

Genetic Component of Noise Sensitivity

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We investigated the genetic component of noise sensitivity using a twin-study design. The study sample consisted of 573 same-sexed twin pairs from the Finnish Twin Cohort. The 131 monozygotic (MZ) and 442 dizygotic (DZ) twin pairs with an age range of 31 to 88 years replied to a questionnaire on noise and health-related items in 1988. The noise sensitivity of respondents was defined as high, quite high, quite low or low. MZ pairs were more similar with regards noise sensitivity than DZ pairs, and quantitative genetic modeling indicated significant familiarity. The best z-fitting genetic model provided an estimate of heritability of 36% (95% CI = .20–.50) and when hearing impaired subjects were excluded this rose to 40% (95% CI = .24–.54). In conclusion, noise sensitivity does aggregate in families and probably has a genetic component.

New disorders, problems and complaints have been taken more seriously by the public and professionals after they have shown to have a significant genetic component (Newlin, 1997). Noise sensitivity is a predictor of noise annoyance (Stansfeld, 1992). It is more likely related to a disposition to react to noise in general rather than to the physical properties of noise (Nivison, 1992). Noise sensitivity has been found to be independent of noise level (Heinonen-Guzejev et al., 2000; Lopez Barrio & Carles, 1993). Noise sensitivity is not merely synonymous with peripheral auditory acuity but it is a function of a more central process (Moreira & Bryan, 1972). It has not been related to either an auditory threshold (Stansfeld, Clark, Turpin, et al., 1985) or to auditory acuity (Ellermeier et al., 2001).

General sensitivity has been shown to have a strong association with noise sensitivity (Stansfeld, Clark, Jenkins, et al., 1985). Noise sensitivity appears to be a self-perceived indicator of vulnerability to stressors in general, and not only noise (Stansfeld, 1992). It has also been associated with neuroticism (Stansfeld, 1992; Stansfeld, Clark, Jenkins, et al., 1985). In our previous study the observed association between neuroticism and noise sensitivity disappeared when other variables (hypertension, emphysema, stress, hostility, use of psychotropic drugs, smoking

and life-time noise exposure) were taken into account (Heinonen-Guzejev et al., in press).

The aim of this study was to investigate the genetic component of noise sensitivity. Different family study designs can be used to estimate the genetic component of a trait, that is, to what extent genetic differences account for interindividual differences in the trait. One of the most common study designs is the comparison of monozygotic (MZ) and dizygotic (DZ) twin pairs (Posthuma et al., 2003). If noise sensitivity has a genetic component, monozygotic twins would be expected to demonstrate greater similarity with regards noise sensitivity than dizygotic twins.

Materials and Methods

Subjects

In 1988 a case-control study was carried out to study the relationship between noise and hypertension. The study was based on the older part of the Finnish Twin Cohort which was compiled in 1974 from the Central Population Registry of Finland. This cohort consisted of all Finnish adult, like-sexed, twin pairs ($n = 17,357$) born before 1958 with both members alive in 1967 (Kaprio & Koskenvuo, 2002) who had all been sent questionnaires in 1975 and 1981. The extensive questionnaire study in 1975 was carried out to confirm twinning, determine zygosity, and obtain data on health-related variables.

Questionnaire

In 1988 a questionnaire was sent to 1005 twin pairs discordant for hypertension. After two reminders, 1495 individuals (688 men, 807 women) replied, an individual response rate of 74.7%. Their mean age was 55.5 years, standard deviation 11.6 years and age range was 31 to 88 years. Among those that replied were 594 same-sexed twin pairs — 137 MZ twin pairs and 457 DZ twin pairs. For 573 twin pairs (131 MZ and 442 DZ), both twins had answered the question on noise sensitivity (Heinonen-Guzejev et al., 2000).

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Noise Sensitivity

Noise sensitivity was investigated by asking 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?' (Heinonen-Guzejev et al., 2000).

Of all respondents, 38.4% were noise-sensitive (high or quite high noise sensitivity), of which 52.5% were women. The overall tendency was that noise sensitivity decreased with age. To evaluate the stability of noise sensitivity, a new questionnaire was sent in 2002 to 405 subjects who had replied to the 1988 questionnaire. Of these, 327 individuals replied; the response rate was 81%. Stability of the noise sensitivity question was found to be adequate. In order to provide more information on the reliability and validity of the question measuring noise sensitivity, the Weinstein's Noise Sensitivity scale (Weinstein, 1978) was also used in the 2002 questionnaire for the same individuals. The scale reliability coefficient was .86. The short question on noise sensitivity and the Weinstein's Noise Sensitivity scale were quite highly correlated ($r = .60$; Heinonen-Guzejev et al., in press).

Hearing Impairment

Hearing impairment was investigated by asking the question: 'Is your hearing impaired?' The response alternatives were: *no*; *yes, slightly*; *yes, notably*; *yes, I am using a hearing-aid*; and *I don't know*.

Twin Zygosity

Twin zygosity was determined by examining the responses of both members of each twin pair to two questions on the similarity of appearance at school age. These items were similar to those used in the other large twin samples (Sarna et al., 1978). A set of decision rules was then used to classify the twin pairs as MZ, DZ or undetermined zygosity. The validity of the zygosity was studied in a subsample of 104 pairs, and the agreement in classification between the questionnaire and 11 blood markers was 100%. The estimated probability of misclassification was 1.7%.

Statistical Methods

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., 2002; Neale & Cardon, 1992). Before further model fitting, confirmation of the central assumptions for the twin analyses was made. These tests provide evidence for the assumption that first and second twins and twins of both zygosity all represent the same population. 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 information 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 the 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., 2002; Neale & Cardon, 1992) fitting directly to the raw ordinal data.

Results

The overall distribution of noise sensitivity was quite similar in MZ and DZ twins (Table 1). The distributions of noise sensitivity within pairs (twin A vs. twin B) and by zygosity did not differ significantly (difference $\chi^2 = 5.87$, $p = .75$ between the fully saturated model and a model where the distributions of the first and second twins were constrained to be equal in both MZ and DZ pairs).

After excluding the pairs in which one or both twins replied *can't say* on the noise sensitivity question (26 MZ and 103 DZ), 105 MZ (43 male) and 339 DZ (160 male) pairs remained for analyses. Table 2 shows the intraclass correlations in MZ and DZ pairs for noise sensitivity among these respondents and for respondents after excluding pairs in which either had severe hearing impairment. Among all respondents, MZ correlations were higher than DZ correlations. Correlations for male and female pairs did not differ significantly statistically. Noise sensitivity was also classified in two other ways by including those replying *can't say* as either midway between *sensitive* and *not sensitive* subjects or by combining the *can't say* answers to the *quite insensitive* group. When this was done, the correlations for both MZ and DZ pairs decreased slightly.

Excluding those pairs in which one or both were hearing impaired did not significantly change the correlations between twins in MZ and DZ pairs, suggesting that the familiarity of noise sensitivity is not explained by hearing impairment to any great extent. The mean age of the pairs after exclusion (mean age 54.9 years, standard deviation 11.6 years) was nearly the same as for all pairs.

Alternative models fitting additive genetic (A) and common (C) and unique (E) environmental sources of variation are shown in Table 3. The E model could be

Table 1
Noise Sensitivity Among MZ and DZ Pairs

Noise sensitivity	MZ N (%)	DZ N (%)	Total
High	26 (8.3)	88 (7.7)	114 (7.8)
Quite high	92 (29.2)	352 (30.9)	444 (30.5)
Quite low	30 (9.5)	150 (13.2)	180 (12.4)
Low	127 (40.3)	406 (35.6)	533 (36.6)
Can't say	40 (12.7)	144 (12.6)	184 (12.7)
Total	315 (100)	1140 (100)	1455 (100)

Table 2

Intraclass Correlations for Noise Sensitivity in MZ and DZ Pairs

Group	MZ correlation (95% CI)	DZ correlation (95% CI)
	<i>N</i>	<i>N</i>
All*	.36 (.16, .52) 105	.19 (.07, .31) 339
Excluding pairs in which either had severe hearing impairment	.42 (.22, .58) 85	.18 (.05, .30) 272

Note: *Excluding the pairs in which one or both twins replied *can't say* on the noise sensitivity question.

rejected meaning that family factors are needed to account for the pairwise distribution of the data ($p < .001$, $\chi^2 20.44$, $df = 2$), in comparison to the ACE model. The remaining models (AE, ACE and CE) provided adequate fit to the data. In the ACE model, the estimate for C was very small (3%), and the fit of the AE model was better than the CE model when either is compared to the ACE model. Hence the best-fitting model was the AE model, which indicates that genetic factors and unique experiences account for the variability in noise sensitivity in the population. The estimate for the proportion of variance accounted for by genetic factors was 36%, with the remainder due to unique environment factors (not shared with family members). When twins with impaired hearing were excluded, the estimate of the proportion of variance accounted for by genetic factors was 40% in an AE model. The CE model was rejected, as it fit significantly worse than the ACE model ($p = .05$).

Discussion

In this study, the genetic component of noise sensitivity was investigated. There are two factors in the study design which may cause an underestimation of the genetic component of noise sensitivity. First, the use of self-report measures in this study can dilute the measured effect. Second, the genetic component of noise sensitivity was studied among twin-pairs selected for discordance for hypertension. Noise sensitivity has been associated with hypertension (odds ratio = 1.47, 95% CI = 1.16, 1.86; Heinonen-Guzejev et al., in press). Thus discordance for hypertension in the study sample may have led to an underestimation of the genetic component of noise sensitivity.

Excluding twins with hearing impairments only marginally increased the heritability from .36 to .40, suggesting that the familiarity of noise sensitivity is not explained by hearing impairment. Noise sensitivity is not merely synonymous with peripheral auditory acuity (Moreira & Bryan, 1972) and it has not been related to auditory threshold (Stansfeld, Clark, Turpin, et al., 1985). 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 judgmental, evaluative predisposition towards the perception of sound (Ellermeier et al., 2001).

Apart from sex and hearing, other variables were not taken into account as the sample size was relatively modest. Other personality, health behavior and health variables that may be associated with noise sensitivity have been found to be only fairly weakly associated with it (Heinonen-Guzejev et al., in press; Nivison, 1992; Otten et al., 1990; Stansfeld, 1992). Hence they

Table 3

Comparison of Alternative Genetic Models Fit to Finnish Twin Data on Noise Sensitivity*

Model	Components of variance estimates and 95% CI			Goodness-of-fit tests		
	Additive genetic effects (A)	Common environment (C)	-2LL	χ^2 change	<i>df</i>	<i>p</i> -value
ACE	.33 (.00, .51)	.03 (.00, .31)	3108.899			
AE	.36 (.20, .50)	—	3108.928	.03	1	.87
CE	—	.24 (.13, .34)	3110.936	2.04	1	.15
Excluding severe hearing impairment:						
ACE	.40 (.00, .54)	.00 (.00, .26)	2938.864			
AE	.40 (.24, .54)	—	2938.864	.00	1	1.00
CE	—	.24 (.14, .35)	2942.726	3.86	1	.05

Note: -2LL: -2 times log-likelihood of data. *Univariate twin analysis for additive genetic effects (A), common environmental effects (C), and unique environmental effects (E). Model-fitting to data on all MZ and DZ pairs with data on noise sensitivity, and after excluding twins with impaired hearing. Chi-square change relative to fit of ACE model.

were unlikely to account for any major portion of the variance accounted for by familial factors.

According to our 1988 and 2002 questionnaire studies, noise sensitivity is a relatively stable trait (Heinonen-Guzejev et al., in press). There is considerable variation between individuals with regards physiological, emotional and behavioral reactions induced by noise. It is suggested that genetic factors, previous experiences and the simultaneous presence of other environmental stimuli play a role in noise sensitivity (Rylander, 2004).

Overlaps have been found in the characteristics of persons reporting chemical and noise sensitivities (Bell et al., 1995). Persons showing high levels of annoyance to noise in their residential area have also displayed annoyance for exposure in the laboratory to the odor of hydrogen sulfide and exposure to environmental tobacco smoke (Winneke & Neuf, 1992). This supports the hypothesis that noise sensitivity also affects reactions to environmental factors other than noise. Multiple chemical sensitivity (MCS) patients often acknowledge hyperreactivity in various other sensory modalities including noise, light and touch (Bell, 1994). A genetic influence on odor identification, as assessed by the University Pennsylvania Smell Identification Test (UPSIT), was demonstrated on a twin study of 39 MZ and 20 DZ pairs (Segal et al., 1992). In a 1995 study of 46 MZ and 37 DZ twin pairs (Segal et al., 1995), a genetic influence on odor identification, as assessed by the UPSIT, was suggested for males, but not for females. Female twins scored significantly higher on the UPSIT than male twins (Segal et al., 1992, 1995). We did not find any significant gender differences in the genetic component of noise sensitivity.

The time-dependent sensitization model proposes that neurobiological amplification underlies the symptoms and phenomenology of MCS patients (Antelman et al., 1988). Differences in serotonin 5-beta hydroxytryptamine 1 (5-HT_{1A}) receptor density may be related to environmental awareness (Borg et al., 2003) and it has been hypothesized that this could be important for individual variation in the reception of sound-mediated information through the central nervous system (Rylander, 2004). Individuals sensitive to both noise and chemicals might be among those most vulnerable to limbic dysfunction and to sensitization of the limbic system and other central nervous system responses by multiple environmental factors (Bell et al., 1995). Further applications of behavioral-genetic designs should be generated for studying the possible role of specific genetic factors in noise sensitivity.

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