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Occupational noise exposure and Raynaud's phenomenon: a nested case–control study

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ABSTRACT

The primary aim of this study was to determine if self-reported occupational noise exposure was associated with Raynaud's phenomenon. In northern Sweden, a nested case–control study was performed on subjects reporting Raynaud's phenomenon (N=461), and controls (N=763) matched by age, sex and geographical location. The response rate to the exposure questionnaire was 79.2%. The study showed no statistically significant association between occupational noise exposure and reporting Raynaud's phenomenon (OR 1.10; 95% CI 0.83–1.46) in simple analyses. However, there was a trend towards increasing OR for Raynaud's phenomenon with increasing noise exposure, although not statistically significant. Also, there was a significant association between noise exposure and hearing loss (OR 2.76; 95% CI 2.00–3.81), and hearing loss was associated with reporting Raynaud's phenomenon (OR 1.52; 95% CI 1.03–2.23) in a multiple regression model. In conclusion, self-reported occupational noise exposure was not statistically significantly associated with Raynaud's phenomenon, but there was a dose–effect trend. In addition, the multiple model showed a robust association between hearing loss and Raynaud's phenomenon. These findings offer some support for a common pathophysiological background for Raynaud's phenomenon and hearing loss among noise-exposed workers, possibly through noise-induced vasoconstriction.

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Introduction

Classification

Raynaud's phenomenon (RP) is a common clinical disorder, defined as episodic, symmetrical vasospasm of mainly the fingers [1]. The condition can be classified as primary, without any associated disease, or secondary, if an associated condition has been diagnosed. Secondary RP can be related to rheumatic diseases, haematological conditions, exposure to certain chemicals and drugs, as well as occupational exposure to hand-arm vibration (HAV) [2–5]. Studies in different countries have reported prevalence rates of RP in the general population between 2% and 30% in women, and 1% and 25% in men [6]. In northern Sweden, the prevalence was recently reported to be 11% for women and 14% for men [7]. Among vibration-exposed Swedish workers, prevalence figures of 3% for women and 15% for men have been described [8]. However, in the same study, the prevalence was 34% for men with noise-induced hearing loss. Suffering from RP can affect both the work ability and the general quality of life [9], and since there is little treatment to offer, primary preventive measures are of utmost importance.

Pathophysiology

RP manifests as vasospasm, mainly of the small arteries of the digits. The vasomotor homoeostasis is based on complex interactions between endothelium, smooth muscle cells, and autonomic and sensory nerves that innervate the vessels [2]. The adrenergic autonomic nervous system has been found to play an important role in the vasoconstrictive response, likely mainly mediated through alpha adrenoceptors [10,11]. However, structural changes, such as atherosclerosis, fibrosis and smooth muscle cell hyperplasia, have also been reported in different forms of secondary RP [12,13].

Occupational risk factors

In an occupational setting, RP can result from exposure to vibrating handheld tools [5]. However, such vibrating equipment usually also has high noise levels, and

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previous studies have shown an increased risk of hearing loss in subjects that develop RP as a result of HAV exposure [14,15]. The mechanisms behind the associations between exposure to noise and HAV in relation to RP and hearing loss are not fully understood. One possible explanation could be that both noise and HAV exposure increases the activity in the sympathetic nervous system, which limits blood flow both to the fingers and the cochleas. In support of this theory, short-term exposure to HAV and noise has been shown to induce a greater decrease in skin temperature in the fingers due to sympathetic vasoconstriction, than just exposure to HAV alone [16–18]. Long-term exposure to occupational noise has also been reported to increase the risk of hypertension and cardiovascular disease, an effect likely mediated by the activation of the sympathetic nervous system [19,20]. However, it is not known with certainty whether noise exposure alone increases the risk of RP, or if the effect is present only in conjunction with simultaneous HAV exposure. Therefore, there is a need to investigate if noise exposure is an independent risk factor for RP.

Aims

The primary aim of this study was to determine if self-reported occupational noise exposure was associated with Raynaud's phenomenon. Secondary aims were to investigate if there was a dose–effect relationship between noise exposure and RP, and if there was an association between occupational noise exposure and hearing loss.

Materials and methods

Study design

The present study was a nested case–control study on subjects reporting RP, and matching controls. The study was part of the Cold and Health in Northern Sweden (CHINS) project, which was originally aimed at investigating cold-related health effects in the working-age population of northern Sweden.

Participants

The first questionnaire (CHINS1) was sent out in February 2015 to 35,144 men and women between 18 and 70 years of age, who were drawn from the national Swedish population register. This survey was used to identify subjects with RP as well as healthy controls, and collected data on general health status, length, weight, occupation and tobacco habits. A follow-up survey (CHINS2) was sent out in October 2015, to collect detailed data on noise and HAV exposure. The data collection of the CHINS project has previously been described in detail [7,21]. The selection of cases was based on a single question: "Does one or more of your fingers turn white when exposed to moisture or cold?". The question was supported by a standardised colour chart, which previously has been shown to increase the accuracy in RP diagnosis [22,23]. Controls were randomly selected, with a ratio of 2:1, among study subjects from the CHINS1 sample, according to the following inclusion criteria: no reported RP; no reported cold sensitivity and matching the case regarding geographical area, sex and age (±2 years). Written informed consent was obtained from all study participants when responding to the questionnaires, and the study protocol was approved by the Regional Ethical Review Board situated at Umeå University (DNR 2015–24-32 M, 2014–286-31 M and 2015–255-32 M).

Variables

Occupational noise exposure was categorised depending on the self-reported voice level needed for workers to be able to hear each other at a distance of one metre. Four categories were used: *using normal voice; using loud voice; needing to scream* at colleagues in order to communicate; or being *unable to talk* due to a noisy environment. For HAV exposure, the study participants were asked to specify if they had recurrent occupational exposure to impact tools (e.g. chipping hammers, rock drills); rapidly rotating tools (e.g. dentist drills, foot files); forestry and gardening tools (e.g. chainsaws, brush cutters); vibrating tools (e.g. drilling machines, circular saws); heavily vibrating tools (e.g. reciprocating saws, oscillating sanders) or vehicles with vibrating controls (e.g. snowmobiles, all-terrain vehicles). The presence of physician-diagnosed and noise-induced hearing loss, first-degree heredity for hearing loss and RP, and tobacco habits were categorised as *yes* or *no*. Questionnaire items for noise and HAV exposure, as well as tobacco use, were then added together to form grouped variables for reporting *any* such exposure. Body mass index (BMI) was dichotomised into *overweight* (>25 kg/m^2) or *not*.

Statistical analyses

Categorical variables were presented as numbers and valid percentages, continuous variables as medians and interquartile ranges (IQR). Age was categorised based on quartiles. The statistical association for each candidate associated factor was assessed separately using

simple conditional logistic regression and presented as an odds ratio (OR) with 95% confidence interval (95% CI) for reporting RP. Then, multiple conditional logistic regression was performed in a *manual forward stepwise* fashion. Binary logistic regression was used when the hearing loss was the dependent variable. A p value below 0.05 was considered statistically significant. All analyses were performed with IBM SPSS Statistics for Windows, version 24.0–27.0 (IBM Corporation, Armonk, NY, USA).

Results

Descriptive data

The CHINS2 questionnaire had a response rate of 79.2%, and the present study consisted of 461 cases reporting RP, and 763 matched controls. The amount of missing data varied between 0.8% and 5.1% between questionnaire items. The study population was dominated by women (61.0% of cases and 59.6% of controls), and the median age was 57 years (IQR 18) and 58 years (IQR 17), respectively. Other descriptive data are presented in Table 1.

Simple logistic regression

Using conditional logistic regression, none of the questionnaire items on self-assessed occupational noise exposure were statistically significantly associated with reporting RP (Table 2). Analyses of occupational HAV exposure revealed significant associations for several vibration sources, where heavily vibrating tools had the highest point estimate (OR 2.42; 95% CI 1.60–3.66). Among other factors, physician-diagnosed hearing loss (regardless of underlying cause) was significantly associated to reporting RP (OR 1.37; 95% CI 1.01–1.86), as was first-degree heredity for RP (OR 4.41; 95% CI 3.17–6.12), and daily snuff use (OR 1.56; 95% CI 1.10–2.20). A BMI exceeding 25 kg/m^2 appeared to have a protective effect (OR 0.44; 95% CI 0.34–0.57). Using binary logistic regression, any occupational noise exposure was associated with reporting hearing loss (OR 2.76; 95% CI 2.00–3.81).

Table 1. Descriptive data for study participants.

	Cases		Controls	
Factor	N	%	N	%
Gender				
Male	180	39	308	40
Female	281	61	455	60
Age group (years)				
18–47	121	26.2	181	23.7
48–56	109	23.6	180	23.6
57–64	114	24.7	187	24.5
65–70	117	25.4	215	28.2
Geographical location				
Alpine	102	22.1	165	21.6
Inland	121	26.2	206	27.0
Coastal	238	51.6	392	51.4
Occupation				
Manual work	117	26.3	214	28.7
Desk work	211	47.4	299	40.1
Retired	97	21.8	192	25.7
Other [a]	20	4.5	41	5.5
Noise exposure sources [b]				
Working in a noisy environment	133	34.7	191	29.3
Shooting in military service	143	31.7	247	32.9
Hunting or sport shooting	97	22.0	150	20.1
Listening to music in headphones	206	46.1	333	44.3
Listening to music at night clubs or concerts	270	60.9	450	60.6
Playing music in bands or orchestras	35	7.8	59	7.8

[a]Studying, unemployment, parental leave or sick leave.
[b]Column percentages for positive responses only.

Multiple logistic regression

In the final multiple model, hearing loss was associated with reporting RP (OR 1.52; 95% CI 1.03–2.23), after adjusting for the following covariates: first-degree heredity for hearing loss; first-degree heredity for RP; any HAV exposure; BMI and any daily tobacco use (Table 3).

Dose–effect trends

There was a dose–effect trend towards higher crude odds ratios for reporting RP with higher occupational noise exposure level (Figure 1). However, these effects were not statistically significant. Acknowledging having previous noise-induced hearing loss showed the highest point estimate (OR 1.58; 95% CI 0.61–4.10).

Studying the associations between occupational noise exposure and hearing loss, there were significant associations at all exposure levels, but no obvious positive trend: using normal voice (OR 1.97; 95% CI 1.47–2.65); using loud voice (OR 3.44; 95% CI 2.51–4.70); needing to scream (OR 3.16; 95% CI 2.20–4.54) or being unable to talk (OR 3.11; 95% CI 2.13–4.55).

Discussion

Main findings and interpretation

The present study did not show any statistically significant association between self-reported occupational noise exposure and the reporting of RP. There was a dose–effect trend between noise exposure levels and the odds ratios for reporting RP, but this was not significant. However, occupational noise exposure was significantly associated with reporting hearing loss, and this was in turn significantly associated to reporting Raynaud's phenomenon in a multiple conditional logistic regression model.

Table 2. Simple conditional logistic regression showing odds ratios (OR) and 95% confidence intervals (95% CI) for reporting Raynaud's phenomenon.

		Cases		Controls			
Factor	Exposure level	N	%	N	%	OR	95% CI
Occupational noise exposure							
Using normal voice	Yes	170	39.2	279	38.9	1.00	0.78–1.29
	No	264	60.8	438	61.1	-	-
Using loud voice	Yes	134	33.8	186	27.7	1.25	0.92–1.69
	No	262	66.2	486	72.3	-	-
Needing to scream	Yes	72	18.2	93	13.9	1.34	0.91–1.98
	No	323	81.8	577	86.1		
Unable to talk	Yes	61	15.6	81	12.2	1.35	0.90–2.04
	No	331	84.4	585	87.8	-	-
Any noise exposure	Yes	188	49.1	295	45.3	1.10	0.83–1.46
	No	195	50.9	356	54.7	-	-
Occupational HAV exposure							
Impact tools	Yes	69	15.2	62	8.3	**1.97**	**1.34–2.91***
	No	384	84.8	688	91.7	-	-
Rapidly rotating tools	Yes	15	3.3	15	2.0	1.44	0.69–3.00
	No	434	96.7	732	98.0	-	-
Forestry and gardening tools	Yes	68	15.2	83	18.3	1.29	0.87–1.90
	No	379	84.8	370	81.7	-	-
Vibrating tools	Yes	83	18.3	86	11.5	**1.79**	**1.25–2.56***
	No	370	81.7	659	88.5	-	-
Heavily vibrating tools	Yes	73	16.1	61	8.2	**2.42**	**1.60–3.66***
	No	380	83.9	686	91.8	-	-
Vehicles with vibrating controls	Yes	62	13.7	75	10.1	**1.54**	**1.04–2.30***
	No	390	86.3	671	89.9	-	-
Any HAV exposure	Yes	121	27.5	157	21.3	**1.52**	**1.10–2.10***
	No	319	72.5	580	78.7	-	-
Other factors							
Physician-diagnosed hearing loss	Yes	99	21.8	138	18.2	**1.37**	**1.01–1.86***
	No	356	78.2	620	81.8	-	-
Noise-induced hearing loss	Yes	35	7.6	40	5.2	1.58	0.61–4.10
	No	426	92.4	723	94.8		
Heredity for hearing loss	Yes	191	42.8	330	44.1	0.91	0.71–1.16
	No	255	57.2	419	55.9		
Heredity for RP	Yes	158	35.6	82	11.2	**4.41**	**3.17–6.12***
	No	286	64.4	653	88.8	-	-
BMI (kg/m^2)	>25	178	39.6	434	58.7	**0.44**	**0.34–0.57***
	≤25	272	60.4	305	41.3	-	-
Daily smoking	Yes	40	8.7	71	9.4	0.98	0.65–1.48
	No	419	91.3	687	90.6	-	-
Daily snuff use	Yes	73	15.9	81	11.8	**1.56**	**1.10–2.20***
	No	387	84.1	664	88.2	-	-
Any tobacco use	Yes	111	24.2	147	19.5	**1.42**	**1.07–1.90***
	No	348	75.8	605	80.5	-	-

*Significant at the 0.05 level. BMI: body mass index. HAV: hand-arm vibration. RP: Raynaud's phenomenon.

The results of the present study offer some support to the hypothesis that there is a common pathophysiological background for RP and hearing loss among noise-exposed workers. One plausible mechanism could be a noise-induced activation of the sympathetic nervous system, decreasing the blood flow to both the fingers and the cochleas. The vasoconstrictive response is likely mediated by neurotransmitters, released mainly from autonomic nerves [2]. HAV exposure is thought to evoke a similar vasoconstrictive response as noise, and a previous study found greater cutaneous vasoconstriction after exposure to a combination of noise and HAV, compared to HAV exposure alone [16]. This additive vasoconstrictive effect of noise among HAV-exposed subjects is in line with the finding in the present paper, where the association between hearing loss and RP was still evident after adjusting for occupational HAV exposure in the multiple model.

Interestingly, the point estimates for reporting RP increased with the level of noise exposure (Figure 1). Stronger statistical associations were found between hearing loss and RP. Although hearing loss can have other backgrounds, it might be thought of as a surrogate marker for noise exposure, supporting the mechanistic hypothesis outlined above. Among the few subjects who had reported having noise-induced hearing loss, the point estimate for RP was even higher, suggesting that the link between hearing loss and RP is indeed noise exposure. The analysis was however lacking in statistical power, and type 2 error cannot be ruled out. Regarding occupational noise exposure and hearing loss, although a clear dose–effect trend was not

Table 3. Final multiple conditional logistic regression model for the association between reporting hearing loss and Raynaud's phenomenon.

		Cases		Controls			
Factor	Exposure level	N	%	N	%	OR	95% CI
Hearing loss	Yes	99	21.8	138	18.2	**1.52**	**1.03–2.23***
	No	356	78.2	620	81.8	-	-
Heredity for hearing loss	Yes	191	42.8	330	44.1	**0.71**	**0.52–0.95***
	No	255	57.2	419	55.9	-	-
Heredity for RP	Yes	158	35.6	82	11.2	**4.43**	**3.06–6.43***
	No	286	64.4	653	88.8	-	-
Any HAV exposure	Yes	121	27.5	157	21.3	**1.47**	**1.00–2.15***
	No	319	72.5	580	78.7	-	-
BMI (kg/m^2)	>25	178	39.6	434	58.7	**0.41**	**0.30–0.55***
	≤25	272	60.4	305	41.3	-	-
Any daily tobacco use	Yes	111	24.2	147	19.5	1.39	0.98–1.96
	No	348	75.8	605	80.5	-	-

*Significant at the 0.05 level. BMI: body mass index. HAV: hand-arm vibration. RP: Raynaud's phenomenon.

discernible, higher point estimates were found at higher exposure levels compared to the lowest level (Section 3.4). These findings were expected, since occupational noise exposure is a well-known risk factor for subsequent hearing loss [24,25] and served to validate the noise exposure questions.

In Table 1, it was evident that many of the cases were of higher age, which is likely related to the fact that many risk factors for RP exert an effect during the course of the working life. The effects of age and gender could not be further investigated, since cases and controls were matched in these aspects. However, there is reason to suspect that the pattern of occupational risk factors may differ between men and women, and this has previously been addressed [21,26]. Risk factor patterns may also differ between primary and secondary RP, but this was not studied in any detail in the present study. Daily snuff use, but not daily smoking, was statistically significantly associated with reporting RP (Table 2). Firstly, snuff use is more common than smoking in northern Sweden. Secondly, nicotine from snuff use is absorbed directly into the blood vessels, and results in higher plasma concentrations compared to cigarette use, which could augment the vasoconstrictive response [27,28]. Occupational exposure to HAV is a well-established risk factor for RP [5], but the association between RP and hearing loss was still present when adjusting for HAV in the multiple model, indicating a possible independent effect of noise exposure. A high BMI appeared protective, which has also been found in previous studies, and suggested to be due to an insulating effect of peripheral adipose tissue [29,30].

Strengths

The present study included several important risk factors for RP in a well-controlled data set, sampled from the general population. The matched case–control design and multiple modelling established hearing loss as a robust associated factor to RP, which is a novel finding. The dose–effect trend between noise exposure levels and RP also offer some support for causality, although this cannot be established with a cross-sectional design. The response rate of the questionnaire for exposure assessment (CHINS2) was

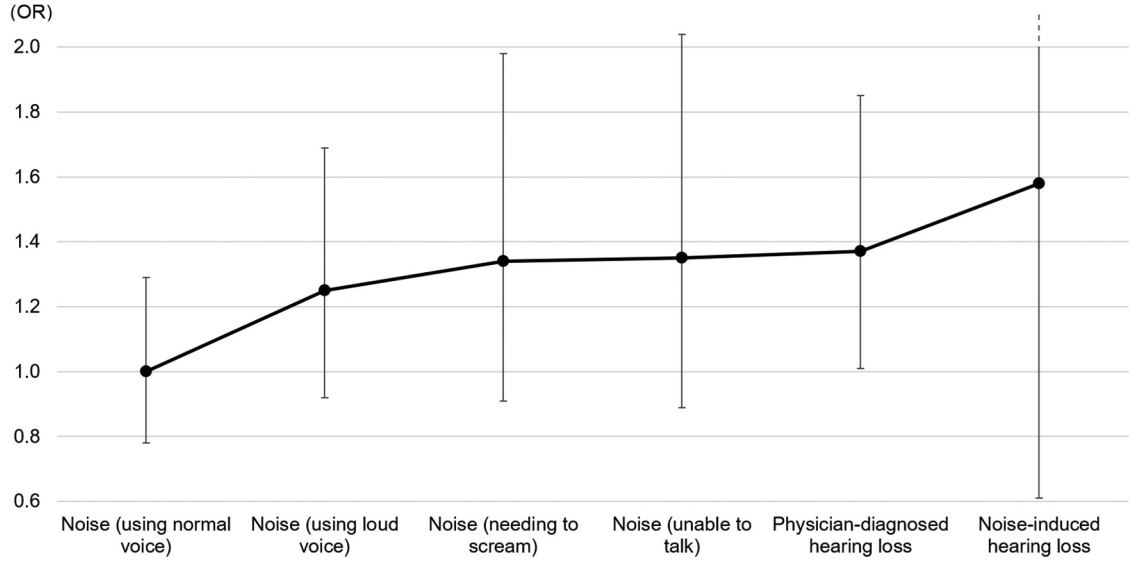

Figure 1. Dose–effect trend based on crude odds ratios (OR) for reporting Raynaud's phenomenon. The error bars depict 95% confidence intervals (95% CI).

acceptable, and the results are considered to be generalisable to other populations in similar climate.

Limits

There are several limitations to the present study. Both the studied associated factors and the outcomes were based on self-assessment in questionnaires. The diagnoses of RP and hearing loss would likely have been better validated if ascertained by health care professionals. In the same manner, noise exposure assessments were rather crude, being based on four questionnaire items on verbal communication. Objective field measurements of noise levels would likely have improved the resolution as well as the validity of noise exposure assessment. However, the use of qualitative exposure assessment of noise made it possible to collect exposure data on many subjects. It was not feasible to objectively measure the individual exposure levels for so many participants. In addition, comparisons between subjective self-reported exposures and objective measurements have suggested that individuals may well be able to make reasonable estimates of their daily noise exposure [31,32]. Although the sample included subjects of working age, there was a large proportion of retired participants (24.3%), which may have attenuated the occupational perspective. Finally, there are likely other explanatory factors for suffering from hearing loss or RP that were not investigated in this study. The results should therefore be regarded as hypothesis-generating, needing to be confirmed in future studies of other designs.

Conclusions

Self-reported occupational noise exposure was not statistically significantly associated with RP, but there was a dose–effect trend. In addition, occupational noise exposure was significantly associated with reporting hearing loss, and this was in turn significantly associated to reporting RP in a multiple conditional logistic regression model. These findings offer some support for a common pathophysiological background for RP and hearing loss among noise-exposed workers, possibly through noise-induced vasoconstriction.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

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Data availability statement

The dataset used for this paper can be made available upon personal request.

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