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Noise sensitivity – medical, psychological and genetic aspects

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ACADEMIC DISSERTATION

To be presented, with the permission of the Faculty of Medicine
of the University of Helsinki, for public examination in
Auditorium XII, University Main Building,
on 23rd January 2009, at 12 am.

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ISSN 0355-7979
ISBN 978-952-10-5179-1 (nid.)
ISBN 978-952-10-5177-7 (PDF)

Cover Photo: Seidi Guzejev
Helsinki University Printing House
Helsinki 2008

Noise sensitivity has been mentioned already in a Hippocratic treatise from the end of the 5th century BC:

> Βραχυπόται, ψόφον καθαπτόμενοι τρομώδεες γίνονται.
> ΠΡΟΡΡΗΤΙΚΟΝ Α, 16.
>
> *"Persons who drink little and are over-sensitive to noise become tremulous."**

*Prorrhetic I, In Hippocrates Volume VIII. Edited and translated by Paul Potter. Loeb Classical Library. Harvard University Press, Cambridge, London, 1995.

Contents

ABSTRACT

Noise can be defined as unwanted sound. It may adversely affect the health and well-being of individuals. Noise sensitivity is a personality trait covering attitudes towards noise in general and a predictor of noise annoyance. Noise sensitive individuals are more affected by noise than less sensitive individuals. The risk of health effects caused by noise can be hypothesized to be higher for noise sensitive individuals compared to those who are not noise sensitive.

The general aim of the present study is to investigate the association of noise sensitivity with specific somatic and psychological factors, including the genetic component of noise sensitivity, and the association of noise sensitivity with mortality.

The study is based on the Finnish Twin Cohort of same-sex twin pairs born before 1958. In 1988 a questionnaire was sent to twin pairs discordant for hypertension. 1495 individuals (688 men, 807 women) aged 31–88 years replied, including 573 twin pairs. 218 of the subjects lived in the Helsinki Metropolitan Area.

Self-reported noise sensitivity, lifetime noise exposure 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. Noise map information (1988–1992) from the Helsinki Metropolitan Area and mortality follow-up 1989–2003 were used. 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.

Of all subjects who had answered the question on noise sensitivity, 38 % were noise sensitive. Noise sensitivity was independent of noise exposure levels indicated in noise maps. Subjects with high noise sensitivity reported more transportation noise exposure than subjects with low noise sensitivity. Noise sensitive subjects reported transportation noise exposure outside the environmental noise map areas almost twice as often as non-sensitive subjects. Noise sensitivity was associated with hypertension, emphysema, use of psychotropic drugs, smoking, stress and hostility, even when lifetime noise exposure was adjusted for. Monozygotic twin pairs were more similar with regards noise sensitivity than dizygotic twin pairs, and quantitative genetic modeling indicated significant familiality. The best fitting genetic model provided an estimate of heritability of 36 %. Follow-up of subjects showed that cardiovascular mortality was significantly increased among noise sensitive women, but not among men. For coronary heart mortality the interaction of noise sensitivity and lifetime noise exposure was statistically significant in women.

Noise sensitivity has both somatic and psychological components. It does aggregate in families and probably has a genetic component. Noise sensitivity may be a risk factor for cardiovascular mortality in women.

TIIVISTELMÄ

Melu on ääntä, joka koetaan epämiellyttävänä tai häiritsevänä, ja joka voi olla haitallista ihmisen hyvinvoinnille ja terveydelle. Meluherkkyys on yksilöllinen ominaisuus, joka kuvaa herkkyyttä kokea melu ja reagoida siihen. Meluherkkyys lisää melun koettua häiritsevyyttä. Voidaan olettaa, että melun terveysvaikutusten, kuten sydän- ja verisuonitautien, riski on suurempi meluherkillä kuin ei-meluherkillä.

Tässä väitöskirjatyössä selvitettiin, onko meluherkkyys riippuvainen melukarttojen osoittamasta liikennemelutasosta, mihin somaattisiin ja psykologisiin tekijöihin meluherkkyys liittyy, selittävätkö perintötekijät meluherkkyyden eroja, ja liittyykö meluherkkyys sydän- ja verisuonitauti-, sepelvaltimotauti- tai kokonaiskuolleisuuteen.

Vuonna 1988 meluaiheinen kysely kohdennettiin Helsingin yliopiston kansanterveystieteen laitoksen aikuisten kaksoskohortin niille kaksospareille, joista vain toisella parin jäsenistä oli todettu verenpainetauti. Kyselylomakkeen palautti 1495 henkilöä (688 miestä ja 807 naista). Vastaajat olivat 31–88-vuotiaita. Kyselyyn vastanneista 218 asui pääkaupunkiseudulla ja heille määriteltiin liikennemelualtistustaso melukarttojen avulla.

Tiedot koetusta melualtistuksesta, melun häiritsevyydestä ja verenpaineesta saatiin vuoden 1988 kyselystä. Tiedot muista sairauksista, lääkkeiden käytöstä ja psykologisista tekijöistä saatiin samoille kaksosille vuonna 1981 tehdystä kyselystä. Geenien ja ympäristötekijöiden yhteyttä meluherkkyyteen arvioitiin kaksosmallinnuksella 573 kaksosparilla. Kohortin kuolleisuutta seurattiin vuosina 1989–2003. Vuonna 2002 lähetettiin otokselle vuoden 1988 kyselyyn vastanneista kysely, jolla selvitettiin mm. ja meluherkkyys-kysymyksen validiteettia.

Tutkituista 38 % oli meluherkkiä. Meluherkkyys oli riippumaton melukarttojen osoittamasta liikennemelualtistuksesta. Erittäin meluherkät raportoivat melua enemmän kuin ei lainkaan meluherkät. Meluherkät raportoivat melua melukartoitusten melualueiden ulkopuolella lähes kaksi kertaa useammin kuin ei-meluherkät. Meluherkkyydellä todettiin somaattinen ja psykologinen komponentti. Se liittyi kohonneeseen verenpaineeseen, uni- ja rauhoittavien lääkkeiden sekä särkylääkkeiden käyttöön, keuhkolaajentumaan, tupakointiin, stressiin ja vihamielisyyteen myös silloin kun elinaikainen itse raportoitu melualtistus oli vakioitu. Samanmunaiset kaksosparit olivat meluherkkyyden suhteen enemmän samankaltaisia kuin erimunaiset kaksosparit. Kvantitatiivinen geneettinen mallinnus osoitti meluherkkyyden osalta huomattavaa familiaalisuutta eli meluherkkyys oli kasautunut perheisiin. 36 % eroista meluherkkyydessä selittyi geneettisillä tekijöillä. Seurantatutkimus osoitti, että meluherkkien naisten sydän- ja verisuonitautikuolleisuus oli tilastollisesti merkitsevästi suurempi kuin ei-meluherkkien naisten. Sepelvaltimotautikuolleisuuden osalta meluherkkyyden ja elinaikaisen melualtistuksen välinen yhteisvaikutus oli naisilla tilastollisesti merkitsevä.

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original articles referred to in the text by Roman numerals I–IV

I Heinonen-Guzejev M, Vuorinen HS, Kaprio J, Heikkilä K, Mussalo-Rauhamaa H, Koskenvuo M. Self-report of transportation noise exposure, annoyance and noise sensitivity in relation to noise map information. J Sound Vib, 234(2), 191–206, 2000.

II Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikkilä K, Kaprio J, Koskenvuo M. Somatic and psychological characteristics of noise-sensitive adults in Finland. Arch Environ Health, 59(8), 410–417, 2004.

III Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikkilä K, Koskenvuo M, Kaprio J. Genetic component of noise sensitivity. Twin Res Hum Genet, 8(3), 245–249, 2005.

IV Heinonen-Guzejev M, Vuorinen HS, Mussalo-Rauhamaa H, Heikkilä K, Koskenvuo M, Kaprio J. The association of noise sensitivity with coronary heart and cardiovascular mortality among Finnish adults. Sci Total Environ, 372, 406–412, 2007.

ABBREVIATIONS

A	additive genetic effects, cumulative allelic effects of several genes
AE model	structural equation model with additive genetic effects and unique environmental effects
C	shared (by family members) environmental effects
CI	confidence interval
D	genetic dominance effects, due to allelic interactions
dB	decibel
E	unique environmental effects, not shared with other family members
DZ	dizygotic
ICD	International Classification of Diseases
MZ	monozygotic
OR	odds ratio
SD	standard deviation

1 INTRODUCTION

Noise is unwanted sound which may adversely affect the well-being and health of individuals. Environmental or community noise is defined as noise emitted from all sources except noise at the industrial workplace. Transportation noise caused by road, rail and air traffic is the main source of environmental noise (Berglund et al. 1999). Noise has been classified as a physical (Pacak and Palkovits 2001), psychosocial (Babisch 2003) and an environmental stressor (Berglund et al. 1999). Epidemiological and laboratory studies have indicated that noise may have both temporary and permanent impacts on physiological functions (Babisch 2002; Babisch 2003; Berglund et al. 1999; Rylander 2004) and thus it can be seen as a stressor challenging cardiovascular and metabolic homeostasis.

A fundamental task of hearing is to warn and to alert. For this purpose, it cannot be turned off and sound is registered in the brain even during sleep. The human auditory system and the varying physiological response to sound are inseparably connected. The auditory pathways of the central nervous system consist of direct pathways from the inner ear to the auditory cortex, and indirect pathways to the reticular activating system that connects to the limbic system and other parts of the brain, to the autonomic nervous system and to the neuroendocrine system. There is a variety of indirect connections from the inner ear to brain centres that control physiological, emotional and behavioural responses of the body (Westman and Walters 1981).

Noise affects alertness, cognition and motor performance (Rylander 2004). A basic behavioural response to sound stimuli is the orientation reflex, which occurs to sounds of low or moderate intensity or significance. It involves ascending and descending auditory cortical pathways. It orients the head and eyes towards the sound and is reflected by an arousal pattern in the EEG (electroencephalogram). The second basic auditory response is startle reflex, which is evoked by sounds of sudden, intense or frightening significance. It has a series of components, such as middle ear muscle and auropalpebral reflexes and flexion of most muscle groups in a freezing posture. The defensive response can occur independently of orientation or startle responses. It is produced by sounds of sufficient intensity, significance or duration to be perceived as threatening and to mobilize "fight or flight" reaction. The defensive response includes alerting of the cerebral cortex, emotional arousal, and preparation of the body for action, and involves largely the sympathetic nervous system but has some parasympathetic aspects. It appears e.g. in the form of skeletal muscle tension, pupillary dilation and acceleration of pulse rate. The defensive response can become

the stress that leads to the general adaptation syndrome. When this takes place the hypothalamic-pituitary-adrenal axis is mobilized resulting in an increase of cortisol and adrenaline output (Westman and Walters 1981). For an immediate triggering of protective coping reactions the information conveyed by noise is often more relevant than the sound level (Ising and Kruppa 2004).

There are direct and indirect acute reactions to noise. Direct reactions are mediated by nervous and/or endocrine transduction to different organs without cortical intermediation (Figure 1). Indirect noise effects are caused by noise-induced disturbances to various activities, provoking different types of cortical response, including psychological stress reactions such as tension and annoyance (Ising and Rebentisch 1993).

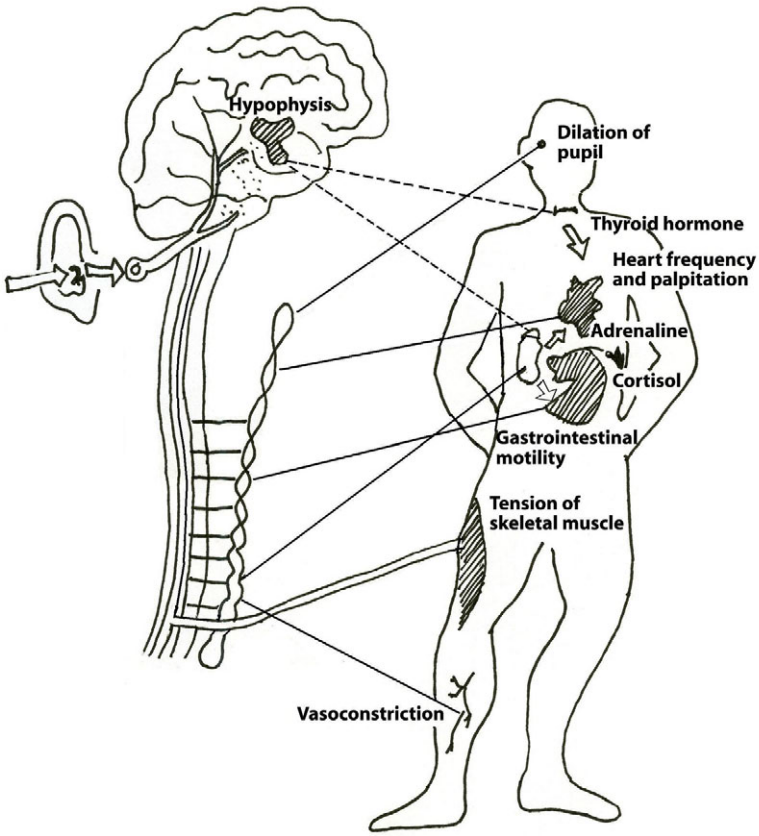

Figure 1. Transmission paths of direct noise effects (adapted from Ising and Rebentisch 1993).

Acute noise exposure activates the autonomic nervous system and endocrine system, which leads to temporary changes such as increased heart rate, vasoconstriction and increased blood pressure (Berglund et al.

1999; Haralabidis et al. 2008; Rylander 2004). After prolonged exposure to high sound levels noise can cause permanent effects, such as hypertension and ischaemic heart disease (Berglund et al. 1999; Eriksson et al. 2007; Rylander 2004).

Noise immissions are processed via central pathways. They activate the neuro-endocrinological systems either by inducing direct effects through instant signal processing in the amygdala, which is linked with cortical, limbic and hypothalamic centres, or by inducing indirect stress effects such as disturbances of concentration and communication (Ising and Kruppa 2004; Spreng 2000a).

Even during sleep noise may be categorized as danger signals and induce the release of stress hormones. The connection between environmental noise and stress reactions during sleep is explained by functions of the amygdala. This region of the brain stem plays an important role in the auditory warning system and is able to differentiate between neutral sounds and those implying danger. The first and fastest signal detection is mediated by the amygdala (Babisch and Ising 2001; Ising and Kruppa 2004).

Noise activates the sympathetic-adrenal-medullary (SAM) axis and the hypothalamic-pituitary-adrenal (HPA) axis (Babisch 2002). The sympathetic-adrenal-medullary system and the hypothalamic-pituitary-adrenal system are the two major stress systems that seem to play an important role in influencing cardiovascular and metabolic functions. Sustained activation of the sympathetic-adrenal-medullary system with overexposure to adrenaline and noradrenaline can contribute to the development of cardiovascular disease. Chronic stress exposure influencing the hypothalamic-pituitary-adrenal-axis is associated with metabolic changes, which increase the risk of cardiovascular disease (Lundberg 1999).

Noise exposure induces increases in levels of stress hormones such as adrenaline, noradrenaline and/or cortisol. Extremely intense acute noise exposure of 105–125 dB has been shown to cause an increased release of cortisol and acute noise exposure of 90–100 dB an increase of adrenaline. Nonhabituated noise has increased primarily the release of adrenaline. Habitual occupational and traffic noise has shown to cause an increase of noradrenaline. In sleeping persons traffic noise has caused significant acute increase of cortisol and chronic noradrenaline increase (Babisch et al. 2001; Babisch and Ising 2001; Ising and Braun 2000).

There is sufficient scientific evidence that noise exposure can induce annoyance, hearing impairment, sleep disturbance, ischaemic heart disease, hypertension, and impaired cognitive performance. For other health effects such as birth defects and changes in the immune system, the evidence is limited (Passchier-Vermeer and Passchier 2000). In children chronic aircraft noise exposure has been associated with higher levels of perceived stress and annoyance, poorer reading comprehension and sustained attention (Haines et al. 2001). It is also assumed that environmental noise may accelerate

and intensify the development of latent mental disorders (Berglund et al. 1999). Road traffic and aircraft noise exposure have been associated with psychological symptoms but not with clinically defined psychiatric disorder (Stansfeld and Matheson 2003; Tarnopolsky et al. 1980).

Many factors play a role in the development of cardiovascular diseases. Noise is an additional risk factor, besides smoking, obesity, lack of physical activity, diabetes, the increase of cholesterol, heredity etc. Epidemiological studies have suggested a higher risk of cardiovascular diseases, including high blood pressure and myocardial infarction, in subjects who were chronically exposed to high levels of transportation noise. Hypertension is a multifactorial disease and the relative contribution by noise is probably quite small compared to other factors. Regarding the association of community noise and hypertension the ratings have been heterogeneous (Babisch 2004; Babisch 2006a; Rylander 2004).

Noise sensitivity constitutes a personality trait covering attitudes to noise in general (Anderson 1971; Stansfeld 1992). It is an important and independent predictor of noise annoyance (van Kamp et al. 2004; Stansfeld 1992). In previous studies noise sensitive individuals have been more affected by noise than less sensitive individuals (Öhrström et al. 1988b). Noise sensitivity has correlated with increased blood pressure (Otten et al. 1990) and health complaints such as cardiac complaints (Nivison and Endresen 1993).

However, determinants and characteristics related to noise sensitivity are not very well known. As noise sensitivity predicts annoyance it can be hypothesized that the risk of health effects caused by noise is higher for noise sensitive individuals compared with non-noise sensitive individuals.

Studies on the role of genetic factors in noise sensitivity have not previously been conducted in humans according to the available literature. Genetic influences in individual susceptibility to noise-induced hearing loss have been investigated (Davis et al. 1999; Davis et al. 2001; Davis et al. 2003; Davis et al. 2007; Di Palma et al. 2001; Dunn et al. 1991).

In the present study the association of noise sensitivity with specific somatic and psychological factors and mortality was investigated. Also the genetic component of noise sensitivity was studied. The study used the subjects from the Finnish Twin Cohort.

2 REVIEW OF THE LITERATURE

2.1 The concept of noise sensitivity

2.1.1 What is noise sensitivity?

2.1.1.1 A short history of the concept

It is difficult to tell exactly when the concept of noise sensitivity was first defined. Terminology has varied. In the earliest studies terms like "noise annoyance susceptibility" (Moreira and Bryan 1972) or "susceptibility to noise" (Griffiths and Langdon 1968) have been used. Hence it is not clear if the earlier studies have investigated noise sensitivity. In those studies the concepts of noise sensitivity and annoyance may have been mixed. These concepts were first distinguished by Anderson (1971). Table 1 lists the main noise sensitivity studies since 1971.

Kryter (1959) was investigating how "noisy" commercial jet aircraft sounded in comparison to commercial aircraft having reciprocating engines. The term "noisiness" was used to designate the wantedness, the acceptability and the annoyingness of the sound. The term noise sensitivity was not used. One aim of the study was to derive a scale or relation between physically measured sound and human reactions to it. The scale of noisiness should be concerned with how wanted or unwanted a sound is considered to be by the average listener. The loudness scale was a numerical scale that indicated how loud the sound was to the listener, and the loudness level scale was a scale of physically measured sound pressure level. The results indicated that the overall sound pressure level and the speech interference level of the sounds from the aircraft as measured on the ground bore little relation to the judgments of noisiness.

Keighley (1966) found that differences in "personal tolerance on noise", the sound pressure level and the extent of momentary fluctuations above the average level were related to differences in noise ratings. Noisiness scale was combined with time factor. Griffiths and Langdon (1968) found that individual dissatisfaction scores correlated poorly with physical measures of noise, which was believed to be the result of wide individual differences in "susceptibility to noise" and "experience of noise", as well as in patterns of living likely to be disturbed by noise.

Based on a cross-sectional study, Anderson (1971) defined that noise sensitivity involves underlying attitudes towards noise in general. According to him annoyance measures attitudes towards a specified noise or noise environment.

In the prospective study of Moreira and Bryan (1972) there were significant differences between subjects in their rating of different types of noise. Subjects most sensitive to noise showed greater initial annoyance. Noise sensitive subjects had a fairly high level of annoyance for quite moderate levels of noise, but their annoyance did not increase very greatly with increasing noise level. There were no correlations of noise sensitivity with age, sex, education level, job responsibility, home background and such personality traits as determined by the EPI (Eysenck Personality Inventory) and the MMPI (Minnesota Multiphasic Personality Inventory). Instead, noise sensitivity was apparently quite strongly related to various measures of personality given by the Rorschach Projection Test. It was proposed that noise sensitive persons show a fair amount of empathy with others, they are creative and have a relatively high intellectual level. Noise sensitive individuals were typically friendly, generous and sociable and they were very much aware of their environment. On the other hand in the prospective study of Griffiths and Delauzun (1977) no personality factors were related to annoyance or to noise sensitivity.

In a cross-sectional study of Langdon (1976 I) residents at 24 sites in and around London were interviewed. The sites were selected by reference to the traffic data of the Greater London Council and by means of exploratory traffic counts and noise measurements. A simple self-rating schedule of noise sensitivity was used. According to the responses 29 % were classified as sensitive, 31 % neutral and 40 % non-sensitive to noise. These subgroups responded in different ways to the range of noise levels. Only the "neutral" group exhibited a sharply graded response. Individual differences in noise sensitivity accounted for the greatest part of the explained variance. Noise sensitivity and dissatisfaction with traffic noise were positively correlated within sites indicating that dissatisfaction is influenced by noise sensitivity.

According to Langdon (1976 III), sensitivity to noise appears to embrace two distinct groups within the population. The first group is "noise sensitive" and the second group has two subgroups "neutral" and "non-sensitive". The three sensitivity groups were analyzed demographically. The changes in the proportions of each, associated with sex, age and occupational class, tended to be confined to respondents from the "neutral" and "non-sensitive" groups. The "sensitive" group persisted unchanged over the range of demographic variation. Physical parameters explained only a small part of the response to noise. The analysis confirmed the overriding importance of individual differences in noise sensitivity. Langdon also suggested that differences in sensitivity to noise are not confined to noise nuisance but extend to other aspects of the perceived environment.

Weinstein (1978) investigated, in a case-control study, differences among college students in their initial reactions to noise and their ability to adapt to noise. Noise sensitive subjects were much more bothered by dormitory noise and became increasingly disturbed. Weinstein defined that noise sensitivity is a personal attribute of sufficient power and generality to permit predictions of reactions to environments encountered for the first time. He developed a noise sensitivity scale (Table 2) that has been largely used in noise sensitivity studies.

2.1.1.2 Definitions

There are many different ways to determine noise sensitivity. According to Anderson (1971) and Stansfeld (1992) it is a measure of attitudes to noise in general. It constitutes a personality trait covering attitudes towards a wide range of environmental noises (Stansfeld 1992; Zimmer and Ellermeier 1999). Noise sensitivity is a predictor of annoyance (Stansfeld 1992).

Noise sensitivity is more likely related to disposition to react to noise in general than to the physical properties of noise (Nivison 1992). It refers to physiological and psychological (also including attitudinal) internal states of any individual, which increase the degree of reactivity to noise in general (Job 1999). Noise sensitive individuals pay more readily attention to noise, perceive more threat from noise and may react more to noise than less sensitive individuals (Stansfeld 1992). Noise sensitivity has been defined as a factor modifying or mediating the effects of noise exposure on the outcome measure, an independent variable, which may be directly related to outcomes such as health status (Smith 2003). It has also been determined as a self-perceived indicator of vulnerability to stressors in general, not only noise (Stansfeld 1992).

As a summary, noise sensitivity can be determined as a personality trait covering attitudes towards noise in general and as a predictor of annoyance. It may also be a self-perceived indicator of vulnerability to other environmental stimuli.

Table 1. Epidemiological studies on noise sensitivity.

First author, Year	Subjects N, sex*	Age in years*	Percentage of noise sensitive subjects *	Study design	Result
Anderson 1971	1) 24 women 2) 10 women 3) 45 male and 26 female students			cross-sectional	Noise sensitivity involves underlying attitudes towards noise in general.
Moreira 1972	1) 34 29 men, 5 women University staff 2) 45	21–55 adults	 20	prospective	Subjects most sensitive to noise were stable in their judgments of annoyance and showed greater initial annoyance, but their annoyance grew less rapidly with increasing noise level. Noise sensitivity did not depend on age, sex, education, job responsibility, home background and such personality traits as determined by the EPI and such personality traits as determined by the EPI and the MMPI, but it was related to various measures of personality given by the Rorschach Projection Test.
Langdon 1976 I	1359 residents of London	16–65+	29	cross-sectional	Individual differences in noise sensitivity accounted for much more of the explained variance than noise. Noise sensitivity and dissatisfaction with traffic noise were positively correlated within sites
Langdon 1976 III	2933 residents of London	16–65+	men 28 women 32	cross-sectional	The noise sensitive group persisted unchanged over the range of demographic variation. Physical parameters explained only a small part of the response to noise. The analysis confirmed the overriding importance of individual differences in noise sensitivity.
Griffiths 1977	1) 413, 2) 325, 3) 25 men and women residents of London and Liverpool			prospective	No personality factor was found to relate to sensitivity to noise in a constant manner.

Weinstein 1978	155, subgroups: 24 and 31 ~50 % men college freshmen	median 18		case-control	Noise sensitive students became increasingly disturbed by noise during the year, but noise insensitive students showed no change. Noise sensitive students were lower in scholastic ability, felt less secure in social interactions, and had greater desire for privacy.
Aniansson 1983	140 men and women cases had hearing loss	30–75		case-control	A higher sensitivity to noise was found in one of the noise induced hearing loss groups compared with men with normal hearing as well as in females compared with males in groups with and without hearing impairment.
Meijer 1985	3445 1507 men, 1938 women	41–43	28	cross-sectional	Noise sensitive subjects had a greater risk of being annoyed by other sounds than road traffic noise (aeroplanes, neighbours, work). They had less appreciation of their living environment. No relationship was found between traffic noise level and noise sensitivity.
Stansfeld 1985 I and II	77 women	21–53		case-control	Noise sensitivity was strongly associated with neuroticism and also associated with general sensitivity to other sensory stimuli. High noise sensitive women exhibited significantly more psychiatric symptoms. Noise sensitivity was not related to auditory threshold. Highly noise sensitive women had a consistently slower heart rate.
Di Nisi 1987	80 40 men, 40 women	mean ~22		case-control	The lower noise sensitivity group showed a lower average amplitude of heart rate response than the high sensitivity group
Peterson 1988	89 men cases had hearing loss	30–75		case-control	Men with noise-induced hearing loss showed higher sensitivity to noise compared to men with normal hearing.
Raw 1988	406 residents of the South of England			prospective	Noise sensitivity was correlated with mean dissatisfaction score and it was independent of noise level.

Öhrström 1988a	93 men and women	18–39		prospective	Annoyance was highly correlated with noise sensitivity. The measured neurophysiological discomfort thresholds for noise, light, cold and heat were significantly intercorrelated.
Öhrström 1988b	1) 24 2) 106		~30	prospective	A significant noise effect on subjective sleep quality was found among sensitive subjects only. No habituation was found for the negative influence of noise on sleep quality, mood and performance. Sensitive individuals were more affected by noise.
Otten 1990	192 95 men, 97 women	20–35		prospective	The increase of blood pressure was correlated with an increase of noise sensitivity and annoyance.
Matsumura 1991	447 men and women	20–64	25	cross-sectional	Noise sensitive individuals were more annoyed by road traffic noise and reported interference with daily activities to higher extent. Noise sensitivity was more common in older age groups.
Nivison (et al.) 1992	1028 458 men, 570 women	men 16–89 women 16–92		cross-sectional	Noise sensitivity and annoyance showed stronger correlations with health and sleep complaints than did objective noise levels. The effect of higher noise levels on persons reporting to be very sensitive to noise significantly increased the number of health complaints.
Stansfeld 1992	77 women	18–50		prospective	Noise sensitivity was associated with neuroticism and psychiatric disorder and it was stable over time. High noise sensitivity was more stable than low noise sensitivity. Noise sensitivity was a predictor of noise annoyance responses. Noise sensitive subjects pay more rapidly attention, perceive more threat from noise and may react more to noise than less sensitive subjects. Noise sensitivity is a self-perceived indicator of vulnerability to stressors in general not only noise.

Lopez Barrio 1993	800 60% women residents of Madrid		all 43 men 40 women 46	cross-sectional	Noise sensitivity is the main predictor of subjective annoyance response
Nivison 1993	82 35 men, 47 women	19–78		cross-sectional	There were strong correlations between the subjective noise responses of annoyance and sensitivity and health complaints. Women revealed a relationship between poor sleep quality and noise sensitivity.
Belojevic (and Jakovljevic) 2001	413 men and women residents of Belgrade	20–60+		cross-sectional	Subjective noise sensitivity was independent of noise exposure. Neuroticism was the most important individual variable significantly influencing subjective noise sensitivity. No significant relations of gender, age and introversion to subjective noise sensitivity were found
Ellermeier 2001	61 28 men, 33 women	19–37		case-control	Noise sensitivity captures evaluative rather than sensory aspects of auditory processing.
Ising (and Michalak) 2004	42 men	24–54		cross-sectional	A positive correlation was found between noise sensitivity and the systolic blood pressure increases during the seminar. However, the correlation between noise sensitivity and systolic blood pressure increases in the laboratory exposure, was negative.

* if reported in the study

2.1.2 Measurement of noise sensitivity

2.1.2.1 Short questions

Noise sensitivity can be measured by short questions such as "Some people are very sensitive to sounds, others are not. In general are you sensitive or insensitive to sounds, or are you in between?" (Meijer et al. 1985). It can also be measured using an open scale (e.g. 100 mm) with end points "not sensitive at all" and "extremely sensitive" (Öhrström et al. 1988a) or using one-item rating scales by asking for the degree of the respondent's self-rating of the susceptibility to noise: an 11-point numerical rating scale with the end points of "not noise sensitive at all" and "very noise sensitive" and self-rating of the susceptibility to sounds: "I am sensitive to noise" with a range of six response options from "strongly" (1) to "disagree" (6) (Zimmer and Ellermeier 1998, 1999) .

2.1.2.2 Questionnaires

Extensive questionnaires have largely been used to measure noise sensitivity. Anderson (1971) developed the General Noise Annoyance Questionnaire. Regardless of its name, it was intended to fill the need for a scale which will help to distinguish between subjects on the basis of their sensitivity to noise in general and to ensure the typicality of their subject groups. The General Noise Annoyance Questionnaire separates subjects' feelings into two factors, those of social awareness of noise and personal sensitivity to noise, which according to Anderson are relatively independent. It consists of two sections of statements aiming to study how the subjects personally feel about noise. In the first section the subjects have to indicate how strongly they agree or disagree with each statement. The second section consists of a list of ordinary everyday activities and the subjects indicate how much they enjoy or dislike the activity. The aim was to distinguish and measure a person's inherent noise sensitivity without being influenced by short-term reactions to a particular noise.

Broadbent-Gregory Annoyance Questionnaire is a 40-item questionnaire which yields two subscales, that of noise annoyance (NA), in fact a measure of noise sensitivity (10 items), and general annoyance (GA) (30 items). The 40 statements describe things and situations which are annoying to many people. The following scale is used in grading each of these things or situations: extremely annoying (3), moderately annoying (2), slightly annoying (1), not annoying (0), have not been in the situation (X) (Anderson 1971; Broadbent 1972).

Weinstein's Noise Sensitivity Scale (Table 2) has been largely used in noise sensitivity studies. It consists of 21 items, which are presented on a 6-point scale rating from "agree strongly" (1) to "disagree strongly" (6). Several items are scored in opposite direction before responses are summed. Last item is

"I am sensitive to noise" which is a short self-rating statement of the person's evaluation of his/her subjective noise sensitivity (Weinstein 1978).

Kishikawa et al. (2006) developed a new noise sensitivity measurement scale named WNS-6B, by excluding, according to them, biased questions from the original Weinstein's Noise Sensitivity Scale (WNS) and applying binary coding to six response options in order to reduce the response bias. To measure subjective noise sensitivity they used the following ten questions from the original Weinstein's Noise Sensitivity Scale in following order: 3, 5, 7, 8, 10, 11, 18, 14, 19 and 21. The degrees of agreement on the statements were asked with six response options ranging from 0 to 5 (from "agree strongly" to "disagree strongly"). The sum of all items (after recoding the 7 items with reverse coding) yielded the respondent's subjective noise sensitivity. A higher score denoted a higher sensitivity to noise (Kishikawa et al. 2006).

Zimmer and Ellermeier (1998) developed a German language noise sensitivity questionnaire which encompasses statements about a wide variety of environmental noises in a range of situations that affect the entire population. The material covers seven content areas: everyday life, recreation, health, sleep, communication, work and noise in general. The 52 items presented in the questionnaire relate to perceptual, cognitive, affective and behavioural responses towards noise in these contexts. An almost equal number of items are scored in each direction. For every item respondents can choose one of four response options ranging from strong disagreement to strong agreement (Zimmer and Ellermeier 1999).

NoiSeQ (the Noise Sensitivity Questionnaire) was recently developed to measure not only global noise sensitivity but also the sensitivity of five domains of daily life. NoiSeQ comprised a total of 35 items from the following five categories: leisure, work, habitation, communication and sleep. The respondents are asked to indicate the extent to which the items applied to their attitudes using the following four-level rating scale: strongly agree (3), slightly agree (2), slightly disagree (1), and strongly disagree (0). To calculate the characteristic value for the global noise sensitivity, the average of the rating values of all 35 items have to be calculated, and for the subscales, the mean value based on the ratings of the corresponding seven items need to be calculated (Schutte et al. 2007; Sandrock et al. 2007).

2.1.2.3 Reliability and validity of noise sensitivity scales

Reliability characterises the repeatability of the measurement and validity characterises the extent to which a measurement procedure is capable of measuring what it is supposed to measure.

Weinstein (1978) stated that the Kuder-Richardson reliability of his noise sensitivity questionnaire was 0.83. A wide variety of the items his noise sensitivity scale were correlated with dormitory noise disturbance.

Kishikawa et al. (2006) have investigated the validity of each question in Weinstein's Noise Sensitivity Scale. According to them Weinstein's Noise

Sensitivity Scale contains some irrelevant questions, which ask about respondents' annoyance toward noise and thus the answers to these questions could be affected by the type of noise exposure. The second problem in Weinstein's Noise Sensitivity Scale is that it adopts Likert scale of six response options, and there is a possibility that the reported relationship between the Weinstein's Noise Sensitivity Score and the subjective reactions to noise are confounded by response bias. Respondents who answer exaggeratedly to one question are likely to answer exaggeratedly to another question, and a correlation between the two questions could be observed. They concluded that their new noise sensitivity measurement scale WNS-6B seemed to be more appropriate to assess noise sensitivity than the original Weinstein's scale.

Öhrström et al. (1988a) found that the Weinstein's Noise Sensitivity Scale and a short noise sensitivity question with an open scale were correlated highly with each other ($r = 0.71$, $p < 0.0001$). Griffiths and Delauzun (1977) used Broadbent-Gregory scale and the self-rating noise sensitivity scale. They found that the reliability of the self-rating noise sensitivity scale was low. Correlation coefficient between Broadbent-Gregory noise sensitivity scale and self-rating of noise sensitivity in noisy and quiet areas of London and Liverpool was between 0.0 and 0.4. Zimmer and Ellermeier (1999) evaluated four German language noise sensitivity measures (one of which was a translation of Weinstein's 1978 Noise Sensitivity Scale). It was found that the one-item ratings did not satisfy established psychometric criteria. They did somewhat worse in matching the pattern of correlations expected with related psychological concepts and did not capture the anger component inherent in increased noise sensitivity.

The assessment of the measurement characteristics of NoiSeQ is based on the generalizability theory, a statistical theory concerning dependability of behavioural measurements. Results of the validity study proved that a single application of NoiseQ is sufficient for determining individual's noise sensitivity. The ratings were age and gender independent. When NoiSeQ was used for measuring global noise sensitivity, the reliability reached a value above 0.90. The validity of the instrument was also proven for the subscales habitation and work, but subscale leisure did not prove satisfactory. A significant difference in annoyance rates was observed between the low and high noise sensitive groups for both the subscales habitation and work. This data supports the validity of NoiSeQ (Schutte et al. 2007; Sandrock et al.).

Table 2. Items of the Noise Sensitivity Scale (Weinstein 1978).

1. I wouldn't mind living on a noisy street if the apartment I had was nice.
2. I am more aware of noise than I used to be. *
3. No one should mind much if someone turns up his stereo full blast once in a while.
4. At movies, whispering and crinkling candy wrappers disturb me. *
5. I am easily awakened by noise.*
6. If it's noisy where I'm studying, I try to close the door or window or move somewhere else.*
7. I get annoyed when my neighbours are noisy.*
8. I get used to most noises without much difficulty.
9. How much would it matter to you if an apartment you were interested in renting was located across from a fire station?*
10. Sometimes noises get on my nerves and get me irritated.*
11. Even music I normally like will bother me if I'm trying to concentrate.*
12. It wouldn't bother me to hear the sounds of everyday living from neighbours (footsteps, running water, etc.).
13. When I want to be alone, it disturbs me to hear outside noises.*
14. I'm good at concentrating no matter what is going on around me.
15. In a library, I don't mind if people carry on a conversation if they do it quietly.
16. There are often times when I want complete silence.*
17. Motorcycles ought to be required to have bigger mufflers.*
18. I find it hard to relax in a place that's noisy.*
19. I get mad at people who make noise that keeps me from falling asleep or getting work done.*
20. I wouldn't mind living in an apartment with thin walls.
21. I am sensitive to noise.*

* Item scored in opposite direction before responses are summed.

(Translation of the scale in Finnish in the Appendix)

2.1.3 Occurrence and stability of noise sensitivity

The percentage of noise sensitive subjects has varied between 20 % and 43 % in previous studies (Table 1). In these studies different scales of noise sensitivity have been used.

The results of different studies of the dependence of noise sensitivity with age have shown conflicting results. Some studies have found that noise sensitivity increases with age (Matsumura and Rylander 1991; Nivison and Endresen 1993). According to Moreira and Bryan (1972) and Belojevic and Jakovljevic (2001) noise sensitivity does not depend upon age. Stansfeld et al. (1985 I) found that noise sensitivity is highest among women of 30–44 years and declines in the older age groups.

In several studies noise sensitivity has not been dependent on sex (Belojevic and Jakovljevic 2001; Langdon 1976 III; Moreira and Bryan 1972). Matsumura and Rylander (1991) found that females had slightly higher noise sensitivity (not statistically significant) in the 20-34 years age group. The difference between the sexes decreased with age.

There are only few studies where the stability of noise sensitivity has been investigated. In the study of Weinstein (1978) the 8-month test-retest correlation on the noise sensitivity scale was 0.63. The change in an individual's score on the noise sensitivity scale was highly correlated with the amount of noise disturbance reported at year's end. Stansfeld (1992) found that high noise sensitivity is more stable than low noise sensitivity and the evidence is strong that noise sensitivity is a stable trait with some consistency across different situations of noise exposure. Zimmer and Ellermeier (1999) compared different noise sensitivity measures. They found that all four noise sensitivity scales measure a homogeneous construct stable over time.

2.2 Noise sensitivity in relation to other factors

2.2.1 Noise sensitivity and sensitivity to other environmental stimuli

Noise sensitivity has been considered a self-perceived indicator of vulnerability to stressors in general, and it is linked to perception of environmental threat and lack of environmental control combined with a tendency to negative affectivity (Stansfeld 1992). Noise sensitive subjects have a predisposition to discriminate environmental conditions and evaluate them (Miedema and Vos 2003). Weinstein (1980) found that some subjects tended to give negative ratings in all investigated categories: noise, privacy, air quality, general neighbourhood satisfaction and neighbourhood amenities, whereas others tended to give consistently positive ratings.

General sensitivity, defined as sensitivity to sensory modalities, such as brightness, colour, pain smell and touch, has correlated significantly with the noise sensitivity measures (Stansfeld et al. 1985 I). Neurophysiological discomfort thresholds for noise, light, cold and heat have been significantly intercorrelated (Öhrström et al. 1988a). Persons highly annoyed by noise have also been annoyed by exposure to odour of hydrogen sulphide and exposure to environmental tobacco smoke (Winneke and Neuf 1992).

Overlaps have been found in the characteristics of persons reporting chemical and noise sensitivities (Bell et al. 1995). Multiple chemical sensitivity encompasses a wide range of subjective symptoms provoked by exposure to low levels of chemicals, foods or other agents in the environment (Woolf 2000). Multiple chemical sensitivity patients often acknowledge hyperreactivity in various other sensory modalities, including noise, light, and touch (Bell 1994).

Some individuals are more sensitive than others and they are more likely to report many complaints at higher intensities (Eriksen and Ursin 2002). It has been suggested that the psychobiological mechanism for this is sensitization in neural loops maintained by sustained attention and arousal. The simplest form of plasticity in the nervous system is that repeated stimulation can lead to habituation (decreased response) or sensitization (increased response) (Eriksen and Ursin 2002). These two processes occur at the synaptic level (Ursin and Eriksen 2001). Sensitization is an increased efficiency in the synapse due to repeated use (Ursin and Eriksen 2001). Cortisol and related hormones may be key participants in sensitization (Bell 1996). Subconvulsive kindling of limbic structures may explain why some individuals get more sensitive than others to a variety of stimuli (Eriksen and Ursin 2004).

It has been suggested that sensitization is a major mechanism for multiple chemical sensitivity (Bell et al. 1996) and that it is a psychobiological mechanism underlying a cluster of illness, referred to as "subjective health complaints", including annoyance to noise, when such complaints become intolerable. Sustained stress responses or sustained arousal may be an important factor for the development of these conditions (Ursin and Eriksen 2001).

In the study of Bell et al. (1995) the group rating high both for illness from chemicals and for noise sensitivity had characteristics predictive of heightened sensitizability on time-dependent sensitization: i.e. higher female to male ratio, increased rates of drug abuse problems in blood relatives (genetic risk factor), the trait of shyness (hyperreactivity to novelty) and carbohydrate craving. They reported the lowest rates of current smoking or personal drug abuse problems and the highest frequency of illness from drinking a small amount of alcohol. Young adults with both chemical and noise sensitivity were more similar to multiple chemical sensitivity patients than their peers with chemical or noise sensitivity alone. The findings suggested that limbic system dysfunction associates more with chemical than with noise sensitivity (Bell et al. 1995).

The central nucleus of amygdala occupies a special position in the organization of stress responses (Pacak and Palkovits 2001). Amygdala is also one of several brain regions that modulate startle reactions to unexpected noise (Davis 1992; Fendt et al. 1994). It is also the most sensitive portion of the brain to chemical stimuli (Bokina et al. 1976).

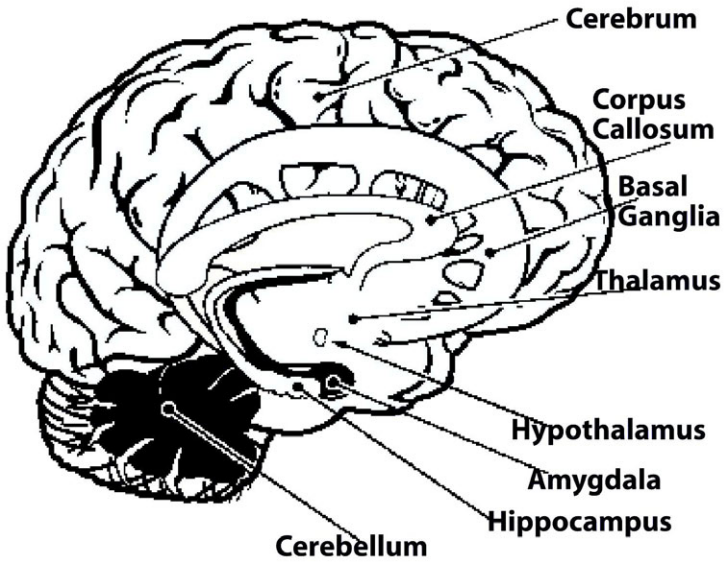

Figure 2. Limbic system (adapted from the website Home of the Carleton University Cognitive Science Student Society).

It has been hypothesized that connections between thalamic structures of the auditory system and subcortical areas (amygdala, hippocampus, hypothalamus – see Figure 2) act as a fast reacting "memory chain" establishing and enhancing adverse excitations during noise exposure. Thalamo-amygdala tract is responsible for full-blown "fear responses" evoked by auditory stimuli as has been shown by several experiments in animals. It can be seen as a fear memory system (Spreng 2000b). A study using fMRI (Functional-Magnetic-Resonance-Imaging) has demonstrated that an amygdalar contribution to conditioned fear learning can be revealed in human subjects (LaBar et al. 1998).

The differences between noise sensitive and non-noise sensitive subjects in the mechanisms of noise perception have apparently not been studied. Differences in serotonin 5-HT_{1A} (5-beta hydroxytryptamine 1A) receptor density may be related to environmental awareness (Borg et al. 2003). Rylander (2004) has hypothesized that this could be important for the individual variation in the reception of sound-mediated information through the central nervous system.

It has also been hypothesized that individual differences in limbic system reactivity and central nervous system sensitizability underlie vulnerability to environmental stimuli (Bell et al. 1992; Bell 1994). Individuals who are sensitive to both chemicals and noise might be among the most vulnerable to limbic dysfunction and to sensitization of limbic and other central nervous system by multiple environmental factors (Bell et al. 1995).

2.2.2 Noise sensitivity and annoyance

The non-auditory effects of noise are generally viewed as stress-related. Annoyance is one of the first reactions to environmental noise (Ouis 2002). It is the most well documented and widespread subjective response to noise. At any noise level there can be individuals who take little notice of the noise and individuals who are very annoyed by it. Noise seems to sort individuals into annoyance categories according to their vulnerability to stress. Annoyance reactions to noise have often been associated with reported interference of noise in everyday activities (Stansfeld 1992). Annoyance is also the most common outward symptom of stress in individuals exposed to noise. These symptoms may be considered as indications for possible more serious health problems (Ouis 2002).

In general annoyance is defined as a feeling of displeasure that is tied to a cause that is believed to affect negatively an individual or a group of individuals. Judging the degree of noisiness caused by a change in sound level contains a cognitive and an emotional component. The cognitive component is concerned with the expectations for the sound to meet some characteristics for an ideal environment. The emotional component is related to the change in mood of the affected person as caused by the exposure to the noise event. Annoyance may be conceived as an emotional process as this reaction is closely tied to the affective experience towards the noise source (Ouis 2002).

Annoyance has also been determined as a multifaceted psychological concept, covering immediate behavioural noise effects aspects, like disturbance and interfering with intended activities, and evaluative aspects like "nuisance", "disturbance", "unpleasantness", and "getting on one's nerves". In spite of the fact that annoyance is related to acoustic variables, they do not play an overwhelming role in the concept of annoyance (Guski et al. 1999).

Noise sensitivity and annoyance are considered to be related but not identical concepts (Nivison 1992). According to Stansfeld (1992), a crucial question is how far noise sensitivity can be viewed as independent of annoyance. The key distinction is that while annoyance is related to noise level, sensitivity is not. Schultz (1978) published a single dose-response relationship for annoyance due to transportation noise from which it is possible to predict the percentage of highly annoyed in different noise levels. From such curve it is not possible to predict which individuals are in the "highly annoyed" group (Weinstein 1980).

Annoyance is correlated with noise sensitivity (Lopez Barrio and Carles 1993; Matsumura and Rylander 1991; Stansfeld 1992; Stansfeld et al. 1993; Öhrström et al. 1988a). Noise sensitivity increases annoyance independently from, and above, level of noise exposure. Noise sensitive individuals are likely to be more annoyed by noise than non-noise sensitive individuals at all noise levels (van Kamp et al. 2004).

Noise sensitivity seems to have a stronger effect on annoyance than noise level (Taylor 1984). Meijer et al. (1985) found considerable differences in the annoyance experience for different categories of noise sensitivity. A negative appreciation of other environmental noises than road traffic noise and of the living environment increased the prevalence of traffic noise annoyance. Noise sensitivity and this appreciation were mutually dependent. Noise sensitive subjects had a greater risk of being annoyed by other sounds than road traffic noise (aeroplanes, neighbours, work) as well.

Thus noise sensitivity is an independent predictor of annoyance response to noise. It is an intervening variable between noise exposure and annoyance and explains much of the variance between noise exposure and individual annoyance responses. Noise sensitivity changes the influence of noise exposure on annoyance. It affects the rate at which annoyance increases when noise exposure gets higher (Miedema and Vos 2003). Highly noise sensitive subjects have demonstrated significantly higher noise annoyance in high noise areas (van Kamp et al. 2004; Stansfeld 1992).

2.2.3 Noise sensitivity and health

There are several studies on health effects of noise, but only a few studies have investigated noise sensitivity and health. Ising and Michalak (2004) investigated reactions to noise-induced communication disturbance during a seminar in men. A positive correlation was found between noise sensitivity and the systolic blood pressure increases during the seminar. However, the correlation between noise sensitivity and systolic blood pressure increases in the laboratory exposure was negative. In field conditions several hours of exposure to road noise at level 60 dB has shown to cause greater blood pressure reactions in noise sensitive subjects than in noise insensitive subjects (Ising 1983 in Ising and Kruppa 2004).

In an experimental study of Di Nisi et al. (1987) the low noise sensitivity group showed lower average amplitude of heart rate response than the high noise sensitivity group. Noise sensitivity has been related to slower habituation of heart rate responses to loud threatening noises (Stansfeld 1992). In a prospective epidemiological study of Otten et al. (1990), blood pressure increases were correlated with self-reported sensitivity to noise. In a cross-sectional study of Nivison and Endresen (1993) individuals with high noise sensitivity had more health complaints, including cardiac, intestinal and allergic complaints than persons with low sensitivity.

Nivison and Endresen (1993) found that individuals with high noise sensitivity had poorer sleep. Noise sensitivity alters self-reported sleep disturbance attributed to noise (Miedema and Vos 2003). It has been significantly correlated with disturbance by noise during sleep. A significant noise effect on subjective sleep quality has been found among noise sensitive subjects. Neither sex, age nor employment rate were related to the different symptoms (Öhrström et al. 1988b). Marks and Griefahn (2007) found correlations between noise sensitivity and subjective sleep quality in terms of worsened restoration, decreased calmness, difficulty to fall asleep, and body movements.

Noise sensitivity has been correlated with anxiety and with nervous complaints in women (Nivison and Endresen 1993). Noise sensitive individuals have had more prominent symptoms of inadequacy, depression, anxiety, sensitivity, anger, tension, inferiority and nervousness (Iwata 1984). In some studies noise sensitivity has been associated with current psychiatric disorder (Stansfeld 1992; Stansfeld et al. 1985 I). However, this does not mean that either the presence of psychiatric morbidity is a necessary prerequisite for reporting high sensitivity to noise or that noise sensitivity is merely a symptom of psychiatric morbidity. An association with the neurotic end of the spectrum of depressive illness has been found. Among depressed patients there has been no evidence to support an association between noise sensitivity and major depression in particular. However, noise sensitivity scores have been considerably higher in the depressed patients compared to the control subjects (Stansfeld 1992). Highly noise sensitive subjects who made some recovery from depression between the two occasions of testing did become less sensitive. This suggests that high noise sensitivity in these patients was partly secondary to current psychiatric state and that it diminishes as psychiatric symptoms lessen (Murata and Sakamoto 1995; Stansfeld 1992).

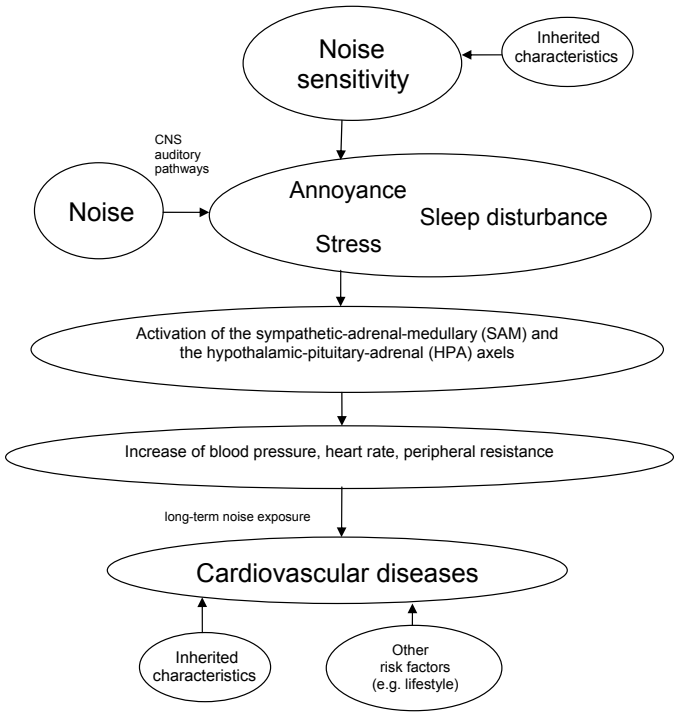

Figure 3. Model of the schematic pathways of the possible relationsship of noise sensitivity and cardiovascular disease.

Figure 3 presents a hypothetical model of the schematic pathways of the possible relationship of noise sensitivity and cardiovascular disease. Noise sensitivity predicts noise annoyance and increases stress and insomnia caused by noise exposure. The noise-induced stress activates the sympathetic and endocrine systems. Prolonged exposure to noise can cause permanent health effects, such as cardiovascular disease.

2.2.4 Noise sensitivity and hearing

The evidence for a sensory component in noise sensitivity is weak. Self-reported noise sensitivity has not been related to auditory acuity, but the effects observed suggest it to reflect a judgemental, evaluative predisposition towards the perception of sounds (Ellermeier et al. 2001). Hearing levels measured by pure tone audiometry have not correlated with noise sensitivity (Stansfeld 1992; Stansfeld et al. 1985 II). There have been no significant differences in noise sensitivity between those with normal hearing and those with impaired hearing according to audiometric thresholds (Stansfeld 1992).

However, a higher prevalence of noise sensitivity has been found among males with noise-induced hearing impairment compared with males having normal thresholds of hearing. No significant differences were found between

females with or without hearing impairment (Aniansson et al. 1983; Peterson and Aniansson 1988).

Noise sensitivity is not a synonym of *hyperacusis*, which can be defined as loudness related hypersensitivity involving a perception of discomfort experienced at sound levels lower than the average loudness discomfort level. In hyperacusis sounds are frequently painful and the uncomfortable loudness level is markedly decreased. Exposure to loud sounds worsens the condition for some time. Pure tone audiograms show normal hearing or a slight high tone loss (Anari et al. 1999). Hyperacusis can appear with many different diseases of the auditory system involving either the ear or the central auditory pathways. The pathogenesis probably involves a central mechanism of efferent auditory pathways (Katzenell and Segal 2001). Prevalence of hyperacusis has varied between 8 % and 15 % in two different studies (Baguley 2003).

Phonophobia is also a different concept than noise sensitivity. Phonophobia means an abnormal strong reaction of anxiety, often in combination with hyperacusis, experienced in high level sound environments. It is regarded to indicate enhanced connections between the auditory and the limbic systems (Jastreboff in Katzenell and Segal 2001).

2.2.5 Psychological characteristics of noise sensitive persons

Results of the personal characteristics of noise sensitive individuals have been inconsistent. Associations have been found between noise sensitivity and introversion. Noise sensitivity has had much in common with a desire for privacy (Weinstein 1978). Noise sensitive individuals have been annoyed by a wide variety of nuisances and they have been less comfortable and effective in social situations, and lower in dominance, capacity for status, sociability and social presence (Weinstein 1978). Belojevic et al. (2001) found that extroverts were less annoyed and had better concentration during mental performance in noise, as compared to introvert subjects. On the other hand Stansfeld et al. (1985 I) did not find that noise sensitivity was related to extroversion.

Noise sensitivity has been associated with a cluster of personal characteristics and subclinical symptoms of neuroticism (Belojevic and Jakovljevic 2001; Job 1999; Stansfeld 1992) or negative affectivity (Job 1999; Stansfeld 1992).

2.2.6 Heritability of noise sensitivity, noise-induced hearing loss and other sensory sensitivities

It has been suggested that genetic factors, previous experiences and simultaneous presence of other environmental stimuli play a role in noise sensitivity (Rylander 2004). Genetic and constitutional individual differences

may increase the likelihood that a particular organ system will respond to noise more than others, and over time lead to a disease (Westman and Walters 1981). Any studies prior to this study on heritability of noise sensitivity have not previously been conducted according to the literature available.

In several animal studies genetic susceptibility to noise-induced hearing loss has been investigated (Davis et al. 1999; Davis et al. 2001; Davis et al. 2003; Davis et al. 2007; Di Palma et al. 2001; Dunn et al. 1991). Individual animals and humans have shown differing susceptibility to noise damage which may be related to unknown genetic components. Workers exposed to the same level of noise exhibit different levels of noise-induced hearing loss. Any gene which weakens the ear functionally or structurally would make the ear more susceptible to noise damage. Gene mutations having a phenotype which is expressed throughout the body would probably shorten the organism's life. A more useful strategy is to look for genes encoding proteins specific to the ear. It is possible to observe these genotypic differences in tissue samples obtained from the living organism by using powerful molecular techniques. There may be many genes which play a role in workers' susceptibility to noise-induced hearing loss (Davis et al. 2003).

A genetic influence on a related trait, odour identification, has been demonstrated in twin studies of Segal et al. (1992) and (1995). In a recent Finnish study the genetic component of olfactory-related traits was studied by performing genome-wide screens utilizing phenotypic data gathered by psychophysical smell testing of humans. Evidence of suggestive linkage was found for an olfactory-related trait, pleasantness of cinnamon odour. Phenotypic variation of pleasantness of the odour was shown to have a strong genetic component that may have an influence on regulation of detection or central processing of the odour signal. It was concluded that perception of odours is potentially modified by genes other than those encoding odorant receptors (Knaapila et al. 2007a).

Taste sensitivity genes have also been studied. PTC (phenylthiocarbamide) and PROP (6-n-Propylthiouracil) carry a chemical group, which is responsible for their characteristic bitter taste. The incidence of taste blindness to PTC/PROP varies from about 3 % in Western Africa to more than 40 % in India (Tepper 1998). Variability in the threshold to PTC is controlled by a major locus with incomplete dominance as well as by a multifactorial component (Reddy and Rao 1989). According to a recent Finnish study individual differences in sweet taste preferences appear to be partly heritable. A locus on chromosome 16 was found to affect the use frequency of sweet foods (Keskitalo et al. 2007). About two thirds of variation in food neophobia (reluctance to eat unfamiliar foods) is genetically determined (Knaapila et al. 2007 b).

3 AIMS OF THE STUDY

The general aim of the present study was to investigate the association of noise sensitivity with specific somatic and psychological factors, including the genetic component of noise sensitivity, and the association of noise sensitivity with mortality.

The specific questions addressed were as follows:

1. Is noise sensitivity independent of noise exposure levels assessed from noise maps? (I)
2. Which personality traits, health-related lifestyle, use of medicines, chronic illnesses and symptoms are associated with noise sensitivity? (II)
3. Does noise sensitivity have a genetic component? (III)
4. Does noise sensitivity increase mortality, especially cardiovascular mortality? (IV)

4 MATERIALS AND METHODS

4.1 Subjects

The study is based on the Finnish Twin Cohort, which was compiled in 1974 from the Central Population Registry of Finland and consists of all Finnish adult same-sex twin pairs (n = 17 357) born before 1958 and with both members alive in 1967 (Kaprio and Koskenvuo 2002). Questionnaires were sent in 1975 and 1981 to the twins of the cohort, as described in detail elsewhere (Kaprio et al. 1978; Kaprio and Koskenvuo 2002).

In 1988 a case-control study was carried out to study the relationship between noise and hypertension. A questionnaire was sent to 1005 twin pairs discordant for hypertension. After two reminders, 1495 individuals (688 men, 807 women) replied, giving a response rate of 74.7 %. The mean age of the respondents was 55.5 years with standard deviation 11.6 years, and the age range was 31–88 years. They were the participants in the first (I) and fourth (IV) substudies.

Twins aged 31–70 years in 1988 who had responded to both questionnaires in 1981 and 1988 were included in the second (II) substudy (N = 1355). Respondents older than 70 years (n = 133) were excluded from the analyses because of the risk of reduced response validity and increased morbidity with age. To evaluate the stability and validity of noise sensitivity, a new questionnaire was sent in 2002 to a sample (n = 405) of the subjects who had replied to the 1988 questionnaire. Of these, 327 individuals replied, yielding a response rate of 81%.

In the third (III) substudy the study sample consisted of 573 same sexed twin pairs (131 monozygotic and 442 dizygotic twin pairs) formed from the respondents to the 1988 questionnaire (both twins had answered the question on noise sensitivity). The age range was 31-88 years.

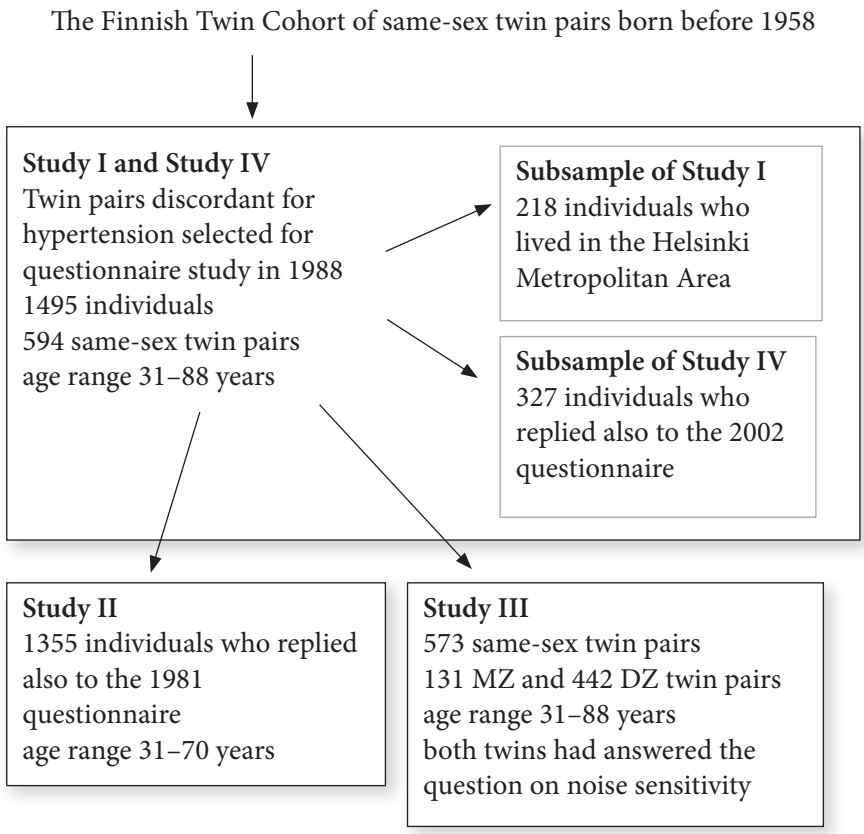

Figure 4. Selection criteria, characteristics and number of subjects in substudies.

4.2 Study designs

Substudy I: Cross-sectional study design
Self-report of noise exposure was compared with the noise exposure levels indicated in noise maps while taking into account measures of self-reported annoyance and noise sensitivity.

Substudy II: Cross-sectional and longitudinal study design
The association of noise sensitivity with health status and psychological factors was investigated as follows:

The 1988 questionnaire was used to assess noise sensitivity, lifetime noise exposure and hypertension. Other health status and psychological factors were obtained from a questionnaire that was administered to the same individuals in 1981. To evaluate the stability of noise sensitivity, a new questionnaire was sent in 2002 to 405 subjects who had replied to the 1988 questionnaire.

Substudy III: Classic twin study design
The genetic component of noise sensitivity was investigated using the classic twin study design comparing similarity of MZ and DZ twin pairs.

Substudy IV: Follow-up study
The association of coronary heart, cardiovascular and total mortality with noise sensitivity and how this association is affected by self-reported lifetime noise exposure was studied. Self-reported noise sensitivity, lifetime noise exposure 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. Data on deaths and causes of death were obtained from record linkage to the nationwide register of death certificates. All deaths that occurred among the study population during the 15 years of follow-up were classified as being due to all causes, to cardiovascular diseases, including the number of deaths due to coronary heart diseases and to causes other than cardiovascular diseases.

4.3 Measures

4.3.2 Questionnaires

4.3.2.1 Questionnaire in 1988
Noise sensitivity was investigated using the question: "People experience noise in different ways. Do you experience noise generally as very disturbing, quite disturbing, not especially disturbing, not at all disturbing or can't say?" Noise sensitivity was determined from the answers in the following way: Subjects answering "very disturbing" and "quite disturbing" were classified as noise sensitive, and subjects answering "not especially disturbing" and "not at all disturbing" were classified as not noise sensitive.

Lifetime noise exposure was measured 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. Noise exposure at home and at work was divided into three exposure categories: not at all (score 1), less than 7 years (2) and 7 years or more (3). Noise exposure during leisure time hobbies was divided into two categories: no noisy leisure time hobby (1) and some noisy leisure time hobby (2). Scores were summed and then subjects were divided into the categories of no lifetime noise exposure (sum score 3) and lifetime noise exposure (sum score 4–8).

Hypertension was elicited by asking: "Has a doctor ever told you that you have elevated blood pressure?" The response alternatives were: no or yes.

4.3.2.2 Questionnaire in 1981

Information on illnesses was obtained by asking "Have you ever been told by a doctor that you have had: Chronic bronchitis; emphysema; asthma; allergic rhinitis, such as hay fever; allergic eczema; epilepsy; high blood pressure; angina pectoris, in other words chest pain due to coronary disease, myocardial infarct, "a coronary", necrosis in the heart muscle; peptic ulcers (stomach or duodenal ulcer); migraine; rheumatoid arthritis; gallstones; Parkinson's disease; psoriasis; any other long or serious illness, which?" The response alternatives for each condition were: no or yes. Allergy included asthma, allergic rhinitis and/or allergic eczema.

The presence of autonomic nervous symptoms was assessed using the question "Have you had any of the following symptoms during last month: Heart palpitation without any physical effort, irregular heart beats, chest pain while angry or emotionally upset, perspiration without physical effort, facial blushing?"

The response alternatives were: daily or almost daily, about once a week, less often and not at all. For analyses, these responses were combined to create a binary variable, such that the first three answers were classified as yes (i.e. any time during past month) and the last answer as no.

Information on the use of medicines was elicited by asking: "During the last year, on how many days together did you use the following types of medicines: fortifying medicines (such as iron or vitamin preparations), pain relievers, antihypertensive drugs, heart drugs, antacids, drugs for skin disorders, sleeping pills, tranquillizers?"

The response alternatives for each class of medicines were: have not used, less than 10 days, 10–59 days, 60–180 days (2–6 months), over 180 days (over 6 months). The first response alternative was classified no use, the other alternatives were classified as any use.

The experienced stress of daily activities was measured on a scale developed by Reeder et al. 1968 on the basis of the four self-reported statements of stress in daily activities (Korkeila et al. 1998). The subjects were divided into the categories of high (4–13) and low stress (14–16).

Extroversion and neuroticism were measured using the abbreviated Eysenck Personality Inventory (EPI) (Flodérus 1974), which includes nine items for the assessment of extroversion and ten items for neuroticism. The subjects were divided into the categories of low extroversion (score 0–4) and high extroversion (5–9) and low neuroticism (0–4) and high neuroticism (5–10).

Hostility was measured using a 3-item scale as described by Koskenvuo et al. (1988). A hostility scale was formed by summing Likert scale responses to these three items: ease of anger arousal, irritability and argumentativeness. The subjects were categorized as having low hostility (score 3–7) and high hostility (8–15).

Life satisfaction was measured on Allardt's four-item scale according to whether respondents found their lives to be interesting, happy, easy or lonely (Koivumaa-Honkanen et al. 2000). The subjects were rating each descriptor on a four or five point scale and a sum scale was formed (range 4–20). They were divided into the categories of satisfied (score 4–6), slightly dissatisfied (7–11) and very dissatisfied (12–20).

Sleep quality was measured by asking "Do you generally sleep well?" The response alternatives were: well (1), fairly well (2), fairly poorly (3), poorly (4) and can't say (5). The subjects were divided into the categories of poor sleep quality (3–4) and good sleep quality (1, 2, 5).

A current cigarette smoker was defined as a person who had smoked at least 5–10 packs of cigarettes in his or her whole life and was smoking daily or nearly daily at the time of the questionnaire study (1981). The subjects were divided into the categories of current, former and never-smokers (Kaprio and Koskenvuo 1988).

Alcohol use was measured by asking the subjects how much beer, wine and spirits they consumed on average per week or month, as well as the frequency of their use (Romanov et al. 1987). The responses were transformed into grams of alcohol from beer, wines and spirits based on frequency, quantity and alcohol content, and then summed to yield total monthly consumption. The subjects were divided into the categories of heavy and not heavy alcohol consumption on the basis of the use of grams of alcohol per month. Heavy alcohol consumption was defined for men as the consumption of more than 900 grams and for women of more than 400 grams monthly.

4.3.3 Noise maps

In the first substudy noise map information of the Helsinki Metropolitan Area was used. Noise maps accumulating the traffic noise information were available for road traffic noise, railway noise and the noise of the Helsinki-Vantaa and Helsinki-Malmi Airports. The noise maps used were chosen so that time of their measurement was as close as possible to 1988.

Only for road traffic noise exposure in Helsinki was a continuous dB-scale available. For other transportation noise sources in Helsinki, and for all transportation noise sources in Vantaa and Espoo, the subjects were grouped into 5-dB categories. Subjects' noise map exposure levels according to the noise maps were for road traffic noise: in Helsinki continuous scale from 55 dB: levels were between 57 dB and 75 dB, in Vantaa: 50–54 dB, 55–59 dB, in Espoo: 55–59 dB, 60–64 dB; for railway noise in Helsinki, Vantaa and Espoo 50–54 dB, 55–59 dB; for aircraft noise around Helsinki-Vantaa airport: 55–59 dB, 60–64 dB and around Helsinki-Malmi airport 45-49 dB, 50–54 dB, 55–59 dB.

The noise map exposure level of each subject was determined by their address at the time of the questionnaire study. The time the subjects lived in a particular residence was known, based on residence records kept by the Central Population Register (CPR) of Finland. Changes of the residence must be reported to the CPR by Finnish law.

4.3.4 Mortality

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.

4.3.5 Twin analysis

The observation of population variation in a certain trait is the starting point for gene finding. The phenotypic variation may be attributed to genetic and environmental causes. When the same variant of a gene differentially affects the phenotype in different environments, genetic and environmental effects are interacting. Within a population many different alleles may exist for the same gene. The different effects of alleles of the same gene are the basis of the model that underlies quantitative genetic analysis (Posthuma et al. 2003).

Monozygotic (MZ) twins derive from a single fertilized egg and therefore inherit identical genetic material. MZ twins reared together share part of their environment and 100% of their genes. Dizygotic (DZ) twins share on average 50% of their segregating genes. Any resemblance between MZ twins is attributed to shared environmental sources or genetic sources. The extent to which MZ twins do not resemble each other is ascribed to unique non-shared environmental factors or to measurement error. Any resemblance between DZ twins due to genetic influences will be lower than for MZ pairs. The extent to which DZ twins do not resemble each other is due to non-shared genetic influences and to non-shared environmental factors (Boomsma et al. 2002; Falconer 1986; Hall 2003; Martin et al. 1997; Posthuma et al. 2003; Zhao et al. 1997).

A widely used study design compares phenotypic resemblance of MZ and DZ twins reared together, which is called the classic twin study design (Boomsma et al. 2002; Posthuma et al. 2003). Comparing the resemblance of MZ twins for a disease or a trait with the resemblance of DZ twins offers

the first estimate of the extent to which genetic variation determines its phenotypic variation (Boomsma et al. 2002).

The classic twin study design allows decomposition of the phenotypic variance into components of additive genetic variance, non-shared environmental variance and either dominant genetic variance or shared environmental variance. The total amount of genetic influence on a phenotype is the sum of the additive (A) and dominance (non-additive) (D) effects of alleles at multiple loci, plus variance due to the interaction of alleles at different loci. At a single locus genetic effects can be partitioned into additive or dominant genetic effects, or their combination. The total environmental effect is partitioned to common (shared) environmental effects (C), which are environmental influences shared by twins reared together, and to unique (non-shared) environmental effects (E) (Figure 5) (Neale and Maes 2006; Posthuma et al. 2003).

In DZ twins the correlation of genetic effects will be on average 0.50, if all contributing alleles act additively and there is no interaction between them within or between loci. If some alleles act in a dominant way the correlation of genetic dominance effects will be 0.25. Thus the presence of dominant gene action reduces the expected phenotypic resemblance in DZ twins relative to MZ twins. Epistasis (describes the interaction between alleles at different loci) reduces this similarity even further (Neale and Maes 2006; Posthuma et al. 2003).

The covariance (or correlation) of DZ twin pairs will be less than the MZ covariance, but greater than one half of the MZ covariance, if both additive genetic effects and shared environmental effects contribute to variation in a trait. The covariance of DZ pairs will be less than one half of the MZ covariance, if both additive genetic effects and dominant genetic effects contribute to variation in a trait. The effects of shared environment and genetic dominance are confounded and cannot be estimated simultaneously in data on twin pairs reared together (Neale and Maes 2006; Posthuma et al. 2003).

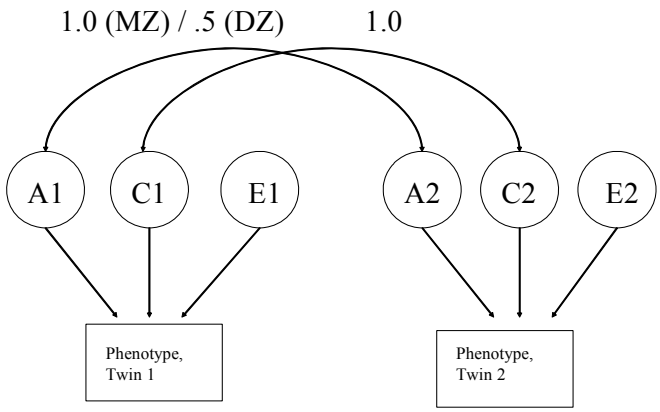

Figure 5. Univariate model for estimation of heritability in the classic twin model.

In the schematic path diagram, A (additive genetic effects), C (shared environmental effects), and E (unique or non-shared environmental effects) represent latent variables, of which A and C are correlated between twins.

4.4 Statistical methods

Cohen's coefficient of agreement for nominal scales (Cohen 1960), Pearson chi-square and logistic regression models were used. Factor analysis (Feinstein 1996), principal components method with varimax rotation, was used to explore the relationship between annoyance, self-reported noise exposure and noise sensitivity.

Assessment of twin similarity was first conducted by computing polychoric correlation coefficients (i.e. noise sensitivity in twin A vs. noise sensitivity in twin B) (Neale et al. 2003; Neale and Cardon 1992; Neale and Maes 2006). Before further model fitting, confirmation of the central assumptions for the twin analyses was made. The distributions of noise sensitivity were studied using the method of maximum likelihood estimation for raw data observations. This method utilizes all available information, including that from pairs in which only one twin has responded.

An initial fully saturated model, in which all the distributions for the first and second twins in both zygosity groups were free to vary, was compared to successively more constrained models by likelihood ratio test. The distributions were first set equal for first- and second-born co-twins and then set equal for MZ and DZ pairs. Standard model fitting methods were

employed using Mx, a program for analysis of twin and family data (Neale et al. 2003; Neale and Cardon 1992) fitting directly to the raw ordinal data.

The Cox proportional hazards regression model was used to evaluate the risk of mortality in relation to noise sensitivity (Cox and Oakes 1984). 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).

Computer analyses were made using the BMDP package (Dixon 1988), Stata (versions 8 and 9) (StataCorp. 2003, 2005), and Mx (Neale et al. 2003).

5 RESULTS

5.1 Measurement, occurrence and stability of noise sensitivity (I)

Noise sensitivity was investigated using a short question on experience of noise. In the 2002 test-retest study questionnaire the Weinstein's Noise Sensitivity Scale (scale reliability coefficient = 0.86) was also used. The short question on noise sensitivity and the Weinstein's Noise Sensitivity Scale were markedly correlated (r = 0.60).

Of all 1455 subjects who had answered the question on noise sensitivity, 38 % were noise sensitive (36 % of women and 41 % of men). In Figure 6 are shown distributions of noise sensitivity in different sensitivity categories in women and in men.

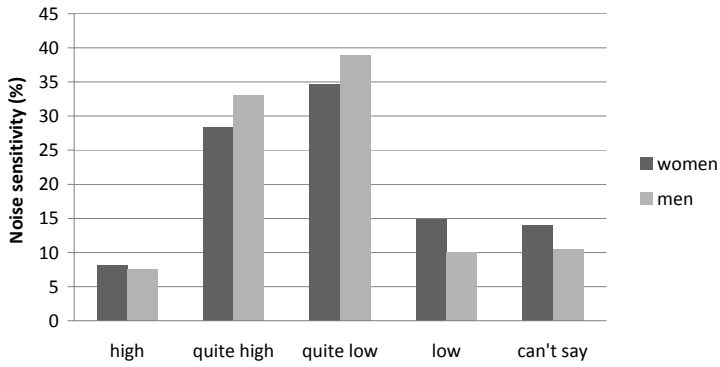

Figure 6. Noise sensitivity among women (n = 677) and men (n = 778) by sensitivity category.

The overall tendency was decreasing noise sensitivity with age (age range 31–70 years) (Figure 7). The age related differences in noise sensitivity were statistically significant among men (p-value 0.003) and women (p-value < 0.001).

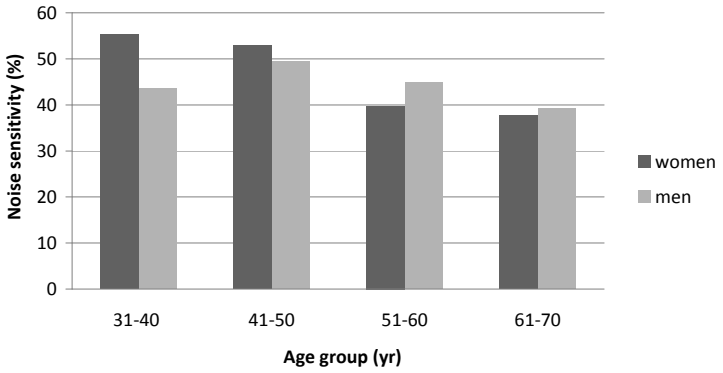

Figure 7. Noise sensitivity among men (n = 645) and women (n = 663) by age category.

According to the 1988 and 2002 questionnaires, noise sensitivity was a quite stable trait over a 14 year period (Cohen's Kappa 0.42, 95% CI 0.31–0.53). Of subjects who were noise sensitive in 1988, 66% were still noise sensitive in 2002, and 75% of those who were not noise sensitive in 1988 were also not noise sensitive in 2002. Among older women (age 46–52 years in 1988 and 60–66 years in 2002) noise sensitive subjects tended to become insensitive to noise. 58% of noise sensitive women in 1988 were still noise sensitive in 2002, whereas 71% of younger noise sensitive women (age 31–45 years in 1988 and 45–59 years in 2002) were still noise sensitive in 2002. Among men there were no such differences.

5.2 Noise sensitivity, self-report of noise exposure and noise map information (I)

In the first substudy some analyses were made using the noise map information of the Helsinki Metropolitan Area. Noise sensitivity was independent of noise exposure levels indicated in transportation noise maps. In the Helsinki Metropolitan Area 46 % of the subjects who determined their noise sensitivity and who lived in the area of noise maps were noise sensitive. 47 % of the subjects who determined their noise sensitivity and who lived outside the area of noise maps were noise sensitive. In factor analysis noise sensitivity was independent of both noise map information and annoyance.

Subjects with high noise sensitivity reported more transportation noise exposure than subjects with low noise sensitivity. Noise sensitive subjects reported exposure outside the environmental noise map areas almost twice as often as non-sensitive subjects. Also in the areas of aircraft, railway and road traffic noise, noise sensitive subjects reported more often noise exposure,

but the difference between these groups was less obvious than outside the noise exposure areas (Figure 8).

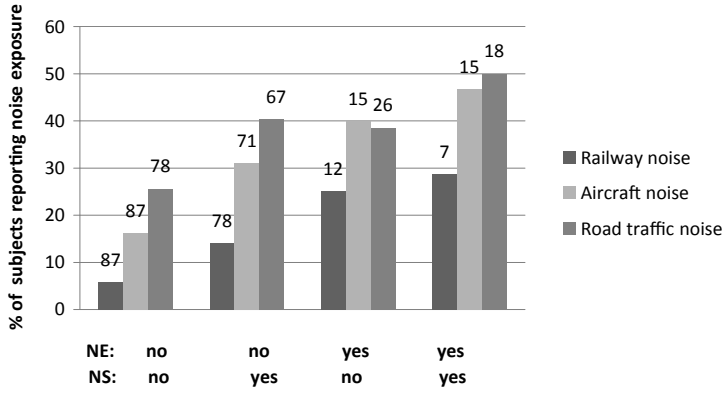

Figure 8. Percentage of subjects reporting noise exposure by noise exposure levels indicated in noise maps (NE) and noise sensitivity (NS).*

* numbers above the columns indicate the number of subjects in different categories

5.3 Association of noise sensitivity with somatic and psychological factors and mortality (II & IV)

Noise sensitivity was significantly associated with hypertension, emphysema, use of psychotropic drugs (sleeping pills, tranquillizers and pain relievers), smoking, stress and hostility, even when lifetime noise exposure was adjusted for (Table 3). The results indicate that noise sensitivity has both somatic and psychological components.

In further analyses separately among men and women, in full models including age, sex, hypertension, emphysema, stress, hostility, use of sleeping pills and tranquillizers, use of pain relievers, smoking and lifetime noise exposure, noise sensitivity was associated significantly with stress (adjusted OR 1.61, 95% CI 1.07–2.42), hostility (adjusted OR 1.09, 95% CI 1.02–1.17) and hypertension (adjusted OR 1.61, 95% CI 1.05–2.46) in women, while in men it was associated with stress (adjusted OR 1.75, 95% CI 1.16–2.63), emphysema (adjusted OR 6.19**,** 95% CI 1.56–24.64) and use of sleeping pills and tranquillizers (adjusted OR 1.53, 95% CI 1.13–2.08) (Table 3).

Cardiovascular mortality was significantly increased among noise sensitive women (hazard ratio 1.80, 95% CI 1.07–3.04). Among men there were no statistically significant effects (Table 4). Coronary heart mortality among noise sensitive women was increased but not statistically significant (age adjusted hazard ratio 1.69, 95% CI 0.89–3.21), with no increase in men.

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.

In Table 5 are shown the interactions of noise sensitivity, lifetime noise exposure and hypertension with coronary heart and cardiovascular mortality in women. Among men there was no statistically significant effect. Hypertensive women with noise sensitivity had increased risk of both coronary heart and cardiovascular mortality. Coronary heart mortality was significantly increased among noise sensitive women reporting lifetime noise exposure, but not among those noise sensitive women 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.

Table 3. Noise sensitivity in relation to other explanatory factors.

		All subjects		Women		Men	
		Age- and sex-adjusted OR	Full model * adjusted OR	Age- and sex-adjusted OR	Full model * adjusted OR	Age- and sex-adjusted OR	Full model* adjusted OR
Hypertension	no	1.00	1.00	1.00	1.00	1.00	1.00
	yes	1.47	1.43	1.59	1.61	1.30	1.29
	95% CI	1.16–1.86	1.07–1.93	1.14–2.22	1.05–2.46	0.93–1.82	0.85–1.96
Emphysema	no	1.00	1.00	1.00	1.00	1.00	1.00
	yes	3.87	4.43	1.42	1.80	4.84	6.19
	95% CI	1.48–10.10	1.51–12.99	0.38–5.27	0.23–14.26	1.55–15.09	1.56–24.64
Stress	low	1.00	1.00	1.00	1.00	1.00	1.00
	high	1.78	1.67	1.70	1.61	1.65	1.75
	95% CI	1.40–2.26	1.25–2.23	1.22–2.36	1.07–2.42	1.19–2.29	1.16–2.63
Hostility	low	1.00	1.00	1.00	1.00	1.00	1.00
	high	1.54	1.06	1.09	1.09	1.08	1.02
	95% CI	1.20–1.98	1.01–1.11	1.03–1.15	1.02–1.17	1.02–1.15	0.95–1.10
Use of sleeping pills and tranquillizers	no	1.00	1.00	1.00	1.00	1.00	1.00
	any use	2.12	1.30	1.18	1.17	1.62	1.53
	95% CI	1.47–3.05	1.09–1.55	1.00–1.39	0.94–1.46	1.27–2.07	1.13–2.08
Use of pain relievers	no	1.00	1.00	1.00	1.00	1.00	1.00
	any use	1.73	1.17	1.19	1.14	1.31	1.21
	95% CI	1.32–2.26	1.02–1.34	1.03–1.37	0.95–1.37	1.11–1.54	0.99–1.48
Smoking	never	1.00	1.00	1.00	1.00	1.00	1.00
	former	1.77	1.48	1.18	1.39	1.83	1.46
	95% CI	1.28–2.44	1.02–2.15	0.69–2.02	0.76–2.56	1.21–2.78	0.90–2.37
	current	1.10	1.00	1.29	1.29	0.99	0.85
	95% CI	0.80–1.51	0.68–1.46 0.82–2.03		0.76–2.18	0.63–1.53	0.50–1.42
Lifetime noise exposure	no	1.00	1.00	1.00	1.00	1.00	1.00
	yes	1.43	1.06	1.21	1.09	1.11	1.06
	95% CI	1.11–1.84	0.94–1.19	1.07–1.38	0.91–1.30	0.98–1.25	0.90–1.24

* Full model includes age, sex, hypertension, emphysema, stress, hostility, use of sleeping pills and tranquillizers, use of pain relievers, smoking and lifetime noise exposure

Table 4. 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

*Adjusted for age, hypertension, lifetime noise exposure, smoking and emphysema

Table 5. Interaction of noise sensitivity, hypertension 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).

a) Noise sensitivity and hypertension

Coronary heart mortality				Cardiovascular mortality			
		Noise sensitivity				**Noise sensitivity**	
		No	Yes			No	Yes
Hypertension				Hypertension			
No				No			
	n/N	5 / 148	2 / 81		n/N	10 / 148	6 / 81
	Hazard ratio	1.0	11.02		Hazard ratio	1.00	5.42
	95% CI		0.21–587.30		95% CI		0.92–32.04
Yes				Yes			
	n/N	15 / 229	16 / 202		n/N	28 / 229	33 / 202
	Hazard ratio	2.63	5.09		Hazard ratio	2.24	3.57
	95% CI	0.98–7.09	1.90–13.64		95% CI	1.10–4.56	1.78–7.17
p for interaction		0.514		p for interaction		0.607	

b) Noise sensitivity and lifetime noise exposure

Coronary heart mortality				Cardiovascular mortality			
		Noise sensitivity				**Noise sensitivity**	
		No	Yes			No	Yes
Lifetime noise exposure				**Lifetime noise exposure**			
No				No			
	n/N	17 / 252	7 / 156		n/N	31 / 252	19 / 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				Yes			
	n/N	3 / 134	11 / 127		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	

5.4 Genetic component of noise sensitivity (III)

The overall distribution of noise sensitivity was quite similar in MZ and DZ twins (Figure 9)[1]. After excluding the pairs in which one or both twins replied "can't say" on the noise sensitivity question 105 MZ (43 male) and 339 DZ (160 male) pairs remained for analyses. 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.

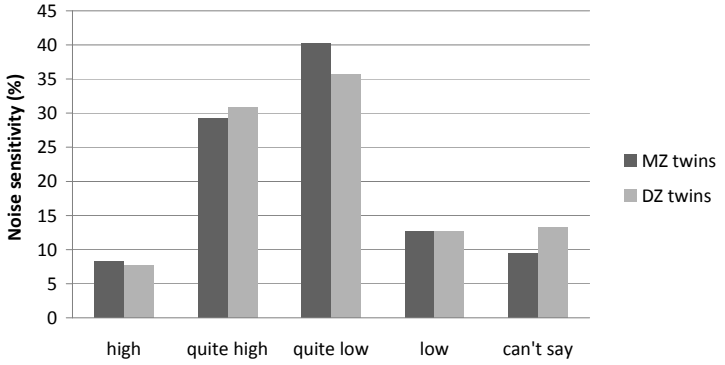

Figure 9. Noise sensitivity among MZ twins (n=315 individuals) and DZ twins (n=1140 individuals).

Best fitting model was the AE model, which indicates that genetic factors and unique experiences account for variability in noise sensitivity in the population. AE model provided an estimate of heritability of 36 % (95 % CI 0.20–0.50) with the remainder due to unique environment factors. Excluding twins with hearing impairment did not significantly change the twin correlations and the heritability estimate was 40% (95 % CI 0.24–0.54). No significant gender differences in the genetic component of noise sensitivity were found.

Monozygotic pairs were more similar for noise sensitivity than dizygotic pairs, and quantitative genetic modelling indicated significant familiality. The study indicates that noise sensitivity aggregates in families and probably has a genetic component.

[1] In the original article in Twin Research and Human Genetics 8 (3), 245–249, 2005, in Table 1 the numbers of the two rows of quite low and low noise sensitivity and can't say had been switched (Erratum in Press).

6 DISCUSSION

6.1 Reliability and stability of short noise sensitivity questions and occurrence of noise sensitivity

The short question on noise sensitivity used in this study and the Weinstein's Noise Sensitivity Scale were markedly correlated. This result is in concordance with the previous study of Öhrström et al. (1988a).

The percentage of noise sensitive subjects was 38 %, which is in accordance with the results of previous studies, in which the percentage has varied between 20 % and 43 %. In these studies different scales of noise sensitivity were used which may explain the large variation in the proportions of noise sensitive subjects (Table1). Thus noise sensitivity is a rather common trait.

Noise sensitivity was a relatively stable trait, which is also in concordance with previous studies. According to Stansfeld (1992) the evidence is strong that noise sensitivity is a stable trait with some consistency across different situations of noise exposure. In the study of Weinstein (1978) the 8-month test-retest correlation on the noise sensitivity scale was 0.63.

The overall tendency was decreasing noise sensitivity with age (age range of the subjects was 31–70 years) which is in concordance with the results of Stansfeld et al. (1985 I). However, some studies have found that noise sensitivity increases with age (Matsumura and Rylander 1991; Nivison and Endresen 1993) or that it does not depend upon age (Moreira and Bryan 1972; Belojevic and Jakovljevic 2001).

6.2 Noise sensitivity and self-report of transportation noise exposure

Noise sensitivity was independent of exposure levels indicated in transportation noise maps, which is in accordance with previous studies (Belojevic and Jakovljevic 2001; Lopez Barrio and Carles 1993; Meijer et al. 1985; Raw and Griffiths 1988).

Subjects with high noise sensitivity reported more transportation noise exposure than subjects with low noise sensitivity. Noise sensitive subjects reported aircraft, railway and road traffic noise exposure outside the transportation noise map exposure areas almost twice as often as non-sensitive subjects. Moreira and Bryan (1972) found that noise sensitive subjects have

a fairly high level of annoyance for quite moderate levels of noise, but their annoyance does not increase very greatly with increasing noise level.

Results of the present study indicate that self-report of noise-related items, such as noise sensitivity and annoyance, can and should be used to supplement noise map information in noise protection. Noise sensitivity should also be taken into account in noise guideline values in noise protection.

6.3 Association of noise sensitivity with medical and psychological factors and cardiovascular mortality

In this study noise sensitivity was associated with hypertension, emphysema, use of psychotropic drugs, stress, hostility and smoking. In previous studies the association of noise sensitivity with hypertension has been found (Ising and Michalak 2004; Otten et al. 1990). The finding of the present study that emphysema is associated with noise sensitivity is new in the literature. It should be noted that the number of subjects with emphysema in this study was low (n = 22). Overall, emphysema was strongly associated with former and current smoking in the 1981 questionnaire study. 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.

In previous studies noise sensitivity has been correlated with sleep quality parameters (Nivison and Endresen 1993; Öhrström et al. 1988b). However, in the study of Nivison and Endresen (1993) noise sensitivity was not related to use of sleeping pills. Noise produces both physical and psychological stress. In previous studies noise sensitivity ratings have been related to the scales for stress (Zimmer and Ellermeier 1999). The Weinstein noise sensitivity scale has been related to anger (Zimmer and Ellermeier 1999). In the present study noise sensitivity was associated with ex-smoking but not with current smoking. In previous studies noise sensitivity has not been associated with current smoking (Bell et al. 1995; Nivison and Endresen 1993). Noise sensitivity seems to be associated with many factors that are also known as a health effect of noise exposure such as hypertension, sleep disturbance and stress. In this study when taking into account lifetime noise exposure these associations did not diminish.

Noise sensitivity has been associated with neuroticism in previous studies (Belojevic and Jakovljevic 2001; Stansfeld 1992). In the present study the age and sex adjusted association of neuroticism with noise sensitivity was quite strong and significant, but it diminished in the multivariate analyses and became non-significant. This weakening of the association may represent adjustment for intermediary variables (stress and hostility) that mediate the relationship between neuroticism and noise sensitivity.

Cardiovascular mortality was significantly increased among noise sensitive women, but not in men. For coronary heart mortality the interaction of

noise sensitivity and lifetime noise exposure was statistically significant in women. Further studies are needed to investigate this interaction. When the interactions were analysed separately in women the number of deaths was quite small and the 95% CI were wide (Table 5). In previous studies cardiovascular complaints have been related to noise sensitivity in women, but not in men (Nivison and Endresen 1993).

The models were adjusted for age, hypertension, lifetime noise exposure, smoking and emphysema (Table 4). Smoking and hypertension are major risk factors for cardiovascular disease. The risk for residual confounding remains, even if statistical corrections are performed.

It was not possible to find any previous studies on the association of noise sensitivity with cardiovascular mortality. There are previous studies on noise and mortality (Davies et al. 2005; Frerichs et al. 1980; Meecham and Shaw 1979, 1993; Melamed et al. 1999), but the results of these studies cannot be compared with the results of the present study as they were not studying the association of noise sensitivity with mortality.

In previous studies mortality has been investigated with respect to occupational (Davies et al. 2005; Melamed et al. 1999) and environmental noise exposure (Frerichs et al. 1980; Meecham and Shaw 1979, 1993) separately. The present study used self-reported lifetime noise exposure, which includes both environmental and occupational noise exposure and also noise exposure of leisure time hobbies. No noise level measurements or other objective estimations of noise exposure were made.

6.3.1 Noise and cardiovascular disease

There are several studies on noise and cardiovascular disease. In a meta-analysis of Babisch (2006a, 2006b) 61 epidemiological noise studies were evaluated regarding the relationship of transportation noise and cardiovascular end points. Both sound level and annoyance were associated with a higher risk of ischaemic heart disease. For hypertension the results were not as consistent as for the ischaemic heart disease. For noise categories above 60 dB a higher ischaemic heart disease risk was relatively consistently found, but statistical significance was rarely achieved. In studies in men an increase in risk for myocardial infarction was found with increasing noise levels above 60 dB showing a dose-response relationship (Babisch 2006a, 2006b; Babisch 2008). Some studies on mortality at the jet noise area have shown conflicting results (Frerichs et al. 1980; Meecham and Shaw 1979, 1993).

A meta-analysis of 43 epidemiologic studies investigating the relation between noise exposure and blood pressure and/or ischaemic heart disease, showed a significant association for both occupational noise exposure and air traffic noise exposure and hypertension (van Kempen 2002). Occupational noise annoyance has been associated with mean blood pressure elevations when combined exposures with other work-related

stress factors were considered (Lercher et al. 1993).

A trend for positive association has been found between past industrial noise exposure and total mortality (Melamed et al. 1999). An increased risk for all-cause mortality has been found in workers who performed complex jobs under high noise exposure levels compared to those who performed simple jobs under low noise exposure (Melamed and Froom 2002). Chronic exposure to noise levels typical for many workplaces has been associated with an excess risk for acute myocardial infarction death (Davies et al. 2005). In a recent Finnish study long-term occupational noise exposure was associated with an increase in coronary heart disease risk in industrially employed men (Virkkunen et al. 2005).

Occupational noise exposure may also have sustained, not transient, effects on vascular properties and it also enhances the development of hypertension. The high-noise-exposed workers had significantly higher systemic vascular resistance than low-noise-exposed workers during work and sleep periods. Low-noise-exposed workers had significantly higher brachial artery compliance, brachial artery distensibility and systemic vascular compliance than high-noise-exposed workers during offduty periods (Chang et al. 2007).

In urban areas inhabitants are exposed to both air pollution and noise, which in the case of road traffic, occur simultaneously. As it is well established that air pollution influences cardiovascular diseases, future epidemiological noise studies should consider other pollutants as confounding if not aggravating factors and take into account the combined effects of noise and air pollution (Schwela et al. 2005).

6.3.2 How to explain the gender differences?

According to the results of the present study, cardiovascular mortality is significantly increased among noise sensitive women, and the interaction of noise sensitivity and noise exposure may increase the risk of coronary heart mortality in women but not in men. There were also differences between men and women in the association of noise sensitivity with somatic and psychological factors. Noise sensitivity was associated significantly with stress, hostility and hypertension in women and with stress, emphysema and use of sleeping pills and tranquillizers in men.

Constitutional and genetic individual differences may increase the likelihood that a particular organ system will respond to stressors, such as noise, more than others and over time lead to a disease (Westman and Walters 1981). It could be hypothesized that the gender and genetic differences found in this study may have to some extent an evolutionary background. Possibly women are more prone to environmental noise because of an evolutionary born alertness to protect their own children against dangers. It is difficult to make a more detailed hypothesis of the connections of noise sensitivity with evolution.

The major risk factors for coronary heart disease for both men and women include hypertension, dyslipidemia, obesity, diabetes mellitus, cigarette smoking, sedentary lifestyle and poor nutrition. Although many risk factors are similar in men and women, gender differences related to dyslipidemia and diabetes mellitus have been reported (Fleury et al. 2000).

Serum total cholesterol and low-density-lipoprotein cholesterol levels predict fatal coronary heart disease in middle-aged (< 65 years) and older men and women (> 65 years). However, the strength and consistency of these relationships in older women were diminished. High-density-lipoprotein cholesterol levels inversely predicted coronary heart disease in middle-aged men and women and in older women, but not in older men (Manolio et al. 1992). A raised non-fasting concentration of triglycerides is an independent risk factor for mortality from coronary heart disease, cardiovascular disease, and any-cause mortality among middle-aged women but not in men (Stensvold et al. 1993). Non-insulin-dependent diabetes mellitus appears to be a stronger risk factor for coronary heart disease in women than in men (Manson and Spelsberg 1996).

In a both cross-sectional and longitudinal industrial noise exposure study (CORDIS) of Melamed et al. (1999) young men (< 44 years) exposed to high noise levels (> 80 dB) had higher total levels of cholesterol and triglycerides, as well as high cholesterol ratio, than young men exposed to low noise levels. In women and in older men (> 45 years) noise did not affect serum lipid or lipoprotein levels. Noise annoyance covaried independently with total cholesterol and high-density lipoprotein levels in young men and with total cholesterol, triglyceride and high-density lipoprotein levels in women under high noise exposure conditions. Noise annoyance and noise exposure levels had an additive effect on cholesterol levels. Young men who scored high on both variables had a 15 mg/dl higher mean cholesterol level than those who scored low on both variables. In women, the corresponding difference was 23 mg/dl. A trend for positive association between past noise exposure and incidence of cardiovascular morbidity, mortality and total mortality was found (in that follow-up part of the study all subjects were men). The trend was statistically significant for total mortality among the extremely high exposed group even after controlling for possible confounders (Melamed et al. 1999).

In an experimental longitudinal study of Maschke et al. (2002) investigating cortisol excretion the study subjects were exposed to electroacoustically simulated aircraft noise. It was found that adaptation to nocturnal aircraft noise was sex specific. Most women belonged to the adaptation type with a stable cortisol trend, whereas the majority of men belonged to the adaptation types with increasing or decreasing cortisol trend. It was concluded that noise-induced health risk for men can be estimated essentially higher than for women.

In men an association has been found between the risk of myocardial

infarction and increasing traffic noise exposure (Babisch et al. 2005), and in women between marginally increased risk of myocardial infarction and annoyance by diurnal environmental noise (Willich et al. 2006). In these studies associations of cardiovascular disease with noise sensitivity were not investigated.

Knipschild (1977) concluded that female subjects use more cardiovascular drugs, especially antihypertensive agents, in areas of aircraft noise compared to quiet areas. In a study on middle-aged men, annoyance due to road traffic noise was associated with a higher incidence of ischaemic heart disease in subjects free of any chronic disease at the beginning of the follow up (Babisch et al. 2003). Significant odds ratios adjusted for age, body mass index and smoking habits have been found for self-reported arterial hypertension and myocardial infarction in very much or extremely noise disturbed male subjects, compared to those who were not annoyed at all, or were slightly annoyed by noise. The respective odds ratios for females were lower and not statistically significant (Belojevic and Saric-Tanaskovic 2002).

There seems to be some differences between men and women in previous studies in the risk factors of cardiovascular disease and in noise effects. However, the results are conflicting and in most studies noise sensitivity has not been investigated. Further studies are needed to investigate differences between men and women in the association of noise sensitivity with cardiovascular and coronary heart mortality found in this study.

6.3.3 Why some persons are noise sensitive and what are the general implications of noise sensitivity?

Noise as a part of modern urbanization is possibly overloading the human sensory environment and individual's processing capacities. The auditory apparatus seems not to be prepared to cope with commonly encountered urban and industrial noise. We are exposed to sound environments that probably overload the auditory system. In addition noise significantly affects the human nervous and endocrine systems (Westman and Walters 1981).

The human auditory system processes the frequencies and intensities relevant to survival in the sound environment of nature, but the evolutionary process has not allowed us enough time to adapt hearing to sounds generated by loud modern sources of noise (Westman and Walters 1981). Previous experiences, simultaneous presence of other environmental stimuli and genetic factors play a part in noise sensitivity (Rylander 2004).

Thalamo-amygdala tract is responsible for full-blown "fear responses" evoked by auditory stimuli (Spreng 2000b). These fear responses evoked by noise could be hypothesized to be more common among noise sensitive subjects than among non-noise sensitive ones.

According to the general stress concept, in subjects chronically exposed to noise repeated autonomic and endocrine responses can result in permanent

functional and metabolic changes (Babisch 2006a). Environmental noise stress has been associated with neuroendocrine physiological reactions. The measurement of stress hormones has been widely used to study the possible increase in cardiovascular risk of noise exposed subjects. In noise studies stress hormones can also be used to identify vulnerable groups (Babisch 2003).

Noise sensitivity is a predictor of noise annoyance and noise sensitive individuals are more affected by noise than less sensitive individuals. Thus it can be hypothesized that the risk of health effects caused by noise, such as hypertension and coronary heart disease, is higher for noise sensitive individuals.

In previous studies general sensitivity has correlated significantly with the noise sensitivity measures (Stansfeld et al. 1985 I). Further investigations are needed to study the association of noise sensitivity with sensitivity to other environmental stimuli. It can be hypothesized that they are connected and there may be similarities in the mechanisms explaining them.

6.4 Does noise sensitivity have a genetic component?

The twin analysis in the present study showed that noise sensitivity does aggregate in families and probably has a genetic component. Monozygotic pairs were more similar for noise sensitivity than dizygotic pairs. The estimate of heritability was 36% with the remainder due to unique environment factors. No significant gender differences in the genetic component of noise sensitivity were found.

The study design had two factors which may have caused an underestimation of the genetic component of noise sensitivity. Firstly the use of self-report measures can dilute the measured effect. Secondly 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 of noise sensitivity.

Familiality can be attributed to the existence of genetic aetiologic mechanism, environmental factors common to family members or a combination of both. If familial aggregation is strong, environmental risk factors alone are unlikely to account for such strong aggregation, unless the presumed environmental risk factors are associated with enormous risk (Guo 2000).

Some genotypes may be more sensitive to the environment than other genotypes (Posthuma 2003). Gene-environment interaction is defined as a different effect of an environmental exposure on disease risk in persons with different genotypes (Ottman 1996). Heritability that is conditional on environmental exposure can indicate the presence of a genotype-environment interaction (Boomsma 2002). This study might have had evidence for gene-

environment interaction, if the heritability of noise sensitivity differed in low and high noise exposure groups. To demonstrate such effects, fairly substantial sample sizes are required and such analyses were not possible in the present study.

Studies on the role of genetic factors in noise sensitivity have not previously been conducted in humans according to the literature available. Heritability of other sensory sensitivities has been studied earlier (Keskitalo et al. 2007; Knaapila et al. 2007a, 2007b; Reddy and Rao 1989; Segal et al. 1992; Segal et al. 1995; Tepper 1998).

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 genetic component of noise sensitivity.

7 SUMMARY AND CONCLUSIONS

A single item noise sensitivity question showed considerable stability and good validity when correlated with Weinstein's multi-item scale. 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.

Noise sensitivity was independent of noise exposure levels indicated in transportation noise maps. Subjects with high noise sensitivity reported more transportation noise exposure than subjects with low noise sensitivity. Noise sensitive subjects reported transportation noise exposure outside the environmental noise map areas almost twice more often than non-sensitive subjects.

Noise sensitivity was associated with hypertension, emphysema, use of psychotropic drugs, smoking, stress, and hostility, even when lifetime noise exposure was adjusted for.

Monozygotic pairs were more similar with regards noise sensitivity than dizygotic pairs, and quantitative genetic modelling indicated significant familiality. The best fitting genetic model provided an estimate of heritability of 36 %.

Follow-up study showed that cardiovascular mortality was significantly increased among noise sensitive women, but not among men. For coronary heart mortality the interaction of noise sensitivity and lifetime noise exposure was statistically significant in women.

Conclusions: Noise sensitivity has both somatic and psychological components. It does aggregate in families and probably has a genetic component. Noise sensitivity may be a risk factor for cardiovascular mortality in women. Noise sensitivity is independent of noise exposure levels indicated in transportation noise maps.

Future research

Health effects of noise have been studied previously, but more studies are necessary to investigate noise sensitivity induced health effects. In further studies noise map information and information of illnesses could be linked and the influence of noise sensitivity on health effects should be investigated. For the city of Helsinki there is now available a new extensive noise mapping of road traffic, railway and aircraft noise concerning the noise situation in 2006, which could be used in further studies of noise exposure, noise sensitivity and cardiovascular mortality. Transportation noise studies should

consider air pollutants as confounding factors and take into account the possible combined effects of noise exposure and air pollution.

In addition gender related associations of noise sensitivity with health effects of noise should be studied. Further studies are needed to investigate the association of noise sensitivity with cardiovascular mortality and the interaction of noise sensitivity and noise exposure which, according to the results of this study, may increase the risk of coronary heart mortality in women. Further studies are also needed to investigate the association of noise sensitivity with emphysema.

The differences between noise sensitive and non-noise sensitive subjects in the mechanisms of noise perception and in the autonomic and endocrine responses to noise exposure should be investigated. Fear responses evoked by noise could be hypothesized to be more common among noise sensitive subjects than among non-noise sensitive ones.

Further investigations are also needed to study the association of noise sensitivity with sensitivity to other environmental stimuli, such as chemical sensitivity. It can be hypothesized that they are connected and there may be similarities in the mechanisms explaining them.

Further large-sample twin studies are needed to investigate the genetic component of noise sensitivity. Applications of behavioural-genetic designs should be generated for studying the possible role of specific genetic factors. Results of the present study can provide new information about the heritability of noise sensitivity that may also help in the search for specific genes underlying noise sensitivity. Fairly substantial sample sizes are also required to demonstrate possible gene-environment interaction.

Practical implications

Noise sensitivity is a rather common trait. Noise contributes to health effects, such as hypertension, ischaemic heart disease and insomnia. It can also impair cognitive functions. According to recent studies noise is also a risk factor for cardiovascular mortality. Noise sensitive individuals are more affected by noise and more vulnerable to noise than less sensitive individuals and the risk of health effects caused by noise can be expected to be higher for them. Noise sensitivity should be taken into account in the risk assessment of transportation noise and in determining the guideline values. Self-report of noise sensitivity and annoyance can supplement noise map information and noise perception surveys should be used in noise protection.

8 ACKNOWLEDGEMENTS

This study was carried out as part of the Finnish Twin Cohort Study at the Department of Public Health of the University of Helsinki, Finland. The Finnish Twin Cohort study is supported by the Academy of Finland Center of Excellence in Complex Disease Genetics.

The study was financially supported by the Finnish Ministry of the Environment, the Finnish Ministry of Social Affairs and Health, The Finnish Work Environment Fund, the Academy of Finland and the City of Helsinki, all of which are gratefully acknowledged.

I am deeply grateful to my supervisor Professor Jaakko Kaprio, Department of Public Health, University of Helsinki and Department of Mental Health and Alcohol Research, National Public Health Institute, for his patience and time in teaching and explaining the twin study methodology. His inspiring research ideas and optimistic attitude have been a great source of motivation. I've always received from him immediate replies to all my e-mail questions in spite of the country where he was when he received the e-mails.

I am extremely grateful to my second supervisor Professor Markku Koskenvuo, Department of Public Health, University of Helsinki, for his sound advice and unending patience while teaching me epidemiology. His friendliness and optimism encouraged me to continue though difficulties.

I want to express my deepest gratitude to my third supervisor Docent Heikki Vuorinen, Department of Public Health, University of Helsinki, who was always ready to patiently and friendlily answer my numerous questions. He was always willing to help me with all issues, even the trivial ones.

I'm warmly thankful to Ph.Lic. Kauko Heikkilä, Department of Public Health, University of Helsinki, who did the hard work with data analysis. During many years he has put long hours into database management. Without his help this work would not have been possible.

I would like to express my sincere gratitude to my co-author Docent Helena Mussalo-Rauhamaa for her encouragement and contribution to this study. Her valuable comments have improved the manuscripts significantly.

I also want to thank the staff and researchers of the Finnish Twin Cohort study for being so supportive. I express my warmest thanks to research assistant Pia Ruokolinna for data entry of the questionnaires. I also want to express my sincere thanks to Eila Voipio, Pirkko Särkijärvi and Sirkka Koskinen for their help with practical matters. I also want to thank Matthew Grainger for revising the language of my thesis.

I want to thank for the collaboration with doctoral students, especially

with Ulla Broms, Anu Raevuori, Suoma Saarni and Kirsi Lillberg for very beneficial conversations and advice.

I am indebted to the reviewers of my thesis, Docent Eeva Sala and Professor Gustav Wikström, whose expertise and supportive attitude I greatly appreciate.

I have spent several pleasant moments discussing current noise issues with Senior Advisor Sirkka-Liisa Paikkala from the Finnish Ministry of the Environment and Docent Tapani Jauhiainen in preparing Finnish publications on health effects of noise. I owe special thanks to Senior Advisor Sirkka-Liisa Paikkala for keeping me up to date in current noise issues and helped me in finding conferences on noise and health. I am very grateful for Docent Tapani Jauhiainen for consulting me about noise and hearing issues and for finding literature especially on those issues.

I also want to thank Helsingin kaupungin Työterveyskeskus and Vantaan työterveys for giving me an opportunity to work as a part-time occupational physician in order to concentrate on my research work.

I am extremely thankful to all my friends for their support and encouragement – you know who you are.

Finally my heartfelt thanks go to my family – my beloved husband Jevgenij, my dear children Anna and Seidi, my mother Helena and my late father Erkki for their loving care and support. I am deeply grateful to my dear daughter Seidi for the graphic design of the first figures and for the cover photo. My family has given me encouragement during all these years.

Helsinki, November 2008 *Marja Heinonen-Guzejev*

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APPENDIX

Main questions used in the study (from questionnaires 1981, 1988 and 2002) in English and Finnish (unofficial translations)

Noise sensitivity measures

Short noise sensitivity question
People experience noise in different ways. Do you experience noise generally as

1 very disturbing
2 quite disturbing
3 not especially disturbing
4 not at all disturbing
5 can't say

Lyhyt meluherkkyys-kysymys
Ihmiset kokevat melun eri tavoin. Koetteko Te melun yleensä

1 hyvin häiritsevänä
2 melko häiritsevänä
3 ei erityisen häiritsevänä
4 ei lainkaan häiritsevänä
5 en osaa sanoa

Weinstein's Noise Sensitivity Scale

Following statements are describing my attitudes to noise. Answer them using the scale from 1 to 6 in following way:

	I totally **agree**					I totally **disagree**
I wouldn't mind living on a noisy street if the apartment I had was nice.	1	2	3	4	5	6
I am more aware of noise than I used to be.*	1	2	3	4	5	6
No one should mind much if someone turns up his stereo full blast once in a while.	1	2	3	4	5	6
At movies, whispering and crinkling candy wrappers disturb me. *	1	2	3	4	5	6
I am easily awakened by noise.*	1	2	3	4	5	6
If it's noisy where I'm studying, I try to close the door or window or move somewhere else.*	1	2	3	4	5	6
I get annoyed when my neighbours are noisy.*	1	2	3	4	5	6
I get used to most noises without much difficulty.	1	2	3	4	5	6
How much would it matter to you if an apartment you were interested in renting was located across from a fire station? *	1	2	3	4	5	6
Sometimes noises get on my nerves and get me irritated.*	1	2	3	4	5	6
Even music I normally like will bother me if I'm trying to concentrate. *	1	2	3	4	5	6
It wouldn't bother me to hear the sounds of everyday living from neighbours (footsteps, running water, etc.).	1	2	3	4	5	6
When I want to be alone, it disturbs me to hear outside noises.*	1	2	3	4	5	6
I'm good at concentrating no matter what is going on around me.	1	2	3	4	5	6
In a library, I don't mind if people carry on a conversation if they do it quietly.	1	2	3	4	5	6
There are often times when I want complete silence.*	1	2	3	4	5	6
Motorcycles ought to be required to have bigger mufflers. *	1	2	3	4	5	6
I find it hard to relax in a place that's noisy.*	1	2	3	4	5	6
I get mad at people who make noise that keeps me from falling asleep or getting work done. *	1	2	3	4	5	6
I wouldn't mind living in an apartment with thin walls.	1	2	3	4	5	6
I am sensitive to noise.*	1	2	3	4	5	6

*Item scored in opposite direction before responses are summed

Weinsteinin meluherkkyys-kysely

Seuraavat väittämät selvittävät tarkemmin suhtautumistanne meluun. Vastatkaa niihin asteikolla 1–6 seuraavasti:

	Olen täysin **samaa** mieltä					Olen täysin **eri** mieltä
Minua ei häiritse meluisan kadun varrella asuminen, jos asunto on hyvä.	1	2	3	4	5	6
Huomaan melua enemmän kuin aikaisemmin.*	1	2	3	4	5	6
Kenenkään ei tulisi välittää siitä, jos joku laittaa stereonsa välillä täysille.	1	2	3	4	5	6
Elokuvissa kuiskaaminen ja karamellipapereiden rapistelu häiritsevät minua. *	1	2	3	4	5	6
Herään helposti meluun. *	1	2	3	4	5	6
Jos huoneessa, jossa keskityn lukemaan, on meluisaa, yritän sulkea oven tai ikkunan tai siirtyä toiseen paikkaan.*	1	2	3	4	5	6
Närkästyn, kun naapurini aiheuttavat melua.*	1	2	3	4	5	6
Totun suurimpaan osaan melua ilman erityisiä vaikeuksia.	1	2	3	4	5	6
En vuokraisi asuntoa, joka sijaitsee paloasemaa vastapäätä.*	1	2	3	4	5	6
Joskus meluäänet käyvät hermoilleni ja saavat minut ärtymään.*	1	2	3	4	5	6
Kun yritän keskittyä, jopa mielimusiikkini häiritsee minua.*	1	2	3	4	5	6
En häiriinny naapureiden jokapäiväisen elämän äänistä (askeleet, juoksevan veden ääni jne.).	1	2	3	4	5	6
Kun haluan olla yksin, minua häiritsevät ulkoa tulevat äänet.*	1	2	3	4	5	6
Olen hyvä keskittymään, tapahtui ympärilläni mitä tahansa.	1	2	3	4	5	6
Kirjastossa en välitä ihmisten keskustelusta, jos he puhuvat hiljaisella äänellä.	1	2	3	4	5	6
On usein aikoja, jolloin toivon täydellistä hiljaisuutta. *	1	2	3	4	5	6
Moottoripyörissä tulisi olla tehokkaammat äänenvaimentimet.*	1	2	3	4	5	6
Minun on vaikea rentoutua meluisassa paikassa.*	1	2	3	4	5	6
Hermostun ihmisiin, jotka metelöivät niin, etten pysty nukahtamaan tai etten saa töitäni tehtyä.*	1	2	3	4	5	6
Voisin asua asunnossa, jossa seinien ääni-eristys on huono.	1	2	3	4	5	6
Olen meluherkkä. *	1	2	3	4	5	6

* Kysymys pisteytetään vastakkaiseen suuntaan ennen kuin tulokset lasketaan yhteen

Lifetime noise exposure

Lifetime noise exposure was measured using three questions about noise exposure at home, at work and noisy leisure time hobbies.

Noise exposure at home:
For how many years during your lifetime have you lived in an apartment where noise has disturbed hearing normal speech? (e.g. transportation noise, machines, or noise inside the apartment such as noisy leisure time hobby)

1 not at all
2 less than a year
3 1–2 years
4 3–6 years
5 7–12 years
6 13–19 years
7 more than 20 years

Noise exposure at work:
For how many years during your lifetime have you been in such work where noise has disturbed hearing normal speech?

1 not at all
2 less than a year
3 1–2 years
4 3–6 years
5 7–12 years
6 13–19 years
7 more than 20 years

Noise exposure during leisure-time hobbies:

During your leisure-time have you hobbies like

shooting, hunting	1	no		
	2	yes	→	For how many years? _____
some noisy motor leisure time hobby	1	no		
	2	yes	→	For how many years? _____
wood or metal work	1	no		
	2	yes	→	For how many years? _____
listening to noisy music	1	no		
	2	yes	→	For how many years? _____
playing in a noisy orchestra	1	no		
	2	yes	→	For how many years? _____
other noisy leisure-time hobby	1	no		
	2	yes	→	For how many years? _____

What is your hobby?_____

Elinaikainen melualtistus

Elinaikainen melualtistus määriteltiin käyttäen kolmea kysymystä melualtistuksesta kotona, töissä ja vapaa-ajan harrastuksissa.

Melualtistus kotona:
Kuinka kauan olette elämänne aikana asunut asunnossa, jossa melu on häirinnyt tavalliseen puheen kuulemista? (esimerkiksi liikenteen melu, koneet, tai asunnon sisäinen melu kuten meluisa harrastus)

1 en lainkaan
2 alle vuoden
3 1–2 vuotta
4 3–6 vuotta
5 7–12 vuotta
6 13–19 vuotta
7 yli 20 vuotta

Melualtistus työssä:
Montako vuotta olette yhteensä olleet sellaisessa työssä, jossa melu on häirinnyt tavalliseen puheen kuulemista?

1 en lainkaan
2 alle vuoden
3 1–2 vuotta
4 3–6 vuotta
5 7–12 vuotta
6 13–19 vuotta
7 yli 20 vuotta

Melualtistus vapaa-ajan harrastuksissa:

Harrastatteko Te vapaa-aikana:

ammuntaa, metsästystä	1	ei			
	2	kyllä	→	Montako vuotta?	_____
jotain meluista moottori-harrastusta	1	ei			
	2	kyllä	→	Montako vuotta?	_____
puu- tai metallitöitä	1	ei			
	2	kyllä	→	Montako vuotta?	_____
äänekkään musiikin kuuntelua	1	ei			
	2	kyllä	→	Montako vuotta?	_____
soittamista äänekkäässä orkesterissa	1	ei			
	2	kyllä	→	Montako vuotta?	_____
muuta meluisaa harrastusta	1	ei			
	2	kyllä	→	Montako vuotta?	_____

Mitä harrastatte? _____

Hypertension

Has a doctor ever told you that you have elevated blood pressure?
(Circle also if you answer no)

1 no
2 yes

Verenpaine

Onko lääkäri koskaan sanonut, että Teillä on tai on ollut korkea verenpaine? (Rengastakaa myös, jos vastaatte ei)

1 ei
2 kyllä

Emphysema

Has a doctor ever told you that you have had?
(Circle also if you answer no)

	no	yes
emphysema	1	2

Keuhkojen laajentuma

Onko lääkäri koskaan sanonut, että Teillä on tai on ollut?
(Rengastakaa myös, jos vastaatte ei)

	ei	kyllä
keuhkojen laajentuma	1	2

Use of medicines

During the last year, on how many days together did you use the following types of medicines? (circle also, even if you have not used any)

	have not used	less than 10 days	10–59 days	60–180 days (2–6 months)	over 180 days (over 6 months)
pain relievers	1	2	3	4	5
sleeping pills	1	2	3	4	5
tranquillizers	1	2	3	4	5

Lääkkeiden käyttö

Kuinka monena päivänä yhteensä **viimeisen vuoden aikana** olette käyttänyt seuraavantyyppisiä lääkkeitä? (Rengastakaa myös, vaikka ette ole käyttänyt)

	en ole käyttänyt	alle 10 päivänä	10–59 päivänä	60–180 päivänä (2–6 kk)	yli 180 päivänä (yli 6 kk)
särkylääkkeitä	1	2	3	4	5
unilääkkeitä	1	2	3	4	5
rauhoittavia lääkkeitä	1	2	3	4	5

Smoking

Have you in your entire life smoked more than 5-10 packs of cigarettes?

1 no
2 yes

Do you still smoke regularly?

1 no → How old were you when you stopped smoking?
____ years old

How many did you smoke on average per day before you stopped?

1 none
2 less than 5 cigarettes
3 10–14 cigarettes
4 15–19 cigarettes
5 20–24 cigarettes
6 25–39 cigarettes
6 over 40 cigarettes

2 yes → How many cigarettes do you smoke daily on average?

1 none
2 less than 5 cigarettes
3 10–14 cigarettes
4 15–19 cigarettes
5 20–24 cigarettes
6 25–39 cigarettes
7 over 40 cigarettes

Tupakointi

Oletteko koko elämänne aikana polttanut enemmän kuin 5-10 rasiaa savukkeita?

1 en ole
2 olen

Poltatteko edelleen savukkeita säännöllisesti?

1 ei → Minkä ikäinen olitte, kun lopetitte?
____ -vuotias

Montako savuketta poltitte keskimäärin ennen kuin lopetitte?

1 ei yhtään
2 alle 5 savuketta
3 10–14 savuketta
4 15–19 savuketta
5 20–24 savuketta
6 25–39 savuketta
7 yli 40 savuketta

2 kyllä → Kuinka monta savuketta poltatte keskimäärin päivittäin?

1 ei yhtään
2 alle 5 savuketta
3 10–14 savuketta
4 15–19 savuketta
5 20–24 savuketta
6 25–39 savuketta
7 yli 40 savuketta

Stress of daily activities

How well do the following statements describe you? Choose the best alternative. The statement describes you well, somewhat, poorly, not at all.

	The statement describes me:			
	Well	Somewhat	Poorly	Not at all
In general, I am unusually tense and nervous	1	2	3	4
There is a great deal of stress connected with my daily activities	1	2	3	4
At the end of the day, I am completely exhausted both mentally and physically	1	2	3	4
My daily activities are extremely trying and stressful	1	2	3	4

Päivittäisiin toimintoihin liittyvä stressi

Kuinka hyvin seuraavat toteamukset sopivat Teihin nähden? Valitkaa sopivin vaihtoehdoista:
Toteamus pitää paikkansa hyvin, melko hyvin, huonosti, ei lainkaan.

	Toteamus pitää paikkansa:			
	Hyvin	Melko hyvin	Huonosti	Ei lainkaan
Yleensä olen tavattoman jännittynyt ja hermostunut	1	2	3	4
Päivittäiseen toimintaani liittyy paljon hermojännitystä	1	2	3	4
Illalla olen aivan uupunut sekä henkisesti että ruumiillisesti	1	2	3	4
Päivittäiset toimintani ovat kovin rasittavia ja painostavia	1	2	3	4

Hostility

Everyone has their own view about themselves. We ask you to describe with the words presented, what kind of a person you think you are. The words are opposites and represent traits and characteristics as far away from each other as possible.

In between the words you can find five lines. Circle one of the lines between the words, nearer by the word you think better describes you.

Examples:

Cheerful — — — (—) sad
This indicates that you are sad.

Blonde — (—) — — dark
This indicates that you are more blonde than brunette.

Please, answer openly, how you really would describe yourself, not by the opinions you think other people have, or how you would perhaps wish to be.

Work fast, do not get too much stuck with any details. Use even the strongest expressions courageously and mark the middle circle only in case both of the characteristics really seem to describe you evenly well or poor.

rarely quarrelsome	—	—	—	—	—	quarrelsome
does not get angry easily	—	—	—	—	—	gets angry easily
gets irritated easily	—	—	—	—	—	doesn't get irritated

Vihamielisyys

Jokaisella ihmisellä on määrätty kuva itsestään. Pyydämme Teitä kuvaamaan esitettyjen sanojen avulla, millaisena pidätte itseänne. Sanat ovat vastakohtaisia ja edustavat ääripäitä jostakin piirteestä tai ominaisuudesta.

Sanojen välissä on viisi viivaa. Rengastakaa jokin sanojen välisistä viivoista lähemmäksi sitä sanaa, jonka katsotte paremmin kuvaavan juuri Teitä.

Esimerkkejä:
hilpeä — — — (—) surullinen
Tällä tavoin osoitatte olevanne surullinen.

vaalea — (—) — — tumma
Tällä tavoin osoitatte olevanne enemmän vaalea kuin tumma.

Vastatkaa avoimesti, millaisena todella pidätte itseänne, ei sen mukaan, millaisena arvelette muiden Teitä pitävän tai millainen kenties haluaisitte olla.

Työskennelkää nopeasti, älkää takertuko liikaa yksityiskohtiin. Käyttäkää rohkeasti ääripäitäkin ja merkitkää ympyrä keskimmäiselle viivalle vain siinä tapauksessa, että molemmat ominaisuudet todella tuntuvat kuvaavan Teitä yhtä hyvin tai yhtä huonosti.

harvoin riitautuva	—	—	—	—	—	melko usein riitautuva
ei suutu helposti	—	—	—	—	—	helposti suuttuva
helposti ärtyvä	—	—	—	—	—	ei ärry helposti