and have important life consequences that extend to work, family and social settings.[5] Recent attention to the potential for early-stage NIHL in young people has emerged with the almost universal availability of personal listening devices. Yet exposures to other recreational and occupational sources of noise that can and do lead to NIHL have received scant attention in populations of young people.

Most established hearing conservation programmes have been developed for specific occupational situations. Such hearing conservation programmes typically focus on the use of hearing protection devices in noisy occupational environments.[6] These initiatives do not necessarily consider recreational noise exposures and lifestyle factors that extend beyond the formal worksite. Under the assumption that non-occupational factors may in some cases substantially impact hearing, testing of new employees, collection of broader exposure data and periodic retesting would all be important with respect to both employee safety and corporate liability.

We had a unique opportunity to study the potential impact of a large variety of occupational and recreational exposures on the development of early-stage NIHL in a cohort of 392 young workers. This cohort was assembled during a randomised controlled trial used to evaluate the efficacy of a school-based hearing conservation intervention.[7] Trial participants were aged 12–16 years when first evaluated and were aged 29–33 years in the most recent assessment. This is a secondary analysis of exposure data and audiometric measures from that trial.

The objectives of the current analysis were to use this cohort to: (1) estimate the strength of associations between occupational and recreational exposures and the occurrence of early-stage NIHL and (2) determine whether relationships between specific exposures to noise and early-stage NIHL were mitigated by the use of hearing protection. These analyses are important because few similar studies of early-stage NIHL exist for young workers, and evidence could further inform the content and targeting of potential countermeasures to prevent early-stage NIHL at young ages.

METHODS

Overview

This study involved a secondary analysis of data collected via exposure history questionnaires and clinical audiometric examinations. Participants were young adults who agreed to take part in a follow-up of a randomised controlled trial. The follow-up study was designed to observe the long-term effects (up to 16 years) of a hearing conservation intervention for high school students.[7] Although most of these young people were originally working in agriculture, many went on to work in other occupations over this time period. The ethics committees of the Marshfield Clinic Research Foundation and the Faculty of Health Sciences at Queen's University approved the study protocol.

Sample

Our target cohort for the 2009–2010 follow-up study was composed of 690 students who had completed the original randomised controlled trial 16 years earlier, with baseline audiometric testing in 1992–1993. Extensive efforts were made to find and contact all members of this cohort.[7] In total, 392 of 690 (57%) participants were successfully located and recruited; 355 (91%) completed both the survey and the clinical audiometric examination, and their records are analysed here.

Exposure assessment

Participants completed an exposure history questionnaire covering the 13 years since last contact in 1996. The questionnaire focused on high noise level occupational and recreational exposures (as informed by the existing biomedical literature) as well as the use of hearing protection devices for each exposure. Exposures to smoking and chemicals with ototoxic potential were also recorded.

All exposure measures were collected using estimates of numeric quantities and time periods relevant to each exposure (eg, cigarettes per day, estimated total shots using guns, estimated total days using chainsaws, etc). Durations of these exposures over the 13-year survey period were also estimated. This permitted development of summary metrics that accounted for both intensity and duration of each exposure.

Distributions of exposures across participants were skewed and typically included a small number of extremely high reported levels of exposure. Based on review of the reported exposures, the study team agreed that the data should be grouped for analysis.

Exposure to smoking was analysed as *ever* versus *never* smoked. Other exposure estimates were each grouped into three categories for analysis, and the primary comparisons contrasted the highest versus lowest exposure categories. Exposures to shooting, high noise recreational activities (overall measure) and high noise occupations (overall measure) were grouped into three equal size groups, and the highest group was compared with the lowest group. For all other exposures where substantial numbers of participants reported no exposure, participants above the median exposure were compared with those reporting no exposure.

The frequency of hearing protection use was reported separately on the questionnaire for each individual exposure. Ordinal scales were used with five possible responses: never (0%), sometimes (1%–33%), often (34%–66%), frequently (67%–99%) or always (100%).

Audiometric assessment

Audiometric examinations were conducted by licensed audiologists in clinical audiology departments under standard operating procedures.[8] Briefly, subjects were evaluated in sound-treated test booths using the modified Hughson–Westlake method of limits. Air conduction testing was performed using standard TDH-50 earphones. An otoscopic inspection of the ear canals was performed, and a structured case history was elicited prior to the test.

Primary outcome measure

NIHL is a common clinical diagnosis that is well recognised to first appear at a single, relatively high frequency (3, 4, or 6 kHz), often presenting with a characteristic audiometric notch.[1 9 10] However, progression of NIHL may lead to a deeper notch and/or broadening of the affected thresholds to lower and/or higher frequencies.

We manually reviewed individual plots showing baseline and follow-up audiometry results for all 355 participants. Many of these participants showed substantial hearing loss at one or more high frequencies at baseline (thresholds up to 70 dB) and/or follow-up (thresholds up to 90 dB), with increases of up to 75 dB over the 16-year period, reflecting substantial hearing loss. However, among those with hearing loss, the audiometric configuration varied substantially, with a minority showing the expected audiometric notch (23% using the method of Niskar *et al*[1]). Based on our desire to have a sensitive measure of NIHL, the maximum change at any high frequency (ΔHF) (3, 4 or 6 kHz) in either ear from baseline to follow-up was used as the primary outcome. This ΔHF measure provided an indicator of the degree of change over time appropriate to the heterogeneous patterns of change represented in this cohort.

Statistical analyses

These results are from secondary analyses of a randomised controlled trial in which a large number of potential risk factors for hearing loss were evaluated. The emphasis in this report is therefore on descriptive summaries, assessment of associations and the consistency of general trends rather than on tests of specific hypotheses.

Ordinal logistic regression models were used to evaluate associations between each individual exposure and the NIHL outcome. To provide adequate numbers for analysis, our measure of NIHL (ΔHF) was grouped into four categories: 0–5, 10, 15 or ≥20 dB. A cumulative logit parameterisation assuming proportional odds[i] was used in a generalised mixed linear model with school as a random effect, in keeping with the original study

[i]The proportional odds model, also called a parallel slopes model, extends the logistic model for a binary response to a response with three or more ordered categories under the assumption that the association between a predictor and the odds of response is the same across all categories. This assumption was believed to provide a good approximation to the observed data based on: (1) face validity, (2) univariate assessment of the raw data and (3) evaluation of formal score tests of proportionality.

randomisation by school. All statistical tests and model results included adjustment for the study intervention and for participant age.

The large number of potentially relevant exposures and the fixed size of the study cohort limited our ability to delineate the independent contribution of each risk factor. In particular, gender and smoking were believed to present unique challenges as potential confounding factors, given their observed associations with both the primary outcome and with other exposures. Gender was not of primary interest in this study since it is not an attribute that can be controlled. After exploring risks by gender, we found it most informative to limit the main aetiological analysis to men, which comprised a large majority of the cohort (68%). Smoking was evaluated for association with ΔHF and was also evaluated for potential confounding of other factors by analysing the subset of participants (68%) who never smoked (analyses not presented here) and by including smoking (ever/never) as a covariate in secondary models. Similarly, the use of hearing protection (or other protective measures as relevant to an exposure) was evaluated as a potential mitigating factor in secondary models with percentage of reported use of the protective factor as a covariate.

Results presented include observed significance levels (p values) for tests of association under the basic model for each individual exposure, as well as ORs with asymptotic confidence limits for models which additionally adjust for (1) smoking and (2) smoking and use of protection.

RESULTS

Table 1 describes the 355 participants in the long-term follow-up study by intervention versus control group membership, basic demographics, smoking and maximum high-frequency change in hearing acuity. This was a relatively young cohort at inception (mean 14.5 years) whose age averaged 31.0 years at the 16-year follow-up. Hearing loss, measured as the maximal change in hearing acuity at high frequencies (ΔHF), was more evident in men versus women. A ΔHF of 15 dB or more was measured in 104/243 (42.8%) men versus 31/112 (27.7%) women, and 25 dB or more in 40/243 (16.5%) men versus 3/112 (2.7%) women.

Table 2 profiles the past exposures to: (1) common sources of recreational noise, (2) work in specific occupations with known hazardous levels of noise and (3) chemicals known or suspected to be implicated in the aetiology of NIHL. For noise exposures, consistent with the documented patterns of ΔHF, men reported substantially higher exposures to power tools and equipment, gun use, vehicles, agriculture, construction and manufacturing jobs, and chemical exposures. These descriptive findings also demonstrate the range and diversity of exposures that could potentially lead to NIHL in this cohort.

A series of logistic regression models were used to evaluate potential associations of recreational, occupational and chemical exposures with the ΔHF outcome measure in male subjects. The primary analyses from these models compared high to low exposure groups, and a summary of the reported exposure levels in these groups is shown in table 3. In table 4, results for unadjusted tests of the associations are presented in the second column ('Unadjusted p'), with those of highest significance being those for gunshots (p<0.001), chainsaws (p=0.001), fuel vapours (p=0.003) and smoking (p=0.004). Columns 3–5 of table 4 summarise the strength of associations with ORs and confidence limits, with adjustment in the models for smoking. The findings demonstrate a remarkable consistency, with 17/18 point estimates of ORs (irrespective of the CIs) in the direction of a positive effect (OR>1.0), and the strongest associations again being those for gunshots (OR=3.05) and chainsaws (OR=3.27). The exception in terms of the direction of association is for personal stereos (OR=0.65), where those reporting highest exposures tended to have smaller increases with respect to ΔHF.

Table 1 Description of study population

	N*	% Yes
Intervention	187	52.7
Control	168	47.3
Men	243	68.5
Technical/college degree	191	53.8
Smoking (ever)	114	32.1

Age (years)	Original trial	16-Year follow-up
Median	14.5	31.0
Minimum	12	29
Maximum	16	33

Maximum high-frequency change baseline to 16 years

	Women		Men	
	N	%	N	%
Maximum change (dB)				
0	17	15.2	20	8.2
5	36	32.1	57	23.5
10	28	25.0	62	25.5
15	22	19.6	45	18.5
20	6	5.4	19	7.8
25+	3	2.7	40	16.5

*Total n=355.

Table 2 Specific exposures to recreational and occupational factors among study participants

	Men		Women	
	N	% Yes	N	% Yes
Recreational noise exposures				
Using power lawn/garden tools	242	95.0	112	81.3
Any gun use	243	84.8	112	27.7
Using other power tools	242	81.0	110	20.0
Using chainsaws	243	79.4	111	15.3
Riding all-terrain vehicles	242	77.7	112	57.1
Attending live music concerts	242	61.2	112	64.3
Riding snowmobiles	243	49.4	111	40.5
Attending auto races	242	35.5	112	32.1
Riding motorcycles	243	35.4	111	19.8
Ear buds (in-ear)	241	32.4	112	50.9
Headphones (over-the-ear)	241	27.0	112	45.5
Playing in a band	242	5.0	112	20.5
Auto racing (driving)	243	2.9	111	0.0
Occupational noise exposures				
Agriculture job	243	81.9	112	67.9
Construction job	242	46.3	112	6.3
Manufacturing job	243	43.2	112	31.3
Occupational hearing test	243	23.5	112	10.7
Chemical exposures				
Fuels or fuel vapours	242	47.9	111	13.5
Motor engine exhaust	242	45.5	111	10.8
Solvents or thinners	243	28.4	110	10.0
Pesticides or herbicides	243	25.9	110	6.4
Heavy metals	243	6.6	112	0.9
Hydrogen cyanide	242	2.5	112	0.0

Table 3 Exposure summary by level of exposure* (men only)

	Lowest exposure group			Highest exposure group		
Exposure†	**N**	**Median**	**Range**	**N**	**Median**	**Range**
Gunshots	81	9	0–137	77	2050	676–131 300
All other recreations noise	81	494	0–870	81	3970	2453–10 452
Motorcycles	157	0	0–0	41	300	150–3190
Snowmobiles	123	0	0–0	58	198	100–600
All-terrain vehicles	54	0	0–0	90	780	390–4355
Auto races	156	0	0–0	41	40	12–660
Chainsaws	50	0	0–0	88	425	140–3900
Other power tools	46	0	0–0	87	1300	390–4745
Music concerts	94	0	0–0	63	48	21–300
Personal stereos	137	0	0–0	53	2080	936–36 400
All occupational noise	81	55	0–261	83	693	650–1452
Agriculture	49	0	0–0	97	468	156–932
Construction	137	0	0–0	53	364	156–676
Manufacturing	144	0	0–0	49	468	160–676
Pesticides/herbicides	180	0	0–0	30	260	90–1500
Engine exhaust	132	0	0–0	49	2970	1170–4745
Solvents	174	0	0–0	33	720	192–4745
Fuel vapours	126	0	0–0	54	2550	1020–4745

*Subjects ranked by each exposure into thirds for common exposures (results presented only for the lowest and highest), and otherwise into those with no exposure and those with exposures greater than the median reported exposure.
†Exposure units are totals over 13 years: total shots for gunshots, total hours for personal stereos, total days for other recreation and for chemicals and total full-time equivalent weeks for occupations.

Reported use of hearing protection (or other protective measures relevant to the exposure) varied widely by exposure category (table 4, column 6 'Use of protection'). After accounting for both smoking and use of protection, most relationships between the specific exposures and ΔHF (table 4, columns 7–9 'Exposure estimates') became more pronounced, although no additional exposures achieved statistical significance. Use of protection for specific exposures generally showed associations with ΔHF in the anticipated direction (table 4, columns 10–12 'Protection estimates', where OR<1.00 shows a protective effect, and statistical significance of the effect is indicated by CIs that do not cross 1.00), but with the exception of work in manufacturing these were not significant. The modest number of male participants (n=49) who reported working in a manufacturing job experienced a significant reduction in the odds of ΔHF in association with use of hearing protection in that occupational context.

Table 4 Findings from mixed logistic regression models of the maximum high frequency change* (men only)

		Adjusted for smoking only				Adjusted for both smoking and protection					
		Exposure estimates†				Exposure estimates			Protection estimates		
Exposure	**Unadjusted p**	**OR**	**Lower**	**Upper**	**Use of protection (%)‡**	**OR**	**Lower**	**Upper**	**OR**	**Lower**	**Upper**
Gunshots	<0.001	3.05	1.57	5.91	51.6	3.35	1.54	7.28	0.95	0.76	1.18
All other recreational noise	0.052	1.67	0.93	3.03	25.6	1.78	0.97	3.25	0.92	0.70	1.21
Motorcycles	0.174	1.48	0.75	2.92	18.1	2.04	0.90	4.61	0.72	0.42	1.25
Snowmobiles	0.090	1.56	0.84	2.92	12.9	1.41	0.73	2.72	1.24	0.79	1.94
All-terrain vehicles	0.269	1.43	0.75	2.72	5.9	1.26	0.64	2.46	2.14	0.93	4.95
Auto races	0.327	1.29	0.64	2.62	28.1	1.38	0.55	3.45	1.00	0.59	1.70
Chainsaws	0.001	3.27	1.59	6.71	41.7	2.99	1.25	7.14	1.06	0.80	1.41
Other power tools	0.022	2.26	1.11	4.59	34.0	3.12	1.33	7.32	0.80	0.57	1.11
Music concerts	0.792	1.07	0.59	1.95	3.8	1.12	0.60	2.10	0.99	0.30	3.25
Personal stereos	0.210	0.65	0.35	1.24	64.3§	0.42	0.04	4.09	1.19	0.51	2.78
All occupational noise	0.753	1.05	0.59	1.88	38.2	1.14	0.63	2.07	0.88	0.68	1.13
Agriculture	0.927	1.03	0.52	2.05	31.3	1.26	0.56	2.88	0.84	0.56	1.25
Construction	0.063	1.55	0.85	2.81	47.0	2.05	0.79	5.33	0.86	0.58	1.28
Manufacturing	0.570	1.10	0.59	2.06	67.3	3.48	0.93	12.94	0.65	0.43	1.00
Pesticides/herbicides	0.508	1.47	0.69	3.13	51.7¶	2.38	0.63	9.02	0.79	0.47	1.34
Engine exhaust	0.023	1.97	1.03	3.78	10.9¶	2.12	1.02	4.40	0.80	0.36	1.78
Solvents	0.102	1.57	0.76	3.23	42.5¶	2.17	0.67	6.96	0.87	0.51	1.47
Fuel vapours	0.003	2.41	1.29	4.50	19.1¶	2.32	1.10	4.91	1.23	0.69	2.21
Smoking	0.004	–	–	–	–	–	–	–	–	–	–

*Ordinal outcome for the maximum high frequency change: 0–5, 10, 15 or ≥20 dB.
†Exposure results are ORs comparing highest to lowest group (excluding middle group), with 95% confidence limits.
‡Use of protection among those reporting exposure.
§For personal stereos, 'protection' is equated with volume control, from 100% ('low') to 0% ('very loud').
¶For chemicals, 'protection' includes respirators, gloves, apron and so on.

DISCUSSION

In this analysis, we had a rare opportunity to examine a diversity of occupational and recreational exposures as potential determinants of hearing loss in a young worker cohort. Over a 16-year period, we documented changes to hearing acuity that exceeded 15 dB at high frequencies in 42.8% of men and 27.7% of women. We demonstrated that members of this young worker cohort were exposed to a large number of recreational, occupational and chemical exposures that have established potential to cause or worsen hearing loss. Finally, analyses of risk factors for NIHL were limited to men, where we found that the risks increased in association with higher levels of almost all common recreational and occupational sources of noise as well as chemical exposures with ototoxic potential.

With respect to use of hearing protection (or other protective measures relevant to the exposure), participants in this study reported a wide variation in the use of protective devices, ranging from 3.8% to 67.3% for specific high-noise contexts and chemical exposures. Hearing protection, although not universal and sometimes modest, appeared to offer some protection to participants who used it. The use of protective devices appeared to mitigate the majority (13/18) of effect estimates in the expected direction (ie, those in table 4, column 10 with the estimated OR for protection <1.0). In manufacturing, which is more likely to be regulated, a statistically significant protective effect was observed (protection OR=0.65, 95% confidence limits 0.43 to 1.0).

Our decision to limit the primary analyses to men was based on two basic considerations. First, over two-thirds of participants in the cohort were men, so analyses specific to women had insufficient statistical power. This lack of power for women was compounded by the fact that exposures to the high-noise occupational and recreational activities that were the focus of this research are much more common in men than in women (table 2). Second, a priori we had strong reasons to believe that the strength and perhaps even the direction of associations may be different in women, which conceptually made it difficult to combine analyses involving men and women. As we (table 1) and others[1 2 10] have reported, even young men show substantially higher audiometric thresholds than do women. We[11] and others[12–15] have also observed that these gender differences are asymmetric, with most if not all the difference attributed to the left ear. Some researchers have in fact extended this observation through the reporting of gender differences in the physiology of the ear and central auditory nervous system,[16 17] which, if true, further raises the concern that fundamental biological differences by gender may exist with respect to some risk factors for NIHL. With both important exposures and thresholds known to differ substantially by gender, and the resulting potential for residual confounding of the effects of various exposures, we limited our primary analyses here to men since they represented 68% of the cohort.

Past longitudinal studies of NIHL and its potential causes are rather limited for young worker cohorts, making this trial and its 16-year audiometric follow-up somewhat unique to the biomedical literature. One notable exception is the contribution of Neitzel *et al*[18] who examined non-occupational exposures to noise as potential determinants of NIHL in construction workers using a person-years analytical approach. An estimated 19% of non-occupational exposures exceeded recommended occupational limits of 85 dB,[17] suggesting that recreational exposures may indeed contribute to overall noise experiences that have the potential to cause damage to hearing. Our findings of strong and consistent associations of specific recreational and occupational exposures with our measure of NIHL (ΔHF) support this contention.

The major strength of our study was its focus on early-stage NIHL in a young worker cohort. At 16 years, the length of longitudinal follow-up of our cohort was considerable and of sufficient latency to begin to identify a large number of young workers who are on a possible trajectory leading to NIHL as they age. Efforts to identify and recruit participants for follow-up were intensive and exhaustive. We also considered the rigour of our audiometric clinical testing to be a core methodological strength. After careful consideration of multiple options for the identification of NIHL, we employed a sensitive method to detect changes in hearing at high frequencies, consistent with the known natural history of early-stage NIHL. Our study also examined a diversity of occupational and recreational exposures simultaneously, with the intentions of identifying those exposures that are likely to be most important in the aetiology of NIHL. We demonstrated a lack of compliance with standard hearing protection practice in a variety of contexts, but we also provide some evidence that hearing protection, when used as intended, is efficacious.

Limitations of this analysis warrant comment. First, because the noise and chemical exposure histories were based on self-reports, there is obvious potential for misclassification that would bias effects estimates towards no effect. Despite this common limitation, our exposure measures were associated with ΔHF in the expected direction on a consistent basis, and as our audiometric measures were obtained in an objective and blinded fashion, this points to the likely validity of our key study measures. Although it is possible that misclassification errors on self-report may be smaller or less frequent for some exposures than for others, all exposures were recorded for the same time frame and in a form which was easy to complete and consistent within an exposure category (eg, occupational). Second, while the sample under study was potentially non-representative, there is no reason to suspect that this selection influenced the strength of relationships between the exposures and audiometric outcomes. Participants and non-participants were similar with respect to all key baseline factors,[7] in addition to short-term audiogram results obtained from the earlier 3-year follow-up. There is therefore no strong evidence of selection bias in this study. Third, while there is always the potential for residual confounding by unmeasured or unknown factors, adjustment was made for major known potential confounders (age, gender, smoking) via restriction and multivariate modelling. Fourth, our sample size provided limited ability to study the potential effects of some factors with precision, especially among women and for additional exposures where few cohort members were exposed. This also limited our ability to control for multiple covariates and many different exposures simultaneously. Finally, in terms of generalisability, the prevalence values for our estimates of NIHL are likely specific to this American Midwest occupational context. However, the presence of a biological effect between the exposures and early-stage NIHL could be generalised to other young worker populations.

CONCLUSIONS

Our study has several implications for occupational and public health. The findings suggest that early-stage NIHL can be detected in young workers using a sensitive and objective measure of high-frequency changes to hearing acuity, measured by audiometry. Such early detection in young workers could be efficacious in preventing further irreversible damage. Second, based on our findings and those of Neitzel *et al*,[18] hearing conservation programmes must expand to focus on a broader range of exposures, whether they occur in occupational or non-occupational settings. Priority exposures include gunshots, chainsaws, power tools, smoking and potentially some chemical exposures. From the employer's perspective, the costs of an expanded hearing conservation programme may be offset by both improvements in employee health and potential reductions in liability. Finally, we observed with some interest that there was little association between ongoing use of personal stereos and the occurrence of NIHL, while many other common exposures were implicated. However, we recognise that our ability to evaluate the long-term effects of personal stereos is limited in this cohort, given the relatively recent growth in their availability and popularity. Nonetheless, based on our results, researchers and practitioners must continue to consider the importance of and potential confounding with more 'traditional' risk factors when considering NIHL.

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Contributors BM, WP and RLB conceptualised and designed this study. BM and RLB supervised the acquisition of data. DJW and SRK evaluated all hearing tests. RLB and JGL conducted all data analyses. BM, WP and RLB interpreted the data and led the writing of the manuscript. All authors reviewed drafts of the manuscript, contributed to critical revision of the manuscript and provided assistance in all other aspects of the study.

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REFERENCES

1. **Niskar AS,** Kieszak SM, Holmes AE, *et al*. Estimated prevalence of noise-induced hearing threshold shifts among children 6 to 19 years of age: the Third National Health and Nutrition Examination Survey, 1988-1994, United States. *Pediatrics* 2001;**108**:40—3.
2. **Henderson E,** Testa MA, Hartnick C. Prevalence of noise-induced hearing-threshold shifts and hearing loss among US youths. *Pediatrics* 2011;**127**:e39—46.
3. **Shargorodsky J,** Curhan SG, Curhan GC, *et al*. Change in prevalence of hearing loss in US adolescents. *JAMA* 2010;**304**:772—8.
4. **National Institute for Occupational Safety and Health,** Centers for Disease Control and Prevention. *Work Related Hearing Loss*. DHHS (NIOSH), Publication number 2001-103. 2001. http://www.ced.gov/niosh/docs/2001-103
5. **Hass-Slavin L,** McColl MA, Pickett W. Challenges and strategies related to hearing loss among dairy farmers. *J Rural Health* 2005;**21**:329—36.
6. **Occupational Safety and Health Administration.** *Hearing Conservation*. OSHA Publication, 3074. U.S. Government Printing Office, Washington, DC. 2002.
7. **Marlenga B,** Linneman JG, Pickett W, *et al*. Cluster randomized trial of a hearing conservation intervention for Rural high school students: long-term outcomes. *Pediatrics* 2011;**128**:e1139—46.
8. **American National Standards Institute (R1997).** *American National Standard Methods for Manual Pure-Tone Threshold Audiometry*. ANSI S3.21—1978. Melville, NY: Acoustical Society of America, 1978. http://books.nap.edu/openbook.php?record_id=11099&page=281 (accessed 1 Apr 2009).
9. **Dobie RA.** Estimating noise-induced permanent threshold shift from audiometric shape: the ISO-1999 model. *Ear Hear* 2005;**26**:630—5.
10. **Rabinowitz PM,** Galusha D, Slade MD, *et al*. Audiogram notches in noise-exposed workers. *Ear Hear* 2006;**27**:742—50.
11. **Berg RL,** Pickett W, Fitz-Randolph M, *et al*. Hearing conservation program for agricultural students: short-term outcomes from a cluster-randomized trial with planned long-term follow-up. *Prev Med* 2009;**49**:546—52.
12. **Kannan PM,** Lipscomb DM. Bilateral hearing asymmetry in a large population. *J Acoust Soc Am* 1974;**55**:1092—4.
13. **Borod J,** Obler L, Albert M, *et al*. Lateralization for pure tone perception as a function of age and sex. *Cortex* 1983;**19**:281—5.
14. **Pirala T,** Jounio-Ervasti K, Sorri M. Left-righ asymmetries in hearing threshold levels in three age groups of a random population. *Audiology* 1992;**31**:150—61.
15. **McFadden D,** Mishra R. On the relation between hearing sensitivity and otoacoustic emissions. *Hear Res* 1993;**71**:208—13.
16. **Sato H,** Sando I, Takahashi H. Sexual dimorphism and development of the human cochlea. Computer 3-D measurement. *Acta Otolaryngol* 1991;**111**:1037—40.
17. **Morlet T,** Perrin E, Durrant JD, *et al*. Development of cochlear active mechanisms in humans differs between gender. *Neurosci Lett* 1996;**220**:49—52.
18. **Neitzel R,** Seixas N, Goldman B, *et al*. Contributions of non-occupational activities to total noise exposure of construction workers. *Ann Occup Hyg* 2004;**48**:463—73.

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