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Road traffic noise, sensitivity, annoyance and self-reported health—A structural equation model exercise

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ABSTRACT

The proposed effect of road traffic noise on hypertension and ischemic heart disease finds mixed empirical support. One problem with many studies is that the directions of the causal relationships are not identified. This is often the case when cross-sectional data and multivariate regression models are utilised. The aim of the study was to explore the relationship between road traffic noise and health. More specifically the relationships between noise complaints, noise sensitivity and subjectively reported hypertension and heart problems were investigated. 1842 respondents in Oslo, Norway were interviewed about their experience of the local environment and their subjective health complaints. The interviews were conducted as part of two surveys. Individual measures of air pollution (NO_2) and noise (Lden) were calculated. The data were analysed using Structural Equation Models. Only sensitivity to noise is related to hypertension and chest pain. No relationships between noise exposure and health complaints were identified. Rather than noise being the causal agent leading to health problems, the results suggest that the noise–health relationships in these studies may be spurious. It is conceivable that individual vulnerability is reflected both in ill health and in being sensitive to noise. The benefit of including more contextual variables in a model of noise–health relationships is supported.

1. Introduction

It has been estimated that more than 30% of EU citizens are exposed to road traffic noise levels above those regarded as acceptable by the World Health Organisation (WHO), and that about 10% report severe sleep disturbance because of transportation noise at night (EEA, 2003). One noise annoyance assessment study suggests that around 24 million people (out of 380 million) in the European Union are highly annoyed by road traffic noise (EEA, 2000). Even with a low impact of noise on health, the large number of EU citizens exposed to road traffic noise implies that the potential population health impacts of road traffic noise can be substantial (de Hollander et al., 1999; de Hollander, 2004). A clear understanding of the causal relationships between noise, noise experience and its potential adverse health effects is therefore crucial for academics, planners and the authorities.

Annoyance and sleep disturbance have been proposed as mediators of the impact of noise on health (Babisch, 2006). The potential role of stress in this relationship is supported by studies suggesting links between noise level and increased noradrenaline concentrations in urine (Babisch et al., 2001), myocardial infarction (Babisch et al., 2005) and hypertension (Aydin and Kaltenbach, 2007; Bluhm et al., 2007; de Kluizenaar et al., 2007; Jarup et al., 2008). However, a meta-analysis of 43 epidemiologic studies found only an effect of *occupational noise* and *air traffic noise (military)* on hypertension, and no effect from *road traffic noise* (van Kempen et al., 2002). The authors concluded that although there was a tendency for increased risk of ischemic heart disease (IHD) and myocardial infarction as a result of *road traffic noise*, the quality of these relationships was in question, mainly due to publication bias and poor noise descriptions in the reviewed studies. Their suggestion that there might be differential effects of road and air traffic noise on IHD was not confirmed. A study looking at noise effects on children found that neither aircraft noise nor road traffic noise had an adverse effect on children's self-reported health status (Stansfeld et al., 2005).

In an attempt to overcome some of the theoretical shortcomings of research on environmental noise and health Lercher (1996) outlines a conceptual framework based on the concept of "embeddedness". This reasoning is based on the work of Cohen (Cohen et al., 1986) who describes an approach where the phenomenon of study (e.g. community noise) is best viewed as systematically surrounded or embedded by a set of events. The embeddedness framework has been applied successfully in previous studies looking at noise exposure and annoyance (Öhrström, 1997; Clench-Aas et al., 2000; Engelien et al.,

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2004; Klæboe et al., 2005; Berglund and Nilsson, 2006), with particular focus on the concept of neighbourhood soundscapes. One important variable to be considered with particular care in such an embedded framework is *noise sensitivity*.

1.1. The role of noise sensitivity on health complaints

Noise sensitivity can be defined as a personality trait that makes certain individuals report more annoyance than their neighbours when exposed to a given noise level (Griffiths and Langdon, 1968). It is a strong predictor of noise annoyance, and moderates the effect of noise exposure on annoyance (Stansfeld, 1992). Noise sensitivity has been associated with subjective health complaints including also cardiac complaints (Nivison and Endresen, 1993). Noise sensitivity has also been linked with a number of medical conditions: hypertension, emphysema, the use of psychotropic drugs, and with behavioural risk factors for disease such as stress, smoking and hostility (Heinonen-Guzejev et al., 2004). A recent study found that cardiovascular mortality is significantly increased among noise-sensitive women. Among men, there were no statistically significant effects (Heinonen-Guzejev et al., 2007). However this study only used a subjective retrospective measure of lifetime noise exposure, and also used a measure of sensitivity where the questionnaire item is more likely to be a measure of general annoyance rather than sensitivity as such ("are you disturbed by noise?").

In an attempt to confirm the role of biological stress mechanisms, laboratory studies have investigated links between levels of the stress hormone cortisol and noise sensitivity. One study looked at noise sensitivity and stress during working conditions when exposed to low-frequency annoying sounds (Waye et al., 2002). The authors concluded that the normal circadian decline in cortisol concentration was significantly affected among subjects who regarded themselves as having a high sensitivity to noise in general. However, the general levels of cortisol were not affected by noise sensitivity or noise exposure during the tasks, implying an interaction between noise, noise sensitivity and time of day on stress response. A recent study aimed at replicating these findings found that cortisol levels, experienced stress or performance during the exposure did not differ between high- and non-sensitive subjects. However, correlations between noise exposure, stress (subjective and objective), and performance were stronger in the highly sensitive group than among the non-sensitive subjects (Ljungberg and Neely, 2007).

1.2. Causal relationships between noise and bad health

The most commonly suggested physiological mechanism for the relationship between noise exposure and detrimental health effects is that noise induces a number of negative outcomes (sleep disturbance, disturbance of daily activities and rest, concentration problems) that results in the chronic activation of the sympathetic nervous and endocrine systems, and elevated levels of physiological risk factors (hypertension, blood lipid levels) that over time give rise to serious health disorders such as cardiovascular disease (Babisch, 2005). In such a model (Fig. 1), annoyance is only described as a psychological side effect. It is however clear from a number of studies that annoyance, rather than actual noise levels, is the factor that has the closest association with cardiovascular diseases (Babisch, 2006). Further, it should be noted that according to psychological theories of stress (Cohen et al., 1986; Levine and Ursin, 1991), it is the persons conscious and cognitive assessment of the stressor (e.g. noise) and its outcomes that is crucial for the stress response. In other words, the potential health effects of noise via stress would have to be mediated by annoyance or some other measure of appraisal.

Stansfeld (1992) discusses the causal relationship between noise sensitivity and psychiatric disorders. He argues that increased sensitivity to noise might be an indicator of increased vulnerability to minor psychiatric disorders. According to this view sensitivity acts as a causal agent for increased degrees of annoyance. The causal relationship with negative (psychiatric) health outcomes is not resolved, but merely described as "associations".

van Kamp et al. (2004) tested the nature of the relationship between sensitivity and annoyance. This study found no evidence of a moderating effect of noise sensitivity on the relationship between exposure and annoyance, but concluded that sensitivity acted as an independent contributor to annoyance. In other words, highly sensitive participants reported higher levels of annoyance regardless of noise level.

Stansfeld's suggestions about vulnerability could be expanded to a model where a third "vulnerability" variable influences sensitivity and also influences health problems. According to such an explanation the proposed relationship between annoyance and health impacts is spurious, and exists because of the impact of noise sensitivity on annoyance.

Stansfeld's model relates vulnerability to mild psychiatric disorders. In the current study we will expand on this and include a range of subjective health complaints as dependent variables in the model (Fig. 2).

2. Objective

The aim of this study is to investigate the hypothesized influence of noise exposure and noise annoyance on subjective health complaints in general, and on reported hypertension and heart problems in particular by the use of an embedded model of the proposed relationships. By exploring the causal pathways between noise, noise sensitivity and reported health outcome the study aims at shedding light on the viability of different causal models for the relationship between noise and health.

3. Method

The studies that were used to analyse the noise–health relationship are two studies conducted in 1987 and 1996 in the city of Oslo (Klæboe et al., 2000). The sub-areas in the study were selected to represent those experiencing increased, decreased and unaltered traffic situations and were not intended to obtain a representative sample of the inhabitants of the area. Within each sub-area we used probability sampling. After quality assurance there were 1842 respondents available for analyses of relationships between noise exposure, sensitivity and health complaints. The respondents were older than 15 years. The mean age of respondents was 42 years, with the oldest respondent being 89 (median 34 years), and 54% were female.

In 1987 face-to-face interviews were carried out in 8 sub-areas. In 1996 we conducted telephone interviews in 14 areas, which along with the original 8 also included 6 sub-areas geographically adjacent. In the analysis, the data from both studies were pooled and treated as one sample of respondents. The response rate was approximately 50% across both surveys.

3.1. Annoyance questions

In both surveys the same wording was used to assess degree of annoyance. Participants were first asked if they heard noise from a certain source, when staying indoors (at home). Thereafter they were asked "Is this noise highly, somewhat or not annoying?" The annoyance scale categories "does not hear" and "hears/not annoyed" were merged before the analyses.

3.2. Sensitivity to noise

Noise sensitivity was measured by a single question using a 3-point scale: "Would you say you are highly, somewhat or not sensitive to noise?"

Table 1
Prevalence of subjective health complaints last 6 months

	Never	Occasionally	Often	Total
Headaches	52	33	16	100
Uncomfortable stomach	82	13	4	100
Sore throat	51	32	17	100
Pain in neck	47	28	25	100
Pain in heart	83	12	5	100
Tiredness	51	30	18	100
High blood pressure	90	5	6	100
Dizziness	81	13	6	100
Annoying rash	73	15	11	100
Nervousness	72	20	8	100
Feel depressed	68	25	7	100
Problems falling asleep	74	16	10	100
Eye irritations	78	14	8	100
Having a cold	56	37	7	100
Breath problems	89	7	4	100

Percentages. $N = 1843$.

3.3. Self-reported health status

We measured health status by asking yes/no questions about chronic conditions (no time specification) and acute health complaints in the last six months. All in all, we recorded 15 acute health problems and five chronic conditions. For the present study we selected six of the acute health problems for analysis. In addition to high blood pressure (hypertension) and pain in the heart (chest pain) we selected three other health complaints based on their assumed relationship with noise exposure or annoyance (problems falling asleep, tiredness and headaches). Sore throat was selected as a control variable since it was assumed to have little direct relationship with noise exposure or annoyance.

3.4. Modifying factors

Age was measured by asking: "What is your age?" The interviewer registered the respondents' sex. Education level was obtained by asking for the number of years of education the interviewee had completed. Other modifying factors, such as employment status and marital status were included in the questionnaire. These were not used in the analysis, as an exploratory multivariate analysis found no significant effect from these variables, and since previous studies have excluded them as significant explanatory variables (see e.g. Fields, 1993; Miedema and Vos, 1999; Fyhri and Klæboe, 2007).

Table 2
Description of study variables

	Non-sensitive	Somewhat sensitive	Sensitive
Mean LA, 24 h+3 dBA	60.0	60.9	61.4
Mean "neighbourhood difference" level, dBA	7.5	7.0	6.7
Mean NO_2 level, ppm	47.0	47.6	48.3
Mean education level, years	11.8	13.0	12.3
Mean age, years	44.4	38.5	45.2
% female	51	53	62
% highly annoyed by road traffic noise indoors	10	23	41
% often having headaches	11	16	33
% often having uncomfortable stomach	3	4	11
% often having a sore throat	12	19	25
% often having pain in neck	18	27	41
% often having pain in heart	5	4	15
% often feeling tiredness	12	20	36
% often having high blood pressure	5	5	10
% often having dizziness	5	5	14
% often having annoying rashes	8	14	15
% often feeling nervous	5	8	21
% often feeling depressed	4	6	22
% often having problems falling asleep	6	9	31
% often having eye irritations	6	8	14
% often having a cold	5	9	12
% often having breathing problems	4	4	8
N	850	778	212

Means or frequencies according to degree of sensitivity to noise.

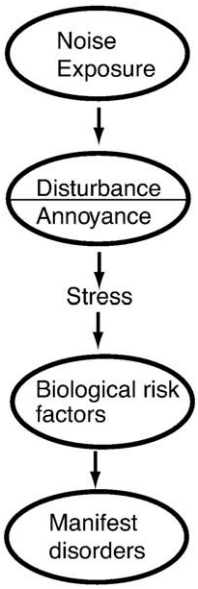

Fig. 1. Original model for testing hypotheses about noise, noise reactions and health in epidemiological research. Based on Babisch et al. (2001). Variables in ellipses are investigated in this article.

3.5. Noise calculations

For each respondent, we calculated the outdoor noise on the most exposed facade, the location of which was determined from survey questions about which streets the different rooms faced.

The 24 h equivalent noise levels at the apartment's most exposed side (LAeq,24h) were estimated using the Nordic calculation method (Public Road Directorate, 1979), with addition of terrain model for one high rise area affected by noise emissions from a larger number of streets. In most

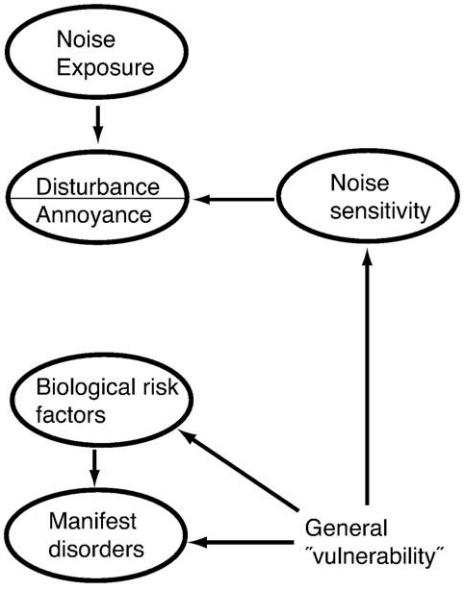

Fig. 2. Proposed alternative model for the relationships between noise, noise reactions and health in epidemiological research. Variables in ellipses are investigated in this article.

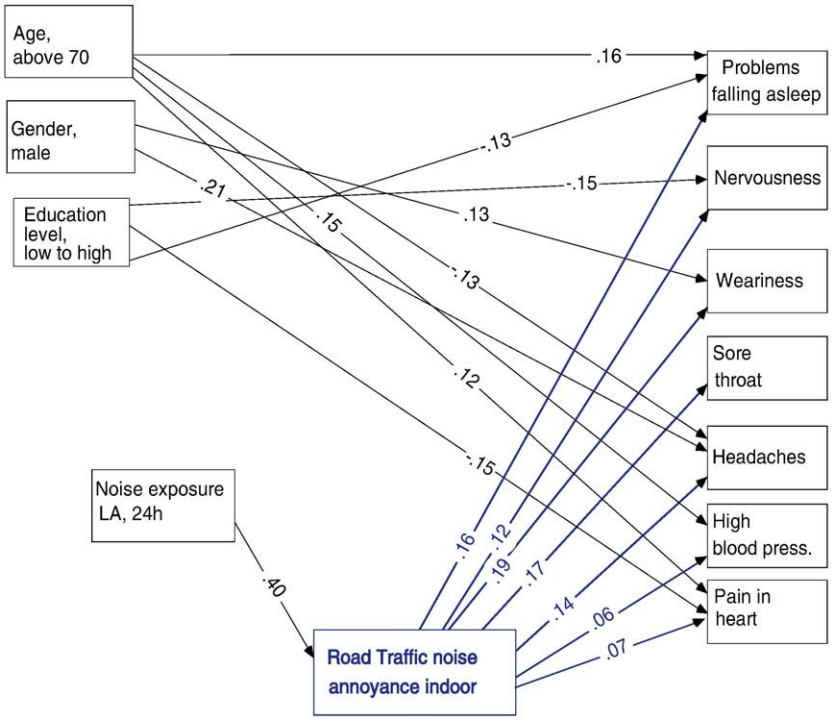

Fig. 3. Model A. Standardized parameter estimates of the relationships between background variables, noise exposure, noise annoyance and subjective health complaints.

cases the noise was calculated from one or two dominating streets. The calculated noise values for each of the surveys were deemed to be within +/−4 dB of the true level. When the calculated noise exposure levels, LAeq,24h, were less than 50 dB, they were set to 50 dB. Maximum noise level was 78 dBA, with a mean of 60.5 dBA. 35% of the dwellings had noise levels at or above 65 dBA, and 74% had noise levels at or above 55 dBA.

3.6. Air pollution calculations

Air pollution exposure was estimated for each resident dwelling location for each hour over a three-month period. NO_2 level (three-month average) was utilised as input to the model periodic mean. NO_2-level at the respondents' addresses ranged from 12 μg/m^3 to

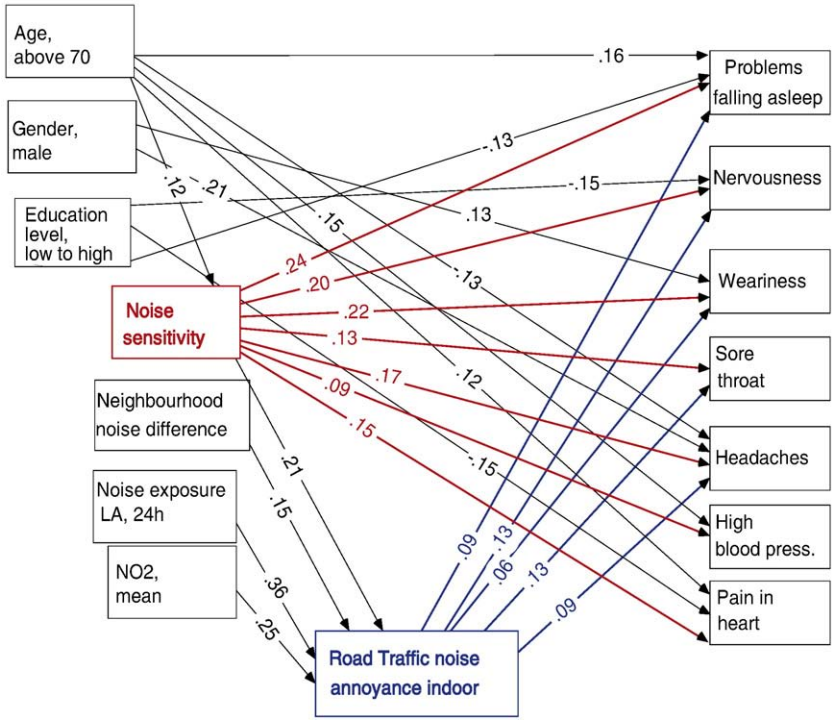

Fig. 4. Model C. Standardized parameter estimates of the relationships between background variables, noise exposure, noise sensitivity, air pollution, noise annoyance and subjective health complaints.

108 μg/m^3 with an average of 47.5 μg/m^3. Further description of these data are given in Clench-Aas et al. (2000).

3.7. Structural Equation Model (SEM)

Regression models are often used for studying relationships between noise exposure and noise annoyance. However, simple regression models only take into account the direct impacts of noise exposure on health and thus neglect the indirect effects. Path models and Structural Equation Models (SEM) are more powerful alternatives to multiple regression analysis. One advantage is that the structural model can be represented as a path model, which allows estimating both indirect and direct effects. In our case, this makes it possible to investigate to what degree noise annoyance is a mediating factor in the noise–health relationship. Further, SEM analyses open for the researcher to evaluate the fit between data and theory. This cannot be done in traditional multiple regression analyses.

In order to analyse causal relationships in the data material we adopted a sequential procedure where we first explored the data with a fairly simple model and then expanded on this model in two steps, inspecting different estimates of model fit at each step. There are a number of ways of assessing model fit for structural models. The first and simplest is to look at the probability level (p). Using the simple probability level as measure of fit has been much debated especially for models based upon large samples (Jöreskog, 1969). In cases such as ours with 1800 respondents, measures of approximate fit are often used to assess the merit of the model failing the simple chi-squared criterion. (Hu and Bentler, 1995). The χ^2 to degrees of freedom ratio has no exact interpretation, but it has been suggested that ratios of 2 and below (the lower the ratio, the better the fit) are indicative of an acceptable fit between the hypothetical model and the sample data (Byrne, 1989).

There are two ways of conducting alterations to models in SEM: all original paths of the altered model can be retained, or alterations to the path structure can be done as suggested by the modification indices provided by the software. The latter approach is considered to be part of an exploratory part of the model fitting, and the results thus need independent confirmation. We followed both procedures.

Possible mediation (indirect effects) was tested by bootstrapping (Shrout and Bolger, 2002).

4. Results

Table 1 presents the frequency distribution of the subjective health complaints in the sample of respondents. The most common health complaints are allergies and pain in the neck. The prevalence of reported hypertension in our sample (10%) is quite comparable with what is found in national data (WHO, 1997). Thus, over-reporting of health problems among low noise participants does not seem to be a problem.

The bivariate relationships between sensitivity to noise and the remaining variables used in the model are shown in Table 2.

The structural models were formulated using the software package AMOS 7.0. For ease of presentation, non-significant paths and paths with small standardized path estimates (<0.1) between the explanatory variables have been removed from the illustration of the model. Also, covariance paths and error estimates for the outcome variables are not shown in the displayed model. However, all estimates (of the final model) are presented in the Appendix.

Model A (Fig. 3) is a fairly simple structural equation model, describing relationships between some background variables, noise level, annoyance and subjective health complaints. The index of model fit ($\chi^2/df = 1.31$) is well within what is deemed acceptable.

In Model B (not displayed) where individual differences in the form of noise sensitivity is included, there is a reduction in model fit. Also, the original path pattern does not provide the best fit. We made alterations to the model but the index of approximate fit ($\chi^2/df = 1.93$) remained inferior to Model A ($\chi^2/df = 1.31$).

In Model C (Fig. 4) where air pollution measures and neighbourhood noise level are included, no path alterations were suggested, resulting in no adjusted model being developed. The inclusion of these contextual variables resulted in an improvement of model fit ($\chi^2/df = 1.47$) above that of Model B, but still inferior to Model A. However, also the model fit of Model C is well within what is deemed acceptable. The path estimates of Models B and C are similar to each other. Model A is distinguished from these two by significant paths between noise annoyance and high blood pressure and between noise annoyance and heart pain. Readers should note that these two path estimates are small (0.06 and 0.07).

In the final model (Fig. 4) road traffic noise exposure has no significant direct relationship with any of the subjective health problems. Annoyance from road traffic is related to tiredness, headaches and having a sore throat. There is no significant relationship between annoyance and hypertension, nor is there any between annoyance and chest pain. The model suggests a significant, but small relationship between noise annoyance and sleeping problems, nervousness, tiredness, sore throat and headaches.

Sensitivity to noise has significant and positive path coefficients (standardized) to all of the subjective health complaints. The strongest of them are with sleeping problems (0.26) and nervousness (0.20). Age is related to all of the health complaints except tiredness. The relationship of age with headaches and sore throat is negative, meaning that the older people are less likely to suffer from these illnesses. The results of the bootstrap analysis of mediation showed that there were few indirect effects (i.e. effects other than those displayed directly in the path diagram) within the model. Further, no indirect effects were found between noise level or noise annoyance and any of the other variables.

5. Discussion

5.1. The structural model

Structural equation modelling of cross-sectional data indicated that there are no direct relationships between noise exposure and any of the self-reported health indicators. The embedded model suggests that there are significant, but small relationships between increased noise annoyance on one side and the variables sleeping problems, nervousness, tiredness, sore throat and headaches. The model suggests that there are no relationships between high blood pressure or heart pain and noise annoyance/noise level. It suggests fairly strong relationships between noise sensitivity and all of the subjective health complaints. If sensitivity is removed from the model, weak but statistically significant paths become established between noise annoyance and high blood pressure and heart pain. This is in line with previous research results where univariate relationships between noise and health disappears when other explanatory variables are included (Babisch et al., 1999; Lercher, 2002).

Importantly the findings indicate a different causal direction than that suggested by previous models of the relationships between noise and health problems (Babisch et al., 2001, 2005; Bluhm et al., 2007) where increased noise levels lead to disturbances that again are associated with increased annoyance that again lead to increased risk of hypertension and myocardial infarctions. Our empirical model finds no support for a causal link between noise exposure, or even noise annoyance, and detrimental health. It does not seem that a model where noise levels and the associated increased annoyance, leads to negative health outcomes is tenable. Rather than noise sensitivity being treated as a moderating variable for annoyance, it could be that noise sensitivity is the influential variable for increased health problems. Thus, increased sensitivity to noise can be associated with subjective health complaints, and increased sensitivity also leads to increased annoyance. It is not within the scope of the current article to decide if the nature of the association between sensitivity and health complaints is causal or spurious. In the presented model the association is described as a causal path running from increased sensitivity to health problems. However, we would like to propose that a model where this association is described as spurious is more likely. Such a model would imply that a third variable, which we could coin “susceptibility”, leads to both increased noise sensitivity and increased health problems. Such an explanation might explain the fact that sensitivity in our data is correlated with all of the health variables, and not only those most likely to be associated with noise experience. The data material available does not warrant any test of these different models, but the results still suggest a different explanatory model than the previously proposed annoyance−>stress−>health model. In other words, it seems like the proposed association between noise/noise reactions and health is spurious, and not due to direct causal links.

In the proposed embedded model, there is no significant path between noise exposure and *sleep disturbances*, and only a fairly weak path (0.09) between annoyance and sleep disturbance. These results might seem surprising, but it should be noted that the question concerning sleeping problems that is included in the model is about a general and lasting condition, and not acute noise-related sleeping problems. The use of the variable “sleep disturbance” as a

negative health outcome *resulting from* annoyance also merits some further comment. It could be argued that sleeping problems are precursors to annoyance rather than resulting from annoyance. In noise–stress–health models (see e.g. Babisch, 2006) this is in fact the causal pathway that is proposed. In the survey we used, there were questions about acute negative consequences of road traffic noise, among them "problems with falling asleep" and "being woken up in the morning". These variables were tested as intermediate variables between noise level and annoyance. However, as the estimated noise–health relationships remained the same and the model became rather complex to interpret, we have not presented it in the current article.

5.2. Data quality

There are inherent limitations to cross-sectional studies such as this when explaining potentially long term effects of environmental stressors on health, e.g. that noise exposure is calculated only around the time of interviews. However, it could be argued that the noise levels assigned to each individual respondent to a certain degree functions as a proxy for long time exposure, since the mean length of residence for the respondents in this sample is 11 years (median 4 years).

The health data that we utilised in the analysis were self-reported health complaints. An argument against self-reports is that they may deviate from what is found through physiological and medical examinations. However, Okura et al. (2004) studied the relationship between self-report questionnaires and medical records, and found a high agreement (kappa 0.75) between self-reports and actual occurrences of hypertension.

In the structural model we used as dependent variables a selection of the specific health complaints reported by the participants. Previous studies looking at the same battery of complaints categorised the whole battery into four factors, and related these to noise exposure and annoyance (Nivison and Endresen, 1993). We calculated a similar factor analysis, which revealed the same factors as previously found, but using the resulting factors in the structural model lead to substantial reductions of model fit.

5.3. Test of model complexity

The test of model complexity indicated that information on the neighbourhood soundscape and air pollution were important modifiers and that they served to explain an important part of the variability that was not captured by the simplified model. Lercher's (1996) proposition about the need for embedded models as crucial for studying non-auditory health effects thus receives some support. An overly simple model, where the importance of individual noise sensitivity was not taken into account, displayed a seemingly good fit to data. However, sensitivity has proven to be a crucial variable for explaining annoyance, both in the current study and in previous research. Thus this result should rather function as a warning against paying too much attention to measures of model fit when assessing overly simple structural models. Results from studies of health effects of noise exposure lacking questions on noise sensitivity (see e.g. de Kluizenaar et al., 2007) will thus have to be interpreted with this in mind.

The other major theoretical proposition made by Lercher, about the need for following a transactional approach with the noise–health relationship being treated as a continuous loop of appraisals, coping efforts and reappraisals, is not tested in the current analysis. This is a promising avenue for increasing the understanding of the noise–health relationship. Future research should therefore include information on individual's differential coping styles, as studied by van Kamp (1990). In the current study we only had data on whether people shut their windows due to road traffic noise. We do not know if such behaviour is a measure of presence of coping skills, or a measure of prioritizing between different environmental nuisances (noise vs. poor indoor air quality). Such issues would have to be resolved if information on coping style is to be of any use.

6. Conclusion

An embedded model of the noise–health relationship found no significant effect of either road traffic noise or noise annoyance on reported hypertension or heart problems. There were weak effects on other self-reported health problems (tiredness, headaches, sore throat). The correlations between noise sensitivity and health problems were generally far stronger than between annoyance and health problems, indicating a different causal direction than previously proposed by researchers. This underlines the necessity of including noise sensitivity as a crucial variable in research on noise–health relationships. The benefit of including contextual variables in a model of noise–health relationships is supported by the model.

Cross-sectional studies such as this have some inherent limitations when it comes to studying effects of long time environmental influences. Therefore, most researchers studying these relationships end up by recommending more large-scale longitudinal studies. Since our research indicates that noise sensitivity has an important role, we will add to this recommendation that future studies should also include information on noise sensitivity. It is highly likely that the role noise sensitivity plays as a general indicator of individual vulnerability in both short-term experience and through residential preferences could be of importance in explaining the noise–health relationship in longitudinal studies.

Appendix A. Estimated relationships (covariances) with standard errors, critical ratios (Estimate/Standard Error) and significance levels between all variables in the structural equation model (Model C)

		Estimate	Standard error	Critical ratio	*p*
Age above 70	<–> Gender	0.017	0.004	4.142	***
Noise exposure	<–> Neighbourhood noise difference	−32.484	1.351	−24.044	***
Noise exposure	<–> Air pollution, NO2	41.363	2.104	19.662	***
Neighbourhood noise difference	<–> Air pollution, NO2	−5.872	1.756	−3.344	***
Age above 70	<–> Education level	−0.503	0.032	−15.692	***
Gender	<–> Education level	−0.094	0.043	−2.199	.028
Sore throat	<–> Tiredness	0.132	0.013	10.460	***
Sore throat	<–> Nervousness	0.074	0.010	7.108	***
Tiredness	<–> Nervousness	0.139	0.011	12.899	***
Sore throat	<–> Headaches	0.105	0.012	8.799	***
Tiredness	<–> Headaches	0.146	0.012	11.963	***
Nervousness	<–> Headaches	0.082	0.010	8.206	***
Headaches	<–> Problems falling asleep	0.069	0.010	6.829	***
Sore throat	<–> Problems falling asleep	0.053	0.010	5.084	***
Tiredness	<–> Problems falling asleep	0.103	0.011	9.634	***
Problems falling asleep	<–> High blood pressure	0.043	0.007	6.048	***
Nervousness	<–> Problems falling asleep	0.148	0.009	15.817	***
Nervousness	<–> High blood pressure	0.030	0.007	4.370	***
High blood pressure	<–> Pain in heart	0.057	0.006	9.617	***
Nervousness	<–> Pain in heart	0.091	0.008	12.027	***
Problems falling asleep	<–> Pain in heart	0.074	0.008	9.724	***
Tiredness	<–> Pain in heart	0.093	0.009	10.479	***
Headaches	<–> Pain in heart	0.085	0.009	9.966	***
Sore throat	<–> Pain in heart	0.050	0.009	5.878	***
Tiredness	<–> High blood pressure	0.030	0.008	3.750	***
Headaches	<–> High blood pressure	0.028	0.008	3.639	***

*** $p<0.0001$.

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