Cigarette- and Snus-Modified Association Between Unprotected Exposure to Noise from Hunting Rifle Caliber Weapons and High Frequency Hearing Loss. A Cross-Sectional Study Among Swedish Hunters

Louise Honeth1,2, Peter Ström3, Alexander Ploner3, Dan Bagger-Sjöbäck1, Ulf Rosenhall1,4, Olof Nyrén3

1Department of Clinical Science, Intervention and Technology, Division of Ear, Nose and Throat Diseases, Karolinska Institutet, Stockholm, 2ENT-center, Cityakuten, 3Department of Medical Epidemiology and Biostatistics, Karolinska Institutet, Stockholm, 4Department of Audiology and Neurotology, Karolinska University Hospital, Solna, Sweden

Abstract

Aim: To investigate in this cross-sectional study among Swedish hunters if tobacco use modifies the previously observed association, expressed as prevalence ratio (PR), between unprotected exposure to impulse noise from hunting rifle caliber (HRC) weapons and high-frequency hearing impairment (HFHI).

Settings and Design: A nationwide cross-sectional epidemiologic study was conducted among Swedish sport hunters in 2012. Materials and Methods: The study was Internet-based and consisted of a questionnaire and an Internet-based audiometry test. Results: In all, 202 hunters completed a questionnaire regarding the hearing test. Associations were modeled using Poisson regression. Current, daily use of tobacco was reported by 61 hunters (19 used cigarettes, 47 moist snuff, and 5 both). Tobacco users tended to be younger, fire more shots with HRC weapons, and report more hunting days. Their adjusted PR (1–6 unprotected HRC shots versus 0) was 3.2 (1.4–6.7), \( P < 0.01 \). Among the nonusers of tobacco, the corresponding PR was 1.3 (0.9–1.8), \( P = 0.18 \). \( P \) value for the interaction was 0.01. The importance of ear protection could not be quantified among hunters with HRC weapons because our data suggested that the HFHI outcome had led to changes in the use of such protection. Among hunters using weapons with less sound energy, however, no or sporadic use of hearing protection was linked to a 60% higher prevalence of HFHI, relative to habitual use. Conclusion: Tobacco use modifies the association between exposure to unprotected impulse noise from HRC weapons and the probability of having HFHI among susceptible hunters. The mechanisms remain to be clarified, but because the effect modification was apparent also among the users of smokeless tobacco, combustion products may not be critical for this effect.

Keywords: Audiometry, effect modifiers, firearms, hunters, internet, noise-induced hearing loss, snus, tobacco, tobacco smokeless

INTRODUCTION

A history of exposure to impulse noise with significant sound energy, such as from unprotected shooting noise from hunting rifle caliber (HRC) weapons, is associated with an increased prevalence of high-frequency hearing impairment (HFHI). Although susceptible individuals who experience acute effects tend to avoid further unprotected shooting with such weapons and thus report no more than a couple of such shots, others seem to withstand similar audio trauma without any adverse sequels; they typically report large numbers of unprotected shots with HRC weapons, but their audiometry results remain largely normal.

We postulated that this variation in susceptibility to impulse noise might be genetically determined, as seems to be the case when the noise is occupational.[2–4] However, there might also be external factors that modify the harmful impact and which are potentially amenable to public health interventions. Research has indicated that tobacco smoking aggravates...
We hypothesized that tobacco use, both in the form of snus/Swedish moist snuff and/or cigarettes, also modifies the detrimental effect of impulse noise.

Twenty percent of the Swedish population in the ages 16–84 years smoke tobacco daily or sporadically. Daily snus use among men is more common (19%), than among women (4%). Snus is typically administered as quids behind the upper lip and involves exposure to nearly the same chemicals as does tobacco smoking, with the important difference that there is no exposure to combustion products such as CO. The integrated nicotine levels during normal use of snus are similar to those measured during normal use of cigarettes. Levels of the harmful tobacco-specific nitrosamines (TSNAs), in modern Swedish snus are relatively low, as snus is based on low-nitrate tobacco and does not undergo any fermentation process. Snus use has been associated with higher blood pressure and endothelial dysfunction, which indicates vascular changes, but there are studies that contradict these results.

To the best of our knowledge, there is no published research on the association between snus use and Noise Induced Hearing Loss (NIHL).

The first aim of this study was to investigate differences between hunters with and without HFHI in regard to patterns of exposure to modifiable external factors. Our second aim was to test the hypothesis that tobacco use effect modifies the association, expressed as prevalence ratio (PR), between a history of unprotected exposure to impulse noise from HRC weapons and probability of having HFHI. The third aim was to investigate if the effect modification, if any, by tobacco smoking looks different from that caused by use of Swedish moist snuff (snus).

**MATERIALS AND METHODS**

In 2012–2013, we conducted an Internet-based cross-sectional study of shooting habits and hearing impairment among Swedish hunters. E-mail invitations were sent to 27,063 hunters with e-mail addresses in the membership roster of the Swedish Hunters Association. Nonresponders received two e-mail reminders. The participants first completed a comprehensive questionnaire with detailed questions about hunting and shooting habits in the preceding 5 years, socio-demographic characteristics, and other possibly confounding factors as listed in Table 1. The questionnaire was followed by a self-administered Internet-based audiometry test (the InternetAudio test).

The InternetAudio test

The InternetAudio test was delivered from the research project's website. The participants used their own home computer with headphones. The setup was first calibrated by using a normal-hearing reference person (RP) as an analogue to the zero decibel reference value. The calibration process compensated for the variation in computer equipment and in the headphones’ frequency span variation, as well as ambient background noise levels.

The RP determined his/her hearing level (HL) with a volume marker on a ruler of 300° on the computer screen. The tone at each frequency was presented to both ears simultaneously to get the reference hearing level (RP-HL) from the better ear. The volume marker had to be adjusted to the level at which the tone was barely audible. For the calibration to be accepted, the following four separate criteria had to be met: (1) The volume marker had to be moved at every frequency; (2) The dB values between all tested frequencies should not exceed a range of 15 dB; (3) The volume marker was not allowed to have exactly the same dB value on more than two frequencies. The latter requirement was to rule out visual positioning of the marker. (4) The process was done twice and should have similar results. The RP-HL was considered to be the zero level decibel for this computer, headphones, and environment. Hence, the hearing test measured the differences in frequency-specific HLs between the tested participant and the RP.

Test persons were presented to tones to each ear separately in random order and at random intervals. Six frequencies were tested (0.5, 1, 2, 4, 6, and 8 kiloHertz (kHz)). The sound level ranged between 0 and 60 decibel-sound pressure levels (dB SPL). The test person pressed the keyboard space bar when hearing. If the tone was not heard, it was presented at a level that was higher by 10 dB. If the tone was heard, it was presented at a level that was lower by 5 dB. The HL was defined as the lowest unambiguously perceptible SPL at the specific frequency. The InternetAudio test has been formally validated, with a clinical pure-tone air-conducted audiometry as gold standard, in 72 volunteers with a broad range of hearing ability, resulting in satisfying results. The test was also further validated under authentic conditions during the present survey with similar results as the formal validation.

**Measurements**

On the basis of the results of our previously published analysis, the main exposure of interest was the number of shots with HRC weapons (generating the highest SPL in sports hunting) fired without hearing protection in the preceding 5 years, categorized into 0, 1–6, and >6. The outcome was the presence of HFHI, ascertained by the InternetAudio test. HFHI, the typical hearing impairment after high-energy impulse noise, was defined as a HL exceeding 20 dB for either 4 or 6 kHz. Because the aim of the investigation was to study a possible interaction with noise, we restricted the analysis of the influence of tobacco to these noise frequencies.

Tobacco use was the suspected effect-modifying factor. An affirmative answer to the question “do you smoke cigarettes daily? yes/no/don’t know,” rendered a classification as “cigarette user.” This class also included participants who reported smoking cessation in the preceding 5 years. Hunters who answered that they had never smoked or had stopped smoking longer than 5 years ago were classified as “cigarette
We considered all variables listed in Table 1 as potential confounding factors. Because of small numbers in some cells in stratified analyses, the response alternatives to a question about use of hearing protection while hunting (always, often, sometimes, rarely, or never) were collapsed into always/often versus sometimes/rarely/never. The responses about the average number of hunting days (0, 1–5, 6–10, 11–20, or >20 days) were dichotomized into 0–20 days versus >20 days per year. Similarly, the responses to an open-ended question about the average number of shots overall per year (HRC, magnum, and small-caliber weapons, during hunting or training and regardless of ear protection) were categorized as 0–50, 51–100, and >100. For shotguns, the response alternatives were already given (0–100, 101–300, 301–500, or >500). Age, reported as a discrete numerical variable, was first categorized as 11–35, 36–55, 56–75, or 76–91 years; but because its relation to HFHI was apparently linear, it was used as a continuous linear term in the final models. Other variables were categorized as in Table 1.

| Table 1: Distributions of significant exposures — both related to shooting noise and to other factors suspected of being related to hearing loss — among hunters with and without high-frequency hearing impairment (HFHI), along with prevalence ratios (PR) and 95% confidence intervals (CI) as measures of age-adjusted associations between the exposures and HFHI. The analysis is stratified according to the main exposure, namely having fired at least one shot in the preceding 5 years with an hunting rifle caliber weapon without ear protection (n = 202) |
|---|
| **Not exposed to the unprotected sound blast from a shot with a hunting rifle caliber weapon in the preceding 5 years (n = 69)** |
| **Exposed to at least one unprotected sound blast from a hunting rifle caliber weapon in the preceding 5 years (n = 133)** |
| **HFHI n/%** | **No HFHI n/%** | **Age-adjusted PR (95% CI)** | **P-value** | **HFHI n/%** | **No HFHI n/%** | **Age-adjusted PR (95% CI)** | **P-value** |
| Overall | 67 | 66 | | | | |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years | | | | | | |
| 0 | 30/100 | 39/100 | n.a. | 0 | 0 | n.a. |
| 1–6 | 0 | 0 | 28/42 | 16/24 | 1.4 (1.1–1.9) | 0.02 |
| >6 | 0 | 0 | 39/58 | 50/76 | 1 (reference) | |
| Number of hunting days per year | | | | | | |
| 0–20 | 18/60 | 29/74 | 0.5 (0.3–0.8) | <0.01 | 28/42 | 19/29 | 1.1 (0.8–1.5) | 0.62 |
| >20 | 12/40 | 10/26 | 1 (reference) | | 39/58 | 47/71 | 1 (reference) | |
| Ear protection use during hunting | | | | | | |
| Always/often | 24/80 | 37/95 | 1 (reference) | 27/40 | 21/32 | 1 (reference) | |
| Sometimes/rarely/never | 6/20 | 2/5 | 1.6 (1.1–2.5) | 0.02 | 40/60 | 45/68 | 0.7 (0.5–0.9) | 0.02 |
| Tobacco use* | | | | | | |
| Yes | 5/17 | 13/33 | 1.0 (0.4–2.2) | 0.97 | 16/24 | 27/41 | 1.0 (0.6–1.5) | 1.0 |
| No | 25/83 | 26/67 | 1 (reference) | 51/76 | 39/59 | 1 (reference) | |
| Sex | | | | | | |
| Men | 25/83 | 30/77 | 1 (reference) | 63/95 | 59/89 | 1 (reference) | |
| Women | 5/17 | 9/23 | 0.8 (0.4–1.7) | 0.62 | 4/75 | 7/11 | 1.1 (0.5–2.5) | 0.82 |
| Age categories (not age-adjusted) | | | | | | |
| 11–35 | 1/3 | 7/18 | 0.2 (0.0–1.1) | 0.06 | 2/3 | 12/18 | 0.2 (0.1–0.7) | 0.02 |
| 36–55 | 4/13 | 23/59 | 0.2 (0.1–0.5) | <0.01 | 23/34 | 40/61 | 0.5 (0.3–0.7) | <0.01 |
| 56–75 | 22/73 | 8/21 | 1 (reference) | 38/57 | 14/21 | 1 (reference) | |
| 76–91 | 3/10 | 1/3 | 1.0 (0.6–1.9) | 0.94 | 4/6 | 0 | 1.4 (1.2–1.6) | <0.01 |
| Family history of impaired hearing | | | | | | |
| Yes | 12/40 | 6/15 | 1.8 (1.1–2.9) | 0.02 | 13/19 | 15/23 | 0.9 (0.6–1.4) | 0.65 |
| No | 5/53 | 29/75 | 1 (reference) | 48/72 | 43/65 | 1 (reference) | |
| Don’t know | 2/7 | 4/10 | | | 6/9 | 8/12 | | |
| Highest education level | | | | | | |
| Primary school | 5/17 | 7/18 | 0.7 (0.3–1.3) | 0.25 | 17/25 | 6/9 | 0.9 (0.6–1.2) | 0.44 |
| High school | 10/33 | 9/23 | 1.5 (0.9–2.5) | 0.10 | 13/21 | 37/56 | 0.6 (0.4–1.0) | 0.03 |
| University | 15/50 | 23/59 | 1 (reference) | 36/54 | 23/35 | 1 (reference) | |

n.a= not applicable; †Column %; *Use of cigarettes or snuff or both currently or in the preceding 5 years. Bold values are significant (<0.05).
**Statistical methods**

We estimated the effect of shooting noise on HFHI, expressed as PR. PRs were estimated using a Poisson model with robust standard errors,\(^2^2\) which provided a less biased estimate than the more commonly used odds ratios from a standard logistic regression model.\(^2^3\) The PRs reported are obtained as the exponential function of the regression coefficients of the Poisson model. After fitting simple age and sex-adjusted models of main effects only, other potential confounding covariates as history of noise at work past or present, noise during army service, tinnitus after army service or tinnitus after leisure activities, history of Menière’s disease, sudden deafness, vestibular schwannoma, ear surgery, chronic otitis media or ear disease as a child, history of myocardial infarction and/or hypertension and/or use of anticoagulants, history of meningitis, migraine, head trauma, epilepsy, joint disease, or cancer were tested in the models. As none of these potential confounders changed the effect estimates of association materially, only age (as a linear term) and sex were retained. Finally, interaction terms corresponding to various aspects of tobacco use and shooting noise exposure were introduced, supplemented by corresponding stratified analyses. As prevalence of hearing impairment is most strongly affected by age, we performed multiple goodness-of-fit analyses to account for possible nonlinear age effects. The statistical analyses were conducted using Statistical Analysis System (SAS) software, version 9.4, of the SAS System for Windows software (SAS Institute Inc., 100 SAS Campus Drive Cary, NC, USA).

The study was approved by the Stockholm Ethics Vetting Board.

**RESULTS**

The numbers of invitees, nonresponders, and excluded hunters are shown in Figure 1. The questionnaire was answered by 1771 hunters (aged 11–91 years), and 203 of them also completed the InternetAudio test. One participant was excluded because of technical evidence that the InternetAudio test had been erroneously executed.

Table 1 highlights the distribution of significant exposures of shooting-related, hereditary, and socio-demographic exposures among hunters with and without HFHI. The table is stratified according to whether or not the hunters had been exposed to the unprotected sound blast from at least one shot with an HRC weapon in the preceding 5 years. Within each stratum, age-adjusted PRs express the crude association between each exposure and HFHI. Although the only truly conspicuous difference between hunters with and without HFHI was markedly differing age distributions, seen in both strata, few other statistically significant differences emerged.

Among hunters exposed to unprotected noise from HRC weapons, a much larger proportion of those with HFHI than of those without had only fired 1–6 such shots. The former were also more inclined to use ear protection during hunting than the latter. Among those who had apparently endured at least one unprotected sound blast from an HRC weapon without HFHI, we observed a somewhat unexpected accumulation of hunters with high school (9–12 years) as their highest attained education, and a corresponding deficit of people with a university education.

Of the hunters who had not been exposed to the unprotected noise from HRC weapons in the past 5 years, those with HFHI were significantly more likely to report many (>20) hunting days per year and to wear ear protection less often during hunting than did those without HFHI. The unexposed hunters with HFHI did also report the existence of a relative with hearing impairment significantly more often than the unexposed hunters without HFHI. In neither of the groups did we observe any direct association between smoking and prevalence of HFHI.

Table 2 particularizes the distributions of relevant exposure variables in categories of tobacco use. Eighty-four hunters had stopped using tobacco (60 cigarettes and 24 snus) more than 5 years ago and were thus classified as nonusers. Additionally, 18 hunters went from cigarettes to snus, and one hunter went from snus to cigarettes. Of the 19 current cigarette smokers, five smokers also used snus daily. The prevalence of snus use was more than twice as high as that of cigarette smoking, but the age distributions were similar in the two groups. Compared to the nonusers, tobacco users tended to be younger (mean age 46.2 versus 55.5 years,
Table 2: Distributions of shooting-related exposure variables as well as sex and age in categories of tobacco use among 202 hunters who underwent the InternetAudio hearing test

| Tobacco use | Overall | No tobacco use n/% | Cigarettes n/% | Snuff n/% | Any tobacco use (cigarettes and/or snuff) n/% |
|-------------|---------|--------------------|---------------|----------|---------------------------------------------|
|             | 202     | 141/100            | 19/100        | 47/100   | 61*/100                                     |

| Exposure to shooting noise from hunting rifle caliber weapons |
|---------------------------------------------------------------|
| Number of unprotected HRC shots in the preceding 5 years      |
| 0                 | 51/36 | 9/47 | 10/21 | 18/29 |
| 1–6               | 35/25 | 1/5  | 9/19  | 9/15  |
| >6                | 55/39 | 9/47 | 28/60 | 34/56 |
| Total number of HRC shots (hunting and training), on average per year in the preceding 5 years, regardless of hearing protection |
| 0–50              | 39/28 | 4/21 | 3/6   | 7/12  |
| 51–100            | 34/24 | 3/16 | 10/21 | 13/21 |
| >100              | 68/48 | 12/63| 34/72 | 41/67 |
| Number of hunting days per year                                |
| 0–20              | 78/55 | 6/32 | 10/21 | 16/26 |
| >20               | 63/45 | 13/68| 37/79 | 45/74 |
| Ear protection use during hunting                               |
| Always/often       | 77/55 | 12/63| 22/47 | 32/52 |
| Sometimes/rarely/never | 64/45 | 7/37 | 25/53 | 29/48 |
| Sex                |
| Men                | 123/87| 14/74| 45/96 | 54/89 |
| Women              | 18/13 | 5/26 | 2/4   | 7/11  |
| Age categories     |
| 11–35              | 14/10 | 2/11 | 7/15  | 8/13  |
| 36–55              | 52/37 | 12/63| 29/62 | 38/62 |
| 56–75              | 67/47 | 5/26 | 11/23 | 15/25 |
| 76–91              | 8/6   | 0    | 0     | 0     |

*Column %.* *Five hunters used both cigarettes and snuff.

P < 0.001, fire more shots with HRC weapons (both unprotected [P = 0.07] and overall [P = 0.02]), and report more hunting days (P < 0.001), compared to the nonusers. There were neither important differences between the groups in regard to reported frequency of hearing protection utilization during hunting, nor was there any difference in type of protection; 86% of hunters in both groups used electronic earmuffs (data not shown).

Table 3 addresses effect modification, by tobacco use, of the PR relationship between self-reported unprotected exposure to the sound blast from an HRC weapon in the preceding 5 years and the probability of having HFHI. Without any stratification for tobacco use, the age- and sex-adjusted PR was 1.5 (1.0–2.1; P = 0.02) in the 1–6 unprotected shots category, relative to the unexposed reference category (data not shown). Stratification according to any tobacco use (upper third of the table) disclosed a lower PR estimate (1.3) among the nonusers of tobacco, but a considerably higher estimate (3.2) among tobacco users, however based on only nine hunters in the exposed category. Despite the small sample size, the interaction term attained statistical significance (P = 0.01). Our attempt to estimate effect modification by cigarette use and snus use separately (lower two-thirds of the Table 3) was hampered by small numbers. Snus use was associated with a higher PR point estimate (2.3) among the users than among the nonusers (1.3), but the snus * shooting interaction term was nonsignificant (P = 0.09). The PR estimate for the 1–6 unprotected shots category in the cigarette smoking stratum was based on only one exposed hunter, prohibiting any meaningful statistical inference. A supplementary analysis of the possible effect modification by snus, excluding the 19 smokers (current and/or stopped smoking in the preceding 5 years), somewhat reinforced the difference between the nonuser and user strata; the PR estimate for the 1–6 unprotected shots category in the non-user stratum was 1.3 (95% confidence interval (CI) 0.9–1.9), and in the user stratum it was 2.7 (95% CI 1.1–6.7) (P for the interaction term = 0.05) (data not shown).

Discussion

In this cross-sectional epidemiological study, we observed evidence for a modifying role of tobacco in the inner ear’s ability to withstand extreme impulse noise, notably unprotected sound blasts from HRC weapons. Among tobacco users, a history of 1–6 unprotected shots was associated with a significant 220% increase in the age-adjusted HFHI PR, relative to 0 such shots, while among tobacco nonusers, the corresponding excess was no more than a nonsignificant 30%. Small numbers prohibited us from statistically verifying any
Effect modification by cigarette smoking alone, but we noted that the effect modification of the PR was nearly significant also among exclusive snus users. Because only a minority among tobacco users smoked, the effect modification was likely driven to a large extent by snus use. This makes us tentatively conclude that combustion products, for example, CO, are not critical.

Tobacco use was not in itself associated with an increased prevalence of HFHI, neither among hunters exposed to unprotected noise from HRC weapons, nor among those who were unexposed to such noise.

Both the main effect of unprotected shooting with HRC weapons and the effect modification were seemingly confined to the 1–6 shot stratum. The majority of hunters exposed to the unprotected noise from HRC weapons reported more than six shots in the preceding 5 years — some substantially more. Less than half of them showed evidence of HFHI upon InternetAudio testing. However, a much larger proportion of exposed hunters with HFHI than exposed hunters without HFHI fell in the 1–6 shots category. This led us to believe that many susceptible hunters who

Table 3: Relative risks (prevalence ratios) of having HFHI (a hearing impairment >20 dB on 4 and/or 6 kHz) by hunting noise exposure status (unprotected sound blast from hunting rifle caliber weapons in the preceding 5 years), stratified according to self-reported tobacco use

| Hunters with HFHI in category n/% | Hunters without HFHI in category n/% | Prevalence ratio with 95% CI (within parentheses) | P-value for interaction term |
|----------------------------------|-------------------------------|-----------------------------------------------|-----------------------------|
|                                  |                               | Age- and sex-adjusted | P-value |
| Overall 202                      | 97                            | 105                           |                |
| Any tobacco use                  |                               |                               |                |
| No 141                           | 76/54                         | 65/46                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 25/49                         | 26/51                         | 1 (reference)   |                |
| 1–6                              | 21/60                         | 14/40                         | 1.3 (0.9–1.8)  | 0.18           |
| >6                               | 30/56                         | 25/45                         | 1.1 (0.8–1.5)  | 0.57           |
| Yes 61                           | 21/34                         | 40/66                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 5/28                          | 13/72                         | 1 (reference)   |                |
| 1–6                              | 7/78                          | 2/22                          | 3.2 (1.4–6.7)  | <0.01          |
| >6                               | 9/26                          | 25/74                         | 1.0 (0.4–2.3)  | 0.95           |
| Cigarettes                       |                               |                               |                |
| No 183                           | 92/50                         | 91/50                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 28/47                         | 32/53                         | 1 (reference)   |                |
| 1–6                              | 27/63                         | 16/37                         | 1.4 (1.0–2.0)  | 0.03           |
| >6                               | 37/46                         | 43/54                         | 1.1 (0.8–1.4)  | 0.69           |
| Yes 19                           | 5/26                          | 14/74                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 2/22                          | 7/78                          | 1 (reference)   |                |
| 1–6                              | 1/100                         | 0                             | 4.2*            |                |
| >6                               | 2/22                          | 7/78                          | 0.9 (0.2–4.8)  | 0.91           |
| Snus                             |                               |                               |                |
| No 155                           | 77/49                         | 78/50                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 26/44                         | 33/56                         | 1 (reference)   |                |
| 1–6                              | 21/60                         | 14/40                         | 1.3 (0.9–1.9)  | 0.11           |
| >6                               | 30/50                         | 31/51                         | 1.1 (0.8–1.5)  | 0.63           |
| Yes 47                           | 20/43                         | 27/57                         |                |
| Number of unprotected shots with hunting rifle caliber in the preceding 5 years |                               |                               |                |
| 0                                | 4/40                          | 6/60                          | 1 (reference)   |                |
| 1–6                              | 7/78                          | 2/22                          | 2.3 (1.0–5.0)  | 0.04           |
| >6                               | 9/32                          | 19/68                         | 0.9 (0.4–2.0)  | 0.78           |

*Too few cases to estimate CI or P-value. Bold values are significant (<0.05).
experienced acute subjective hearing impairment after an unprotected shot with HRC weapons likely stopped such unprotected shooting, while those with resistant ears could continue. Consistent with this interpretation is the greater inclination among the former to use ear protection during hunting compared to the latter, leading to a false impression that sporadic use of ear protection is more protective than frequent use. This paradox – basically attributable to reverse causation – was not observed in the group of hunters who negated a history of unprotected shooting with HRC weapons in the preceding 5 years; on the contrary, such unexposed hunters who used ear protection only sporadically during hunting had a significant 60% higher prevalence of HFHI than unexposed hunters who used ear protection always or often. Likewise, the prevalence of HFHI among unexposed hunters reporting >20 hunting days per year was twice as high as among those reporting 0–20 hunting days. As shooting without protection occurs only during hunting and essentially never during training, this suggests that unprotected shooting with non-HRC weapons may also entail some risk for HFHI.

The main limitation of this study is the low participation, leading to both an increased risk of selection bias and poor precision of observed estimates. Specifically, the small number of participants prevented deeper statistical analysis in subgroups of tobacco users. Participation, not the least execution of the hearing test, was demanding and deemed to be too time consuming by the majority of the invited hunters. Selection bias occurs when the participation or response inclination is linked to both exposure and outcome or their causative factors. A brief questionnaire to the nonparticipants did not signal any important differences between participants and nonparticipants in terms of shooting exposure or subjective hearing ability, but the response rate was insufficient to ensure representativeness vis-à-vis all nonparticipants. The prevalence of cigarette smoking and snus use among participants was well in line with Swedish statistics. Our overall evaluation was that the observed effect modification was not likely to be attributable to selection bias, but our results must be cautiously interpreted.

Given the cross-sectional design, involving retrospective exposure recollection over a 5-year period, the risk of reverse causation, recall bias, and nondifferential misclassification (e.g., number of shots per caliber, use of hearing protection, and number of hunting days), must be kept in mind. Although nondifferential misclassification would generally lead to underestimation of studied associations, the direction of errors caused by reverse causation and recall bias is less predictable.

The accuracy of the InternetAudio test is clearly inferior to that of a clinical audiogram, but two attempts to validate the test against clinical pure-tone audiograms suggest that the accuracy is acceptable. However, both validation studies showed that the InternetAudio test tends to underestimate the hearing threshold levels. Moreover, if the test person would simply abandon the test and leave the home computer running without any responses, the result could be interpreted as a severe hearing loss on all frequencies. Another concern is that some test persons could have acted as their own RPs in the calibration, contrary to the instructions. The test result would then be interpreted as normal hearing regardless of the person’s hearing ability. Because these causes of misclassification of the outcome are not very likely to be linked to the studied exposure, the error is in all probability nondifferential. Such errors tend to attenuate studied associations.

Abundant evidence links noise-induced hearing loss to smoking, but available data concern continuous noise. Some of the observational studies on continuous noise and smoking in humans reported a dose–response pattern. In this study, it was found that the interactive effect of tobacco also includes impulse noise.

Mechanisms behind the smoking-associated potentiation of the detrimental effects of continuous noise on hearing are debated and probably multifactorial. Tobacco contains, apart from nicotine, different types of TSNAs. TSNAs are mainly known as potent carcinogens, by damaging the cell’s deoxyribonucleic acid (DNA). This cytotoxic effect can be potentiated in cell cultures by free radicals. Cigarette smoke contains carbon monoxide (CO), which has been shown to induce hearing loss in high doses. CO and nicotine are both factors influencing the microcirculation in the organ of Corti, leading to a decrease of the local circulation as well as affecting the formation of free radicals adding to the oxidative stress, which is already caused by noise exposure. Moreover, smoking causes a general inflammatory process in the body, which also might involve the cochlea. However, human provocation experiments have revealed a smaller temporary threshold shift (TTS) upon noise exposure among smokers than among nonsmokers after exposure noise but not after chewing a nicotine gum, which had an opposite effect on nonsmokers (larger TTS). Profound and dose-dependent damages in guinea pigs’ cochlear hair cells and in the supportive cells have been reported after subcutaneous injections of nicotine and examination with electron microscopy. Our results, in which the effect modification seems to be driven mainly by snus (as the majority of the tobacco users were snus users), seem to contradict the hypothesis that combustion products, such as CO, are necessary for the tobacco-associated vulnerability to high-energy impulse noise. Both smoking and snus use reportedly induce vascular and hemodynamic changes that might result in cochlear hypoxia. Cochlear hypoxia could possibly add to the oxidative stress in the cochlea, which is known to already be caused by noise. It has been demonstrated that it is possible to “protect” against blast-induced cochlear damage with an antioxidant cocktail that enhances the TTS recovery and reduces the permanent threshold shift (probably by reducing the oxidative stress caused by noise). Thus the notion that tobacco use

could have an opposite effect and increase oxidative stress, making the cochlea even more vulnerable among susceptible individuals, is not far-fetched.

To conclude, this study suggests that tobacco use, at least snus use, brings about important modification of the association between exposure to unprotected impulse noise from HRC weapons and the probability of having HFHI – an association that is seen only among susceptible individuals. The mechanisms remain to be clarified. Although the importance of hearing protection could not be quantified as a result of post-trauma-induced changes in the use of such protection (reverse causation) among hunters using HRC weapons, there were indications that habitual use of hearing protection might be protective among hunters using weapons with less sound energy.

Acknowledgments

The authors wish to thank Ann Johansson, audiologist, Division of Audiology, Karolinska University Hospital, the Tysta Skolan Foundation, the Acta Oto-Laryngologica Foundation, and the Swedish Research Council.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Honeth L, Ström P, Ploner A, Bagger-Sjöback D, Rosenhall U, Nyren O. Shooting history and presence of high-frequency hearing impairment in Swedish hunters: A cross-sectional Internet-based observational study. Noise Health 2015;17:273-81.
2. Kowalski TJ, Pawelczyk M, Rajkowska E, Dudarowicz A, Sliwinska-Kowalska M. Genetic variants of CDH23 associated with noise-induced hearing loss. Otol Neurotol 2014;35:358-65.
3. Konings A, Van Laer L, Van Camp G. Genetic studies on noise-induced hearing loss: A review. Ear Hear 2009;30:151-9.
4. Sliwinska-Kowalska M, Pawelczyk M. Contribution of genetic factors to noise-induced hearing loss: A human studies review. Mutat Res 2013;752:61-5.
5. Rosenhall U, Sixt E, Sundh V, Svansborg A. Correlations between presbyacusis and extrinsic noxious factors. Audiology 1993;32:234-43.
6. Cruickshanks KJ, Klein R, Klein BE, Wiley TL, Nondahl DM, Tweed TS. Cigarette smoking and hearing loss: The epidemiology of hearing loss study. JAMA 1998;279:1715-9.
7. Agrawal Y, Platz EA, Niparko JK. Risk factors for hearing loss in US adults: Data from the National Health and Nutrition Examination Survey, 1999 to 2002. Otol Neurotol 2009;30:139-45.
8. Fransen E, Topsakal V, Hendricks JJ, Van Laer L, Huyghe JR, Van Eyken E, et al. Occupational noise, smoking, and a high body mass index 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;9:264-76.
9. Mohammadi S, Mazhari MM, Mehrparvar AH, Attarchi MS. Effect of simultaneous exposure to occupational noise and cigarette smoke on binaural hearing impairment. Noise Health 2010;12:187-90.
10. Tao L, Davis R, Heyer N, Yang Q, Qiu W, Zhu L, et al. Effect of cigarette smoking on noise-induced hearing loss in workers exposed to occupational noise in China. Noise Health 2013;15:67-72.
11. Mehrparvar AH, Mirmohammadi SJ, Hashemi SH, Davari MH, Mostaghaci M, Mollasadeghi A, et al. Concurrent effect of noise exposure and smoking on extended high-frequency pure-tone thresholds. Int J Audiol 2014;1-7.
12. Tobacco Habits. https://www.folkhalsomyndigheten.se/folkhalsorrapp ortering-statistik/statistikdatabaser-och-visualisering/nationella-folk halsenkaten/levnadsvanor/tobaksvanor/. [Last accessed 2016 Mar 4].
13. Agewall S, Persson B, Lindstedt G, Fagerberg B. Smoking and use of smokeless tobacco in treated hypertensive men at high coronary risk: Utility of urinary cotinine determination. Br J Biomed Sci 2002;59:145-8.
14. Lee PN. Summary of the epidemiological evidence relating snus to health. Regul Toxicol Pharmacol 2011;59:197-214.
15. Bolinder G, Norén A, Wahren J, De Faire U. Long-term use of smokeless tobacco and physical performance in middle-aged men. Eur J Clin Invest 1997;27:427-33.
16. Cranberry MC, Smith ES 3rd, Troillet RD, Eitd JF. Forearm endothelial response in smokeless tobacco users compared with cigarette smokers and nonusers of tobacco. Pharmacotherapy 2003;23:974-8.
17. Skaug EA, Nes B, Aspnes ET, Ellingsen O. Non-smoking tobacco affects endothelial function in healthy men in one of the largest health studies ever performed; The Nord-Trøndelag Health Study in Norway; HUNT3. PLOS ONE 2016;11:e0160205.
18. Eliasson M, Lundblad D, Hägg E. Cardiovascular risk factors in young snuff-users and cigarette smokers. J Intern Med 1991;230:17-22.
19. Ashlund K. Smokeless tobacco and cardiovascular disease. Prog Cardiovasc Dis 2003;45:383-94.
20. Standardisation IIOf. ISO 8253-1, Acoustics – Audometric Test Methods – Part 1: Basic Pure Tone Air and Bone Conduction Threshold Audiometry. Geneva: ISO; 1989.
21. Honeth L, Bexelius C, Eriksson M, Sandin S, Litton JE, Rosenhall U, et al. An Internet-based hearing test for simple audiometry in nonclinical settings: Preliminary validation and proof of principle. Otol Neurotol 2010;31:708-14.
22. Pou G. A modified poisson regression approach to prospective studies with binary data. Am J Epidemiol 2004;159:702-6.
23. Barros AJ, Hirakata VN. Alternatives for logistic regression in cross-sectional studies: An empirical comparison of models that directly estimate the prevalence ratio. BMC Med Res Methodol 2003;3:21.
24. Bexelius C, Honeth L, Ekman A, Eriksson M, Sandin S, Bagger-Sjöback D, et al. Evaluation of an Internet-based hearing test – Comparison with established methods for detection of hearing loss. J Med Internet Res 2008;10:e32.
25. Norberg M, Malmberg G, Ng N, Brostrom G. Who is using snus? – Time trends, socioeconomic and geographic characteristics of snus users in the ageing Swedish population. BMC Public Health 2011;11:929.
26. Danielsson M, Gilljam H, Hemström O. Tobacco habits and tobacco-related diseases: Health in Sweden: The National Public Health Report 2012. Chapter 10. Scand J Public Health 2012;34(Suppl):197-210.
27. Ohgami N, Kondo T, Kato M. Effects of light smoking on extra-high-frequency auditory thresholds in young adults. Toxicol Ind Health 2011;27:143-7.
28. Ahotupa M, Bussacchini-Griot V, Béréziat JC, Camus AM, Bartsch H. Rapid oxidative stress induced by N-nitrosamines. Biochem Biophys Res Commun 1987;146:1047-54.
29. Sheweita SA, El-Bendery HA, Mostafa MH. Novel study on N-nitrosamines as risk factors of cardiovascular diseases. BioMed Res Int 2014;2014:817019.
30. Arimilli S, Damratoski BE, Bomnick B, Borgerding MF, Prasad GL. Evaluation of cytotoxicity of different tobacco product preparations. Regul Toxicol Pharmacol 2012;64:350-60.
31. Hoffmann D, Djordjevic MV. Chemical composition and carcinogenicity of smokeless tobacco. Adv Dent Res 1997;11:322-9.
32. Weitberg AB, Corvese D. The effect of epigallocatechin gallate and sarcophytol A on DNA strand breakage induced by tobacco-specific nitrosamines and stimulated human phagocytes. J Exp Clin Cancer Res 1999;18:433-7.
33. Weitberg AB, Corvese D. Oxygen radicals potentiate the genetic toxicity of tobacco-specific nitrosamines. Clin Genet 1993;43:88-91.
34. Shahbaz Hassan M, Ray J, Wilson F. Carbon monoxide poisoning and sensorineural hearing loss. J Laryngol Otol 2003;117:134-7.
35. Csiszar A, Podlutsky A, Wolin MS, Losonczy G, Pacher P, Ungvari Z. Oxidative stress and accelerated vascular aging: Implications for cigarette smoking. Front Biosci (Landmark Ed) 2009;14:3128-44.
36. Csordas A, Wick G, Laufer G, Bernhard D. An evaluation of the clinical evidence on the role of inflammation and oxidative stress in smoking-mediated cardiovascular disease. Biomark Insights 2008;3:127-39.
37. Dengerink HA, Lindgren FL, Axelsson A. The interaction of smoking and noise on temporary threshold shifts. Acta Otolaryngol 1992;112:932-8.
38. Abdel-Hafez AM, Elgayar SA, Husain OA, Thabet HS. Effect of nicotine on the structure of cochlea of guinea pigs. Anat Cell Biol 2014;47:162-70.
39. Quirk WS, Shivapuja BG, Schwimmer CL, Seidman MD. Lipid peroxidation inhibitor attenuates noise-induced temporary threshold shifts. Hear Res 1994;74:217-20.
40. Ewert DL, Lu J, Li W, Du X, Floyd R, Kopke R. Antioxidant treatment reduces blast-induced cochlear damage and hearing loss. Hear Res 2012;285:29-39.
41. Lu J, Li W, Du X, Ewert DL, West MB, Stewart C, et al. Antioxidants reduce cellular and functional changes induced by intense noise in the inner ear and cochlear nucleus. J Assoc Res Otolaryngol 2014;15:353-72.