ehp ENVIRONMENTAL HEALTH PERSPECTIVES
http://www.ehponline.org

# Residential Road Traffic Noise and High Depressive Symptoms after Five Years of Follow-up: Results from the Heinz Nixdorf Recall Study

**Ester Orban, Kelsey McDonald, Robynne Sutcliffe, Barbara Hoffmann, Kateryna B. Fuks, Nico Dragano, Anja Viehmann, Raimund Erbel, Karl-Heinz Jöckel, Noreen Pundt, and Susanne Moebus**

http://dx.doi.org/10.1289/ehp.1409400

**Received: 30 October 2014**
**Accepted: 14 September 2015**
**Advance Publication: 25 November 2015**

*Environ Health Perspect* DOI: 10.1289/ehp.1409400
Advance Publication: Not Copyedited

# Residential Road Traffic Noise and High Depressive Symptoms after Five Years of Follow-up: Results from the Heinz Nixdorf Recall Study

Ester Orban,[1] Kelsey McDonald,[1] Robynne Sutcliffe,[1] Barbara Hoffmann,[2,3] Kateryna B. Fuks,[2] Nico Dragano,[4] Anja Viehmann,[5] Raimund Erbel,[6] Karl-Heinz Jöckel,[5] Noreen Pundt,[5] and Susanne Moebus[1,5]

[1]Centre for Urban Epidemiology (CUE), Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), University Hospital Essen, Essen, Germany; [2]IUF - Leibniz Research Institute for Environmental Medicine, Düsseldorf, Germany; [3]Medical School, Deanery of Medicine, the Heinrich Heine University of Düsseldorf, Düsseldorf, Germany; [4]Institute for Medical Sociology, Centre for Health and Society, Medical Faculty, University of Düsseldorf, Düsseldorf, Germany; [5]Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), University Hospital Essen, Essen, Germany; [6]Department of Cardiology, University Hospital Essen, Essen, Germany

**Address correspondence to** Ester Orban, Centre for Urban Epidemiology (CUE), Institute for Medical Informatics, Biometry and Epidemiology (IMIBE), Hufelandstr. 55, 45147 Essen, Germany. Telephone: 0049 201 92239 238. E-mail: ester.orban@uk-essen.de

**Running title:** Residential road traffic noise and depressive symptoms

**Competing financial interests:** The authors declare they have no actual or potential competing financial interests.

## ABSTRACT

**Background:** Traffic noise affects a large number of people especially in urbanized areas. Noise causes stress and annoyance, but less is known about the relationship between noise and depression.

**Objective:** To investigate the association of residential road traffic noise and depressive symptoms using five-year follow-up data from a German population-based study.

**Methods:** We analyzed data from 3,300 participants of the Heinz Nixdorf Recall study, aged 45-75 years and without depressive symptoms at baseline (2000-2003). Depressive symptoms were defined based on the CES-D 15-item questionnaire (total score ≥17) and antidepressant medication intake. Road traffic noise was modeled per European Parliament/Council Directive 2002/49/EC. High noise exposure was defined as annual mean 24-hour noise levels >55dB(A). Poisson regression with robust variance was used to estimate relative risks (RR), adjusting for the potential confounders 1) age, sex, socioeconomic status (SES), neighborhood-level SES, traffic proximity, 2) additionally adjusting for body mass index and smoking, and 3) additionally adjusting for the potential confounders/intermediates co-morbidities and insomnia.

**Results:** Overall 35.7% of the participants were exposed to high residential road traffic noise levels. At follow-up (mean = 5.1 years after baseline), 302 participants were classified as having high depressive symptoms, corresponding to an adjusted RR of 1.29 (95% confidence interval 1.03, 1.62; model 1) for exposure to >55 versus ≤55 dB(A). Adjustment for potential confounders/intermediates did not substantially alter the results. Associations were stronger among those who reported insomnia at baseline (RR = 1.62; 1.10, 2.59 vs. RR = 1.21; 0.94, 1.57) and appeared to be limited to those with ≤13 years of education (RR = 1.43; 1.10, 1.85 vs. 0.92; 0.56, 1.53 for >13 years).

**Conclusion:** Our results suggest that residential road traffic noise exposure increases the risk of depressive symptoms.

## INTRODUCTION

Noise is a psychosocial stressor that may affect health, even at low levels (Babisch 2002). A large number of people in urban settings are exposed to traffic noise, and the World Health Organization (WHO) considers environmental noise to be an important public health issue (WHO Regional Office for Europe 2011). Beyond annoyance, exposure to traffic noise has been associated with stress-related and cardiovascular outcomes such as hypertension and myocardial infarction (Barregard et al. 2009; Fuks et al. 2011; Willich 2005). Recently, an association of long-term exposure to traffic noise with incident diabetes mellitus type 2 has been reported (Sørensen et al. 2012). Until now, epidemiologic research on noise has focused mainly on cardiovascular effects and less is known about the relationship between traffic noise and mental health problems such as depression.

Depression is a common mental disorder and an increasing public health concern (Weissman et al. 1992). It is a leading cause of disability worldwide. According to results from the Global Burden of Diseases, Injuries, and Risk Factors Study 2010, mental and substance use disorders contributed 7.4% to the total global burden of disease (as measured in disability adjusted life years, DALYs) in 2010, which of 40.5% were attributable to depressive disorders (Whiteford et al. 2013). Individuals affected by depression not only experience reduced quality of life due to suffering, but may also be unable to cope with everyday life including occupational activities, which results in increased sick leave (Wedegaertner et al. 2013).

The etiology of depression is multi-factorial and complex. Psychological, social and biological factors may be involved, most likely in combination (WHO 2012). The potential influence of noise on mental health has been examined, but findings from studies of noise and mental health outcomes have been inconsistent (Crombie et al. 2011; Floud et al. 2011; Hardoy et al. 2005;

Niemann et al. 2006; Schreckenberg et al. 2010; Sygna et al. 2014). This may be attributed to differences in study design, investigated populations (children, adults), exposures (aircraft and road traffic noise, and subjective noise annoyance as opposed to objectively modeled/measured noise) and outcomes (various psychological symptom measures/questionnaires, diagnoses, medication intake, mental hospital admissions). Few studies have examined the association between road traffic noise and depressive symptoms in adults and there is a particular lack of evidence from prospective studies. To our knowledge, there is only one prospective study examining this association (Stansfeld et al. 1996). This study was conducted in Caerphilly, south Wales, and found no association between traffic noise levels at baseline and depression scores after five years of follow-up; however, exclusively men (n=1,725) were included.

There are several potential pathways supporting the hypothesis that chronic noise exposure may be related to depressive symptoms. Sleep disturbance conditions such as insomnia, which may be caused by traffic noise (Halonen et al. 2012), have been shown to be associated with depression in previous studies (Franzen and Buysse 2008; Riemann and Voderholzer 2003; Roberts et al. 2000). Thus, decreased quality of sleep represents one possible link between noise exposure and mental health. A recent cross-sectional study analyzing survey data of 2,778 adults from an age- and sex-stratified population registry sample in Oslo, Norway, found a weak association between road traffic noise and mental health as measured by the Hopkins Symptom Checklist, but only in participants with poor quality of sleep (Sygna et al. 2014). Further, acute noise events cause biological stress reactions (Babisch 2002). Such stress reactions may in turn promote onset of depression (Anisman and Merali 2002; Wager-Smith and Markou 2011); however, single acute noise events are unlikely to cause depression. Thus it is an open question, whether repeated or chronic noise exposure has long-term effects on depressive illness.

The aim of this study is to investigate the association of long-term exposure to objectively measured road traffic noise and depressive symptoms within a population-based cohort of middle-aged men and women living in the highly urbanized metropolitan Ruhr area in Germany.

## METHODS

### Study population

We analyzed baseline and five-year follow up data from the ongoing prospective Heinz Nixdorf Recall study (HNR) conducted in three large adjacent cities (Bochum, Essen and Mülheim/Ruhr) located in the west of Germany. The study design has been described in more detail elsewhere (Schmermund et al. 2002). Baseline examinations were performed between 2000 and 2003 and included 4814 participants aged 45–75 years who were randomly selected from population registries. Individuals were eligible if their address was valid, they were not institutionalized, had sufficient knowledge of the German language, were not severely ill and able to be interviewed. Further, pregnant women (though not a priority considering the investigated age group) and relatives of study personnel were excluded. The baseline response calculated as recruitment efficacy proportion was 55.8% (Stang et al. 2005). The follow-up examinations were performed between 2005 and 2008. Our analyzed sample is depicted in Figure 1 and further described in the statistical analysis section of the methods. The study maintains extensive quality management procedures, including a certification according to DIN ISO 9001:2000/2008. The HNR was approved by the local ethics committees and all participants gave informed consent prior to participation.

**Outcome**

Depressive symptoms during the previous week were assessed using the 15-item short form questionnaire of the Center for Epidemiologic Studies Depression scale (CES-D) (Radloff 1977, Hautzinger and Bailer 1993), which was handed out at the baseline and 5 year-follow-up visit at the study center (and mailed to participants that did not attend the examinations). The CES-D is a screening tool for measuring depressive symptoms which has been validated in different populations and settings and is frequently applied in health research (Radloff 1977). Possible scores of the 15-item version range from 0 to 45, with higher levels indicating more and/or more frequent depressive symptoms. It is considered an indicator of a probable depressive episode, but does not replace a face-to-face physician diagnosis. Antidepressant medication was also included in the outcome definition, because it is indicative of depressive symptoms being treated (even if off-label use may occur) and may affect CES-D results in depressive individuals, as treated participants may show fewer symptoms. Assessment of all medication intake was performed by asking participants to bring along all medication (including packages) taken in the last 7 days to the baseline and follow-up visit, respectively. Intake of antidepressant medication out of the Anatomical Therapeutic Chemical (ATC) groups N06A or N06CA and/or a CES-D score ≥17 according to Hautzinger and Bailer (1993) were used to define high depressive symptoms.

**Exposure**

Road traffic noise was modeled according to the Directive 2002/49/EC of the European Parliament and Council (European Parliament and Council of the European Union 2002) for the year 2006 as a weighted day-evening-night (24-hour) average sound level ($L_{den}$) in 5-dB(A) categories (isophones). The following factors were considered in the noise level modeling:

small-scale topography of the area, dimensions of buildings, noise barriers, street axis, vehicle-type-specific traffic density, speed limit and type of road surface. Noise exposure data was assigned to the geographic residence location of the study participants at baseline using the geographic information system ArcGIS, assuming average noise levels to be relatively stable over time. High noise exposure was defined as noise levels of $L_{den}$ >55 dB(A) based on the maximum community noise levels recommended by the WHO (Berglund et al. 1999). Data on nighttime noise ($L_{night,}$ 10 p.m.–6 a.m.) was available and analyzed as well, defining nighttime noise levels >50 db(A) as high noise exposure.

**Covariates**

Socioeconomic (e.g. income), demographic (e.g. age), behavioral (e.g., smoking: current, former, or never-smoker), and medical history data were assessed via standardized computer-assisted personal interviews at the baseline examination. Education, income and economic activity were applied as indicators of socioeconomic status (SES) (Shavers 2007; Galobardes et al. 2007). Education was defined combining school and vocational training as total years of formal education according to the International Standard Classification of Education (UNESCO 1997) and categorized into four groups (≤10, 11 to 13, 14 to 17, and ≥18 years). Income was measured as the monthly household equivalent income calculated by dividing the total household net income by a weighting factor for each household member and divided into four groups using sex-specific quartiles. Economic activity was categorized into three groups (employed, inactive [retired, homemaker, etc. but not unemployed] and unemployed). Information on whether participants have/have ever had myocardial infarction, heart failure, stroke, diabetes mellitus, emphysema, asthma, cancer, rheumatism, slipped disc, or migraine (yes/no) at baseline was used to create a categorical variable indicating the number of co-morbidities (0, 1 or ≥2). In addition,

participants were asked to indicate if they have/have ever had depression. Insomnia was assessed based on three insomnia symptoms: difficulties falling asleep, difficulties maintaining asleep, and early morning arousals (Riedel et al. 2012). If participants reported all of these were present at least two times per week during the last four weeks, participants were classified as having insomnia. One example of the three insomnia questions is "How often, during the last 4 weeks, did you have difficulties to fall asleep?", with possible answers 'never', 'sometimes (one time per week or less)', 'often (at least 2 times per week)' or 'almost every night'. Height and weight were obtained by standardized anthropogenic measurements during the clinical examination. The body mass index (BMI) was calculated as (weight in kg/[height in m]$^2$).

We applied the 2001 unemployment rate in the respective city unit (German terms: in Essen 'Stadtteil', in Bochum and Mülheim/Ruhr 'statistischer Bezirk') as an indicator of neighborhood-level SES. This data was obtained from the local census authorities of the respective cities of Bochum, Essen and Mülheim/Ruhr.

Residential distance to the nearest major road was calculated as a marker of traffic proximity using ArcGIS. A major road was defined as one falling into the upper quartile of mean daily traffic density (>22,980 vehicles per day, year 2000). There was a weak negative correlation between traffic proximity and noise in our study (Pearson r=-0.22). We included this variable in the analysis to control for non-acoustic factors of traffic and the physical environment of the neighborhood, which might affect mental wellbeing, e.g. aesthetic aspects and perceived safety.

**Statistical analyses**

From the full HNR sample (n=4,814), we excluded 432 participants with missing information on depressive symptoms (CES-D and/or antidepressant medication) and further 593 participants

with prevalent high depressive symptoms at baseline (Figure 1). Of the remaining 3,789 participants, 154 died during follow-up, 312 were excluded because they did not attend the follow-up examination (when medication use and CES-D was assessed) or complete the mailed non-attendee follow-up questionnaire (including the CES-D), and 23 were excluded because they did not complete the CES-D and were not identified as using anti-depressant medication at the follow-up visit (Figure 1). Five of the included participants did not attend the follow-up visit, but were classified as having high depressive symptoms based on the mailed non-attendee follow-up CES-D. The final analysis sample hence included 3,300 participants (87.1% of the 3,789 eligible participants).

We used Poisson regression with a robust variance to estimate crude and adjusted effects of high road traffic noise on depressive symptoms after five years (Spiegelman and Hertzmark 2005; Zou 2006). The adjustment sets were selected *a priori* based on a directed acyclic graph (see Supplemental Material, Figure S1) created with DAGitty (Textor et al. 2011). In model 1, we adjusted for age (continuous), sex, SES (education, income, economic activity), neighborhood-level SES (unemployment rate, continuous) and traffic proximity (continuous). In model 2, we additionally adjusted for the potential confounders BMI (continuous) and smoking, and in model 3 the potential confounders/intermediates co-morbidities (0, 1, or ≥2) and insomnia (yes/no) were added. Observations with any missing covariate data were automatically excluded in the respective analysis (complete case analysis). All analyses were also stratified by sex to investigate potential sex-specific differences. In addition to modeling road traffic noise as a binary variable [$L_{den}$ >55 vs. ≤55 dB(A)], we estimated associations with three noise exposure categories [$L_{den}$: >55 to ≤60 dB(A), >60 to ≤65 dB(A), >65 dB(A)] compared to the reference group with $L_{den}$ ≤55 dB(A) noise exposure.

We conducted exploratory analyses by stratifying the participants by (i) education level (≤13 vs. >13 years of formal education), (ii) movers vs. non-movers between baseline and five-year follow-up, (iii) insomnia (yes/no), and (iv) city of residence. Further sensitivity analyses were conducted by (v) additionally excluding participants who reported to have/have ever had depression at baseline, (vi) using a cutoff $L_{den}$ >65 dB(A) to define very high noise exposure, (vii) using CES-D score ≥17 exclusively to define high depressive symptoms at baseline and follow-up and (viii) using antidepressant medication intake exclusively to define high depressive symptoms at baseline and follow-up, respectively.

All analyses were conducted with SAS version 9.4.

**RESULTS**

Baseline characteristics of the analyzed population by noise exposure are shown in Table 1. Participants with high and low noise exposure were similar regarding sex and mean age, whereas proportions of insomnia, low education, low income, unemployment and active smoking were higher in participants exposed to high noise levels. There was only little missing covariate data (max. 15, for insomnia), with exception of the income variable with a total of 196 values missing (Table 1). There were further 605 missing values for the variable indicating reported (lifetime) prevalence of depression, which was applied in one of the sensitivity analyses. At follow-up (5.1 years after baseline, on average), 302 participants [9.2%, including 201/1,585 women (12.7%) and 101/1,715 men (5.9%)] were classified as having high depressive symptoms based on a CES-D score ≥ 17 (n=179), use of antidepressant medication (n=97), or both (n=26) in the previous week (Figure 1). Participants who were excluded from the analysis due to depressive symptoms/missing depressive symptoms data at baseline (drop out 1), or death or missing outcome data at follow-up (drop out 2), were similar to the analysis sample with regard to sex,

age, and other baseline characteristics (see Supplemental Material, Table S1). However, they were more likely to have been current smokers (26–31% vs. 20–24%), and had more co-morbidities (36–37% with ≥2, vs. 29–31%), lower education (19% ≤10 years vs. 8–9%), and lower income (33–34% in the lowest quartile vs. 21–27%). Participants excluded because of prevalent depressive symptoms at baseline/missing depressive symptoms data were more likely to have reported insomnia at baseline (22% vs. 8–11%) and were less likely to be male (40% vs. 52%).

Of the included study population, 35.7% (n=1,179) were exposed to high 24-hour traffic noise levels [$L_{den}$ >55 dB(A)] and 25.8% (n=850) were exposed to high traffic noise at night [$L_{den}$ >50 dB(A)]. Distributions of annual mean noise exposures (overall and at night) were positively skewed (see Supplemental Material, Figure S2).

The results of the regression analysis (Table 2) revealed an adjusted RR (model 1) of 1.29 (95% CI: 1.03, 1.62) for high depressive symptoms at follow-up in participants exposed to high noise levels compared with the low noise exposure group. Estimates for men and women combined were similar for models 2 and 3 and the unadjusted estimate (Table 2). Unadjusted associations were stronger for men than women, but were similar between men and women after adjustment for socio-demographic covariates (model 1) and BMI and smoking (model 2). Adjusting for potential intermediates (co-morbidities and insomnia, model 3) slightly reduced the RR toward the null for men, but did not influence the association for women. We excluded participants with missing income data (n=196) and found that this had no substantial influence on the results, yielding a crude total RR of 1.39 (95% CI: 1.11, 1.74, n=3,104), a RR of 1.43 (95% CI: 0.97, 2.10, n=1,652) in men and a RR of 1.36 (95% CI: 1.03, 1.78, n=1,452) in women (data not shown in Table 2). In general, associations between depression and exposure to noise at night

[$L_{night}$ >50 vs. ≤50 dB(A)] were similar to associations with average 24-hour noise exposure (model 1 RR = 1.29; 95% CI: 1.01, 1.64 for men and women combined), though associations were weaker for men (RR = 1.19; 95% CI: 0.77, 1.82) than for women (RR = 1.36; 95% CI: 1.01, 1.82) (see Supplemental Material, Table S2).

Associations between noise and depressive symptoms did not increase with increasing noise when exposure was categorized into four groups (Figure 2). When compared with ≤55 dB(A), the association was strongest for the middle exposure category [>60 to ≤65 dB(A), RR = 1.52; 95% CI: 1.11, 2.07] and equally weaker for the highest and lowest exposure groups (RR = 1.19; 95% CI: 0.85, 1.68 and RR = 1.19; 95% CI: 0.86, 1.65, respectively) (Figure 2). Similarly, for nighttime road traffic noise there was no evidence of a monotonic dose-response relation, but the pattern differed, with the middle exposure category [>55 to ≤60 dB(A)] having the weakest association compared with the ≤50 dB(A) reference group (RR = 1.14; 95% CI: 0.78, 1.65) (see Supplemental Material, Figure S3).

Table 3 shows the results of additional analyses. We estimated a positive association between noise exposure and high depressive symptoms at follow-up among 2,115 participants with ≤13 years of education (model 1 RR = 1.43; 95% CI: 1.10, 1.85), in contrast with a weak negative association among 1,185 participants with >13 years of education (RR = 0.92; 95% CI: 0.56, 1.53). As expected, a higher effect estimate was found in the subgroup with insomnia at baseline (model 1 RR = 1.62; 95% CI: 1.01, 2.59; n=281), compared to those without insomnia at baseline (RR 1.21; 95% CI: 0.94, 1.57; n=2,803) (Table 3). The association between traffic noise and depressive symptoms did not change remarkably when excluding participants who reported to have/have ever had depression at baseline (n=176) or had missing data on depression (n=605), yielding a RR of 1.24 (95% CI: 0.97, 1.59; model 1). Using a higher cutoff value for defining

high noise exposure [$L_{den}$ >65 vs. ≤65 dB(A)] resulted in a RR of 1.07 (95% CI: 0.77, 1.49), which is in accordance with the results shown in Figure 2. Using either only a CES-D score ≥17 (n=244 cases at follow-up) or only intake of antidepressant medication (n=157 cases at follow-up) to define the outcome did not lead to different results compared with the combined outcome definition (Table 3). Generally, additional analyses for the association of nighttime traffic noise exposure >50 dB(A) vs. ≤50 dB(A) with high depressive symptoms at follow-up showed similar results as those for 24-hour noise exposure, with the possible exception of the analysis using antidepressant medication use to define outcome (see Supplemental Material, Table S3).

## DISCUSSION

Our prospective study provides support for the hypothesis that long-term exposure to road traffic noise may increase the risk of depressive symptoms.

In our study population as a whole, high depressive symptoms at follow-up were about 25-30% more frequent in study participants exposed to road traffic noise levels >55 dB(A) compared with ≤55 dB(A). The association remained stable after adjustment for various covariates, which underlines the robustness of the results when considering potential confounding factors. Our findings are in line with results from previous cross-sectional studies on road traffic noise and depression. A study in Serbia (Stošić and Blagojević 2011) with 911 participants aged 18-80 years found that subjects living in a noisy city area of Niš (daily period noise ≥55dB(A) and night noise ≥45dB(A)) reported 'feeling depressed' more frequently than the control subjects living in two quiet city areas (daily period noise ≤ 55dB(A) and night noise ≤ 45 dB(A)). A similar small Swedish study compared 151 persons in a quiet city area and 97 persons in an area exposed to noise (Öhrström 1991). This study used mailed questionnaires to assess psycho-social well-being, including depression and found that people living in the noisy area more often felt

depressed. In another questionnaire-based study of 366 women (20-60 years) living in Tokyo (Yoshida and Osada 1997), an unadjusted OR of 2.9 ($p<0.05$) for high responses to depression-related questions was found in women exposed to residential road traffic noise levels >70 dB(A) compared with those exposed to 45 to ≤70 dB(A). Importantly, none of these cross-sectional studies reported controlling for potential confounding factors. Sygna et al. (2014) found an association (controlled for confounders) of road traffic noise and psychological distress, including depressive symptoms, but only in a subgroup of 274 participants with low sleep quality (OR 1.40, 95% CI: 0.99–1.98, per 10dB increase). The Caerphilly study (Stansfeld et al. 1996) is the only previous prospective study of traffic noise and depressive symptoms we know of and analyzed data from 1,725 men living in Caerphilly (age 50-64). This men-only study found no association between traffic noise levels at baseline (in four 5-dB(A) categories ranging from 51-55 dB(A) to 66-70 dB(A)) and mean depression scores from the general health questionnaire at five-year follow-up, adjusting for age, social class, noise sensitivity and depressive symptoms at baseline (n=1,587), but did find an association with mean anxiety scores, which significantly differed across the noise categories (p for heterogeneity=0.03, n=1,584) (Stansfeld et al. 1996). In summary, most previous studies on road traffic noise and depressive symptoms found an association, and our study adds to the existing body of evidence by prospectively analyzing a comprehensive cohort including both men and women, while accounting for potential confounding factors.

Sex-specific analyses revealed no differences between men and women. It is noticeable though, that high depressive symptoms at follow-up were far more common in women than in men (12.7% vs. 5.9%). This result is consistent with existing epidemiologic research, where a higher prevalence of depression has been observed in women than in men, with an estimated

female:male ratio of 2.3 (Wittchen et al. 2011). It has been argued that these differences in prevalence may not be real, as depression symptoms may vary between men and women (Azorin et al. 2014; Rutz 1999; Schuch et al. 2014), but commonly applied diagnostic criteria focus on symptoms rather typical for women and men are believed to display a less pronounced help-seeking behavior than women (Piccinelli 2000; Schuch et al. 2014). Thus, a potential for measurement error due to gender-insensitive diagnostic criteria and varying prescribing patterns needs to be considered and gender-specific associations deserve further attention.

When investigating different categories of road traffic noise, relative risks did not increase linearly with increasing noise levels and we found that elevated risks of high depressive symptoms were strongest not in the highest but in the intermediate exposure group for 24-hour noise exposure. However, number of participants in the noise categories was small, overall incidence of depressive symptoms low and we consider this analysis primarily explorative for future research aims. Previous studies also failed to identify a linear trend (Yoshida and Osada 1997; Stansfeld et al. 1996). An explanation for this missing dose-response relationship may be that measures of noise mitigation (e.g. noise protection windows) and behavioral prevention (i.e. closed windows, choice of quiet sleeping room, earplugs) may be more common in areas with a very high noise exposure. A non-linear relationship of exposure and outcome may also contribute to the inconsistencies of results from previous studies.

We found a stronger association of traffic noise and high depressive symptoms in less educated participants and a weak negative association in the higher educated (Table 3). Also, a higher proportion of study participants with low income, low education and who were unemployed had high traffic noise exposure (Table 1), supporting previous observations of a socially inequitable distribution of environmental burden (Braubach and Fairburn 2010). A previous analysis of the

German Socio-Economic Panel also found that low household income was associated with higher perceived noise exposure (Kohlhuber et al. 2006).

The association of noise and depression-related outcomes found in the HNR and previous studies seems biologically plausible. Stratified analyses revealed a stronger association between high noise exposure and high depressive symptoms in participants with insomnia at baseline and the same was found in a previous study (Sygna et al. 2014). This is in line with the hypothesis of impaired sleep as a possible pathway (Baglioni et al. 2011). However, insomnia may also be a symptom of depression rather than a contributing factor, thus, an association between depression and insomnia at the same point in time may be bi-directional. Our results suggest that individuals with pre-existing sleep disturbances might be more vulnerable to effects of noise on depressive symptoms. However, we do not know the underlying causes of insomnia in our study population. Another factor linking noise and depression may be noise induced stress reactions of the body. Acute noise stimuli cause the central nervous system to initiate warning/alert reflexes that are beyond individual control and affect muscle tension and pulse rate, for example (Rylander 2004). Repeated exposure to noise over longer times is usually considered unpleasant or annoying when it interferes with activities of living such as communication, tasks that require concentration or recreational activities like sleep and rest. Habituation to noise does hardly occur and chronic exposure to noise that causes negative physiological stress reactions may lead to a stage where the acute effects, such as increase in blood pressure, become permanent (Rylander 2004). It has further been discussed that exposure to stressors promotes neurochemical and endocrine changes that may be involved in the provocation of depressive disorder (Anisman and Merali 2002; Wager-Smith and Markou 2011). Chronic stress due to noise may lead to involuntary defeat reactions characterized by e.g. decreased motor function, decreased secretion of cortisol and

adrenalin and suppression of the immune system, with depression of mood as a possible consequence. However, the extent to which noise causes such defeat reactions may differ individually, depending on the possibility to escape noise, e.g. by closing the windows or choosing a bedroom facing away from the street (Rylander 2004). Increased stress hormone levels due to noise are a frequent finding (Ising and Kruppa 2004), which may be an explanation for our observed results when considering physiological stress as a factor in the pathway from noise exposure to depression. It is also possible that the observed association of noise and depressive symptoms is in part mediated by other stress-related or chronic diseases such as cardiovascular diseases, which have been found to be associated with both noise and depression (Münzel et al. 2014; Hare et al. 2013), however, accounting for co-morbidities by adjustment did not change the RR estimate in our study.

Strengths of this study include a high quality noise exposure model and residential addresses at baseline to accurately assess exposure. Depressive symptoms were assessed by a widely used and well established instrument. The prospective design allows for investigation of long-term noise effects, assuming that mean noise levels modelled for 2006 and assigned to the baseline (2000-2003) residence location were constant over the five-year follow-up period. We were able to investigate a large number of randomly selected participants, to consider potential confounding factors in our analyses and to study noise effects in different subgroups.

Considering limitations, exposure misclassification is a main concern in environmental epidemiology. Noise exposure assessment in this study includes residential road traffic noise only; other sources of residential noise, for instance air or railway traffic noise, or noise caused by neighbors, were not included. Nevertheless, road traffic is considered the major source of noise pollution in urban metropolitan contexts such as the investigated Ruhr area (Omidvari and

Nouri 2009) and most neighborhoods of our study population are not affected by aircraft noise. Further, we had no information on time spent at the residence and non-residential noise exposures such as occupational noise. Individual characteristics such as room ventilation patterns, hearing ability, noise protection windows, etc. were not accounted for in the analysis, but may also contribute to misclassification of noise exposure. As subjects exposed to (very) high levels of noise may make more use of noise avoidance strategies, this may lead to an underestimation of the effect that would be observed without these measures – which may in part explain our findings of a lower RR in the highest noise category. Subjects exposed to high and low levels of noise may differ in some characteristics relevant to the development of depressive symptoms and even though we were able to take a range of these factors into account in our analyses, unknown confounding cannot be ruled out. Additional bias due to missing data is possible, however, income was the most common missing data and yet excluding those missing income from the crude model did not change the results. Potential air pollution effects were only accounted for indirectly by adjusting for traffic proximity. Modeling the average noise level as done here does not reflect potential peaks, extreme noise events or single sleep disturbing noise events in otherwise quiet areas, all of which are of special relevance in terms of physiological stress reactions to noise (Rylander 2004; Babisch 2002). Also, noise was modeled for 2006 and the assumption of unchanged noise exposure during the study period may not hold. Severity and presence of depressive symptoms varies over time; hence additional CES-D assessments (e.g. yearly instead of every five years) would have allowed for a more precise outcome measurement. We investigated a general population sample of middle-aged and older men and women living in a German metropolitan area and hence our results cannot be generalized to populations from other countries or children, young adults and populations residing in rural areas.

## CONCLUSION

Our results suggest that exposure to residential traffic noise may increase the risk of high depressive symptoms in middle-aged and older adults. Also, our study offers preliminary evidence that those with a low socioeconomic status and sleep disturbances may be particularly vulnerable to noise effects. Further prospective research is needed to confirm the results of our study and to extend the generalizability of our findings to other populations. Studies including measures of stress and subjective noise annoyance may also extend our knowledge into the mechanisms of noise-induced depression. However, there is already evidence of adverse health effects from noise, stressing the necessity of protecting populations from noise pollution, especially with regard to environmental justice as our results indicate that traffic noise may be unequally distributed across social strata.

## REFERENCES

Anisman H and Merali Z. 2002. Cytokines, stress, and depressive illness. Brain Behav Immun 16(5):513–524.

Azorin J, Belzeaux R, Fakra E, Kaladjian A, Hantouche E, Lancrenon S et al. 2014. Gender differences in a cohort of major depressive patients: Further evidence for the male depression syndrome hypothesis. J Affect Disord 167:85–92.

Babisch W. 2002. The noise/stress concept, risk assessment and research needs. Noise Health 4(16):1–11.

Baglioni C, Battagliese G, Feige B, Spiegelhalder K, Nissen C, Voderholzer U et al. 2011. Insomnia as a predictor of depression: A meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord 135(1-3):10–19.

Barregard L, Bonde E, Ohrstrom E. 2009. Risk of hypertension from exposure to road traffic noise in a population-based sample. Occup Environ Med 66(6):410–415.

Berglund B, Lindvall T, Schwela DH. 1999. Guidelines for community noise. Geneva:World Health Organization.

Braubach M and Fairburn J. 2010. Social inequities in environmental risks associated with housing and residential location-a review of evidence. Eur J Public Health 20(1):36–42.

Crombie R, Clark C, Stansfeld SA. 2011. Environmental noise exposure, early biological risk and mental health in nine to ten year old children: a cross-sectional field study. Environ Health 10(39):doi: 10.1186/1476-069X-10-39.

European Parliament and Council of the European Union. 2002. Directive 2002/49/EC of the European Parliament and of the Council of 25 June 2002 relating to the assessment and management of environmental noise - Declaration by the Commission in the Conciliation Committee on the Directive relating to the assessment and management of environmental noise. Available: http://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=CELEX:32002L0049:EN:NOT [accessed 03 December 2013].

Floud S, Vigna-Taglianti F, Hansell A, Blangiardo M, Houthuijs D, Breugelmans O et al. 2011. Medication use in relation to noise from aircraft and road traffic in six European countries: results of the HYENA study. Occup Environ Med 68(7):518–524.

Franzen PL and Buysse DJ. 2008. Sleep disturbances and depression: risk relationships for subsequent depression and therapeutic implications. Dialogues Clin NeurosCI: 10(4):473–481.

Fuks K, Moebus S, Hertel, Viehmann A, Nonnemacher M, Dragano N et al. 2011. Long-Term Urban Particulate Air Pollution, Traffic Noise, and Arterial Blood Pressure. Environ Health Perspect 119(12):1706–17011.

Galobardes B, Lynch J, Smith GD. 2007. Measuring socioeconomic position in health research. British Medical Bulletin 81-82(1):21–37.

Halonen JI, Vahtera J, Stansfeld S, Yli-Tuomi T, Salo P, Pentti J et al. 2012. Associations between Nighttime Traffic Noise and Sleep: The Finnish Public Sector Study. Environ Health Perspect 120(10):1391–1396.

Hardoy MC, Carta MG, MarCI: AR, Carbone F, Cadeddu M, Kovess V et al. 2005. Exposure to aircraft noise and risk of psychiatric disorders: the Elmas survey. Soc Psychiatry Psychiatr Epidemiol 40(1):24–26.

Hare DL, Toukhsati SR, Johansson P, Jaarsma T. 2013. Depression and cardiovascular disease: a clinical review. Eur Heart J 35(21):1365–1372.

Hautzinger M and Bailer M. 1993. ADS—General Depression Scale. Manual [German]. Weinheim: Beltz Test, 1993.

Ising H and Kruppa B. 2004. Health effects caused by noise : Evidence in the literature from the past 25 years. Noise Health 6(22):5–13.

Münzel T, Gori T, Babisch W, Basner M. 2014. Cardiovascular effects of environmental noise exposure. Eur Heart J 35(13):829–836.

Niemann H, Bonnefoy X, Braubach M, Hecht K, Maschke C, Rodrigues C et al. 2006. Noise-induced annoyance and morbidity results from the pan-European LARES study. Noise Health 8(31):63–79.

Öhrström E. 1991. Psycho-social effects of traffic noise exposure. J Sound Vib 151(3):513–517.

Omidvari M and Nouri J. 2009. Effects of noise pollution on traffic policemen. Int J Environ Res 3(4):645–652.

Piccinelli M. 2000. Gender differences in depression: Critical review. BJP 177(6):486–492.

Radloff LS. 1977. The CES-D Scale: A Self-Report Depression Scale for Research in the General Population. Appl Psych Meas 1(3):385–401.

Riedel N, Fuks K, Hoffmann B, Weyers S, Siegrist J, Erbel R et al. 2012. Insomnia and urban neighbourhood contexts – are associations modified by individual social characteristics and change of residence? Results from a population-based study using residential histories. BMC Public Health 12(810):doi: 10.1186/1471-2458-12-810.

Riemann D and Voderholzer U. 2003. Primary insomnia: a risk factor to develop depression? J Affect Disord 76(1-3):255–259.

Roberts RE, Shema J, Kaplan GK, Strawbridge WJ. 2000. Sleep Complaints and Depression in an Aging Cohort: A Prospective Perspective. Am J Psychiatry 157(1):81–88.

Rutz W. 1999. Improvement of care for people suffering from depression: the need for comprehensive education. Intern Clin Pharmacol 14(suppl 3):S27-S33.

Rylander R. 2004. Physiological aspects of noise-induced stress and annoyance. J Sound Vib 277(3):471–478.

Schmermund A, Möhlenkamp S, Stang A, Grönemeyer D, Seibel R, Hirche H et al. 2002. Assessment of clinically silent atherosclerotic disease and established and novel risk factors for predicting myocardial infarction and cardiac death in healthy middle-aged subjects: Rationale and design of the Heinz Nixdorf RECALL Study. Am Heart J 144(2):212–218.

Schreckenberg D, Meis M, Kahl C, Peschel C, Eikmann T. 2010. Aircraft Noise and Quality of Life around Frankfurt Airport. International Journal of Environmental Research and Public Health 7(9):3382–3405.

Schuch JJ, Roest AM, Nolen WA, Penninx BW, Jonge P de. 2014. Gender differences in major depressive disorder: Results from the Netherlands study of depression and anxiety. J Affect Disord 156:156–163.

Shavers VL. 2007. Measurement of Socioeconomic Status in Health Disparities Research. Journal of the National Medical Association 99(9):1013–1023.

Sørensen M, Andersen ZJ, Nordsborg RB, Becker T, Tjønneland A, Overvad K et al. 2012. Long-Term Exposure to Road Traffic Noise and Incident Diabetes: A Cohort Study. Environ Health Perspect 121(2):217–222.

Spiegelman D and Hertzmark E. 2005. Easy SAS Calculations for Risk or Prevalence Ratios and Differences. Am J Epidemiol 162(3):199-200.

Stang A, Moebus S, Dragano N, Beck EM, Möhlenkamp S, Schmermund A et al. 2005. Baseline recruitment and analyses of nonresponse of the Heinz Nixdorf recall study: Identifiability of phone numbers as the major determinant of response. Eur J Epidemiol 20(6):489–496.

Stansfeld SA, Gallacher J, Babisch W, Shipley M. 1996. Road traffic noise and psychiatric disorder: prospective findings from the Caerphilly study. BMJ(313):266–267.

Stošić L and Blagojević L. 2011. Environmental noise and mental disturbances in urban population. Acta Med Median 50(3):34–39.

Sygna K, Aasvang GM, Aamodt G, Oftedal B, Krog NH. 2014. Road traffic noise, sleep and mental health. Environmental Research 13117–24.

Textor J, Hardt J, Knüppel S. 2011. DAGitty: A Graphical Tool for Analyzing Causal Diagrams. Epidemiology 22(5):745.

United Nations Educational, Scientific and Cultural Organization (UNESCO). 1997. International standard classification of education: ISCED 1997. Available: http://www.uis.unesco.org/Library/Documents/isced97-en.pdf [accessed 28 October 2014].

Wager-Smith K and Markou A. 2011. Depression: A repair response to stress-induced neuronal microdamage that can grade into a chronic neuroinflammatory condition? NeurosCI: Biobehav Rev 35(3):742–764.

Wedegaertner F, Arnhold-Kerri S, Sittaro N, Bleich S, Geyer S, Lee WE. 2013. Depression- and anxiety-related risk of permanent disability and mortality in the working population in Germany: a cohort study. BMC Public Health 13(145):doi:10.1186/1471-2458-13-145.

Weissman MM, Wickramaratne P, Greenwald S, Hsu H, Ouellette R, Robins LN et al. 1992. The Changing Rate of Major Depression: Cross-National Comparisons. JAMA Intern Med 268(21):3098–3105.

Whiteford HA, Degenhardt L, Rehm J, Baxter AJ, Ferrari AJ, Erskine HE et al. 2013. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. Lancet 382(9): 1575-1586.

WHO Regional Office for Europe. 2011. Burden of disease from environmental noise: Quantification of healthy life years lost in Europe. Available: http://www.euro.who.int/__data/assets/pdf_file/0008/136466/e94888.pdf [accessed 23 February 2015].

Willich SN. 2005. Noise burden and the risk of myocardial infarction. Eur Heart J 27(3):276–282.

Wittchen H, Jacobi F, Rehm J, Gustavsson A, Svensson M, Jönsson B et al. 2011. The size and burden of mental disorders and other disorders of the brain in Europe 2010. Eur Neuropsychopharm 21(9):655–679.

World Health Organization (WHO). 2012. Depression: Fact sheet N°369. Available: http://www.who.int/mediacentre/factsheets/fs369/en/ [accessed 28 October 2014].

Yoshida T and Osada Y. 1997. Effects of Road Traffic Noise on Inhabitants of Tokyo. J Sound Vib 205(4):517–522.

Zou G. 2004. A modified Poisson regression approach to prospective studies with binary data. Am J Epidemiol 159(7):702–706.

**Table 1** Characteristics of the analyzed Heinz Nixdorf Recall study population (n=3,300), by 24-hour road traffic noise.

| | **$L_{den}$ >55 dB(A)** | **$L_{den}$ ≤55 dB(A)** |
|---|---|---|
| | N (%), mean ± SD, or median (Q1, Q3) | N (%), mean ± SD, or median (Q1, Q3) |
| **Baseline** | | |
| N(%) | 1,179(35.7) | 2,121(64.3) |
| Men | 610(51.7) | 1105(52.1) |
| Age (years) | 59.1±7.7 | 59.3±7.6 |
| Insomnia | 124(10.5) | 177(8.4) |
| N missing | 3 | 12 |
| Number of co-morbidities[a] | | |
| 0 | 440(37.3) | 830(39.1) |
| 1 | 374(31.7) | 687(32.4) |
| ≥2 | 365(31.0) | 604(28.5) |
| Reported (lifetime) prevalence of depression | 70(7.3) | 106(6.1) |
| N missing | 225 | 380 |
| Body mass index | 27.9±4.7 | 27.7±4.5 |
| N missing | 6 | 4 |
| Smoking | | |
| current | 288(24.4) | 423(19.9) |
| former | 419(35.5) | 778(36.7) |
| never | 472(40.0) | 920(43.4) |
| Distance to nearest major road (meters) | 532.4(220.0,1083.1) | 987.7(552.8,1620.7) |
| N missing | 0 | 5 |
| Unemployed in neighborhood (%) | 12.8±3.3 | 12.0±3.3 |
| Education[b] | | |
| ≤10 years | 111(9.4) | 165(7.8) |
| 11–13 years | 703(59.6) | 1135(53.5) |
| 14–17 years | 251(21.3) | 525(24.8) |
| ≥18 years | 114(9.7) | 295(13.9) |
| N missing | 0 | 1 |
| Household net income | | |
| Quartile 1 (low) | 300(27.0) | 420(21.1) |
| Quartile 2 | 257(23.1) | 473(23.8) |
| Quartile 3 | 290(26.1) | 502(25.2) |
| Quartile 4 (high) | 266(23.9) | 596(29.9) |

| | | |
|---|---|---|
| N missing | 66 | 130 |
| Economic activity | | |
| employed | 503(42.7) | 937(44.2) |
| inactive | 591(50.2) | 1,078(50.8) |
| unemployed | 84(7.1) | 106(5.0) |
| N missing | 1 | 0 |
| City of residence | | |
| Mülheim/R | 467(39.6) | 772(36.4) |
| Bochum | 334(28.3) | 654(30.8) |
| Essen | 378(32.1) | 695(32.8) |
| **Follow-up** | | |
| CES-D ≥17 and/or antidepressant medication | 127(10.8) | 175(8.3) |
| CES-D ≥17 | 89(7.6) | 116(5.5) |
| antidepressant medication | 56(4.8) | 67(3.2) |
| N missing[c] | 2 | 3 |
| Moved between baseline and follow-up | | |
| yes | 214(18.2) | 314(14.8) |
| no | 965(81.9) | 1,807(85.2) |

Q1 and Q3=quartile 1 (25$^{th}$ percentile) and quartile 3 (75$^{th}$ percentile)
[a] Of the following: myocardial infarction, heart failure, stroke, diabetes, emphysema, asthma, cancer, rheumatism, slipped disc, migraine
[b] Combining school and vocational training
[c] These participants were identified to have high depressive symptoms by CES-D and were therefore included.

**Table 2** Relative risks (with 95% confidence intervals) of high depressive symptoms at follow-up in study participants exposed to residential road traffic noise ($L_{den}$) >55 dB(A) compared with ≤55 dB(A).

| Model | N cases | N total[a] | RR (95% CI) |
|---|---|---|---|
| **Unadjusted** | | | |
| total | 302 | 3,300 | 1.31(1.05,1.62) |
| men | 101 | 1,715 | 1.46(1.00,2.13) |
| women | 201 | 1,585 | 1.23(0.95,1.60) |
| **Model 1**[a] | | | |
| total | 279 | 3,098 | 1.29(1.03,1.62) |
| men | 98 | 1,650 | 1.29(0.87,1.92) |
| women | 181 | 1,448 | 1.30(0.98,1.72) |
| **Model 2**[b] | | | |
| total | 278 | 3,089 | 1.28(1.02,1.61) |
| men | 98 | 1,644 | 1.28(0.85,1.94) |
| women | 180 | 1,445 | 1.28(0.97,1.69) |
| **Model 3**[c] | | | |
| total | 276 | 3,075 | 1.26(1.00,1.58) |
| men | 97 | 1,637 | 1.21(0.81,1.82) |
| women | 179 | 1,438 | 1.28(0.97,1.70) |

[a] Adjusted for age, sex (except in the sex-stratified analysis), education, income, economic activity, neighborhood-level socioeconomic status, traffic proximity
[b] Additionally adjusted for body mass index, smoking
[c] Additionally adjusted for co-morbidities, insomnia

**Table 3** Results of the sensitivity analyses, showing relative risks (with 95% confidence intervals) of high depressive symptoms at follow-up in study participants exposed to residential road traffic noise ($L_{den}$) >50 dB(A) compared with ≤50 dB(A).

| **Subgroup** | **N cases** | **N total**[a] | **RR (95% CI)**[b] |
|---|---|---|---|
| Education | | | |
| ≤13 years | 214 | 1,968 | 1.43(1.10,1.85) |
| >13 years | 65 | 1,130 | 0.92(0.56,1.53) |
| Moved during follow up | | | |
| yes | 61 | 502 | 1.17(0.72,1,88) |
| no | 218 | 2,596 | 1.33(1.02,1,72) |
| Insomnia | | | |
| yes | 55 | 281 | 1.62(1.01,2.59) |
| no | 222 | 2,803 | 1.21(0.94,1.57) |
| City of residence | | | |
| Mülheim/R | 99 | 1,162 | 1.21(0.83,1,76) |
| Bochum | 89 | 927 | 1.51(1.00,2.29) |
| Essen | 91 | 1,009 | 1.16(0.77,1.74) |
| Excluded lifetime prevalence of depression at baseline[c] | 189 | 2,382 | 1.34(1.01,1.76) |
| Noise cutoff $L_{den}$ >65 dB(A) | 279 | 3,098 | 1.07(0.77,1.49) |
| CES-D ≥17 only to define outcome | 227 | 3,469 | 1.24(0.96,1.61) |
| Antidepressant medication only to define outcome | 144 | 3,467 | 1.28(0.92,1.80) |

[a] max. total N in model 1=3,098, numbers differing from those in table 1 reflect missing covariate data (in model 1)
[b] Adjusted for age, sex, education (not in the education-stratified analysis), income, economic activity, neighborhood-level socioeconomic status and traffic proximity (model 1); no substantial differences were in unadjusted and model 2 and 3 results (data not shown)
[c] Excluded 176 who reported having/having ever had depression and 605 with missing data

## FIGURE LEGENDS

**Figure 1** Flow chart of study participants in the Heinz Nixdorf Recall study. Missing information=missing information on depressive symptoms (CES-D, antidepressant medication use); prevalent depressive symptoms=CES-D ≥17 and/or antidepressant medication use.

**Figure 2** Relative risks and 95% confidence intervals of high depressive symptoms at follow-up in association with exposure to different categories of 24-hour noise compared with the lowest noise category [≤55 dB(A); n=1,986], adjusted for baseline age, sex, education, income, economic activity, neighborhood-level socioeconomic status and traffic proximity (model 1).

Figure 1.

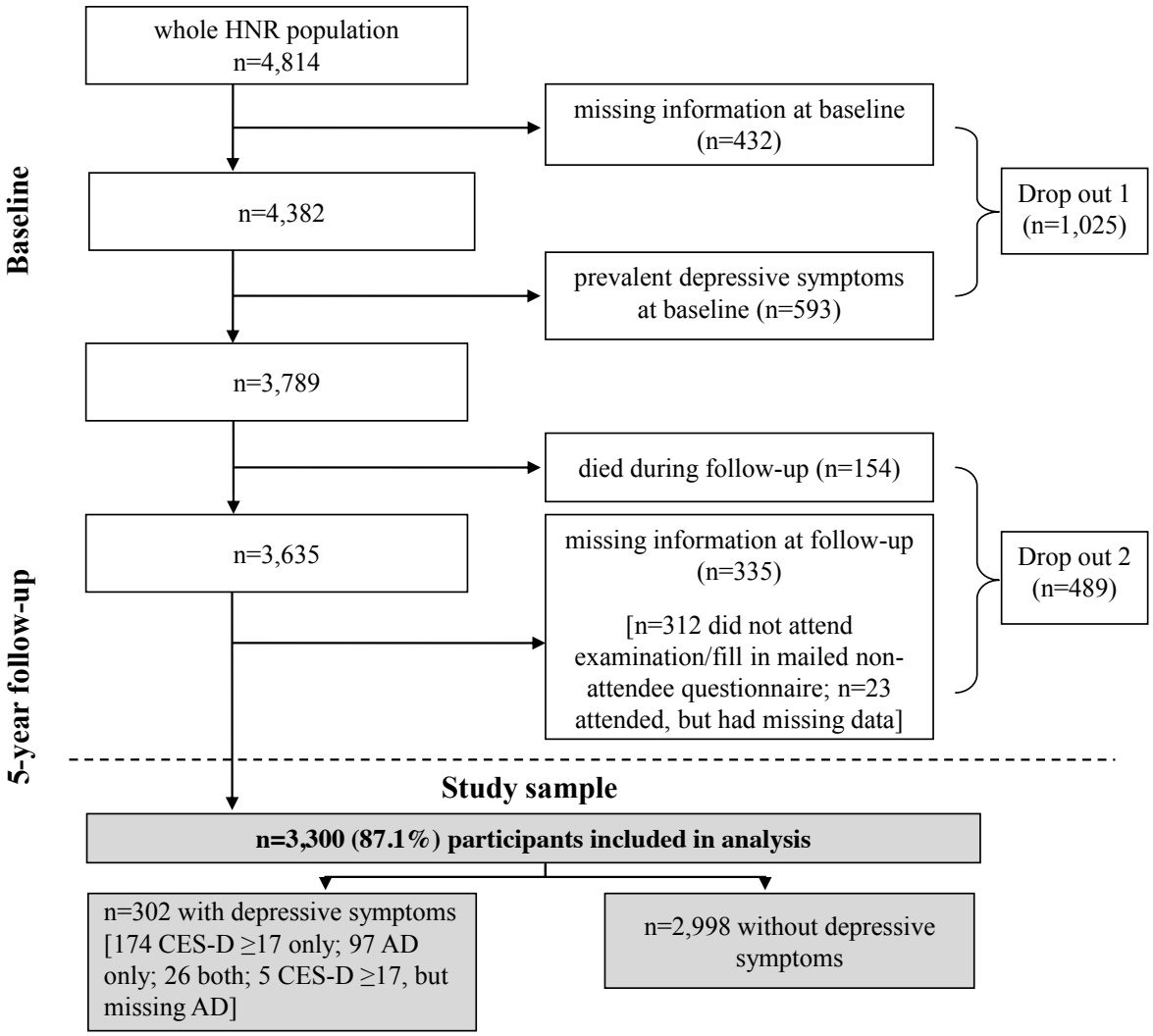
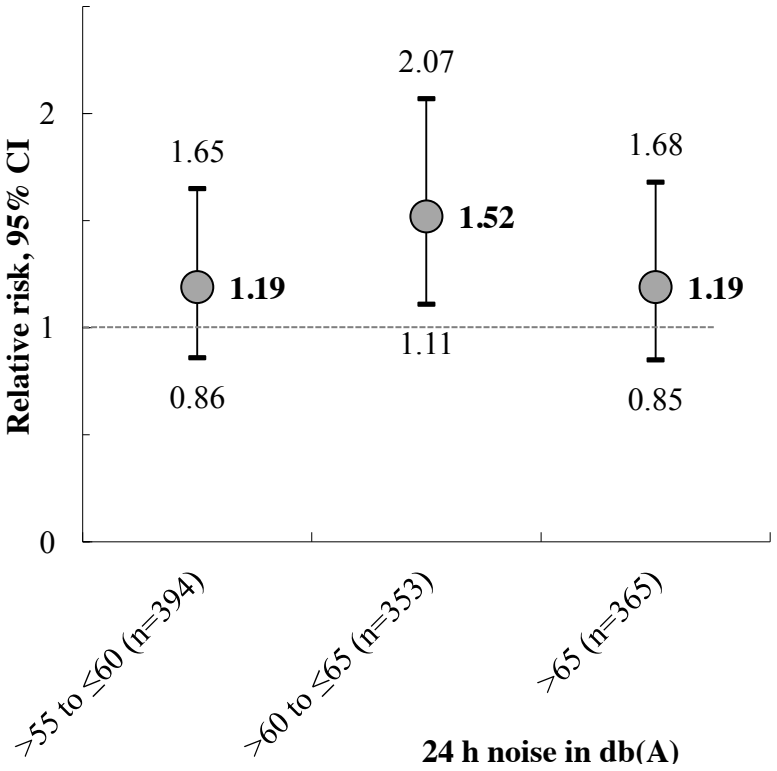

whole HNR population
n=4,814
missing information at baseline (n=432)
Drop out 1 (n=1,025)
Baseline
n=4,382
prevalent depressive symptoms at baseline (n=593)
n=3,789
died during follow-up (n=154)
n=3,635
missing information at follow-up (n=335)
[n=312 did not attend examination/fill in mailed non-attendee questionnaire; n=23 attended, but had missing data]
Drop out 2 (n=489)
5-year follow-up
Study sample
n=3,300 (87.1%) participants included in analysis
n=302 with depressive symptoms [174 CES-D ≥17 only; 97 AD only; 26 both; 5 CES-D ≥17, but missing AD]
n=2,998 without depressive symptoms

Figure 2.


Relative risk, 95% CI
2
1
0
1.65
1.19
0.86
2.07
1.52
1.11
1.68
1.19
0.85
>55 to ≤60 (n=394)
>60 to ≤65 (n=353)
>65 (n=365)
24 h noise in db(A)