Science of the Total Environment 408 (2010) 4935–4942

Contents lists available at ScienceDirect

Science of the Total Environment

journal homepage: www.elsevier.com/locate/scitotenv

# Noise, sleep and poor health: Modeling the relationship between road traffic noise and cardiovascular problems

Aslak Fyhri [a,*,1], Gunn Marit Aasvang [b]

[a] Institute of Transport Economics, Norway
[b] The Norwegian Institute of Public Health, Norway

ARTICLE INFO

*Article history:*
Received 14 January 2010
Received in revised form 21 June 2010
Accepted 24 June 2010
Available online 12 August 2010

*Keywords:*
Hypertension
Noise annoyance
Sleep disturbance
Subjective health complaints
Noise sensitivity

ABSTRACT

Several adverse effects have been associated with exposure to traffic noise. Studies supporting a noise–stress–health model have suggested links between noise level and increased noradrenalin concentrations in urine, hypertension and myocardial infarction. Among the more commonly documented effects, sleep disturbances have been regarded as being the most serious. Both noise annoyance and sleep disturbance have been proposed as important mediators of the impact of noise on health. The present paper investigates the relationships among long-term noise exposure, annoyance, sleeping problems and subjective health complaints by the use of a structural equation model. Further, it aims at giving insight into how noise sensitivity is related to sleep disturbances from road traffic noise. Finally, it examines whether any effect of noise exposure or response to noise can be detected on prevalence of cardiovascular problems, when information on sleep disturbances is included in a model. Data from a questionnaire survey conducted among a population sample in Oslo (N = 2786) are combined with nighttime noise levels calculated from outside each respondents dwelling, at the bedroom façade. The results of the analysis showed significant relationships between noise annoyance at night and sleeping problems. The model also showed strong links among pseudoneurological complaints, annoyance and sleeping problems, thus pointing to the importance of including information on psychosomatic disorders and mild psychological problems in future studies looking at potential health effects of noise. The analysis showed no relationship between neither noise exposure nor response to noise and cardiovascular problems.



## 1. Introduction

### 1.1. Noise and health

Environmental noise may lead to adverse health effects through a complex web of pathways where both situational and personal factors act as mediators and moderators. It is well documented that environmental noise affects health and well-being by disrupting basic activities such as sleep, rest, communication, concentration and cognition; and it may also lead to a general feeling of annoyance (Berglund et al., 1999; Muzet, 2007; Stansfeld et al., 2005). 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 Organization (WHO), and that about 10% report severe sleep disturbance because of transportation noise at night (Eea, 2003). In Norway, it is estimated that about 1.5 million people (1/3 of the total population) are exposed to noise from transport that exceeds the recommended values (Engelien et al., 2004). Road traffic noise is the main contributor to environmental noise.

Adverse effects of noise on human health are related to noise as an environmental stressor influencing behavioral, psychological and physiological processes (Babisch, 2008; Babisch et al., 2001; Babisch et al., 2002; Berglund et al., 1999). Auditory information is continuously processed by the central nervous system (CNS) acting as a constant guardian to signal danger or significant sounds (Velluti, 1997). Acute noise exposure has been shown to induce physiological responses such as increased blood pressure and heart rate (Carter et al., 2002; Haralabidis et al., 2008) as well as endocrine changes, including the levels of catecholamines and glucocorticoids (Ising et al., 1980; Miki et al., 1998). Since acute exposure to noise has been linked to transient increases in blood pressure and levels of stress hormones in experimental settings, it is hypothesized that long-term exposure to noise may have adverse effects on health (Babisch, 2000). There is some support in the literature for the notion that prolonged exposure to noise from aircraft and road traffic can increase the risk of hypertension and myocardial infarction (Babisch et al., 2005; Bluhm et al., 2007; Jarup et al., 2008; Rosenlund et al., 2006). However, the evidence for a causal relation between noise and cardiovascular problems is still inconclusive due to methodological limitations in

* Corresponding author. Tel.: +47 22573822.
*E-mail address:* af@toi.no (A. Fyhri).
[1] Postal address: Gaustadalléen 21, 0349 Oslo, Norway.

0048-9697/$ – see front matter © 2010 Elsevier B.V. All rights reserved.
doi:10.1016/j.scitotenv.2010.06.057

exposure characterization, cross-sectional design and lack of adjustment for potential confounders.

### *1.2. Sleep as a factor in the noise health relationship*

In western countries, sleep problems are increasing owing to lifestyle and environmental factors. Sleep disturbances are regarded as being among the most serious effects of environmental noise and one of the most common reasons for noise complaints (Guski, 1977). Experimental sleep research has revealed that partial sleep deprivation has negative effects on sleepiness, performance, and mood (Dinges et al., 1997), as well as on some metabolic, hormonal, and immunological variables (Irwin et al., 1996; Spiegel et al., 1999); thus, it may have serious long-term health effects (Ferrara and De Gennaro, 2001). Some of these effects are also observed in subjects after exposure to high levels of nighttime traffic noise, i.e. reduced subjective sleep quality, changed cortisol levels and reduced performance on reaction time tests (Griefahn et al., 2006; Maschke et al., 2002; Öhrström and Rylander, 1982).

One hypothesis is that noise exposure during the night leads to both sleep disturbances and activation of the sympathetic nervous system, thus increasing the risk of cardiovascular disease in the long run. Laboratory studies have documented immediate effects of traffic noise, such as EEG (Electroencephalographic) arousals, awakenings and sleep stage shifts (Basner et al., 2008) and consequently on overall sleep architecture (Basner and Samel, 2005; Griefahn et al., 2006). Furthermore, cardiovascular responses to noise during sleep are observed with little or no evidence of habituation (Carter et al., 2002; Griefahn et al., 2008). Thus, after years of exposure these changes may lead to dysregulation and permanent physiological changes that increase the risk of cardiovascular disease. However, only a limited number of epidemiological studies provide information on nighttime noise exposure. Thus, the potential impact of long-term exposure to nighttime noise on cardiovascular endpoints has hardly been studied. Results from a study by Jarup et al. (2008) showed an increased association between aircraft noise and hypertension for those exposed to nighttime noise. A second Swedish study found an elevated association between road traffic noise and myocardial infarction among those annoyed by noise in the bedroom, although this was not found among participants reporting sleep disturbances due to noise (Selander et al., 2009). Other studies have reported a stronger relationship between cardiovascular outcomes and noise induced sleep disturbances than with other subjective responses to noise, such as general annoyance (Babisch et al., 2005; Babisch et al., 2003). Thus, the results reported are few and inconsistent.

Moreover, inaccuracy in the assessment of nighttime noise further hampers the epidemiological knowledge on the noise–sleep–health relationship. Large-scale field studies have tended to employ noise levels assessed at the most exposed façade of the house when determining individual noise exposure. However, not all people have their bedroom facing that side of the house exposed to the most noise. It would be more appropriate to assess noise levels at the bedroom façade or inside the bedroom when analyzing the negative effect of noise-induced sleep disturbance on cardiovascular health.

In an attempt at empirically modeling the relationship between noise and various negative health outcomes (Fyhri and Klaeboe, 2009) the analysis revealed that there was no significant relationship between noise exposure and self-reported *sleep disturbance*, and only a fairly weak relationship between annoyance and sleep disturbance. However, the measure of sleeping problems used was a general one i.e. all types of sleeping problems were encaptured, rather than those due solely to traffic noise. A further analysis in which the specific relationship between noise annoyance and sleeping difficulty is examined more closely by differentiating between causes of sleeping problems is therefore required.

The proposed causal pathway in the noise–stress–health model (Babisch, 2006) goes from sleeping problems to noise annoyance. Such a model is based on the assumption that annoyance is a generalized assessment of a range of more specific disturbances due to noise exposure, and that interference with sleep is one such specific disturbance. As an empirical model this causal chain needs some refinement. Sleep is generally affected by factors such as age, gender and various physical and psychological health conditions (Bixler et al., 2009; Redeker et al., 2004; Reyner et al., 1995). Hence, the amount and quality of sleep varies considerably among both individuals and age groups (Siegel, 2003). The noise annoyance reaction is hypothetically only related to one specific type of sleeping disturbance, namely that due to noise. Thus it can be proposed that annoyance is a mediating variable between the specific sleep disturbance from noise and a general assessment of sleep quality.

Insomnia is common in individuals with depression and anxiety disorders (Sandor and Shapiro, 1994). Neuroticism is found to be associated with higher sensitivity to noise (Stansfeld, 1992b; Öhrström et al., 1988). Self-reported noise sensitivity has been found to be highly influential on annoyance due to noise (Fields, 1993). Field and laboratory studies indicate that noise sensitivity also influence subjectively reported sleep disturbances due to noise (Aasvang et al., 2008; Belojevic et al., 1997; Marks and Griefahn, 2007; Öhrström et al., 1990). Belojevic et al. (1997) found that several aspects of sleep disturbances, e.g. difficulty falling asleep and tiredness in the morning, were correlated with both noise sensitivity and neuroticism. Hence, in addition to sleep quality it seems important to take both noise sensitivity and psychological distress into account when studying the effect of noise on health.

### *1.3. Objective of study*

The main aim of the present paper was to investigate the relationship among variables measuring noise, sleeping problems and health problems. More specifically we wanted to examine any links between noise exposure or annoyance due to noise and prevalence of cardiovascular disease, when sleep disturbances are accounted for. In order to achieve this, a model of the relationship between noise and sleeping problems was developed. Our additional aim is to provide insight into how noise sensitivity is related to sleep disturbances from road traffic noise.

## **2. Method**

### *2.1. Sample and survey*

The study included inhabitants living in Oslo. Data on residential addresses were obtained from the Norwegian Public Roads Administration in connection with their ongoing work on noise mapping. Selection of the study areas was based on traffic density maps and crude data on noise levels recorded at the most exposed façade of the residential buildings. From the home addresses, an age- and gender-stratified sample of 5390 persons in Oslo, the largest city of Norway was selected using the central Norwegian person registry (49% female and 51% male >18 years). A self-administered questionnaire together with an introductory letter was mailed to the population sample in October 2000. To avoid possible bias in responses to the noise questions, we presented the study as a general investigation into health and quality of life, with no specific focus on noise. A total of 3262 respondents (60.5 %) answered and returned the questionnaire. 145 respondents who had either moved or whose home address was not verifiable were excluded from the analyses. A total of 3117 respondents (51.1% female and 48.9% male) were used for the further analyses. The study was approved by The Regional Committee for Medical Research Ethics in Norway.

The study environment consisted of randomly located dwellings in all parts of the city of Oslo (587 000 inhabitants). The final sample consisted of 36% single family houses, 39% semi-detached/row houses and 25% apartment blocks. 35% of the dwellings had nighttime equivalent noise levels ($L_{p,\mathrm{A,eq,night}}$) below 40 dB, and 10 % had levels above 55 dB. None of the addresses in the sample were included in the railway noise mapping program of the Norwegian National Rail Administration, and thus, not exposed to high levels of railway noise. Oslo airport Gardermoen is located 47 km north of the city. Nighttime aircraft noise exposure was thus considered negligible. Most of the respondents (69%) were long-term residents (>5 years).

### 2.2. Questionnaire items

The questionnaire consisted of items concerning perceived sleep quality and sleep disturbances, sleep problems due to external noise, orientation of the bedroom (towards noise source or garden/ backyard), and personal characteristics such as year of birth, gender, total income, education, duration of residence and noise sensitivity. The general sleep questions were adopted from the Basic Nordic Sleep Questionnaire (Partinen and Gislason, 1995). Additional details regarding the questionnaire items can be found in Aasvang et al. (Aasvang et al., 2008).

The annoyance questions complied with Fields et al. (2001) and the recommendations of ISO (Iso/Tc43/Wg, 2003). A similar wording was used to assess nighttime annoyance. However, our questions referred to the last three months rather than the (more usual) last twelve months. This time frame was chosen to comply with that used for the sleep quality and sleep disturbance questions. The last question of Weinstein's noise sensitivity scale (Weinstein, 1978) was used to assess noise sensitivity: “I am sensitive to noise”. This statement was presented with a six-point response scale, ranging from “disagree strongly” to “agree strongly”.

Subjective health complaints were measured using the SHC inventory (Eriksen et al., 1999). The original version of SHC consists of 29 questions concerning severity and duration of subjective somatic and psychological complaints. 27 of these items were used (two questions concerning sleeping problems were omitted, as more specific questions on sleeping problems were asked elsewhere in the questionnaire). Severity was scored on a four-point scale, from 0 — no complaints to 3 — severe complaints. Information on duration was also collected, but this was not used in the current analysis, as tests indicated that it did not provide any information above that of severity. The SHC was categorised into five sub-scales, and a test of reliability of these factors (Chronbach's alpha) revealed scores exceeding those obtained previously: *musculoskeletal pain* ($\alpha = 0.87$), *pseudoneurological complaints* ($\alpha = 0.89$), *gastrointestinal problems* ($\alpha = 0.90$), *allergy* ($\alpha = 0.85$) and *flu* ($\alpha = 0.80$). Using all the five factors in SEM model along with all the noise and sleep variables would have led to a rather complex model. Thus it was decided that only one SHC factor was to be included in the analysis. As the *pseudoneurological complaints* factor has previously been shown to have the highest correlation with sleeping problems (Pallesen et al., 2005), it was selected for the final SEM model. The factor *pseudoneurological complaints* consists of the following items: *palpitation, heat flushes, dizziness, anxiety* and *depression*.

The 25 item Hopkins Symptom Checklist (HSCL) was used to measure psychological distress (Derogatis, 1983). In the present survey we used the Norwegian version of this well-established measuring instrument aimed at tapping anxiety and depression. The SCL-25 has proved to have satisfactory validity and reliability as a measure of psychological distress (Derogatis et al., 1974; Glass et al., 1978). Four alternative responses are given to each question, ranging from *not at all* to *extremely*.

Prevalence of cardiovascular problems was measured by three items: previous or existing diagnosis of myocardial infarction, previous or existing diagnosis of angina pectoris and previous or existing diagnosis of hypertension. These three variables are summarized as one dichotomous variable, *cardiovascular problems*.

### 2.3. Noise exposure assessment

The *nighttime equivalent noise levels* ($L_{p,\mathrm{A,eq,night}}$) were calculated according to The Nordic Prediction Method for road traffic noise (Norden, 1996). Assessment of individual noise exposure was conducted by using digital maps and geographical coordinates of the address of each respondent in the survey. The software program CadnaA (DataKustik, 2004) was employed for the noise exposure calculations. CadnaA applies digitalised terrain data, buildings and noise screens in three dimensions. Quantitative data for road traffic in the study areas (traffic accounts, % heavy vehicles, speed, diurnal distribution), representative for the survey period, were obtained from the Norwegian Public Roads Administration and the City Council of Oslo. Additional on-site traffic counts were conducted on some low trafficked roads for which no data on traffic volume were available. The effects of distance from receiver to the noise source, air absorption, ground properties, topography, and screens were included as major sound propagation parameters.

The nighttime equivalent noise levels were calculated by integrating the sound energy from all noise events over an 8-hour nighttime period, from 23.00 to 07.00. $L_{p,\mathrm{A,eq,night}}$ was calculated outside each respondents dwelling, at the bedroom façade. For each address, we used information provided on the questionnaire about which side of the dwelling the bedroom faced and the elevation (floor) of the bedroom. When the bedroom was reported as facing a major road, the calculation of the noise level representing the most exposed façade was used. In those cases in which the bedroom was reported as facing a garden or backyard, the calculation points represent the façade with the lowest noise exposure. The outdoor noise levels are given as free-field values.

### 2.4. Structural Equation Model (SEM)

Regression models are often used for studying the relationship 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 both indirect and direct effects to be estimated. In our case, this makes it possible to investigate to what degree noise annoyance and self-reported sleep disturbances are mediating factors in the noise–health relationship. Further, SEM analysis allows the researcher to evaluate the fit between data and theory. This cannot be done in traditional multiple regression analyses. The goodness-of-fit index (GFI), the adjusted root mean square error of approximation (RMSEA) and the Chi Square ($\chi^2$) were used to assess the fit of the model to the observed data.

Due to missing values on questionnaire items, the sample size for the SEM analysis ($N = 2786$) is lower than the total sample size of traffic noise exposed respondents ($N = 3117$).

The structural model was analyzed using the software package AMOS 16.0. For ease of presentation, non-significant paths and paths with small standardized path estimates (<0.09) 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 Appendix A.

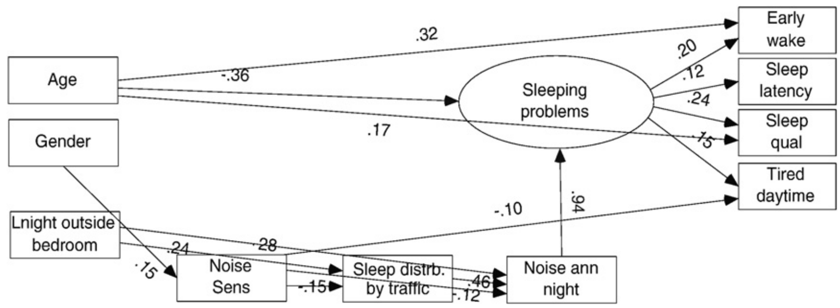


**Fig. 1.** Structural model relationship between noise and sleeping problems. Values on paths are standardized regression weights. All displayed paths are significant ($p<0.05$). Non-significant paths and paths with values below 0.09 are removed from the displayed figure. $N=2786$.

The model was developed in two stages. First a model was formulated analyzing the relationships among individual background variables, noise related variables and sleep variables (Fig. 1). Secondly, information on self-reported health problems was included in the model (Fig. 2). Education was also included in the model at this stage, as a measure of socioeconomic status. A pilot test revealed that the omitted SHC factors provided virtually no explanation of sleep disturbances, cardiovascular problems or noise variables when controlling for pseudoneurological complaints and HSCL.

## 3. Results

Table 1 presents the distribution and mean scores of the study variables according to self-reported prevalence of cardiovascular problems (hypertension, myocardial infarction or angina pectoris).

### 3.1. SEM model for road traffic noise

In the noise-sleep model (Fig. 1) one of the indices of model fit (AGFI = 0,912) was within normally accepted limits. The RMSEA was somewhat higher than normally accepted limits (RMSEA = 0.084). The chi square/degree of freedom ratio test also failed ($\chi^2$/DF = 20.68).

The model indicated a significant relationship between nighttime noise exposure and nighttime noise annoyance (path estimate 0.24), and a significant relationship between nighttime noise annoyance and reporting transportation noise as a cause for sleeping disturbances (path estimate 0.24). Those respondents reporting sleep disturbances from transportation noise were far more likely to report being annoyed (standardized path estimate 0.46) by noise during night. In this version of the model, being annoyed by traffic noise at night was a very strong and significant predictor of the sleep problems (standardized path estimate 0.94). The latent variable sleeping problems had rather low scores on the path estimates to all its measured variables.

There was a significant but rather weak relationship (standardized path estimate 0.15) between noise sensitivity and noise annoyance during night, and a separate path from sensitivity to feeling tired (standardized path estimate 0.10). Age had a strong negative relation to the latent variable sleeping problems (standardized path estimate −0.36). Age also had a significant path estimate directly to the item early awakenings (standardized path estimate 0.32) and to subjective assessment of sleep quality (standardized path estimate 0.17).

Fig. 2 shows the structural equation model for road traffic noise, sleep disturbances and subjective health complaints. Education is also included in the model as a measure of socioeconomic status. Two of the indices of model fit (AGFI = 0,986 and RMSEA = 0.024) were well within acceptable limits. The chi square/degree of freedom ratio test indicated that the model fit was somewhat lower than it should be ($\chi^2$/DF = 2.66).

In both models modification indices suggested that allowing for all error terms for the latent variable *sleep* to covariate would bring this indicator well below the suggested level of 2. This alteration was tested, leading to some minor changes to parameter estimates elsewhere in the model. However, as all error terms in a latent variable are not supposed to covary, the suggested model was rejected.

The second model indicated rather similar relationships among the noise and sleep variables as the first model, with a few notable exceptions: being annoyed by traffic noise at night was still a

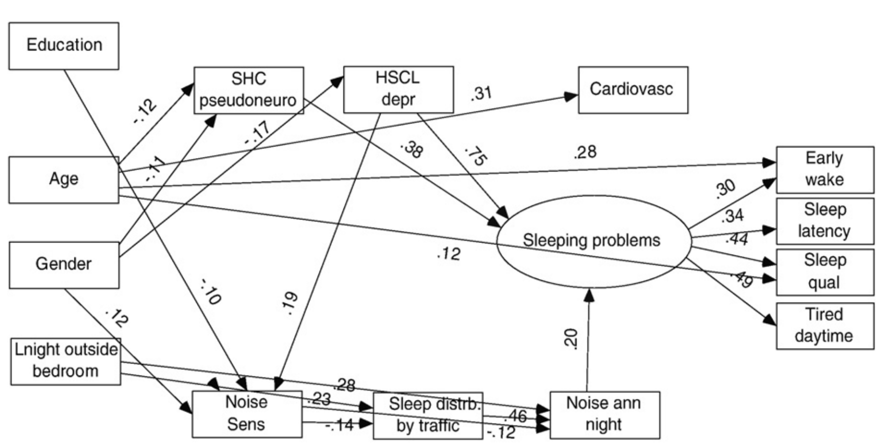


**Fig. 2.** Structural model relationship between noise, sleeping problems and health complaints. Values on paths are standardized regression weights. All displayed paths are significant ($p<0.05$). Non-significant paths and paths with values below 0.09 are removed from the displayed figure. $N=2786$.

**Table 1**
Description of study variables. Distributions and mean scores according to prevalence of cardiovascular problems.

| | Cardiovascular problems | |
|---|---|---|
| Distributions | No (2294) | Yes (492) |
| % male*** | 47.3 | 57.7 |
| % rather/very noise sensitive | 32.1 | 32.7 |
| % rather/very annoyed by traffic noise at night | 10.8 | 11.8 |
| % sleep disturbed by nocturnal traffic noise | 8.7 | 9.3 |
| % rather/very poor sleep quality*** | 8.9 | 14.2 |
| % early woken almost every morning*** | 2.9 | 6.1 |
| % tired daytime more than once/week* | 45.2 | 51.4 |
| % with $L_{p,A,eq,night}$ > 55 dB, outside bedroom | 10.8 | 8.5 |
| Mean scores | | |
| Age*** | 46 | 59 |
| Education*** | 4.1 | 3.7 |
| $L_{p,A,eq,night}$, outside bedroom | 43.3 | 43.7 |
| SHC, pseudoneurological complaints [a] | 1.2 | 1.2 |
| Anxiety/depression, HSCL** [a] | 1.38 | 1.43 |
| Sleep latency, minutes* | 23.9 | 27.2 |

[a] Scores from 1 to 4.
* $p<.05$.
** $p<.01$.
*** $p<.001$.

significant predictor of the sleep problems, but this relationship was weaker than in the first version of the model (standardized path estimate 0.20). There was no longer any significant path between sensitivity and feeling tired. The latent variable sleeping problems was now mostly influenced by feeling tired (standardized path estimate 0.49) and least influenced by early awakenings (standardized path estimate 0.30). The size of the path estimates between age and sleeping problems was also less.

Having a high score on the SHC pseudoneurology scale was a strong predictor (standardized path estimate 0.75) of sleep problems, as was having a high score on the HSCL anxiety /depression scale (standardized path estimate 0.38).

High scores on the HSCL anxiety/depression scale was a significant predictor of noise sensitivity (standardized path estimate 0.19). Cardiovascular problems were predicted by age (standardized path estimate 0.31), gender (standardized path estimate 0.08), pseudoneurological complaints (standardized path estimate 0.07) and education (standardized path estimate −0.07).

### 3.2. Logistic regression on cardiovascular problems

In the model the independent variable cardiovascular problems is a dichotomous summary variable reflecting three measured dichotomous variables (measuring whether or not respondent had been diagnosed with the angina pectoris, hypertension or myocardial infarction). Initially, SEM assumes that the data are continuous or interval in nature and that they are normally distributed. The model violates this assumption, especially with the inclusion of the cardiovascular problems variable. However several procedures and estimation techniques are available that allow for the use of ordinal level data in SEM, the most common being to test the robustness of the estimations using bootstrapping (Arbuckle, 2008). This procedure was tested on the current SEM model, but it revealed no significant differences to the parameter estimates presented. As the presented model still violated the assumptions of SEM, a logistic regression analysis was performed on the same variables included in the model and with cardiovascular problems as independent variable. The model fit was moderate (Nagelkerke $R^2=0.13$). The results of the regression analysis confirmed that age was the most important predictor of cardiovascular problems (odds ratio = 1.07 per increasing year). However, it differed somewhat from the SEM analysis in that the variable pseudoneurological problems (odds ratio = 2.22) was a far better predictor of cardiovascular problems than gender (odds ratio = 1.74). Neither noise exposure nor sensitivity to noise was a significant predictor of cardiovascular problems. The relationship between noise annoyance during night time and cardiovascular problems was close to significance ($p=0.065$). Intriguingly, this relationship was negative (odds ratio = 0.76).

## 4. Discussion

The main aim of this study was to investigate whether sleep disturbances can help explain the relationship between noise and health problems. In order to achieve this main goal a secondary goal was to establish a model of the relationship between noise and sleep disturbance. This model was formulated with Structural Equation Modeling (SEM) software. The SEM analysis shows that even if sleep interruption due to transportation noise was a strong predictor of being annoyed by road traffic noise at night, quite a number of respondents were annoyed without being interrupted in their sleep. Further, those that were annoyed by noise scored higher on the sleeping problems variable. The inclusion of information on subjective health problems gives a significant contribution to the model. Without these variables in the model, noise annoyance seemed to be a very strong predictor of sleeping problems. However, when information on pseudoneurological complaints was included the *health problems* factor became the strongest predictor of sleeping problems, on behalf of noise annoyance. In the original version of the questionnaire (Ihlebaek et al., 2002) this factor also consisted of the items *sleep problems* and *tiredness.* In order to avoid circularity, these two items were left out of the current questionnaire. The suggestion is that respondents with psychological or psychosomatic problems are more prone to being disturbed in their sleep from road traffic noise than others. However, for these respondents the health problems themselves are far more important causal factors for sleeping problems than road traffic noise is.

In order to answer the main research question of the study, information on cardiovascular problems were included in a complete SEM model with noise, sleep and health variables. The results of this analysis show that when all variables are considered together, age, gender and pseudoneurological problems are the most important predictors of cardiovascular problems. None of the noise-related variables predicted cardiovascular problems. Thus, our data does not support the hypothesis that noise-related sleeping problems or annoyance is the "missing link" in the proposed noise health relationship. These results are in contrast to some previous studies that have linked annoyance with cardiovascular problems (Aydin and Kaltenbach, 2007; Babisch, 2006; Babisch et al., 2001; Bluhm et al., 2007; de Kluizenaar et al., 2007; Jarup et al., 2008). Other studies (Fyhri and Klaeboe, 2009; Stansfeld et al., 2005) and systematic reviews (van Kempen et al., 2002) support the present findings.

It should be noted that there are no bivariate correlations between either noise or response to noise and cardiovascular problems in the current data material. Thus, it is not surprising that the multivariate relationships do not exist. However, it could be that a further inspection of the web of causal pathways in this data material would provide insights into why some studies suggest such relationships. In the SEM analysis pseudoneurological complaints are related to reacting negatively to road traffic noise at nighttime. Pseudoneurological complaints are also related to noise sensitivity via their relationship with psychological distress (anxiety/depression). There is also a significant, albeit weak, relationship between pseudoneurological complaints and cardiovascular problems. Thus, it can be speculated that any increased risk of cardiovascular problems among respondents annoyed by noise is due to a higher occurrence of psychosomatic disorders (pseudoneurological complaints) among these people).

The health data that we utilized in the analysis were subjectively reported health complaints. It could be argued that self-reports may deviate from what is found through physiological and medical examination. For example, some studies have found that self-reported hypertension and coronary heart disease tend to be underestimated (Bowlin et al., 1993; Heliovaara et al., 1993; Molenaar et al., 2007). Others have found quite high agreement between self-reported disease and medical records of, for instance, hypertension (Okura et al., 2004). Although biometrical data on diseases are more reliable than self-reported data, self-reporting methods are less expensive than those used to gather objective data. In a cardiovascular study from Norway, questionnaire information on myocardial infarction showed a high degree of correlation with corresponding data from medical records, but the same study found that participants under-reported strokes (Tretli et al., 1982). Self-reported myocardial infarction is probably less influenced by reporting bias than angina pectoris. Both over- / under-reporting and sampling procedures may potentially lead to deviances of reported health problems in the sample compared with the prevalence in the population. In Norway there are no national registry data of prevalence of cardiovascular problems. In order to compare with the general population we therefore used data from CONOR. CONOR is a collection of health data and blood samples from several Norwegian health surveys, and includes three different measures of hypertension: *hypertension* according to the WHO criteria (systolic blood pressure $\geq$170 and /or diastolic blood pressure $\geq$100 mmHg) *moderate hypertension* according to the WHO criteria (systolic blood pressure between 140 and 170 and/or diastolic blood pressure between 90 and 100 mmHg) and use of antihypertensives. Reported hypertension in the current sample is somewhere between prevalence of hypertension and moderate hypertension in CONOR, for all age groups (Graff-Iversen et al., 2007). Reported hypertension is quite comparable with frequencies of use of antihypertensives, except for the oldest age group, who report *less hypertension* than the comparable number of users of antihypertensives in CONOR. The prevalence of myocardial infarction is fairly comparable for all age groups, although there is a slight tendency for a somewhat higher prevalence than reported in the national data for older males: 21% of the males in the age group 72–78 reported to have suffered MI in the current study versus 15% (males, 75 years of age) in CONOR (Graff-Iversen et al., 2007). Self reported occurrence of angina pectoris is quite comparable with self-report measures from CONOR, although there is a small tendency for a *lower* than normal prevalence among the eldest in the sample: 5% among females in the age group 72–78 in the current study versus 12% among 75 year old females CONOR (Graff-Iversen et al., 2007). In sum, the occurrence of cardiovascular problems in the current sample seems to be quite comparable to that of the general population. Still, misclassification of true prevalence of disease might have occurred. However, any potential misclassification is most probably not related to the noise exposure and would thus dilute any association in our study.

Socioeconomic status (SES) is operationalised as education in the current analysis. The respondents were also asked other questions related to SES, such as income or employment status. It could be argued that including these variables in the model, for instance as a latent SES-variable, might have improved the analysis, since such variables are known to co-vary with both somatic and psychosomatic health. Such a model was tested and it did reveal a slightly stronger relationship between SES and the health variables. However, the main aim of the study was to investigate noise–sleep–health relationships. As the path estimates among these variables remained the same, and the model became rather complex to interpret, we do not include it in the current article. It should also be noted that a test of an intermediary model without education revealed that none of the changes in path estimates between model 1 and model 2 could be attributed to the inclusion of this variable.

In most studies where noise sensitivity is included it comes out as a strong predictor of noise annoyance (Miedema and Vos, 2003; Stansfeld, 1992a) . In the current study sensitivity was a significant predictor, but the relationship was not very strong. One explanation for our finding is that the inclusion of variables such as sleeping problems and health complaints in the model cancels out the relationship between sensitivity and annoyance. However, a recent study using SEM analysis (Fyhri and Klaeboe, 2009), and also including variables on sleep and subjective health complaints, found a relationship between noise sensitivity and annoyance that was more in line with previous research. The major difference between these two SEM analyses is that in the current analysis annoyance is measured as annoyance from road traffic noise *during night time*, whereas in the previous study it was measured as annoyance at any time of day. If the night time annoyance question is replaced by a more general road traffic annoyance question, the relationship with noise sensitivity increases to 0.16, which is more in line with previous results. In other words, as road traffic noise at nighttime is generally seen as more annoying than at day time, the “distinguishing power” of sensitivity is reduced in the nighttime situation.

The strength of this study is that it utilizes information from the questionnaires about bedroom orientation to assess nighttime noise levels at the bedroom façade, instead of noise level outside of the most exposed façade. Such an approach is highly feasible since the majority of the study population (66%) reported to have their bedroom oriented towards a garden or a backyard, and the average difference in the A-weighted noise level between the most exposed and the bedroom façade, when the bedroom was oriented towards a garden/backyard, was 8 dB in case of road traffic noise exposure.

A further strength of this study is that it combines the data on individual noise exposure with standardized questionnaire items on a range of individual factors, in particular sleep quality, annoyance due to noise and health outcomes. All selected participants are included, rather than, for example, excluding participants with hearing loss. Even though the sample size of the current study is lower than that found in some epidemiologic studies, it is far larger than that normally found in similar studies of detrimental noise effects.

## 5. Conclusion

A structural equation analysis including information on sleeping problems and subjective health indicators found significant relationships between noise annoyance at night and sleeping problems for a community sample. The results of the analysis confirmed that sleep is an important negative effect of road traffic noise, even if other factors contribute more to sleeping problems. We found no direct or indirect relationships among any of the noise variables and cardiovascular problems. As pseudoneurological complaints are related to both annoyance and sleeping problems it is important to account for this factor when investigating noise–sleep–health relationships. Also, when looking at the potential ill health effects of noise in the future, it will be important to include information on psychosomatic disorders and mild psychological problems, as these are found to have an influence on somatic disorders.

## Appendix A

**Appendix A1**
Estimated relationships ("⟵" = unstandardized regression weights; "⟷" = covariances) with standard errors, critical ratios (Estimate/Standard Error) and significance levels between all variables in the structural equation mode.

| | | | Estimate | S.E. | C.R. | P |
|---|---|---|---|---|---|---|
| Anxiety/ depression | ⟵ | Gender | −.136 | .015 | −8.994 | *** |
| Anxiety/ depression | ⟵ | Age | −.001 | .000 | −2.414 | .016 |
| Anxiety/ depression | ⟵ | $L_{night}$ outside bedroom | .004 | .001 | 4.568 | *** |
| Anxiety/ depression | ⟵ | Education | −.020 | .004 | −4.364 | *** |
| Noise sensitivity | ⟵ | Anxiety/ depression | −.792 | .076 | −10.418 | *** |
| Noise sensitivity | ⟵ | Gender | .413 | .062 | 6.660 | *** |
| Noise sensitivity | ⟵ | Age | −.008 | .002 | −4.142 | *** |
| Noise sensitivity | ⟵ | Education | −.102 | .019 | −5.239 | *** |
| Pseudoneurology | ⟵ | Age | −.004 | .001 | −6.244 | *** |
| Pseudoneurology | ⟵ | Gender | −.115 | .019 | −5.991 | *** |
| Sleep disturbance by traffic | ⟵ | $L_{night}$ outside bedroom | .007 | .001 | 12.844 | *** |
| Sleep disturbance by traffic | ⟵ | Noise sensitivity | −.024 | .003 | −7.721 | *** |
| Sleep disturbance by traffic | ⟵ | Pseudoneurology | .042 | .010 | 4.159 | *** |
| Noise annoyance night | ⟵ | Pseudoneurology | .117 | .025 | 4.735 | *** |
| Noise annoyance night | ⟵ | Sleep disturbance by traffic | 1.379 | .046 | 29.825 | *** |
| Noise annoyance night | ⟵ | $L_{night}$ outside bedroom | .026 | .001 | 18.587 | *** |
| Noise annoyance night | ⟵ | Noise sensitivity | −.061 | .008 | −7.904 | *** |
| Sleeping problems | ⟵ | Pseudoneurology | .235 | .022 | 10.813 | *** |
| Sleeping problems | ⟵ | Anxiety/ depression | .589 | .039 | 15.054 | *** |
| Sleeping problems | ⟵ | Noise annoyance night | .076 | .011 | 6.990 | *** |
| Cardiovascular disease | ⟵ | Pseudoneurology | .050 | .014 | 3.669 | *** |
| Cardiovascular disease | ⟵ | Age | .007 | .000 | 16.891 | *** |
| Cardiovascular disease | ⟵ | Gender | .058 | .014 | 4.182 | *** |
| Early Awakening | ⟵ | Sleeping problems | 1.000 | | | |
| Sleep latency | ⟵ | Sleeping problems | 31.758 | 2.375 | 13.371 | *** |
| Sleep quality | ⟵ | Sleeping problems | 1.392 | .080 | 17.402 | *** |
| Tired at daytime | ⟵ | Sleeping problems | 1.889 | .124 | 15.216 | *** |
| Early awakening | ⟵ | Age | .019 | .001 | 16.364 | *** |
| Sleep quality | ⟵ | Age | .008 | .001 | 7.454 | *** |
| Sleep latency | ⟵ | Education | −1.280 | .322 | −3.977 | *** |
| Early awakening | ⟵ | Noise annoyance night | .079 | .021 | 3.743 | *** |
| Cardiovascular disease | ⟵ | Education | −.017 | .004 | −3.917 | *** |
| Gender | ⟷ | $L_{night}$ outside bedroom | −.216 | .086 | −2.506 | .012 |
| Gender | ⟷ | Age | .470 | .150 | 3.140 | .002 |
| Age | ⟷ | Education | −3.165 | .478 | −6.626 | *** |
| Gender | ⟷ | Education | .071 | .015 | 4.743 | *** |
| Err. term Pseudoneurology | ⟷ | Err. term Anxiety/ depress. | .072 | .004 | 17.906 | *** |
| Err. term Sleep Latency | ⟷ | Err. term Sleep Quality | 8.430 | .509 | 16.574 | *** |
| Err. term Early Awakening | ⟷ | Err. term Sleep Quality | .383 | .018 | 20.810 | *** |
| Err. term Early Awakening | ⟷ | Err. term Sleep Latency | 4.286 | .531 | 8.077 | *** |
| Err. term Sleep Quality | ⟷ | Err. term Tired at Daytime | .165 | .018 | 9.265 | *** |
| Err. term Early Awakening | ⟷ | Err. term Tired at Daytime | .148 | .020 | 7.400 | *** |

*** $p<.001$.

## References


Aasvang GM, Moum T, Engdahl B. Self-reported sleep disturbances due to railway noise: exposure–response relationships for nighttime equivalent and maximum noise levels. J. Acoust. Soc. Am. 2008;124:257–68.
Arbuckle JL. AMOS 16 User's guide; 2008.
Aydin Y, Kaltenbach M. Noise perception, heart rate and blood pressure in relation to aircraft noise in the vicinity of the Frankfurt airport. Clin. Res. Cardiol. 2007;96: 347–58.
Babisch W. Traffic noise and cardiovascular disease: epidemiological review and synthesis. Noise Health 2000;2:9-32.
Babisch W. Transportation Noise and Cardiovascular Disease. Berlin: Umweltsbundesamt; 2006.
Babisch W. Road traffic noise and cardiovascular risk. Noise Health 2008;10:27–33.
Babisch W, Beule B, Schust M, Kersten N, Ising H. Traffic noise and risk of myocardial infarction. Epidemiology 2005;16:33–40.
Babisch W, Fromme H, Beyer A, Ising H. Increased catecholamine levels in urine in subjects exposed to road traffic noise — the role of stress hormones in noise research. Environ. Int. 2001;26:475–81.
Babisch W, Ising H, Gallacher J. Health status as a potential effect modifier of the relation between noise annoyance and incidence of ischaemic heart disease. Occup. Environ. Med. 2003;60:739–45.
Babisch W, Keil T, Stallman M, Wegscheider K, Schust M, Stark H, et al. The Naromi Study: a new study on the relationship between noise and risk of myocardial infarction — first results. Epidemiology 2002;13:S89–90.
Basner M, Glatz C, Griefahn B, Penzel T, Samel A. Aircraft noise: effects on macro- and microstructure of sleep. Sleep Med. 2008;9:382–7.
Basner M, Samel A. Effects of nocturnal aircraft noise on sleep structure. Somnologie - Schlafforschung und Schlafmedizin 2005;9:84–95.
Belojevic G, Jakovljevic B, Aleksic O. Subjective reactions to traffic noise with regard to some personality traits. Environ. Int. 1997;23:221–6.
Berglund B, Lindvall T, Schwela DH. Guidelines for community noise. Geneva: World Health Organisation; 1999.
Bixler EO, Papaliaga MN, Vgontzas AN, Lin HM, Pejovic S, Karataraki M, et al. Women sleep objectively better than men and the sleep of young women is more resilient to external stressors: effects of age and menopause. J. Sleep Res. 2009;18:221–8.
Bluhm GL, Berglind N, Nordling E, Rosenlund M. Road traffic noise and hypertension. Occup. Environ. Med. 2007;64:122–6.
Bowlin SJ, Morrill BD, Nafziger AN, Jenkins PL, Lewis C, Pearson TA. Validity of cardiovascular disease risk factors assessed by telephone survey: the Behavioral Risk Factor Survey. J. Clin. Epidemiol. 1993;46:561–71.
Carter N, Henderson R, Lal S, Hart M, Booth S, Hunyor S. Cardiovascular and autonomic response to environmental noise during sleep in night shift workers. Sleep 2002;25:457–64.
DataKustik. CadnaA. Computer Aided Noise Abatement; 2004.
de Kluizenaar Y, Gansevoort RT, Miedema HME, de Jong PE. Hypertension and road traffic noise exposure. J. Occup. Environ. Med. 2007;49:484–92.
Derogatis LR. SCL-90-R, Administration, scoring and procedures manual. Baltimore: MD: Procedures Psychometric Research; 1983.
Derogatis LR, Lipman RS, Rickels K, Uhlenhuth EH, Covi L. The Hopkins Symptom Checklist (HSCL): a self-report symptom inventory. Behav. Sci. 1974;19:1.
Dinges DF, Pack F, Williams K, Gillen KA, Powell JW, Ott GE, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. Sleep 1997;20:267–77.
Eea. Europe's environment: The third assessment. Copenhagen, Denmark: European Environment Agency (EEA; 2003.
Eriksen HR, Ihlebaek C, Ursin H. A scoring system for subjective health complaints (SHC). Scandinavian Journal of Public Health 1999;27:63–72.
Engelien E, Haakonsen G, Steinnes M. Noise annoyance in Norway. Results from a first generation model for calculating the number of noise exposed persons and the mean noise annoyance. Norway, Oslo: Statistics; 2004.
Ferrara M, De Gennaro L. How much sleep do we need? Sleep Med. Rev. 2001;5:155–79.
Fields JM. Effect of personal and situational variables on noise annoyance in residential areas. J. Acoust. Soc. Am. 1993;93:2753–63.
Fields JM, De Jong RG, Gjestland T, Flindell IH, Job RFS, Kurra S, et al. Standardized general-purpose noise reaction questions for community noise surveys: research and a recommendation. Journal of Sound and Vibration 2001;242:641–79.
Fyhri A, Klaeboe R. Road traffic noise, sensitivity, annoyance and self-reported health — a structural equation model exercise. Environ. Int. 2009;35:91–7.
Glass RM, Allan AT, Uhlenhuth EH, Kimball CP, Borinstein DI. Psychiatric screening in a medical clinic — evaluation of a self-report inventory. Arch. Gen. Psychiatry 1978;35:1189–95.
Graff-Iversen S, Jenum AK, Grøtvedt L, Bakken B, Selmer RM, Søgaard AJ. Risk factors for myocardial infarction, stroke and diabetes in Norway. The Journal of the Norwegian Medical Association 2007;128:2537–41.
Griefahn B, Brode P, Marks A, Basner M. Autonomic arousals related to traffic noise during sleep. Sleep 2008;31:569–77.
Griefahn B, Marks A, Robens S. Noise emitted from road, rail and air traffic and their effects on sleep. J. Sound Vib. 2006;295:129–40.
Guski R. An analysis of spontaneous noise complaints. Environ. Res. 1977;13:229–36.
Haralabidis AS, Dimakopoulou K, Vigna-Taglianti F, Giampaolo M, Borgini A, Dudley ML, et al. Acute effects of night-time noise exposure on blood pressure in populations living near airports. Eur. Heart J. 2008;29:658–64.
Heliovaara M, Aromaa A, Klaukka T, Knekt P, Joukamaa M, Impivaara O. Reliability and validity of interview data on chronic diseases. The Mini-Finland Health Survey. J. Clin. Epidemiol. 1993;46:181–91.

Ihlebaek C, Eriksen HR, Ursin H. Prevalence of subjective health complaints (SHC) in Norway. Scand. J. Public Health 2002;30:20–9.

Irwin M, McClintick J, Costlow C, Fortner M, White J, Gillin JC. Partial night sleep deprivation reduces natural killer and cellular immune responses in humans. Faseb J. 1996;10:643–53.

Ising H, Dienel D, Gunther T, Markert B. Health-effects of traffic noise. Int. Arch. Occup. Environ. Health 1980;47:179–90.

Iso/Tc43/Wg. Assessment of noise annoyance by means of social and socio-acoustic surveys. ISO; 2003.

Jarup L, Babisch W, Houthuijs D, Pershagen G, Katsouyanni K, Cadum E, et al. Hypertension and exposure to noise near airports: the HYENA study. Environ. Health Perspect. 2008;116:329–33.

Marks A, Griefahn B. Associations between noise sensitivity and sleep, subjectively evaluated sleep quality, annoyance, and performance after exposure to nocturnal traffic noise. Noise Health 2007;9:1–7.

Maschke C, Harder J, Ising H, Hecht K, Thierfelder W. Stress hormone changes in persons exposed to simulated night noise. Noise Health 2002;5:35–45.

Miedema HME, Vos H. Noise sensitivity and reactions to noise and other environmental conditions. J. Acoust. Soc. Am. 2003;113:1492–504.

Miki K, Kawamorita K, Araga Y, Musha T, Sudo A. Urinary and salivary stress hormone levels while performing arithmetic calculation in a noisy environment. Ind. Health 1998;36:66–9.

Molenaar EA, Van Ameijden EJ, Grobbee DE, Numans ME. Comparison of routine care self-reported and biometrical data on hypertension and diabetes: results of the Utrecht Health Project. EUROPEAN JOURNAL OF PUBLIC HEALTH 2007;17:199–205.

Muzet A. Environmental noise, sleep and health. Sleep Med. Rev. 2007;11:135.

Norden. Road traffic noise — Nordic Prediction Method; 1996.

Okura Y, Urban LH, Mahoney DW, Jacobsen SJ, Rodeheffer RJ. Agreement between self-report questionnaires and medical record data was substantial for diabetes, hypertension, myocardial infarction and stroke but not for heart failure. J. Clin. Epidemiol. 2004;57:1096–103.

Pallesen S, Nordhus IH, Omvik S, Sivertsen B, Matthiesen SB, Bjorvatn B. Pittsburgh Sleep Quality Index. Tidsskrift for Norsk Psykologforening; 2005. p. 42.

Partinen M, Gislason T. Basic Nordic Sleep Questionnaire (Bnsq) — a quantitated measure of subjective sleep complaints. J. Sleep Res. 1995;4:150–5.

Redeker NS, Ruggiero JS, Hedges C. Sleep is related to physical function and emotional well-being after cardiac surgery. Nurs. Res. 2004;53:154–62.

Reyner LA, Horne JA, Reyner A. Gender- and age-related differences in sleep determined by home-recorded sleep logs and actimetry from 400 adults. Sleep 1995;18:127–34.

Rosenlund M, Berglind N, Pershagen G, Hallqvist J, Jonson T, Bellander T. Long-term exposure to urban air pollution and myocardial infarction. Epidemiology 2006;17:383–90.

Sandor P, Shapiro CM. Sleep patterns in depression and anxiety: theory and pharmacological effects. J. Psychosom. Res. 1994;38(Suppl 1):125–39.

Selander J, Nilsson ME, Bluhm G, Rosenlund M, Lindqvist M, Nise G, et al. Long-term exposure to road traffic noise and myocardial infarction. Epidemiology 2009;20:272–9.

Siegel JM. Why we sleep. Sci. Am. 2003;289:92–7.

Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435–9.

Stansfeld SA. Noise, noise sensitivity and psychiatric-disorder — epidemiologic and psychophysiological studies. Psychol. Med. 1992a:1.

Stansfeld SA. Noise, noise sensitivity and psychiatric disorder: epidemiological and psychophysiological studies. Psychol. Med. 1992b;22 1-44+I.

Stansfeld SA, Berglund B, Clark C, Lopez-Barrio I, Fischer P, Öhrström E, et al. Aircraft and road traffic noise and children's cognition and health: a cross-national study. Lancet 2005;365:1942–9.

Tretli S, Lund-Larsen PG, Foss OP. Reliability of questionnaire information on cardiovascular disease and diabetes: cardiovascular disease study in Finnmark county. J. Epidemiol. Community Health 1982;36:269–73.

van Kempen EEMM, Kruize H, Boshuizen HC, Ameling CB, Staatsen BAM, de Hollander AEM. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ. Health Perspect. 2002;110:307–17.

Velluti RA. Interactions between sleep and sensory physiology. J. Sleep Res. 1997;6:61–77.

Weinstein ND. Individual differences in reactions to noise: a longitudinal study in a college dormitory. J. Appl. Psychol. 1978;63:458–66.

Öhrström E, Bjorkman M, Rylander R. Noise annoyance with regard to neurophysiological sensitivity, subjective noise sensitivity and personality variables. Psychol. Med. 1988;18:605–13.

Öhrström E, Björkman M, Rylander R. Effects of noise during sleep with reference to noise sensitivity and habituation. Environ. Int. 1990;16:477–82.

Öhrström E, Rylander R. Sleep disturbance effects of traffic noise — a laboratory study on after effects. J. Sound Vib. 1982;84:87-103.