Noise sensitivity - medical, psychological and genetic aspects

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INTRODUCTION

Noise sensitivity refers to physiological and psychological states of any individual, which increase the degree of reactivity to noise (Job 1999). It constitutes a trait covering attitudes to noise in general and it is a predictor of annoyance (van Kamp et al. 2004; Stansfeld 1992). Noise sensitivity may be directly related to outcomes of noise exposure (Smith 2003). A significant correlation has been found between subjective health and noise exposure in the noise-sensitive group, while no significant correlation were observed in the insensitive group. Thus, the adverse health effects may exist especially in the sensitive group (Kishikawa et al. 2009). In women noise sensitivity has been related to slower habituation of heart rate responses to loud threatening noises (Stansfeld 1992) and to cardiac health complaints (Nivison & Endresen 1993). Blood pressure increase has correlated with self-reported sensitivity to noise (Otten et al. 1990). In field conditions several hours of exposure to road noise at level 60 dB has shown to cause more pronounced blood pressure reactions in noise sensitive subjects than in noise insensitive subjects (Ising 1983 in Ising & Kruppa 2004). However, in Austrian Tyrol area studies noise sensitivity was non-significantly or even inversely associated with blood pressure readings or self-reported hypertension or treatment (Lercher et al. 2011). A significant noise effect on subjective sleep quality has been reported among noise sensitive subjects (Öhrström et al. 1988) and noise sensitivity has altered self-reported sleep disturbance attributed to noise (Miedema & Vos 2003). Correlations have been found between noise sensitivity and subjective sleep quality (Marks & Griefahn 2007; Nivison & Endresen 1993) in terms of difficulty to fall asleep, body movements, poor restoration and decreased calmness (Marks & Griefahn 2007).

Noise sensitivity has been associated with health-related quality of life and annoyance and sleep disturbance have mediated the effects of noise sensitivity on health (Shepherd et al. 2010). The risk of health effects caused by noise may be higher for noise sensitive individuals compared with non-noise sensitive individuals. A cardiovascular disease may be an example of outcomes (Figure 1).

However, determinants and characteristics related to noise sensitivity are not very well known. There are studies on noise and mortality, but previous studies on the association of noise sensitivity with mortality were not available according to the literature available. Studies on the role of genetic factors in noise sensitivity prior to this study were not available, but heritability of acoustic startle response (Brocke et al. 2006) has been studied. The aim of the present study was to investigate the association of noise sensitivity with hearing ability, specific somatic and psychological factors and mortality and to study the genetic component of noise sensitivity. The central

results presented here have been published previously (Heinonen-Guzejev 2008; Heinonen-Guzejev et al. 2004, 2005, 2007, 2011).

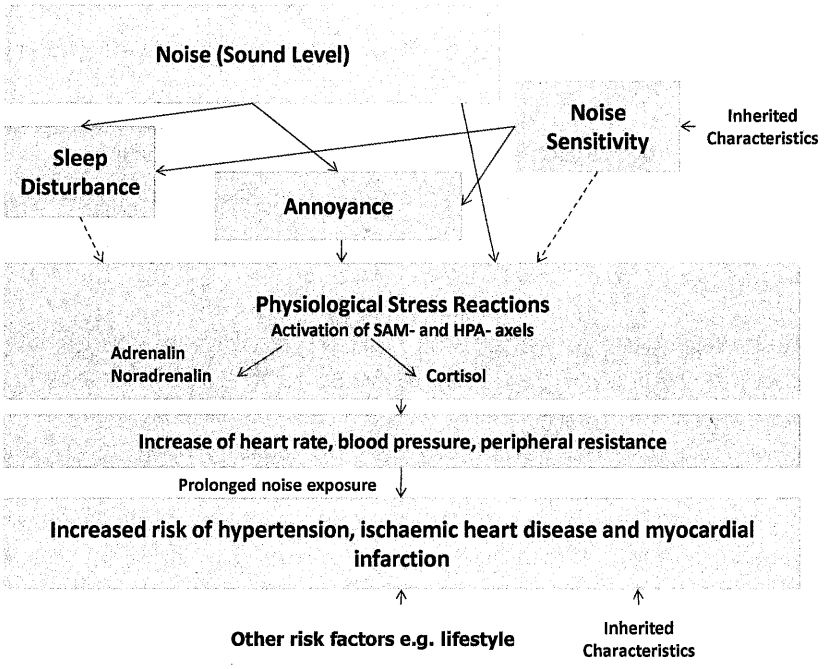

Figure 1: Model of the schematic pathways of the possible relationship of noise sensitivity with development of cardiovascular disease

MATERIALS AND METHODS

The study is based on the older Finnish Twin Cohort of same-sex twin pairs born before 1958 with both members alive in 1967 who had all been sent questionnaires in 1975 and 1981. In 1988 a questionnaire on noise and related factors was sent to 1005 twin pairs discordant for hypertension. 1495 individuals (688 men, 807 women) aged 31-88 years replied. For 573 twin pairs (131 MZ and 442 DZ) both twins had answered the question on noise sensitivity. Self-reported noise sensitivity, hearing ability and hypertension were obtained from the questionnaire study in 1988 and other somatic and psychological factors from the questionnaire study in 1981 for the same individuals. To evaluate the stability and validity of noise sensitivity, a new questionnaire was sent in 2002 to a sample of the subjects who had replied to the 1988 questionnaire. A subsample of thirty-eight elderly women with noise sensitivity response from 1988 had audiometric data from 2000 to 2001 (Heinonen-Guzejev et al. 2011).

Noise sensitivity was investigated using a short question (Heinonen-Guzejev et al. 2004, 2005, 2007). In the 2002 questionnaire the Weinstein's Noise Sensitivity Scale was also used. Lifetime noise exposure was evaluated using three questions about noise exposure at home, at work and noisy leisure time hobbies. A lifetime noise exposure scale was formed by summing these three items (Heinonen-Guzejev 2008). Noise map information was available only for 218 subjects who lived in the Helsinki Metropolitan, but it was not used in these analyses. In our previous study aircraft noise maps were consistently associated with the self-report of noise exposure (Hei-

nonen-Guzejev et al. 2000). All questions used are presented in detail elsewhere (Heinonen-Guzejev 2008; Heinonen-Guzejev et al. 2004, 2011).

Data on deaths and causes of death were obtained from record linkage to the nationwide Finnish death register at Statistics Finland using the unique personal identity numbers given to all residents of Finland. All deaths that occurred among the study population from 1 January 1989 to 31 December 2003 were classified as being due to all causes (n = 382), to cardiovascular diseases (ICD 9 codes 390–459, ICD10 I00–I99) (n = 193), to coronary heart disease (ICD 9 codes 410–414, ICD10 codes I20–I25) (n = 111) and to other causes than cardiovascular diseases (n =189). Autopsy had been made for 19 % of the deceased (Heinonen-Guzejev et al. 2007).

Cohen's coefficient of agreement for nominal scales, Pearson chi-square and logistic regression models were used. The Cox proportional hazards regression model was used to evaluate the risk of mortality in relation to noise sensitivity (Heinonen-Guzejev 2008; Heinonen-Guzejev et al. 2007). To take into account the sampling of twin pairs, the possible lack of statistical independence of twins in a twin pair, robust estimators of variance were computed with the cluster option in Stata to derive correct confidence intervals (Williams 2000). Standard model fitting methods were employed using Mx, a program for analysis of twin and family data (Neale et. al. 2003) fitting directly to the raw ordinal data.

RESULTS

The short question on noise sensitivity showed good validity when correlated with the Weinstein's multi-item noise sensitivity scale. Noise sensitivity was relatively stable between 1988 and 2002 questionnaires. Of all subjects who had answered the question on noise sensitivity, 38 % were noise sensitive (36 % of women and 41 % of men). The overall tendency was decreasing noise sensitivity with age (age range 31–70 years). The age related differences in noise sensitivity were statistically significant among men and women (Heinonen-Guzejev 2008; Heinonen-Guzejev et al. 2004).

Noise sensitivity and hearing disability

Noise sensitivity was associated with self-reported hearing disability among all subjects (adjusted OR 1.55, 95% CI 1.14–2.12) and among women (adjusted OR 1.55, 95% CI 1.19–3.04), but no-more significantly among men (adjusted OR 1.55, 95% CI 0.86–1.98). Noise sensitivity was associated with self-reported hearing disability with evidence for a dose-response relationship. The association was primarily seen among younger subjects (50 years or less) (Heinonen-Guzejev et al. 2011).

However, noise sensitivity was not associated with auditory acuity. The average of thresholds at frequencies of 0.5–4 kHz in the better ear among elderly noise sensitive women was 27.6 dB (95% CI 20.0–35.3) and among non-noise sensitive women 31.5 dB (95% CI 27.3–35.6). The difference between these two groups was not statistically significant (Pr = 0.18). However, statistical significance was reached for the threshold differences at frequencies of 0.125 and 8 kHz. An analysis of mean BEHL (better ear hearing levels) at different frequencies showed that noise sensitive female subjects tended to have somewhat better hearing thresholds than non-noise sensitive ones (Heinonen-Guzejev et al. 2011).

Noise sensitivity did not modify the association of self-reported hearing disability with the self-reported history of occupational noise exposure. Self-reported history of noise exposure during leisure time hobbies was associated with self-reported hearing disability among younger non-noise sensitive subjects (Heinonen-Guzejev et al. 2011).

Noise sensitivity and somatic and psychological factors

Noise sensitivity was significantly associated with hypertension, stress, hostility, use of sleeping pills, tranquillizers and pain relievers, former smoking and emphysema, even when lifetime noise exposure was adjusted for. The age and sex adjusted association of neuroticism with noise sensitivity was significant, but in the multivariate analyses became non-significant. This weakening of the association may represent adjustment for intermediary variables (stress and hostility). The results indicate that noise sensitivity has both somatic and psychological components (Heinonen-Guzejev et al. 2004).

In analyses performed separately among women and men, in women noise sensitivity was associated significantly with stress, hostility and hypertension, while in men it was associated with stress, emphysema and use of sleeping pills and tranquillizers (Heinonen-Guzejev et al. 2004).

Noise sensitivity and mortality

Cardiovascular mortality was significantly increased among noise sensitive women (hazard ratio 1.80, 95% CI 1.07–3.04). Among men there was no statistically significant effect (hazard ratio 0.80, 95% CI 0.45–1.43) (Table 1). Both in women (hazard ratio 1.13, 95% CI 0.66-1.92) and in men (hazard ratio 1.19, 95% 0.63-2.25) reporting lifetime noise exposure, cardiovascular mortality was increased, but not statistically significant (Table 1) (Heinonen-Guzejev et al. 2007).

Table 1: Adjusted hazard ratios for cardiovascular mortality among women and men

		Women		Men	
		Age-adjusted hazard ratio	Full model*	Age-adjusted hazard ratio	Full model*
Noise Sensitivity	No	1.00	1.00	1.00	1.00
	Yes	1.75	**1.80**	0.88	0.80
	95% CI	1.15-2.67	**1.07-3.04**	0.54-1.44	0.45-1.43
Hypertension	No	1.00	1.00	1.00	1.00
	Yes	2.06	**2.58**	1.71	1.44
	95% CI	1.32-3.21	**1.33-4.98**	1.06-2.77	0.81-2.56
Lifetime Noise Exposure	No	1.00	1.00	1.00	1.00
	Yes	1.21	1.13	0.93	1.19
	95% CI	0.81-1.81	0.66-1.92	0.59-1.47	0.63-2.25

*Adjusted for age, hypertension, smoking and emphysema

Coronary heart mortality was increased, but not statistically significant among noise sensitive women (hazard ratio 2.03; 95% C.I. 0.94-4.37), with no evidence for an increase in noise sensitive men (Table 2). In men reporting lifetime noise exposure coronary heart mortality was increased but not statistically significant (hazard ratio 1.52; 95% CI 0.73-3.18), and it was higher than in women (hazard ratio 1.08, 95% CI 0.48-2.44) (Table 2) (Heinonen-Guzejev et al. 2007).

Taking into account factors known to affect mortality in general (education, body mass index, physical activity, alcohol consumption, passing out due to alcohol use more than once in a year) did not change the results for any of the cause of death categories (data not shown) (Heinonen-Guzejev et al. 2007).

Table 2: Adjusted hazard ratios for coronary heart mortality among women and men

		Women		Men	
		Age-adjusted hazard ratio	Full model*	Age-adjusted hazard ratio	Full model*
Noise Sensitivity	No	1.00	1.00	1.00	1.00
	Yes	1.69	2.03	0.89	0.89
	95% CI	0.89-3.21	0.94-4.37	0.49-1.60	0.45-1.73
Hypertension	No	1.00	1.00	1.00	1.00
	Yes	1.89	2.32	1.59	1.29
	95% CI	1.02-3.51	0.91-5.91	0.90-2.82	0.67-2.51
Lifetime Noise Exposure	No	1.00	1.00	1.00	1.00
	Yes	1.24	1.08	1.33	1.52
	95% CI	0.69-2.23	0.48-2.44	0.77-2.33	0.73-3.18

*Adjusted for age, hypertension, smoking and emphysema

Table 3 shows the interactions of noise sensitivity and lifetime noise exposure with coronary heart and cardiovascular mortality in women. Among men there was no statistically significant effect (data not shown). **Coronary heart mortality** was significantly increased among noise sensitive women reporting lifetime noise exposure, but not among those not reporting lifetime noise exposure. For coronary heart mortality the interaction of noise sensitivity and lifetime noise exposure was statistically significant (p for interaction 0.022). **Cardiovascular mortality** was significantly increased among noise sensitive women both reporting and not reporting lifetime noise exposure, and the point estimate of the hazard ratio was higher among women reporting lifetime exposure. The interaction was not statistically significant (p for interaction 0.076) (Heinonen-Guzejev 2008; Heinonen-Guzejev et al. 2007).

Table 3: Interaction of noise sensitivity and lifetime noise exposure with coronary heart and cardiovascular mortality (age adjusted) in women and number of deaths (n) and total number of female subjects (N)

Coronary heart mortality				**Cardiovascular mortality**			
Lifetime Noise Exposure		**Noise Sensitivity**		**Lifetime Noise Exposure**		**Noise Sensitivity**	
		No	Yes			No	Yes
No	n/N	17/252	7/156	No	n/N	31/252	17/156
	Hazard ratio	1.00	1.36		Hazard ratio	1.00	**2.10**
	95% CI		0.48-3.83		95% CI		**1.03-4.28**
Yes	n/N	3/134	11/127	Yes	n/N	9/134	20/127
	Hazard ratio	0.64	**3.11**		Hazard ratio	0.84	**2.93**
	95% CI	0.20-2.11	**1.19-8.10**		95% CI	0.38-1.82	**1.39-6.19**
p for interaction		**0.022**		p for interaction		0.076	

Genetic component of noise sensitivity

Monozygotic twin pairs were more similar with regards noise sensitivity than dizygotic twin pairs. The intraclass correlations for noise sensitivity in MZ pairs was 0.36 (95% CI 0.16–0.52) and in DZ pairs 0.19 (95% CI 0.07–0.31). Excluding those pairs in which one or both were hearing impaired did not significantly change the correlations between twins in MZ and DZ pairs. Correlations for male and female pairs did not differ significantly statistically (Heinonen-Guzejev et al. 2005).

Quantitative genetic modeling indicated significant **familiality**. The E model could be rejected meaning that family factors are needed to account for the pairwise distribution of the data. The remaining models (AE, ACE and CE) provided adequate fit to the data. In the ACE model, the estimate for C was very small (3 %), and the fit of the AE model was better than the CE model when either is compared to the ACE model. Hence the best-fitting model was the AE model, which indicates that genetic factors (A) and unique environmental factors (E) (not shared with family members) account for the variability in noise sensitivity in the population. The estimate for the proportion of variance accounted for by genetic factors was 36 %, with the remainder due to unique environment factors (Figure 2) (Heinonen-Guzejev et al. 2005).

When twins with impaired hearing were excluded, the estimate of the proportion of variance accounted for by genetic factors was 40% in an AE model (Figure 2) and the CE model was rejected, as it fit significantly worse than the ACE model ($p = 0.05$) (Heinonen-Guzejev et al. 2005).

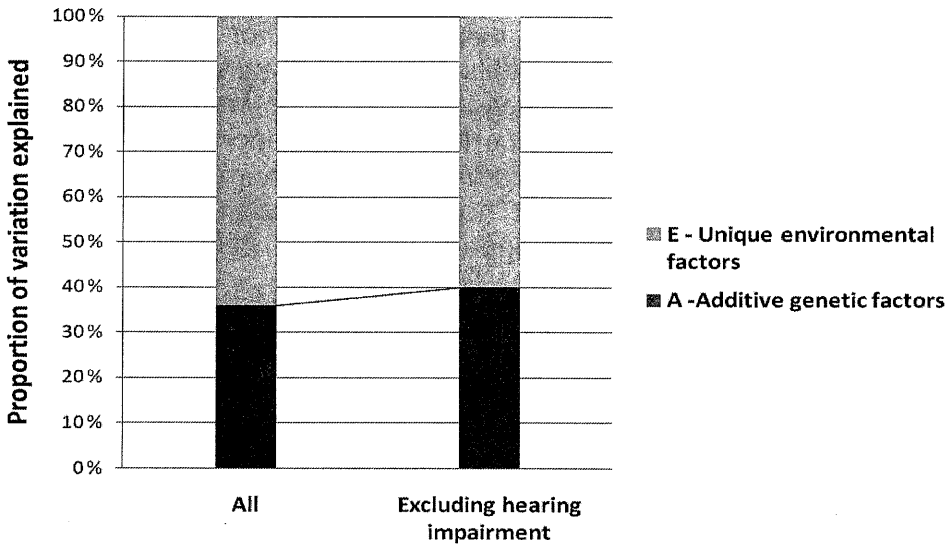

Figure 2: Proportional estimates of additive genetic factors (A) and unique environmental factors (E) on noise sensitivity in Finnish Twins.

DISCUSSION

In this study, noise sensitivity was not associated with auditory acuity among elderly women. However among younger subjects (50 years or less), it was associated with self-reported hearing disability, with evidence for a dose-response relationship. In previous studies noise sensitivity has not either been associated with auditory acuity (Ellermeier et al. 2001; Stansfeld 1992). The effects observed previously have suggested noise sensitivity to reflect a judgemental, evaluative predisposition towards

the perception of sounds (Ellermeier et al. 2001). Highly noise sensitive women have rated the loudest tones as louder and the softest tones as softer than low noise sensitive women (Stansfeld et al. 1985). The lack of an association between noise sensitivity and auditory acuity in elderly subjects in this study may also indicate an increasing role of neural presbycusis with age, whereby the loudness function may turn to a gentler one.

In previous studies, too, noise sensitivity has been associated with hypertension (Ising 1983 in Ising & Kruppa 2004; Otten et al. 1990) and stress (Zimmer & Ellermeier 1999). It has been associated with sleep quality (Marks & Griefahn 2007; Nivison & Endresen 1993; Öhrström et al. 1988). However, our finding that emphysema is associated with noise sensitivity is new. Overall, emphysema was strongly associated with former and current smoking. Noise sensitivity was also associated with former smoking. The cross-sectional nature of this study does not permit the resolution of the causal nature of this association.

Noise sensitivity was associated significantly with hypertension in women, but not in men. Cardiovascular mortality was significantly increased among noise sensitive women, but among men there were no statistically significant effects. Previous studies have also found some gender differences in the association of noise sensitivity with cardiovascular disease. In women cardiovascular problems have been related to noise sensitivity (Nivison & Endresen 1993), but in men noise sensitivity has not increased the risk of ischaemic heart disease (Babisch et al. 1999), which is in accordance with the present study.

Genetic factors and unique environmental factors account for the variability in noise sensitivity in the population. The AE model provided an estimate of heritability of 36 %. Noise sensitivity aggregates in families. The genetic component of noise sensitivity was studied among twin pairs discordant for hypertension, and noise sensitivity was associated with hypertension, which may have led to an underestimation of the genetic component. Results of the present study can provide new information about the heritability of noise sensitivity that may help in the search for specific genes or sets of genes underlying noise sensitivity. Further large-sample twin studies are needed to investigate the nature of the genetic component of noise sensitivity.

CONCLUSIONS

Noise sensitivity is not associated with auditory acuity in elderly subjects, but it is associated with self-reported hearing disability particularly among younger subjects. It does not modify the association of self-reported hearing disability with the history of occupational noise exposure. Noise sensitivity has both somatic and psychological components and it may be a risk factor for cardiovascular mortality in women, but not in men. Noise sensitivity aggregates in families and probably has a genetic component.

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Patterns of physiological and subjective responses to vehicle pass-by noises, depending on age, gender, and personality traits

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INTRODUCTION

In former laboratory studies, we compared the perceived sound quality of pairs of noise recordings (traffic noises as well as single pass-by noises) adjusted to the same L_{eq} of 83 dBA that resembled each other except for one aspect, e.g. the same car passing by either with petrol or diesel engine (Notbohm et al. 2002; Schwarze et al. 2003). In most pair-wise comparisons, stronger physiological responses were accompanied by more negative judgments on the respective sound. But there were a few exceptions in which the sound creating a stronger physiological arousal was judged more favorable (Gärtner et al. 2003).

This seemingly contradictive result might be understood better in terms of the psychological model of affective reaction to external stimuli illustrated in Figure 1: Any sensory stimulus triggers physiological and cognitive responses which can be assigned to the categories "activation" and "pleasantness" resulting in four different tendencies of judgment (Bradley & Lang 2000; Västfjäll et al. 2002). Most traffic noises can probably be perceived as activating *and* unpleasant, but it is evident that there are single pass-by noises (e.g. from sport or luxury cars) that at least by some people are judged to be activating as well as pleasant. With regard to our studies mentioned above carried out with young male students, we concluded from some remarks that the young subjects perceived these specific car noises as sounding more "sportive" or "powerful" leading to a rather pleasant activation.

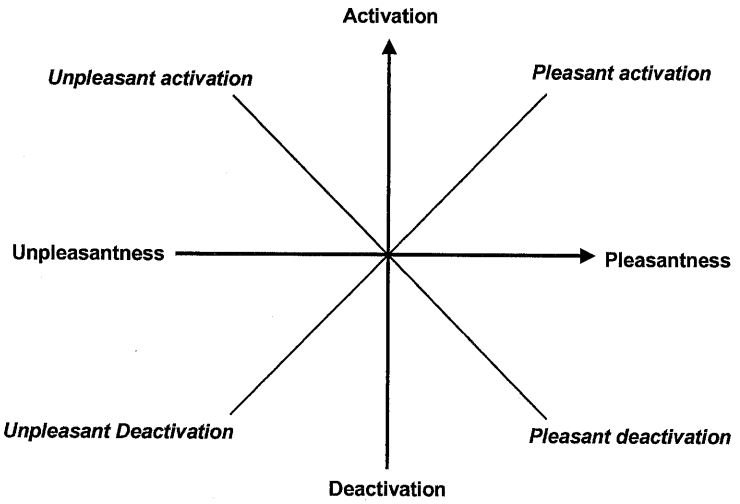

Figure 1: Two-dimensional model of affective reactions to external stimuli – activation and pleasantness

The present study is outlined to examine the effects of age and gender on the type of affective responses to vehicle pass-by noises more systematically using sounds from the previous experiments. However, far more factors are involved in shaping the individual response to sound. Already on the physiological level, there is much evidence