Sick of Noise: the Health Effects of Loud Neighbours and Urban Din

Diana Weinhold *†

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Abstract Residential neighbour noise is ubiquitous and a major source of stress, but its effects are relatively under-researched and often controlled under nuisance, rather than environmental health, regulations. We analyze the health effects of residential noise annoyance using a high quality longitudinal survey of over 5000 adults in the Netherlands between 2008 and 2013 that includes a broad variety of socio-economic, demographic, and health information. To address identification concerns about selection we also collect data on home moving and construct a psychometric measure of sensitivity to stress, allowing us to examine to what extent moving and sensitivity are associated with noise annoyance in both the cross section and the time series. Additional robustness tests including matching regression and panel fixed effects estimation that condition only on initially healthy respondents to mitigate concerns of reverse-causality. Overall we find surprisingly strong and robust effects of residential noise annoyance on a variety of health outcomes, including cardio-vascular symptoms, auto-immune conditions associated with joint and bone disease, headache, and fatigue. The relationship is not only statistically significant but also of a meaningful magnitude. Controlling for sleep disruption only provides a partial explanation, suggesting additional physiological mechanisms. Finally, we find neighbour noise to be relatively more damaging to health than street noise.

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†Department of International Development, London School of Economics, London UK. Email: d.weinhold@lse.ac.uk.

1 Introduction

As many an urban dweller can attest, residential noise is a particularly vexing aspect of city life. In the seven months between the 10th of January and the 3rd of August, 2014, New Yorkers logged a total of 79,971 noise complaints to the city's '311' citizen complaint number (about 8% of all complaints). Of these, 37% were for loud music/parties, 13% were for loud talking, and 15% were for engine/automobile related noise. News stories about urban noise generate an eager readership; a search of the *New York Times* archive turns up 18,700 results for 'neighbor noise,' (about as many as for 'African poverty,' at 19,200, and apparently about a quarter as interesting as 'Middle East peace,' at 78,400). A recent survey in Denmark found that almost a quarter of respondents had experienced conflicts with neighbours during the previous five years, and of those fully half were related to noise (Hansen, 2016). Ethnographic research in Santiago Chile reveals how city dwellers perceive neighbour noise as personal intrusions, and in turn how some use noise themselves to stake out their territory (Ureta, 2007).

For such a ubiquitous urban irritant, however, there is surprisingly little policy action aimed at reducing neighbour noise (Hammer et al., forthcoming, 2017). This may be partially due to the intrinsic difficulties associated with regulating private social behaviour, but a scarcity of evidence on the effects of neighbour noise is likely also a contributing factor. Despite the lack of direct evidence, however, there is growing body of literature on the health effects of stress, and neighbour noise is arguably one of the most common sources of urban stress.

Segerstrom and Miller (2004) conduct a meta-analysis of studies on the relationshiop between stress and and the immune system, noting that:

> The most chronic stressors were associated with the most global immunosuppression, as they were associated with reliable decreases in almost all functional immune measures examined. Increasing stressor duration, therefore, resulted in a shift from potentially adaptive changes to potentially detrimental changes, initially in cellular immunity and then in immune function more broadly.

Just as importantly, the authors note that individual differences in subjective experience are critically important; "people's cardiovascular and neuroendocrine responses to stressful experience are dependent on their appraisals of the situation and the presence of intrusive thoughts about it." (Segerstrom and Miller, 2004)

Nevertheless, where urban din can be objectively measured, such as for traffic and airport related noise, studies have indeed found links to both lowered subjective well-being (Praag and Baarsma, 2005) and health effects such as stress, cardio-

vascular problems, stroke, and sleep disruption (Sørensen et al., 2011; Babisch, 2011, 2014; Evans et al., 1998, 2001; Ising and Braun, 2000). Indeed, in their review of the evidence, Hammer et al. (2014) estimate that residential noise puts tens of millions of Americans at risk of heart disease and other noise-related health effects.

Where noise is less easily measured, however, there has been much less research. Unlike traffic or airport noise, loud neighbours are often unpredictable and not generally *ex ante* observable. Thus the presence of loud neighbours may not manifest itself in the form of property values or other economic tangibles, making it difficult to indirectly impute a monetary valuation ((Weinhold, 2013) makes one desperate attempt). Furthermore, until recently there was little theoretical reason to distinguish between noise pollution from loud neighbours and that from other urban sources. The relative lack of evidence has led most national noise regulation to focus on (easily observed) airport, construction, traffic and work-related exposure to noise (Hammer et al., 2014; Nelson et al., 2005). Enforcement of neighbour noise laws, where they exist, often falls under the purview of local nuisance laws, rather than under environmental health authorities, and is largely left to local governments with varying degrees of prioritization and effectiveness (Hammer et al., forthcoming, 2017).

Nevertheless there are good theoretical reasons to suspect that neighbour noise could additionally affect health through channels not captured in studies of traffic and airports; noise from loud neighbours is quite different than that from streets in that it is less predictable and often has a very high informational content, even if the decibel level is similar or even lower (Niemann et al., 2006). Thus the potential biological and psychological mechanisms that link each type of noise to health are distinct.

This paper contributes to a small but growing growing body of evidence on the effects of residential noise on health by analyzing the effects of self-reported neighbour and street noise annoyance in a high quality, large longitudinal survey of over 5000 adults in the Netherlands between 2008 and 2013. While a quasi-experimental research design would be preferable, it is difficult to imagine how such a framework could be operationalized, so a good first step is to explore existing observational data while remaining mindful of the methodological limitations to causal inference. The Dutch survey in particular allows us to control for a much broader variety of socio-economic, demographic and health variables than the single previous quantitative analysis (Niemann and Maschke, 2004) on the topic. Furthermore, the breadth and longitudinal nature of the data allow this to be the first study, to the author's knowledge, to quantitatively examine the issue of the health effects of neighbour noise while taking identification concerns seriously. In particular, endogenous sensitivity correlated with health and noise annoyance, and/or health- and noise sensitivity- moderated selection in moving house could poten-

tially both constitute sources of endogeneity bias in the 'naive' baseline model specification. Thus we collect data on both indiviudal psychometric measures of 'sensitivity to stress' and on home moving activity that allow us to both directly control for these variables in the analysis, as well as further explore different dimensions of these challenges to causal inference through direct modelling of home moving and subjective satisfaction. In the event, the coefficients on neighbour noise remain stable and robust to inclusion of these additional variables and our exploratory analysis of home-moving, sensitivity, and subjective well-being produces little evidence that unobservable selection is likely to be driving observed health effects of noise. Finally, the primary benchmark results we find with regression analyses remains robust when we estimate average treatment effects using propensity score matching and panel fixed effects models that mitigate the likelihood of reverse causality by conditioning only on initially healthy respondents. Thus although we cannot entirely rule out the possibility that (unobservable) sensitivity- and/or selection- related endogeneity could have an effect, our evidence suggests these are unlikely to exert a first-order impact on the observed relationship.

We find that neighbour noise displays a distinct pattern of association with health outcomes; for diseases unrelated to stress we find no relationship, but for those health outcomes that have been theoretically and empirically linked to stress we find a strong and consistent relationship, including with cardio-vascular disease, auto-immune diseases such as arthritis and bone disorders, as well as with fatigue and headache. Not only are these effects statistically significant, but they are of a meaningful magnitude, with neighbour noise annoyance having approximately the same effect on overall self-reported health as a history of smoking. We find little evidence of selection via moving house, and the estimated effects remain stable and robust to the inclusion of our psychometric measure of sensitivity. We then control for sleep disruption, finding (as expected) that this is a primary mechanism linking noise with fatigue and headache. Nevertheless the relationship between neighbour noise with auto-immune disease, and to some extent cardio-vascular disease, persists, suggesting that sleep disruption is only one mechanism through which the noise-health relationship may operate. Furthermore, in striking contrast to the relatively greater attention that both policymakers and scholars pay to more objectively measured sources of noise, we consistently find neighbour noise to be more harmful to health than street noise. Overall our analysis strongly suggests that everyday urban residential noise annoyance, especially from noisy neighbours, could contribute to a surprising variety of health disorders.

The paper proceeds as follows: in section 2 we briefly review the existing literature on the health effects of noise; in section 3 we describe the data and estimating method; and in section 4 we discuss the benchmark cross sectional results. Section 5 presents the first set of robustness exercises, including estimating propensity score matching that avoids some of the parametric linearity assumptions of regression, and fixed effects panel models that condition only on initially healthy

respondents to mitigate potential reverse causality. Section 6 extends the robustness tests to further explore potential problems of identification related to selection on sensitivity and moving. Section 7 summarizes the findings and concludes with a discussion of the implications both for urban and public health policy and for future research. Regression results are presented in the Tables Appendix.

2 Noise and Health

Residential noise is a common source of stress and studies have shown it to be associated with lower overall life satisfaction (Weinhold, 2013; Praag and Baarsma, 2005). However less is known about how urban din could affect physical health. Early research on the relationship focused on the effects of work-related exposure to noise on hearing (Olishijski and Harford, 1975; Schori, 1976; Nelson et al., 2005). This literature generally concluded that exposures below about 80 dB(A) (approx. the noise of a garbage disposal) are safe, with these consensus thresholds reflected in workplace noise standards adopted in many countries (e.g. ISO 1999).

More recently there has been growing evidence that that the physiological effects of noise potentially extend to many more dimensions of health, and at much lower dB(A) levels of exposure, than had previously been recognized. For example, Sørensen et al. (2011) explores the link between road traffic noise and stroke, while Sørensen et al. (2013) analyze its effect on diabetes. Babisch (2014) provides a meta-analysis of 14 recent cross-sectional and case control studies of traffic noise and coronary heart disease, finding a statistically significant 8% increase in risk for every additional increase of 10 dB(A) traffic noise (within the range of 52-77 dB(A)). Evans et al. (1998, 2001) and Ising and Braun (2000) find significant increases in stress hormones released during sleep in both children and adults exposed to moderate street noise (over 60 db(A)). More broadly, a number of medical studies link mental stress to immunological and cardiovascular reactions in humans (see Niemann et al. (2006) for a good survey) and an expanding literature demonstrates the negative effects of noise annoyance on reported well-being (Weinhold, 2013). WHO (2011) synthesized the existing evidence in order to estimate the years of healthy life (DALYs) lost to noise in Europe; the peer-reviewed chapters (each authored by experts in that field) estimate 61,000 years lost due to ischemic heart disease; 45,000 years lost to cognitive impairment in children; 903,000 DALYs lost due to sleep disturbance; 22,000 years lost to tinnitus; and 654,000 years lost due to noise induced annoyance.

When it comes to noise caused by neighbours the relationship to health may be even more complex. Leventhall (2004) points out that low frequency noise can be a particular problem for people in their homes, with ”learned aversion” easily leading to annoyance and stress at decibel levels that may not fall within existing

regulations. Furthermore, as Niemann et al. (2006) explain,

> Usually, neighbourhood noises are sounds with high information content such as language, music or also the noise of footsteps. It is in the nature of humans to have their attention drawn to such informative sounds, even if the sound level is relatively low. The annoyance potential of neighbourhood noise is therefore relatively high also at low noise levels and is heightened by the hearer's knowledge of the sound producer and other things causing the noise. (p. 64)

Thus the established relationship between noise, sleep-disruption induced endocrine abnormalities, and stress hormone reactions all provide plausible biological mechanisms linking noise disturbances, even at relatively low decibel levels, to a host of health problems, including cardiovascular, immunological, and even blood sugar regulation issues (Babisch, 2003; Ising and Kruppa, 2004; Hammer et al., 2014). However, as these mechanisms may operate very differently from the kinds of physiological effects caused by specific levels of artificial noise observed in laboratory settings, it is methodologically challenging to gauge the prevalence, if any, of the effects of real-life everyday residential noise annoyance on health. Indeed, to date the only large epidemiological study on the health effects of chronic annoyance by neighborhood noise was carried out in 2003-2004 by Niemann and Maschke (2004)(with a follow up in Niemann et al. (2006)) for the World Health Organization (WHO) as part of its Large Analysis and Review of European Housing and Health Status (LARES). The authors examined the cross-sectional relationship between noise exposure and health outcomes in a sample of about 8000 adults and children across Europe, finding elevated risks of exposure to neighborhood noise associated not only with psychological depression, but also in the cardiovascular, respiratory and musculoskeletal systems, with the particular risks displaying a strong dose-response effect that varied significantly between children, adults and the elderly (but independent of socio-economic and housing conditions).

This paper extends this small existing literature on neighbour noise and health by not only including a broader set of environmental and socio-economic control variables in the analysis, but also by addressing identification concerns about selection by exploring and explicitly controlling for both observed home-moving and individual sensitivity to stress. In addition we conduct a number of robustness tests, including estimating average treatment effects using propensity score matching, and estimating panel fixed effects models that condition only on initially healthy respondents in order to mitigate the possibility of reverse causality.

3 Data and Method

3.1 Data

Data for the analysis come from the Longitudinal Internet Studies for the Social Sciences (LISS) panel administered by CentERdata (Tilburg University, The Netherlands). The LISS CentER data is based on an internet-based longitudinal survey from 2007-2013 of over 8000 individuals that was designed for "scientific, policy or socially relevant research." The quality and the coverage of the sample was of prime concern; participants were identified using a true probability sample drawn from the Dutch population registers by Statistics Netherlands and recruitment was by repeated contact via phone and/or in person, resulting in an enrollment rate of 48% of the total initial sample, including households with no internet connection as a computer and connection were provided as needed. Scherpenzeel (2009) conducts a detailed evaluation of the sampling method and resulting representativeness of the LISS panel, finding that the LISS sample compares favorably to high-standard traditional surveys (for more detail on the LISS panel, see Scherpenzeel and Das (2010) or visit www.lissdata.nl).

There are only 65 observations in the LISS data on children born before 1990 so we omit this category and focus only on those respondents over 17 years of age in 2007 when the survey started, ending up with a total sample size of 5440 (though not all respondents answer all questions, or respond in all years, so sample size varies by regression). Although the sampling is done at the individual level, some individuals may reside in the same household so we cluster all standard errors at the household level (in practice this is a very minor adjustment). The LISS is an ongoing survey with multiple waves of question 'modules' sent to participants throughout the year. The primary modules used for this analysis was the Housing module, collected in June and July, the Health module, collected in November and December, and the Personality and Income modules, collected in May-June and June-July, respectively. In the Health module of the LISS respondents were asked both general and specific questions about their health. *Health Level* is the self-reported overall level of health, from 1 (poor) to 5 (excellent), from the question "How would you describe your health, generally speaking?" Respondents were also asked about many more objective, factual health problems and diseases by having them select from a list of possibilities to address such questions as, "Do you regularly suffer from:"; "Are you currently taking medicine at least once a week for:"; and "Has a physician told you this last year that you suffer from the following diseases/problems?". Respondents were coded with a specific health problem if they indicated in the affirmative with respect to that health problem to any of these questions. Specifically, health problems were coded as cardiovascular; joints & bones (including arthritis and skeletal problems); lung disease (including bronchitis), asthma, diabetes, blood pressure, cholesterol, fatigue and headache

(including migraine). In addition respondents noted whether they suffered from sleep disturbance (from any cause). Unfortunately the Health module was not run in 2014, so our data extends through 2013.

The main explanatory variables of interest are binary responses from the Housing module to the question "Are you ever confronted with the problems listed below in your home environment?" *Neighbor Noise* takes the value 1 if respondents indicated 'noise annoyance caused by neighbors', and 0 otherwise. *Street Noise* takes the value 1 if respondents indicated 'noise annoyance caused by factories, traffic or other street sounds,' and 0 otherwise. Residential noise is ubiquitous; almost 20% of respondents report suffering from street sounds and fully 33% report loud neighbours. Although a reliance on self-reporting may seem at first glance to be a handicap, in may ways the subjective nature of the data is also a strength. In particular, the theoretical biological mechanisms linking residential noise to health outcomes operate primarily through sleep disturbance and stress (including unconscious stress) that are related not only to the decibel level of noise but also to its timing, frequency, and information content. Biological and psychological triggers could be set off by different degrees of these various characteristics of noise for different people. In the presence of such heterogeneous responses to any given (homogenous) noise, self-reported subjective noise annoyance in effect subsumes the underlying heterogeneity within the responses themselves. In other words, unobserved sensitivity to noise is already captured in the reported noise measure since a person needs to be exposed to the noise and, at the same time, sensitive enough to notice the noise to report being disturbed by it. The reported noise measure should thus be considered as an interaction of "being exposed to noise" and "sensitive to noise."

As we discuss below in section 3.2 and in depth in section 6, the possibilty of unobserved heterogenous sensitivity to noise, either in the cross section or the time series, creates a number of potential empirical challenges given the baked-in nature of sensitivity in our self-reported noise variables. Thus a very useful feature of the LISS survey is that it also collects psychometric data in the Personality module, allowing us to extract information on individual susceptibility to stress and irritability, a key control variable throughout the analysis. In particular, the survey asked respondents to rank from 1 (very inaccurate) to 5 (very accurate) whether statements about personality characteristics describes them. Of these we focus on: (a) 'Gets stressed out easily'; (b) 'Am easily disturbed'; and (c) 'Gets irritated easily'. We create an index of sensitivity, *Stress Index*, as the average of these three responses and use this as a robustness control in the analysis. In addition we construct a measure of both overall 'happiness' or life satisfaction as the average across responses (on a scale from 0 to 10) to two questions from the Personality module; "On the whole, how happy would you say you are?" and "How satisfied are you with the life you lead at the moment?", and one question from the Income module, "Can you indicate, on a scale from 0 to 10, to what degree you

consider yourself happy?". The composite *Life Satisfaction* variable maximizes sample size, with a Cronbach Alpha of 0.9132. From the Housing module we also collection information on satisfaction with housing (*Dwelling Satisfaction*) as the response to the question, "Please indicate on a scale from 0 to 10 how satisfied you are with the dwelling that you currently inhabit." Finally, in order to test whether (sensitivity-induced) moving home could be driving any observed correlation we obtained additional data from CentERdata on whether a respondent moves home in any given year (*Moved*) and whether a respondent has moved at any time during the survey period (*Moved Ever*).

In addition to the key variables of interest the LISS also provides a large number of socio-economic, demographic, housing and neighbourhood characteristics that can be controlled for in the analysis. These include information on gender, age, whether the respondent has ever smoked, whether they consume more than one alcoholic drink per day, body mass index (BMI), education level (from primary to university level, 1-4), marital status, labor market status, number of hours worked, monthly household income, number of children in the household, and whether the respondent is religious. There is no location information in the LISS survey; we do not know *where* respondents live and therefore cannot control for neighbourhood fixed effects. However the survey does include an extensive set of variables describing the respondents' neighborhood and dwelling characteristics, including whether the neighborhood is very urban, moderately urban (control), or rural, whether the respondent has experienced vandalism or crime at home, and whether the respondent finds their dwelling to be too small, too dark, too damp, too cold, has a leaking roof, or has rotten window frames or floors. To control for poor air quality associated with being near a busy road or factory, *Air Quality* takes the value 1 if respondents indicate their dwelling suffers from 'stench, dust or dirt, caused by traffic or industry,' and 0 otherwise. Finally, additional information provided by CENTeRData allow us to construct an annual indicator of whether a respondent has moved residence. Table 1 presents summary statistics for all variables used in the analysis.

3.2 Estimation strategy

Our benchmark estimation strategy consists of 'between' (time-averaged) cross sectional estimates of the relationship between neighbour noise and health outcomes. Most of the health variables of interest are binary; respondents either have the condition in a particular year, or they do not. To be consistent with the health literature and facilitate comparability with others' results we model the probability that an individual will develop a health condition as a logistic function and estimate the model using maximum likelihood logistic regression. The conclusions we draw are robust to this choice of functional form - linear probability methods

(LPM) give similar results, but the LPM coefficient estimates are not easy to compare to the existing literature. Thus the model takes the form:

$$(1) \qquad logit(p_i) = log(p_i/(1-p_i)) = \beta_0 + \beta_1 X_{1i} + ... + \beta_k X_{ki} + u_i$$

where p is the probability of disease, X_{1i}- X_{ki} are the *k* possible explanatory and control variables, and β_0- β_k and the *k* coefficients. As explained above standard errors are clustered at the household level. The monotonic transformation $OR = e^{\hat{\beta}_k}$ yields the odds ratio (OR) of variable X_i which gives the ratio of the likelihood of disease with and without the factor X_i (if *X* is dichotomous), or for a one-unit increase in a factor X_i (if *X* is continuous). Odds ratios thus range from 0 to infinity, with values below 1 indicating that the factor x_i has *lowered* the odds of disease, and values above 1 indicating that it has *increased* it. For example, in regression (1) of table 3a we report that the odds ratio of exposure to neighbour noise for cardio-vascular disease is 1.38, meaning that, all else equal, the odds of cardio-vascular disease for those with irritatingly loud neighbours is 1.38 times the odds for those with more polite folks nearby. In the same regression we find an odds ratio for body mass index (BMI) of 1.05, meaning that the odds of cardio-vascular disease will increase by 5% for every one unit increase in BMI. On the other hand, higher household income, with a coefficient of less than 1 (0.70), is associated with a *lower* risk of cardiovascular disease.

Initially we estimate equation (1) using time-averaged RHS variables across the panel, so that binary variables can take values between 0 and 1 depending on the proportion of years respondents indicated the variable was present. This measure can loosely be interpreted an indication of the 'intensity' of treatment; survey respondents who report noise in a greater fraction of the years they participated will be considered to have been subjected to relatively more noise than those who report fewer incidences. In our robustness exercises presented in section 5 we additionally report the cross sectional results setting all binary values to 1 if their time-averaged value is greater than 0.

As discussed above, one reason the literature has focused on road and airport noise is that it is easier to obtain objective measures for these compared to, for example, intermittently loud neighbours. This study, on the other hand, uses subjective noise data and thus several problems for identification could arise from selection issues. First, unobserved individual characteristics associated with sensitivity (such as being easily irritated, stressed, or disturbed) could be a driver of both self-reported noise annoyance as well as health outcomes. Second, another potential threat to inference in our baseline estimates arises from selection related to home-movers. For example, in theory people may prefer to move away from noisy neighbours, but if less healthy people find it more difficult to move house, then we could observe a negative association between noisy neighbours and health outcomes. On the other hand, if more sensitive people successfully self-select away from noisy homes and also suffer (or report) more health problems, this could in-

duce a bias towards finding no effect. Finally, the analysis could suffer from reverse causality if illness leads to noise sensitivity.

We address these identification concerns using multiple approaches. In the baseline estimates we attempt to mitigate potential selection concerns by directly controlling for our psychometric measure of stress, *Stress Index* and comparing estimates with and without this variable. Then, in section 6, we then further investigate the degree to which selection concerns could be driving the results by directly modelling the cross sectional and time series relationships between noise, stress, and subjective well-being.

When we consider home moving, our primary response is to argue that, especially in Europe, there are a number of reasons why people may not be able to self-select away from noisy neighbours so easily. First, unlike street noise, neighbour noise is not an *ex-ante* observable characteristic of housing stock; thus any move might result in a metaphorical leap from the pot into the fire. Moreover, in many areas moving is difficult and costs are very high; for example in the UK a 'stamp duty' of up to 15% of the purchase price of homes reduces property transactions by between 8-20% (*Property Taxes: Welcome to New London*, 2015). Moving costs in the Netherlands are especially high; a UNHSP report found 33 of 35 European countries surveyed employ some kind of property transfer tax, and among these the transfer tax adopted in the Netherlands was rated 'High' (Un-Habitat, 2013). Moving rental homes is also difficult and expensive in the Netherlands; most properties are offered unfitted and unfurnished - they lack not only furniture, but also basics like carpets, light fittings, and major appliances. Furthermore, almost three quarters of Dutch rental properties are social housing that are highly rationed and can have waiting lists that approach 7 years (*Government of the Netherlands website*, (Accessed: February 25, 2016). Indeed, Praag and Baarsma (2005) investigated the impact of airplane noise on housing prices in Amsterdam and found that moving was so difficult that the disamenity of airplane noise was absorbed not in housing prices but exclusively as a 'residual' in the life satisfaction of homeowners. Indeed, among respondents in our own LISS data just under 23% moved at least once in the six years between 2008-2013, significantly fewer than the typical 35%-45% of the population that move homes in any five year period in the U.S. (Ihrke and Faber, 2012).

Nevertheless, while there are good reasons to suspect that moving home is sufficiently difficult in the Netherlands to mitigate this type of selection, in the end this is an empirical question, so our more direct strategy to address this potential concern is to control for observable home-moving in the analysis. Of course, this will not capture home-moving that occured before the survey begain, so we supplement this with additional analyses in section 6 that directly models the determinants of home-moving and explores the degree to which either the cross sectional or time series variation in stress and noise play a role.

The inclusion of our psychometric measure of sensitivity, *Stress Index* should also mitigate potential reverse causality concerns, but we could still face a problem if illness leads to unobserved noise sensitivity that is uncorrelated with the observed sensitivity captured by *Stress Index* and other controls. While the observational nature of the data preclude us from eliminating this (arguably small) possibility entirely, we again employ several strategies to investigate the likelihood and to build confidence in the results. In particular, the timing of LISS survey modules ensures psychological separation of the noise and health questions (thus avoiding priming biases) as noise questions are asked in the spring while health questions follow in the fall. We also examine a wide variety of health outcomes, so to the extent that we observe a theoretically consistent (e.g. connected to stress) pattern of association between noise exposure with some illnesses, but not with others, this heterogeneity of effects provides further indirect support of a causal path from noise exposure to health rather than the reverse. Finally, as part of our robustness exercises in section 5.4 we estimate panel fixed effects analyses in which we condition only on initially healthy respondents to further reduce the likelihood of reverse causality.

4 Results

4.1 Cross sectional analysis

Table 2 reports the cross sectional OLS results of the relationship between residential noise annoyance and self-reported health level (from 1 to 5, worst to best) controlling for our standard set of socio-economic, dwelling, and demographic variables. In column (1) we find both neighbour and street noise have a negative and statistically significant correlation with health, with neighbour noise having almost twice the effect of street noise. Indeed, computing standardized betas (not reported) we find that the effect of exposure to neighbour noise on self-reported health is of approximately the same order of magnitude as ever having smoked. Consistent with existing studies we find BMI and age to be major factors in declining health level, while household income is strongly and significantly positive. In column (2) we additionally control for air quality and the effect of street noise loses statistical significance, a result consistent with Lipfert et al. (2006) who show that road noise is highly correlated with air quality.

As discussed above, an especially sensitive personality might both report more noise annoyance and lower health. To test this hypothesis in table 2 column (3) we introduce our psychometric control *Stress Index* and the coefficient on *Neighbour Noise* falls moderately but remains highly statistically significant. While we cannot rule out that some other (orthogonal) unobservable characteristic could still create a source of endogeneity, the fact that the inclusion of *Stress Index* has such a

moderate effect on the coefficient of *Neighbour Noise* suggests that some excessive sensitivity, or a related personality condition, is unlikely to be driving the correlation. Finally in column (4) we control for whether the respondent has moved home during the sample period; this variable is not significant and does not much affect the coefficients on noise.

Finally, as discussed above, one of the mechanisms through which residential noise could contribute to health outcomes is through sleep disruption. Thus in Table 2 column (5) we control for this possibility (though poor health could also lead to sleep disruption, so this relationship could be endogenous). We find *Sleep Disruption* to have a very large and highly statistically significantly negative effect on self-reported health, and while its inclusion leads to a small reduction in the magnitude the estimated effect of *Neighbour Noise*, the latter remains large and highly statistically significant. Overall we find residential noise to have a meaningfully large relationship with self-reported health, with neighbour noise relatively more important than street noise. While some of this impact may operate through sleep disruption, the evidence is consistent with more direct channels as well.

Despite the use of *Stress Index* as a control there may still be concern about the use of self-reported noise annoyance to explain self-reported health. Thus in the remainder of the paper we turn to more objectively (though still self-reported) measures of health from respondents answers to questions about specific health problems, doctor's advice, and medications they are taking.

Tables 3a, 3b, and 3c present the odds ratios (OR) from logistic regressions analyzing the association of residential noise sources with various kinds of disease categories. We control for *Moved Ever* in all specifications to address the possibility that respondents who move homes may be both differentially affected by noise and be either healthier or sicker on average (though as discussed in section 6.2 we find no evidence of selection of this sort). In addition all regressions include our full set of socio-economic and demographic controls (not reported for parsimony, but available upon request) and for each category we report results both with and without *Stress Index* as a control. *Stress Index* is highly statistically significant across a wide variety of health conditions, but we find it to have only a relatively minor effect on the noise coefficients (with a slightly larger impact on the estimated neighbour noise coefficients in the regressions for fatigue and headache). Although our psychometric control may not perfectly capture some unobservable sensitivity that could drive an endogeneity bias between noise and health, we find it unlikely that the observable and unobservable components would be so uncorrelated. Thus we conclude from this exercise that it is unlikely that the correlation between noise annoyance and health outcomes is being spuriously driven by excessive sensitivity, but nevertheless explore this possibility in more depth below in section 6.1.

More specifically, in table 3a column (2) we find neighbour noise (but not street

noise) to be significantly associated with both Cardiovascular disease (columns 1 and 2) and with odd-ratios that, at 1.3 (conservatively, from column 2), are almost identical to the results obtained by Niemann and Maschke (2004) for 'moderate annoyance' of neighbourhood noise in adults for 'cardio-vascular symptoms' (OR=1.3) and slightly less than those obtained for 'strong annoyance' (OR=1.6). Similarly we find effects of neighbour noise on Lung disease (table 3b column 4 (OR=1.35) that are comparable to those found by Niemann and Maschke (2004) for 'bronchitis' (OR=1.0 (not statistically significant) for 'moderately annoyed' and 1.9 for 'strongly annoyed'). In table 3b column (6) and table 3c column(6) we find significant positive effects of neighbour noise on 'Joints & Bones,' and Headache, respectively, with odds ratios of 1.54 and 1.28 that again fall in between the estimates obtained by Niemann and Maschke for moderate and strong annoyance of neighbourhood noise on 'arthritis symptoms' (OR=1.3 and 2.3 respectively), and 'migraine' (OR= 1.2 and 1.8). As there is no analogue in the Niemann and Maschke (2004) results for 'fatigue' we cannot compare, but the significant OR of 1.25 that we obtain would not come as any surprise to anyone who has dealt with loud neighbour problems...

The estimated Odds Ratios for neighbour noise effects on cholesterol, blood pressure, and asthma are greater than or close to 1 but not statistically significant, which is again mostly consistent with results from Niemann and Maschke (2004)(although their estimate for blood pressure ('hypertension') is small but significant). Also consistent with the WHO-LARES study we find effects of noise on diabetes and stroke (not reported, available upon request) to be statistically insignificant and less than 1.

Thus overall our results, using different data and superior controls, are remarkably similar to those obtained by Niemann and Maschke (2004). We find statistically significant relationships between neighbour noise annoyance and cardiovascular symptoms, lung disease, joint and bone problems, fatigue, and headache. Our point estimates of the associated odds ratios fall just between the Niemann and Maschke's OR estimates for 'moderate' and 'strong' annoyance to neighbourhood noise, and both our studies fail to find statistically significant effects on diabetes incidence, stroke (not reported), or asthma.

4.2 Sleep disturbance

As discussed above, one of the likely mechanisms through which noise can impact health is through sleep disturbance (Ising and Braun (2000); Niemann et al. (2006)). In table 4 we explore whether sleep disturbance could explain the health outcomes reported in tables 3a, 3b and 3c. Column (1) of table 4 shows that, consistent with the literature (and experience), neighbour noise and street noise annoyance are both large and highly significant predictors of sleep disturbance. Although

we interpret the results with caution due to the possibility of reverse causality between health and sleep, columns (2)-(11) explore whether controlling for sleep disturbance (from any cause) can eliminate the correlation between residential noise and health outcomes. Consistent with the literature we find that sleep disturbance displays a large and significant correlation with many poor health outcomes, being associated with a doubling or trebling of the odds for just about everything.

As expected, controlling for sleep disturbance has a large impact on the results, lending strong suggestive support to the hypothesis that a major channel through which neighbour noise impacts health is through sleep disruption. In particular once we have controlled for sleep disturbance the statistical significance of the relationship between residential noise and cardiovascular, lung, and headache problems fall slightly (with p= .09, .05, and .058 respectively) to levels just above conventional cut-offs. Autoimmune disorders of the joints and bones however do still remain robust and statistically significantly associated with neighbour noise. Notably, controlling for sleep disturbance eliminates the effects of street noise on all disease outcomes. To this author's knowledge this is the first analysis to directly separate the health effects of noise above and beyond the effects of related sleep disturbance (from noise or any other cause). We find that the (high-information) noise caused by neighbours seems to have a stronger additional effect on health, especially on disorders related to immune system, compared to the relatively information-free noise caused by traffic, providing some additional support to the idea that some kinds of relatively low-decibel noise sources can nevertheless be detrimental both to well-being and health.

5 Robustness: Alternative Empirical Specifications

The benchmark logistic regressions presented in sections 4.1 and 4.2 provide strong suggestive evidence of an impact from residential noise to health outcomes. The results are consistent with the limited theoretical and empirical literature on residential noise and health, while providing added confidence in the findings by controlling for individuals' degree of sensitivity, moving history, as well as a host of additional socio-economic, demographic, and neighbourhood variables. The evidence further suggests that the impact of neighbour noise may operate on disorders of the immune system through mechanisms other than, and additional to, that of sleep disturbance. To investigate the robustness of these results we run a series of alternative tests, investigating to what extent the benchmark results are sensitive to sensible alternative specifications, and exploiting the (albeit limited) time series element of the panel to examine the relationship between noise and health over time for individual respondents.

5.1 Noise and health in a simple cross section

As discussed above, the regressors in the benchmark regressions explored in sections 4.1 and 4.2 were calculated as time-averages for each individual in the panel, allowing those who experienced more frequent noise problems to be coded as having higher 'treatment intensity.' In Tables 5a - 5c and Table 6 we reproduce the analysis in 'pure' cross section form, defining each RHS variable as taking the value 1 if its time-averaged value is greater than zero. Thus respondents are coded as suffering from neighbour noise if they reported this problem in *any* of the years in which they participated in the survey.

Overall defining the qualitative cross sectional variables as simple binaries does not change the pattern of the benchmark results but does generally strengthen the statistical significance. Notably in column (2) of Table 6 the effect of neighbour noise on cardiovascular health is now statistically significant (p=.01) even when we control for sleep disturbance.

5.2 Noise, health, and the elderly

A further concern about the analysis thus far is that, although we control for a quadratic effect of age, the elderly could still be disproportionately driving the relationship if the quadratic control is inadequate and they are the most likely to experience health problems in response to exposure to neighbour noise. Certainly the elderly are more likely to experience many health problems, so to address this concern we re-run the benchmark regressions, restricting the sample to respondents under the age of 67 in 2007. However, as the results in Table 7 illustrate, we find no disproportionate effect; among the younger sample the pattern between neighbour noise and health outcomes is almost identical to that in the full sample.

5.3 Propensity score matching

An alternative method to control for potential confounding variables in observational data is to use propensity score matching, originally developed by Rosenbaum and Rubin (1983), in which an estimated probability (propensity score) of being 'treated' (i.e. being subjected to neighbour noise in our case) is first generated based on observable characteristics, with treated and untreated observations then matched on the basis of their propensity scores. In this way each treated observation is compared only with the most similar untreated observation(s), avoiding the linear parametric assumptions of regression analysis. Although propensity score matching cannot directly control for unobservable confounders, to the extent that unobservables are clustered in patterns similar to observables, the matched ob-

servations may arguably constitute a more robust counterfactual. In tables 8 and 9 we estimate average treatment effects (ATE) for both neighbour noise and street noise using propensity scores estimated from our full set of socio-economic, demographic, dwelling and neighbourhood control variables, including both *Stressindex* and *Moved Ever*. In each table we present results both for matching on the sole best propensity score ('nearest neighbour') and on the two closest matches. In Table 8, consistent with previous results, we find statistically significant effects of neighbour noise on cardiovascular health, auto-immune diseases of the bones and joints, and headache, with estimated effects for lung disease falling just short of statistical significance. However one difference between the benchmark results and the matching estimates is that in the latter not only do we not find any statistically significant effect of neighbour noise on Fatigue (column (8)), but the point estimate is negative. Fatigue is by far the most common health issue (with 76% of the sample reporting it), which may make it difficult for matching to detect an effect. In Table 9 we find the average treatment effects of street noise are also consistent with those from regression analysis; we find statistically significant effects on auto-immune disease of the bones and joints, and on headache. Thus overall the results from the matching exercise closely mirror those from the regression analyses.

5.4 LPM with fixed effects analysis

Although we include a broad array of controls we cannot entirely eliminate the possibility of unobservable characteristics (uncorrelated with *Stress Index* and our other controls) driving both noise annoyance and health. Furthermore, it could be that illness itself could increase annoyance and/or sensitivity to noise above and beyond our ability to control for sensitivity with *Stress Index*. To attempt to address these concerns we exploit the fact that the LISS is a longitudinal survey, with respondents answering questions on health and noise in up to six different years (with an average for the sample of between 2 and 4 for most disease outcomes). Thus we are able to look to see whether the likelihood of disease outcomes increases when individuals are exposed to noise, compared to when *the same individuals* are not exposed to noise. By controlling for individual fixed effects, we effectively eliminate the effect of unobservable time-invariant characteristics that could confound the cross-sectional analysis. In the time-series analysis we only extract information from changes over time in our variables of interest for the same individual, so the chronically annoyed person will report noise in all time periods and information from their observations will thus be dropped from the analysis. Furthermore, by conditioning only on respondents who have positively reported themselves to be healthy in the past with respect to the health condition of interest, we can mitigate the likelihood of reverse causality, where illness occurs first and itself leads to increased sensitivity to noise (later).

While attractive in theory, in practice estimating a FE panel model using the LISS data raises a number of difficulties. In particular, compared to the cross sectional analysis, the effective sample size for the LPM fixed effects estimates is greatly reduced as information can only be extracted from respondents whose health status has changed, and not all respondents answered all survey modules in all years. Thus we modify our definition of 'exposure to noise' to include whether a respondent reports exposure to noise in either the current or previous years, so if they reported neighbour noise in 2008, skipped the Housing module in 2009, and then reported a health problem in 2010, they will be coded in 2010 as having been exposed to neighbour noise whether or not they reported neighbour noise in 2010. This seems sensible not only to increase the sample size, but it may be that the health impact of new noise exposure manifest over time with a lag and this will allow us to capture such effects. To mitigate problems of reverse causality we restrict the sample to only those individuals who were initially healthy (with respect to the particular health outcome of interest) by conditioning only on observations for which the respondent has indicated they were healthy (i.e. a '0' for that health outcome) at least once in a previous year, and with no prior indications of illness (i.e. no prior '1' for that health outcome). Thus we model whether respondents who have experienced a change in noise exposure are more likely to develop a *new* health problem compared to those years in which there was no change in their noise exposure. Data from respondents who experience noise continuously before their health changes (or do not experience noise at all) will not contribute to the estimates, nor will information from respondents who fall ill before a noise change, or who are ill (or healthy) throughout the sample period.

There is a lively theoretical and empirical literature on the advantages and disadvantages of alternative fixed effects models with binary dependent variables (Allison, 2009). As ours is the first study (to our knowledge) to analyze noise and health in panel data, unlike with the cross sectional results it is not as important to generate comparable estimates. Thus to keep the analysis as straightforward as possible we adopt a simple linear probability model approach (the results are not sensitive to this choice; we find almost identical outcomes with conditional fixed effects logistic regression, available upon request). Thus we have:

$$(2) \quad H_{it}|_{t''=0} = \beta_i + \beta_{1X1it'} + ... + \beta_k X_{kit'} + u_{it}$$

where β_i denotes the individual-specific fixed effect, t'' denotes all previous years, and t' denotes the current and previous years.

All time-invariant confounders, including unobservable personality characteristics, are controlled for by the fixed effects and thus we include as controls other time-varying variables that could be correlated with changes in health and exposure to noise, namely whether a respondent moved, exposure to poor air quality, marital status and labour market status. We also control for the full set of dwelling

characteristics in order to control for changes in housing quality.

Table 10 presents the results of the LPM fixed effects estimation; for each health outcome we present the results both with and without controlling for whether the respondent has moved in the current or past periods (i.e. defined in the same way as the noise variables). First, we note that the coefficient estimates do not tend to change with the inclusion of our control for moving, consistent with our conclusions from section 6.2 that this is unlikely to be creating a selection problem. In columns (1) and (2) we find exposure to neighbour noise (but not street noise) is still associated with the onset of cardiovascular problems, although when we control for moving the statistical significance of this result fall to just over 5%. As with the benchmark cross section analyses, we continue to find significant effects of neighbour noise on auto-immune disorders of the bones and joints, fatigue and headache (though the significance of the latter also falls just over the critical value of 5% when we control for moving). Unlike the cross sectional we do not find any correlation with lung disease, however. In addition, in columns (3)-(6) we also now also find a statistically significant effect of neighbour noise on cholesterol and blood pressure. There were too few observations to estimate effects for diabetes.

In Table 11 we explore whether sleep disruption as a mechanism could explain the within-individual health results we found in table 10. When it appears on the RHS we define *Sleep Disruption* as equal to 1 if the respondent has experience sleep disruption in either this or the previous years (as with the noise variables as explained above), and when it appears on the LHS in column (1) we also condition on only initially 'healthy' respondents who earlier had reported no sleep disruption problems. As in the cross sectional results we find sleep disruption to have a strong negative effect on most health outcomes (except, as one would expect, asthma). In column (2) we find that sleep disruption increases the chance of cardiovascular problems but has little impact on the magnitude of the coefficient of neighbour noise, although it does slightly reduce the statistical significance to just above 5%. The results for the effects of neighbour noise on other health outcomes remain robust as well to the inclusion of *Sleep Disruption*, with the exception of headache, which is now significant only at just over 10%.

6 Robustness: Potential Issues Related to Selection

As discussed in section 3.2, the "naive" baseline estimates face several selection-related threats to identification both from sensitivity to stress and home moving. Although we attempt to control for *observable* sensitivity to stress and home moving to mitigate these concerns, in this section we explore to what extent it is likely that *unobservable* selection could still be playing a role.

6.1 Sensitivity to noise

We begin by exploring the relationship between the observed components of sensitivity and reported noise annoyance, in Table 12 columns (1)-(2) we estimate linear probability models (LPM) of the likelihood of reporting neighbour noise, controlling for each component of our composite psychometric index of stress as well as the full set of demographic and socio-economic control variables. We find, as expected, that in the cross-section those who indicate they are more easily stressed, disturbed, and/or irritated are more likely to report both neighbour and street noise annoyance (F-tests of joint significance soundly reject with $p < 0.01$). These results not only confirm the importance of controlling for sensitivity in any analysis of the effects of self-reported noise, but also point to an important benefit of controlling for both neighbour and street noise annoyance in the same regressions, namely that reported street noise will help control for unobservable sensitivity when reported neighbour noise is the coefficient of interest, and vice-versa. In column (3)-(4) we explore the within-individual relationship between sensitivity and noise annoyance, modelling a fixed effects LPM of the likelihood of an individual reporting noise annoyance as a function of their time-varying sensitivity and other time-varying socio-economic controls, with the fixed effects controlling for all time-invariant individual characteristics. As the components of the sensitivity index are designed to capture general personality characteristics we do not expect much variation over time in their values for given individuals, but if certain internal or external life circumstances do change reported sensitivity, this could in turn precipitate more noise annoyance and give rise to time-varying reverse causality. However as the results in table 12 columns (3)-(4) report, we find no evidence of a significant time-varying correlation between the components of our stress index and reported noise annoyance (the F-tests of joint significance of the sensitivity components robustly fail to reject that they are all equal to zero for both neighbour and street noise models).

As we find evidence that, at least in the cross section, those respondents that are more easily stressed may report more noise annoyance, we want to further investigate the extent to which individual sensitivity, rather than noise itself, could be a primary determinant of observed correlations beween reported noise annoyance and observed outcomes of interest. As the underlying sensitivity captured in the reported noise annoyance variables is unobserved, we explore this possibility by examining the stability of coefficient estimates on the noise variables between alternative models that do and do not control for our observed measure of sensitivity to stress. If the unobserved sensitivity component of the noise variables are largely responsible for the observed correlations, then controlling directly for (observed) sensitivity should, to the extent that the unobserved and observed sensitivity are correlated, significantly affect the magnitude and perhaps the statistical significance of the estimated coefficients on noise. If, on the other hand, we find that the coefficients on the noise variables remain largely stable across alter-

native specifications that do and do not control for observed sensitivity, this provides some reassurance that it is unlikley that endogenous (unobserved) sensitivity is driving the results; this conclusion would only fail if the salient observed and unobserved components of sensitivity were uncorrelated, which we find unlikely. Indeed, throughout the baseline estimates we report results with and without *Stress Index* for this very purpose and, as we discussed above, find that coefficient magnitudes and levels of statistical significance are reasonably robust.

If endogenous, unobservable sensitivity is indeed influencing the coefficient estimates on noise, it should be most apparent when exploring the relationship between noise and an outcome variable that we would most expect to also be affected by unobservable sensitivity. In our data the variables that best fulfill this description are those related to self-reported levels of subjective well-being. Thus to take this exercise even further, in Tables 13 and 14 we explore whether endogenous (unobserved) sensitivity could be driving any observed relationship between noise annoyance and both self-reported life- and dwelling-satisfaction by examining whether the estimated relationships significantly change when we additionally control for our psychometric measure of sensitivity to stress. As discussed above, if the relationship between life- and dwelling-satisfaction and reported noise is primarily driven by unobserved endogenous sensitivity, we should observe instability of the regression coefficients in the alternative models. On the other hand, if the $objective$ noise component of the reported noise variables is more important, then controlling for observed sensitivity to stress should not have as much of an impact on the magnitude and/or significance of the estimated coefficients.

In Table 13 we model the cross sectional relationship of both life- and dwelling-satisfaction with neighbour and street noise, our full set of socio-economic and demographic controls, and with and without our psychometric measure of sensitivity. Consistent with the findings in Weinhold (2013), in columns 1 and 3 we see that both neighbour noise and street noise lower both life- and dwelling- satisfaction, but interestingly we find that the impact of loud neighbours lowers dwelling satisfaction by almost twice as much (with the coefficient = -0.55) as does street noise (-0.29). In columns (2) and (4) we then additionally control for our psychometric measure of sensitivity to stress. In column (2) the coefficient on neighbour noise in the life-satisfaction regression falls to -0.20, but remains highly statistically significant. The coefficients on neighbour noise are even closer in the two models of dwelling-satisfaction; for the model in column (4) controlling for sensitivity the coefficient falls only slightly, to -0.52. For reported street noise the coefficient estimates with and without controlling for sensitivity are even more robustly consistent across alternative models. Thus overall it does not appear that the relationship between reported noise and life- and dwelling-satisaction is being primarily driven by heterogeneous sensitivity; especially with respect to dwelling-satisfaction the evidence suggests that the relationship is driven more by actual noise leading to lower subjective satisfaction.

Second, in Table 14 we again exploit the longitudinal dimension to estimate panel LPM fixed effects models to examine the within-individual effects of time-varying noise exposure on time-varying life and dwelling satisfaction, both including and excluding our observed time-varying psychometric index of sensitivity as a control. In other words, we estimate whether reported life- or dwelling- satisfaction changes over time for the same individual when that individual's reported exposure to noise changes. As the fixed effects control for time-invariant individual characteristics we include only time-varying controls. In column (1) we find that neither changes in neighbour or street noise are statistically significant in explaining changes in overall life-satisfaction. However in column (3) changes in neighbour noise (but not street noise) are highly statistically significantly correlated with lower dwelling satisfaction. In order to test whether these within-individual relationships between noise and life- and dwelling- satisfaction might be driven by endogenous time-varying sensitivity to stress, in columns (2) and(4) we additionally control for time-varying *Stress Index*. The effect of street noise on Life Satisfaction strengthens slightly, but overall the coefficients on the noise variables are remarkably stable between alternative specifications. Thus overall, consistent with the results from Table 12, we find little evidence that time-varying sensitivity plays a role in generating the observed correlation between changes in reported noise annoyance and reported life- and dwelling-satisfaction.

The results from Tables 13 and 14 confirm that residential noise lowers subjective well-being, and more importantly for this analysis, we find that the effects of neighbour noise and street noise are distinct. Chronic exposure (as captured in the cross sectional analysis) to both neighbour and street noise lowers overall life satisfaction. However only neighbour noise lowers dwelling satisfaction; presumably street noise was observable and predictable when residents moved in, so it comes as no surprise now. Noisy neighbours, however, may be considered an unexpected, unlucky, and unwelcome feature of their current home, and the magnitude of associated dissatisfaction is also significant - the presence of noisy neighbours lowers dwelling satisfaction by more than the presence of criminality nearby. This interpretation is supported by the panel within-individual analysis; a new appearance of noisy neighbours has an immediate negative effect on dwelling satisfaction that is again of same order of magnitude as a new occurrence of crime in a formerly peaceful neighbourhood. Over time, if the neighbour problem persists, the cross sectional results suggest that the effect will spill over to overall life satisfaction as well.

Perhaps more importantly for this paper, however, we find little evidence that endogenous sensitivity is driving the observed effects of noise on subjective well-being, either across individuals or over time. Those respondents who rate themselves as being particularly susceptible to stress do report more noise annoyance and do have lower levels of both life- and dwelling- satisfaction, but the estimated impact of noise on subjective well-being remains relatively stable and robust to

the inclusion of observed measures of sensitivity in the regressions. This result could only be consistent with sensitivity-driven selection bias if the unobservable and observable components of sensitivity were uncorrelated, which of course we cannot prove but believe is highly unlikely. Nevertheless throughout the analysis we have presented all primary regressions on various health outcomes with and without controlling for our psychometric measure of sensitivity to stress, to the extent that observe the noise coefficients to remain robust and stable to the inclusion of *Stress Index*, this builds confidence that the results are unlikley to be driven by other unobservable sensitivity-related confounders.

6.2 Moving

As discussed earlier, another potential threat to inference in our baseline estimates arises from selection related to home-movers. As we outlined in section 3.2, our primary argument against this critique is that noisy neighbours are often *ex-ante* unobservable, and home-moving tends to be extremely costly, especially in Europe. So, while selection via home-moving may play a role, we are skeptical that this form of selection could be a primary driver of the results. Nevertheless since this is an empirical question we delve deeper by investigating the determinants of moving within our sample. Of course, this approach cannot rule out the possibility that unobservable selection by moving could have occured some time before the sample period, but as we control for current household income and housing characteristics already in the analysis, if selection by moving is a major determinant of the observed correlations between health and noise we should be able to detect it during the sample period. Columns (1) and (2) of Table 15 presents the cross sectional estimates of whether respondents moved at any time during the sample period as a function of neighbour noise, street noise, overall health level, other dwelling characteristics, and our full set of socio-economic, demographic and labour market controls. Consistent with expectations, we find that older, married respondents, and those with more children, are less likely to move house. Those with homes that are too small or too cold are more likely to move, and we find that exposure to *street* noise is also strongly predictive of moving. Importantly, however, neither neighbour noise nor overall health level is statistically significant in this regression. In column (2) we additionally control for our psychometric index of sensitivity to stress, *Stress Index* (discussed above in section 6.1). Moving itself can be highly stressful, so to the extent that stressful events might induce respondents to rate themselves more susceptible to stress, the variable is likely endogenous. In the event, *Stress Index* is not significant, and combined with the fact that its inclusion does little to change the coefficient on neighbour noise, suggests that there is little interaction between moving house, susceptibility to stress, and neighbour noise (at least amongst our sample in the Netherlands).

In columns (3) and (4) of Table 15 we exploit the time series dimension of the data to estimate the within-individual relationship of noise exposure and the likelihood of moving in any given year by estimating a linear probability (LPM) panel fixed effects model. Regression (3) conditions only contemporaneous variables, and thus tests whether respondents are more likely to move in years in which they report neighbour noise or their health changes. However, as respondents do not answer all questions in all years, and because finding a new home takes time and moving could occur one or more years after a problem was reported, in column (4) we condition on whether the respondent has reported neighbour noise, street noise, or any other dwelling problem *any time* in the current or previous periods. We find very few significant predictors of moving, and notably neither current or past exposure to neighbour or street noise seem to play a role. We do find weak evidence that respondents whose health has deteriorated may be *more* likely to move in a given year.

Overall both the cross section and the panel fixed effects results suggest that neighbour noise problems are unlikely to be a major driver of moving house among the survey respondents. Nor is there evidence of strong selection related to health, and to the extent that health may matter, it does so in a way that would tend to nudge our results *away* from finding a correlation with noise. Nevertheless throughout the analysis we control for whether a respondent has moved during the sample period. If there are some cases where respondents have moved and experienced neighbour noise (though on average we saw these were not correlated), then the inclusion of *Move Ever* could bias our results against finding an impact of noise on health, so inclusion of the variable is the more conservative option.

7 Discussion

Loud and/or rude neighbours are an under-appreciated cause of misery and, apparently, health problems for many urban residents. Unlike other more visible dwelling characteristics, like street noise, the presence (or new appearance) of loud neighbours cannot be easily observed or predicted in advance when purchasing or renting a new place to live. Faced with noisy neighbours and unsympathetic regulators, choices are few; we find that neighbour noise is significantly correlated with lower life- and dwelling- satisfaction in the cross section, and that changes in neighbour noise are significantly associated with lower individual dwelling-satisfaction in the time series. Beyond constituting a source lowered life- and dwelling- satisfaction, however, we observe large, statistically significant correlations between residential noise exposure and myriad health outcomes.

This paper extends the small existing body of literature on residential noise and health by exploiting a high quality longitudinal survey that allows us to explore

potential threats to causal inference associated with individual sensitivity to stress and selection in moving home. Our analysis is based on observational survey data and subjective assessments of noise, and thus we cannot rule out the possibility of unobservable sensitivity or selection playing a role. However we argue that the existing evidence suggests these potential unobservable sources of endogeneity are unlikely to be primary drivers of the observed correlations, and perhaps more importantly, the underlying research question should be understood not to be 'what are the health effects of neighbour noise', but rather 'what are the health effects of neighbour noise on those who are sensitive to it. Existing medical studies (e.g. Segerstrom and Miller (2004)) strongly suggest that people's biological responses to stressful circumstances are heavily dependent on their subjective perspective and emotional response to it;

In the cross section we find, as expected, that more sensitive individuals are more likely to report noise annoyance. However across the analysis the inclusion of our psychometric sensitivity measure rarely has much impact on the stability of the coefficients on the noise variables, suggesting that unobserved sensitivity is unlikely to be driving the results. Furthermore we find no evidence that changes in individual observable sensitivity to stress are associated with changes in the likelihood of noise complaints. Selection via moving home does also not seem to play a major role during the sample period. Moving is extremely costly in the Netherlands, and we find that in the cross section neither neighbour noise nor health level is a significant predictor of moving house. Furthermore, for individuals over time, neither current or past exposure to neighbour or street noise increase the individual likelihood of moving. Finally, in further robustness tests we exploit the within-individual variation in the panel to estimate the impact of changes in noise exposure over time, conditioning on initial healthy respondents to mitigate concerns about both unobservable time-invariant confounders and reverse causality.

Overall we find strong suggestive evidence that residential noise annoyance, and specifically neighbour noise, can contribute to a number of health problems. Our results indicate that neighbour noise annoyance is associated with increased likelihood of cardiovascular disease, auto-immune diseases of bones and joints, fatigue and headaches, and perhaps blood pressure and cholesterol levels. We find sleep disruption can explain some, but not all, of the effect, leaving room for theoretical mechanisms that link stress to immune system disorders to potentially provide some explanatory power.

The larger conclusion of this paper, however, is that much more research is needed. For all its ubiquity, residential noise pollution receives relatively little attention from policy-makers and regulators, due largely we suspect to the difficulty in "objectively" measuring the problem. While a quasi-experimental research design would be ideal, until it is feasible to operationalize such a framework, rather than declining to investigate the problem we would argue that a good alternative

is to explore existing observational data while remaining mindful of the methodological limitations to causal inference. This paper shows that this is feasible using surveys that rely on self-reporting of noise annoyance, and while concerns remain about possible endogeneity in the use of subjective data, we view this relatively low-cost study as a first step on the road to further research. The LISS CentER data used in this analysis was not designed *ex ante* to study noise pollution; a much more focused survey design could achieve much greater precision and address some of the lingering concerns about the accuracy of self-reported data and unobservable missing variables. As urbanization spreads across the world, residential noise pollution deserves much more academic and policy attention.

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8 Tables

Table 1: Summary Statistics

Continuous Variables					
Variable	Obs	Mean	St. Dev.	Min	Max
Health Level	5440	3.12	0.644	1	5
Stress Index	5406	2.61	0.727	1	5
BMI	5440	25.67	4.35	11	50
Age	5440	49.30	14.57	17	85
HH #kids	5440	0.809	1.062	0	6
HH Income	5440	2969	2796	250	126110
Hours	5440	32.3	13.1	0	110

Dichotomous Variables
(take the value 1 if the variable ever took the value 1 during the sample period)

Variable	Obs	Frequency	Variable	Obs	Frequency
Neighbour Noise	5440	0.330	Unemployed	5440	0.090
Street Noise	5440	0.192	Housewife	5440	0.399
Moved Ever	5195	0.227	Student	5440	0.073
Bad Air	5440	0.103	Retired	5440	0.328
Dwelling small	5440	0.134	Religious	5440	0.459
Dwelling dark	5440	0.040	Crime in Area	5440	0.160
Dwelling cold	5440	0.060	Urban Area	5440	0.592
Dwelling eaky	5440	0.039	Rural Area	5440	0.252
Dwelling damp	5440	0.081	Cardio-vascular	5440	0.147
Dwelling rotten	5440	0.034	Fatigue	5440	0.763
Smoke Ever	5440	0.657	Headache	5440	0.277
Daily Drinker	5440	0.248	Blood Pressure	5440	0.236
Male	5440	0.501	Cholesterol	5440	0.183
Primary Ed	5440	0.015	Bones&Joints	5440	0.634
Secondary Ed	5440	0.165	Lung Disease	5440	0.136
Post-Secondary Ed	5440	0.684	Asthma	5440	0.058
Terciary Ed	5440	0.174	Diabetes	5440	0.074
Married	5440	0.778			

Table 2: Self-reported level of health from 1 (poor) to 5 (excellent) and residential noise, Cross Section OLS

	(1) Health Level	(2) Health Level	(3) Health Level	(4) Health Level	(5) Health Level
Neighbour Noise	-0.149***	-0.146***	-0.107***	-0.110***	-0.089***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Street Noise	-0.079*	-0.059	-0.026	-0.038	-0.020
	(0.018)	(0.088)	(0.430)	(0.255)	(0.536)
Air Quality		-0.091	-0.067	-0.061	-0.051
		(0.060)	(0.151)	(0.198)	(0.265)
Stress Index			-0.227***	-0.228***	-0.190***
			(0.000)	(0.000)	(0.000)
Move Ever				0.004	0.011
				(0.864)	(0.601)
Sleep Disruption					-0.384***
					(0.000)
Smoke Ever	-0.110***	-0.110***	-0.106***	-0.106***	-0.101***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
BMI	-0.027***	-0.027***	-0.027***	-0.027***	-0.025***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Age	-0.010*	-0.010*	-0.014**	-0.014**	-0.012*
	(0.048)	(0.047)	(0.004)	(0.007)	(0.014)
Male	0.054*	0.055*	-0.003	-0.004	-0.027
	(0.014)	(0.013)	(0.879)	(0.856)	(0.213)
Married	-0.008	-0.008	0.006	-0.001	-0.011
	(0.735)	(0.745)	(0.791)	(0.964)	(0.639)
University	0.187***	0.187***	0.153***	0.143***	0.131***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)
HH Income	0.149***	0.148***	0.132***	0.142***	0.126***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
N	5386	5386	5356	5119	5104
R^2	0.136	0.137	0.198	0.203	0.239

*R*obust p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Dwelling and neighbourhood characteristics, agesq, religious status, alcohol consumption, other educational levels, labor market status, and number of children.

Table 3a: Odds Ratios for cross-sectional logistic regressions of residential noise on disease outcomes

	(1) Cardio-vascular	(2) Cardio-vascular	(3) Choles-terol	(4) Choles-terol	(5) Blood Pressure	(6) blood Pressure
Neighbour Noise	1.384**	1.294*	1.026	0.964	1.013	0.966
	(0.007)	(0.037)	(0.842)	(0.777)	(0.913)	(0.772)
Street Noise	0.982	0.941	1.165	1.124	1.175	1.138
	(0.912)	(0.715)	(0.335)	(0.457)	(0.283)	(0.386)
Air Quality	1.243	1.197	1.307	1.264	1.246	1.215
	(0.341)	(0.436)	(0.199)	(0.264)	(0.290)	(0.353)
Move Ever	0.900	0.878	0.875	0.852	0.859	0.834
	(0.365)	(0.268)	(0.254)	(0.176)	(0.152)	(0.089)
Stress Index		1.478***		1.390***		1.311***
		(0.000)		(0.000)		(0.000)
Smoke Ever	1.176	1.176	1.380***	1.388***	1.144	1.149
	(0.096)	(0.099)	(0.001)	(0.001)	(0.124)	(0.115)
BMI	1.048***	1.048***	1.076***	1.076***	1.141***	1.142***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Age	1.017	1.024	1.260***	1.272***	1.239***	1.254***
	(0.526)	(0.374)	(0.000)	(0.000)	(0.000)	(0.000)
Age^2	1.000	1.000	0.998***	0.998***	0.999***	0.999***
	(0.389)	(0.518)	(0.000)	(0.000)	(0.000)	(0.000)
Male	1.284*	1.416**	1.640***	1.751***	1.089	1.158
	(0.028)	(0.003)	(0.000)	(0.000)	(0.399)	(0.150)
Married	1.286*	1.268*	1.230	1.214	1.055	1.031
	(0.029)	(0.040)	(0.059)	(0.079)	(0.608)	(0.771)
University	0.738	0.775	0.667*	0.700	0.678*	0.713
	(0.130)	(0.207)	(0.031)	(0.057)	(0.029)	(0.057)
HH Income	0.701**	0.717**	0.797*	0.818	1.020	1.038
	(0.002)	(0.003)	(0.029)	(0.052)	(0.849)	(0.718)
N	5147	5119	5145	5117	5145	5117
pseudo R^2	0.116	0.126	0.182	0.188	0.204	0.209

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, alcohol consumption, educational level, labor market and religious status, number of children.

Table 3b: Odds Ratios for cross-sectional logistic regressions of residential noise on disease outcomes

	(1) Asthma	(2) Asthma	(3) Lung Disease	(4) Lung Disease	(5) Bones& Joints	(6) Bones& Joints
Neighbour Noise	1.195	1.164	1.463**	1.345*	1.699***	1.542***
	(0.342)	(0.418)	(0.002)	(0.016)	(0.000)	(0.000)
Street Noise	1.059	1.042	1.362	1.303	1.332*	1.272
	(0.815)	(0.867)	(0.050)	(0.093)	(0.022)	(0.060)
Air Quality	0.707	0.696	1.514*	1.477	1.377	1.281
	(0.341)	(0.319)	(0.047)	(0.061)	(0.086)	(0.190)
Move Ever	1.265	1.253	1.076	1.053	1.236**	1.224*
	(0.130)	(0.146)	(0.516)	(0.654)	(0.007)	(0.012)
Stress Index		1.166		1.581***		1.678***
		(0.069)		(0.000)		(0.000)
Smoke Ever	0.949	0.942	1.436***	1.434***	1.343***	1.316***
	(0.699)	(0.659)	(0.000)	(0.000)	(0.000)	(0.000)
BMI	1.056***	1.056***	1.069***	1.070***	1.057***	1.059***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)
Age	1.003	1.005	0.951*	0.959	1.018	1.029
	(0.930)	(0.893)	(0.043)	(0.100)	(0.389)	(0.191)
Age^2	1.000	1.000	1.001**	1.001*	1.000	1.000
	(0.909)	(0.933)	(0.004)	(0.011)	(0.407)	(0.635)
Male	0.572***	0.595***	0.774*	0.862	0.647***	0.729***
	(0.000)	(0.001)	(0.020)	(0.186)	(0.000)	(0.000)
Married	1.106	1.092	1.245	1.208	1.127	1.097
	(0.565)	(0.613)	(0.063)	(0.108)	(0.158)	(0.282)
University	1.077	1.096	0.739	0.769	0.727*	0.768
	(0.773)	(0.722)	(0.129)	(0.192)	(0.018)	(0.057)
HH Income	0.898	0.911	0.640***	0.671***	0.853*	0.885
	(0.475)	(0.528)	(0.000)	(0.000)	(0.047)	(0.142)
N	5142	5114	5147	5119	5147	5119
pseudo R^2	0.028	0.030	0.075	0.088	0.068	0.087

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, alcohol consumption, educational level, labor market and religious status, number of children.

Table 3c: Odds Ratios for cross-sectional logistic regressions of residential noise on disease outcomes

	(1) Diabetes	(2) Diabetes	(3) Fatigue	(4) Fatigue	(5) Headache	(6) Headache
Neighbour Noise	0.806	0.793	1.372**	1.246*	1.409***	1.280*
	(0.244)	(0.214)	(0.003)	(0.041)	(0.000)	(0.012)
Street Noise	0.902	0.886	1.338*	1.268	1.362*	1.284*
	(0.657)	(0.602)	(0.040)	(0.101)	(0.014)	(0.049)
Air Quality	2.109**	2.060**	1.274	1.184	1.189	1.159
	(0.007)	(0.008)	(0.257)	(0.436)	(0.346)	(0.422)
Move Ever	0.822	0.813	0.810*	0.799*	1.233*	1.227*
	(0.279)	(0.252)	(0.022)	(0.017)	(0.012)	(0.016)
Stress Index		1.222*		1.695***		1.750***
		(0.016)		(0.000)		(0.000)
Smoke Ever	1.417*	1.444*	1.337***	1.346***	1.053	1.050
	(0.014)	(0.010)	(0.000)	(0.000)	(0.482)	(0.519)
BMI	1.156***	1.155***	1.026**	1.028**	1.025**	1.026***
	(0.000)	(0.000)	(0.002)	(0.001)	(0.001)	(0.001)
Age	1.165***	1.169***	0.848***	0.856***	1.000	1.005
	(0.001)	(0.000)	(0.000)	(0.000)	(0.995)	(0.826)
Age^2	0.999*	0.999*	1.001***	1.001***	1.000	1.000
	(0.013)	(0.010)	(0.000)	(0.000)	(0.532)	(0.473)
Male	1.580**	1.640**	0.645***	0.724***	0.359***	0.405***
	(0.003)	(0.001)	(0.000)	(0.001)	(0.000)	(0.000)
Married	1.186	1.169	0.829*	0.811*	1.210*	1.165
	(0.289)	(0.333)	(0.050)	(0.031)	(0.048)	(0.119)
University	0.670	0.656	0.704*	0.759	0.581***	0.619**
	(0.161)	(0.146)	(0.022)	(0.078)	(0.001)	(0.004)
HH Income	0.824	0.836	0.897	0.921	0.759**	0.792*
	(0.219)	(0.254)	(0.219)	(0.360)	(0.003)	(0.013)
N	5145	5117	5147	5119	5131	5104
pseudo R^2	0.172	0.175	0.063	0.082	0.081	0.104

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Dwelling and neighbourhood characteristics, alchohol consumption, educational level, labor market and religious status, number of children.

Table 4: Odds Ratios for cross-sectional logistic regressions of residential noise and sleep disturbance on disease outcomes

	(1) Sleep Disruption	(2) Cardio-vascular	(3) Choles-terol	(4) Blood Pressure	(5) Asthma Asthma
Neighbour Noise	1.494***	1.230	0.919	0.923	1.134
	(0.000)	(0.095)	(0.521)	(0.509)	(0.506)
Street Noise	1.359*	0.899	1.093	1.102	1.009
	(0.015)	(0.525)	(0.576)	(0.517)	(0.971)
Move Ever	1.370***	0.859	0.842	0.825	1.233
	(0.000)	(0.199)	(0.150)	(0.076)	(0.179)
Stress Index	2.052***	1.350***	1.308***	1.219***	1.102
	(0.000)	(0.000)	(0.000)	(0.000)	(0.252)
Sleep Disruption		2.185***	1.702***	1.878***	1.595**
		(0.000)	(0.000)	(0.000)	(0.005)
N	5104	5104	5102	5102	5099
pseudo R^2	0.105	0.136	0.193	0.215	0.033

	(6) Lung Disease	(7) Bones & Joints	(8) Diabetes	(9) Fatigue	(10) Headache
Neighbour Noise	1.274	1.505***	0.769	1.183	1.208
	(0.050)	(0.000)	(0.165)	(0.123)	(0.058)
Street Noise	1.231	1.223	0.860	1.238	1.231
	(0.195)	(0.118)	(0.520)	(0.147)	(0.109)
Move Ever	1.026	1.206*	0.803	0.788*	1.204*
	(0.827)	(0.020)	(0.227)	(0.012)	(0.032)
Stress Index	1.422***	1.572***	1.159	1.571***	1.576***
	(0.000)	(0.000)	(0.085)	(0.000)	(0.000)
Sleep Disruption	2.471***	2.087***	1.582**	2.656***	2.844***
	(0.000)	(0.000)	(0.004)	(0.000)	(0.000)
N	5104	5104	5102	5104	5104
pseudo R^2	0.102	0.093	0.178	0.094	0.123

Exponentiated coefficients; Robust p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, smoking history, air quality, BMI, age, agesq, gender, marital and religious status, alcohol consumption, educational level, labor market status, number of children.

Table 5a: Robustness: Odds Ratios for cross-sectional logistic regressions of residential noise on disease outcomes, Simple Cross Section (X_i=1 if $\sum_t \frac{X_{it}}{T} > 0$)

	(1) Cardio-vascular	(2) Cardio-vascular	(3) Choles-terol	(4) Choles-terol	(5) Blood Pressure	(6) Blood Pressure
Neighbour Noise	1.384***	1.321**	1.059	1.013	1.057	1.020
	(0.000)	(0.003)	(0.533)	(0.892)	(0.519)	(0.816)
Street Noise	0.969	0.940	1.021	0.994	1.122	1.096
	(0.789)	(0.599)	(0.851)	(0.958)	(0.264)	(0.376)
Air Quality	1.083	1.059	1.278	1.255	1.158	1.143
	(0.571)	(0.685)	(0.067)	(0.089)	(0.257)	(0.302)
Move Ever	0.874	0.859	0.884	0.865	0.857	0.835
	(0.254)	(0.201)	(0.291)	(0.221)	(0.145)	(0.091)
1em] Stress Index		1.470***		1.391***		1.311***
		(0.000)		(0.000)		(0.000)
N	5147	5119	5147	5119	5147	5119
pseudo R^2	0.117	0.127	0.182	0.188	0.203	0.208

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, alcohol consumption, educational level, labor market and religious status, number of children.

Table 5b: Robustness: Odds Ratios for logistic regressions of residential noise on disease outcomes, Simple Cross Section (X_i=1 if $\sum_t \frac{X_{it}}{T} > 0$)

	(1) Asthma	(2) Asthma	(3) Lung Disease	(4) Lung Disease	(5) Bones& Joints	(6) Bones& Joints
Neighbour Noise	1.225	1.201	1.354**	1.268*	1.561***	1.453***
	(0.145)	(0.192)	(0.001)	(0.013)	(0.000)	(0.000)
Street Noise	1.044	1.030	1.154	1.117	1.311**	1.277**
	(0.796)	(0.859)	(0.196)	(0.317)	(0.001)	(0.005)
Air Quality	0.786	0.780	1.256	1.231	1.263*	1.215
	(0.275)	(0.258)	(0.090)	(0.120)	(0.037)	(0.086)
Move Ever	1.214	1.204	1.021	1.006	1.174*	1.171
	(0.215)	(0.233)	(0.857)	(0.957)	(0.044)	(0.050)
Stress Index		1.157		1.570***		1.662***
		(0.081)		(0.000)		(0.000)
N	5147	5119	5147	5119	5147	5119
pseudo R^2	0.029	0.030	0.076	0.088	0.074	0.091

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, alcohol consumption, educational level, labor market and religious status, number of children.

Table 5c: Robustness: Odds Ratios for logistic regressions of residential noise on disease outcomes, Simple Cross Section (X_i=1 if $\sum_t \frac{X_{it}}{T} > 0$)

	(1) Diabetes	(2) Diabetes	(3) Fatigue	(4) Fatigue	(5) Headache	(6) Headache
Neighbour Noise	0.806	0.793	1.372**	1.246*	1.409***	1.280*
	(0.244)	(0.214)	(0.003)	(0.041)	(0.000)	(0.012)
Street Noise	0.902	0.886	1.338*	1.268	1.362*	1.284*
	(0.657)	(0.602)	(0.040)	(0.101)	(0.014)	(0.049)
Air Quality	2.109**	2.060**	1.274	1.184	1.189	1.159
	(0.007)	(0.008)	(0.257)	(0.436)	(0.346)	(0.422)
Move Ever	0.822	0.813	0.810*	0.799*	1.233*	1.227*
	(0.279)	(0.252)	(0.022)	(0.017)	(0.012)	(0.016)
Stress Index		1.222*		1.745***		1.720***
		(0.016)		(0.000)		(0.000)
N	5145	5117	5147	5119	5131	5104
pseudo R^2	0.172	0.175	0.063	0.082	0.081	0.104

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Dwelling and neighbourhood characteristics,
smoking history, BMI, age, agesq, gender, alcohol consumption
educational level, marital, labor market and religious status, number of children.

Table 6: Robustness: Odds Ratios for logistic regressions of residential noise and sleep disturbance on disease outcomes, Simple Cross Section (X_i=1 if $\sum_t \frac{X_i}{T} > 0$)

	(1) Sleep Disruption	(2) Cardio-vascular	(3) Choles-terol	(4) Blood Pressure	(5) Asthma
Neighbour Noise	1.303***	1.268*	0.980	0.983	1.180
	(0.000)	(0.011)	(0.832)	(0.847)	(0.238)
Street Noise	1.263**	0.902	0.965	1.060	1.006
	(0.007)	(0.389)	(0.750)	(0.574)	(0.973)
Move Ever	1.309**	0.827	0.846	0.816	1.179
	(0.001)	(0.114)	(0.164)	(0.057)	(0.292)
Stress Index	2.022***	1.346***	1.301***	1.211***	1.105
	(0.000)	(0.000)	(0.000)	(0.000)	(0.238)
Sleep Disruption		1.900***	1.618***	1.768***	1.378*
		(0.000)	(0.000)	(0.000)	(0.012)
N	5119	5119	5119	5119	5119
pseudo R^2	0.102	0.138	0.194	0.216	0.033

	(6) Lung Disease	(7) Bones& Joints	(8) Diabetes	(9) Fatigue	(10) Headache
Neighbour Noise	1.210*	1.419***	0.924	0.857	1.178*
	(0.047)	(0.000)	(0.553)	(0.052)	(0.034)
Street Noise	1.059	1.249*	0.817	1.017	1.131
	(0.609)	(0.011)	(0.197)	(0.865)	(0.179)
Moved	0.954	1.137	0.810	0.792*	1.138
	(0.688)	(0.114)	(0.252)	(0.014)	(0.141)
Stress Index	1.407***	1.534***	1.180	1.629***	1.525***
	(0.000)	(0.000)	(0.052)	(0.000)	(0.000)
Sleep Disruption	2.231***	1.958***	1.292*	1.839***	2.570***
	(0.000)	(0.000)	(0.043)	(0.000)	(0.000)
N	5119	5119	5119	5119	5119
pseudo R^2	0.107	0.103	0.175	0.089	0.129

Exponentiated coefficients; Robust *p*-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$
Included in regession but not shown: Dwelling and neighbourhood characteristics, smoking history, air quality, BMI, age, agesq, gender, marital and religious status, alcohol consumption, educational level, labor market status, number of children.

Table 7: Robustness: Odds Ratios for logistic regressions of residential noise on disease outcomes, respondents age 17-67 in 2007

	(1) Cardio-vascular	(2) Choles-terol	(3) Blood Pressure	(4) Asthma	(5) Lung Disease
Neighbour Noise	1.307*	0.994	0.972	1.171	1.359*
	(0.035)	(0.964)	(0.819)	(0.412)	(0.015)
Street Noise	0.983	1.111	1.190	1.192	1.347
	(0.921)	(0.528)	(0.259)	(0.475)	(0.070)
Stress Index	1.488***	1.399***	1.311***	1.160	1.581***
	(0.000)	(0.000)	(0.000)	(0.097)	(0.000)
Air Quality	1.121	1.148	1.201	0.624	1.537*
	(0.648)	(0.549)	(0.405)	(0.234)	(0.048)
Move Ever	0.915	0.876	0.843	1.351	1.158
	(0.470)	(0.283)	(0.121)	(0.060)	(0.208)
N	4833	4831	4831	4828	4833
pseudo R^2	0.102	0.182	0.198	0.032	0.081

	(6) Bones& Joints	(7) Diabetes	(8) Fatigue	(9) Headache
Neighbour Noise	1.576***	0.754	1.246*	1.262*
	(0.000)	(0.154)	(0.045)	(0.020)
Street Noise	1.220	0.945	1.272	1.307*
	(0.125)	(0.818)	(0.109)	(0.040)
Stress Index	1.688***	1.260*	1.699***	1.744***
	(0.000)	(0.011)	(0.000)	(0.000)
Air Quality	1.215	1.876*	1.121	1.103
	(0.312)	(0.041)	(0.611)	(0.611)
Move Ever	1.211*	0.773	0.830	1.257**
	(0.018)	(0.183)	(0.055)	(0.008)
N	4833	4831	4833	4818
pseudo R^2	0.084	0.182	0.085	0.105

Exponentiated coefficients; robust *p*-values in parentheses: $^{*}\ p < 0.05$, $^{**}\ p < 0.01$, $^{***}\ p < 0.001$
Included in regession but not shown: Dwelling characteristics, neighbourhood characteristics, smoking history, BMI, age, agesq, gender, marital and religious status, alcohol consumption, educational level, labor market status, and number of children.

Table 8: Propensity Score Matching: Average Treatment Effects

	(1) Cardio-vascular	(2) Choles-terol	(3) Blood Pressure	(4) Asthma	(5) Lung Disease
			matching on nearest neighbour		
Neighbour Noise	0.033*	-0.003	0.009	0.008	0.025
	(0.023)	(0.868)	(0.568)	(0.388)	(0.058)
			matching on 2 nearest neighbours		
Neighbour Noise	0.030*	-0.007	0.007	0.003	0.021
	(0.018)	(0.620)	(0.642)	(0.679)	(0.078)
	(6) Bones& Joints	(7) Diabetes	(8) Fatigue	(9) Headache	
			matching on nearest neighbour		
Neighbour Noise	0.097***	-0.008	-0.027	0.045**	
	(0.000)	(0.368)	(0.105)	(0.006)	
			matching on two nearest neighbours		
Neighbour Noise	0.088***	-0.006	-0.025	0.045**	
	(0.000)	(0.469)	(0.111)	(0.002)	
N	5119	5119	5119	5119	5119

*N*ote: Robust Abadie-Imbens p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 9: Propensity Score Matching: Average Treatment Effects

	(1) Cardio-vascular	(2) Choles-terol	(3) Blood Pressure	(4) Asthma	(5) Lung Disease
			matching on nearest neighbour		
Street Noise	-0.027	-0.004	0.018	-0.002	0.016
	(0.140)	(0.810)	(0.367)	(0.878)	(0.271)
			matching on 2 nearest neighbours		
Street Noise	-0.011	-0.003	0.015	0.003	-0.001
	(0.512)	(0.879)	(0.416)	(0.793)	(0.917)
	(6) Bones& Joints	(7) Diabetes	(8) Fatigue	(9) Headache	
			matching on nearest neighbour		
Street Noise	0.066**	-0.013	-0.004	0.045*	
	(0.007)	(0.184)	(0.871)	(0.039)	
			matching on two nearest neighbours		
Street Noise	0.065**	-0.017	-0.001	0.040*	
	(0.003)	(0.064)	(0.949)	(0.035)	
N	5119	5119	5119	5119	5119

*N*ote: Robust Abadie-Imbens p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Table 10: Robustness: LPM FE Regression, Conditioning on initially healthy respondents age 17-67 in 2007

	(1) Cardio-vascular	(2) Cardio-vascular	(3) Choles-terol	(4) Choles-terol	(5) Blood Pressure	(6) Blood Pressure
Neighbour Noise	0.012*	0.011	0.021**	0.020*	0.022*	0.020*
	(0.045)	(0.055)	(0.010)	(0.012)	(0.013)	(0.027)
Street Noise	0.004	0.003	0.002	0.001	0.012	0.010
	(0.624)	(0.655)	(0.772)	(0.819)	(0.265)	(0.369)
Moved		0.006		0.005		0.034**
		(0.351)		(0.344)		(0.006)
N	9316	9297	8764	8746	8202	8183
R^2	0.004	0.004	0.005	0.005	0.007	0.009
Individuals	3550	3533	3383	3367	3208	3191
	(7) Asthma	(8) Asthma	(9) Lung Disease	(10) Lung Disease	(11) Bones& Joints	(12) Bones& Joints
Neighbour Noise	0.007	0.006	0.005	0.004	0.052*	0.047*
	(0.138)	(0.153)	(0.296)	(0.415)	(0.021)	(0.036)
Street Noise	-0.002*	-0.002*	0.009	0.008	0.130***	0.125**
	(0.020)	(0.014)	(0.153)	(0.215)	(0.001)	(0.001)
Moved		0.003		0.017*		0.051
		(0.323)		(0.038)		(0.056)
N	10039	10019	9355	9338	4371	4363
R^2	0.003	0.003	0.007	0.008	0.032	0.034
Individuals	3708	3690	3528	3512	1965	1957
	(13) Fatigue	(14) Fatigue	(15) Headache	(16) Headache		
Neighbour Noise	0.056**	0.051**	0.014*	0.012		
	(0.002)	(0.005)	(0.038)	(0.079)		
Street Noise	0.107**	0.102**	0.004	0.002		
	(0.001)	(0.002)	(0.655)	(0.834)		
Moved		0.075**		0.037**		
		(0.002)		(0.002)		
N	5997	5979	7872	7858		
R^2	0.031	0.033	0.007	0.010		
Individuals	2411	2394	3002	2989		

Robust clustered p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Dwelling and neighbourhood characteristics, marital status, labor market status.

Table 11: Robustness: LPM FE Regression, Conditioning on initially healthy respondents age 17-67 in 2007

	(1) Sleep Disruption	(2) Cardio-vascular	(3) Choles-terol	(4) Blood Pressure	(5) Asthma
Neighbour Noise	0.025*	0.011	0.020*	0.019*	0.006
	(0.024)	(0.055)	(0.012)	(0.032)	(0.157)
Street Noise	0.018	0.003	0.001	0.010	-0.002*
	(0.209)	(0.682)	(0.836)	(0.360)	(0.015)
Sleep Disruption		0.020*	0.025*	0.024*	0.003
		(0.019)	(0.018)	(0.041)	(0.509)
N	7731	9238	8696	8133	9960
R^2	0.022	0.005	0.007	0.010	0.003
Individuals	3031	3502	3343	3168	3661

	(6) Lung Disease	(7) Bones& Joints	(8) Fatigue	(9) Headache
Neighbour Noise	0.004	0.045*	0.051**	0.011
	(0.490)	(0.041)	(0.005)	(0.105)
Street Noise	0.008	0.130***	0.101**	0.001
	(0.237)	(0.001)	(0.002)	(0.877)
Sleep Disruption	0.034**	0.205***	0.211***	0.059***
	(0.005)	(0.000)	(0.000)	(0.000)
N	9277	4303	5979	7858
R^2	0.011	0.048	0.048	0.018
Individuals	3482	1924	2394	2989

*R*obust clustered p-values in parentheses; * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Moved, dwelling characteristics, neighbourhood characteristics, air quality, marital and labour market status.

Table 12: Reported Noise Annoyance and Sensitivity to Stress

	(1) noise_neighbour	(2) noise_street	(3) noise_neighbour	(4) noise_street
	cross section OLS		Fixed Effects LPM panel	
stressed	0.021**	0.009	0.007	0.006
	(0.005)	(0.113)	(0.362)	(0.296)
disturbed	0.002	-0.005	0.008	0.004
	(0.832)	(0.398)	(0.266)	(0.481)
irritated	0.025***	0.014*	0.005	-0.003
	(0.001)	(0.011)	(0.469)	(0.630)
Prob <F	0.0000	0.0005	0.2801	0.6005
N	4943	4943	9205	9205
R^2	0.141	0.171	0.007	0.010

p-values in parentheses

$^{*}\ p < 0.05$, $^{**}\ p < 0.01$, $^{***}\ p < 0.001$

Included in regession but not shown: Dwelling and neighbourhood characteristics, alcohol consumption, smoking history, education, labor market, marital and religious status, air quality, BMI, age, agesq, gender, constant.

Table 13: Cross Section Relationship between Noise, Life- and Dwelling- Satisfaction

	(1) Life Satisfaction	(2) Life Satisfaction	(3) Dwelling Satisfaction	(4) Dwelling Satisfaction
constant	4.851***	6.697***	6.078***	6.634***
	(0.000)	(0.000)	(0.000)	(0.000)
Noise	-0.291***	-0.203***	-0.549***	-0.517***
	(0.000)	(0.000)	(0.000)	(0.000)
Street Noise	-0.237***	-0.179**	-0.241**	-0.230**
	(0.000)	(0.004)	(0.001)	(0.002)
Stress Index		-0.474***		-0.134***
		(0.000)		(0.000)
Air Quality	0.010	0.062	-0.318**	-0.302*
	(0.910)	(0.467)	(0.009)	(0.014)
dwell_small	-0.224**	-0.177*	-1.527***	-1.517***
	(0.002)	(0.012)	(0.000)	(0.000)
dwell_dark	-0.312*	-0.278*	-0.695***	-0.686***
	(0.012)	(0.018)	(0.000)	(0.000)
dwell_cold	-0.246*	-0.178	-0.891***	-0.878***
	(0.046)	(0.120)	(0.000)	(0.000)
dwell_leaky	-0.486*	-0.409*	-0.276	-0.253
	(0.016)	(0.042)	(0.306)	(0.345)
dwell_damp	-0.378***	-0.340***	-0.648***	-0.629***
	(0.000)	(0.000)	(0.000)	(0.000)
dwell_rotten	0.027	0.005	-0.436**	-0.450**
	(0.809)	(0.963)	(0.007)	(0.006)
crime	-0.174*	-0.133*	-0.322***	-0.316***
	(0.013)	(0.049)	(0.000)	(0.001)
N	5354	5354	5342	5342
R^2	0.237	0.237	0.294	0.294

p-values in parentheses; sym* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Also included but not shown: BMI, Age, Agesq, Gender, Smoke Ever, Hours, Rural, Urban, Labor & Marital status, Education level, Alcohol consumption, Income, Religous status, Kids.

Table 14: Within-Individual (Panel Fixed Effects) Relationship between Noise, Life- and Dwelling-Satisfaction

	(1) Life Satisfaction	(2) Life Satisfaction	(3) Dwelling Satisfaction	(4) Dwelling Satisfaction
Neighbour Noise	0.030	0.031	-0.222***	-0.230***
	(0.255)	(0.366)	(0.000)	(0.000)
Street Noise	-0.055	-0.086*	-0.036	-0.038
	(0.083)	(0.047)	(0.491)	(0.566)
Stress Index		-0.229***		-0.040
		(0.000)		(0.236)
Air Quality	-0.030	-0.028	-0.175**	-0.243**
	(0.502)	(0.688)	(0.004)	(0.008)
dwell_small	0.003	0.045	-1.106***	-1.113***
	(0.956)	(0.475)	(0.000)	(0.000)
dwell_dark	-0.090	-0.014	-0.510***	-0.454*
	(0.232)	(0.894)	(0.001)	(0.021)
dwell_cold	0.050	-0.005	-0.464***	-0.537***
	(0.460)	(0.950)	(0.000)	(0.000)
dwell_leaky	-0.013	0.082	-0.120	-0.069
	(0.878)	(0.461)	(0.339)	(0.696)
dwell_damp	-0.014	-0.035	-0.393***	-0.381**
	(0.808)	(0.626)	(0.000)	(0.002)
dwell_rotten	0.032	-0.009	-0.356***	-0.459***
	(0.573)	(0.890)	(0.000)	(0.000)
Married	0.254**	0.275**	0.094	0.054
	(0.003)	(0.003)	(0.312)	(0.640)
unemployed	-0.211**	-0.176*	-0.117	-0.211
crime	-0.003	0.042	-0.195***	-0.152*
	(0.939)	(0.356)	(0.000)	(0.028)
N	15339	10606	15318	10494
R^2	0.008	0.031	0.075	0.083
Individuals	5514	5156	5520	5129

p-values in parentheses: $^{*}\ p < 0.05$, $^{**}\ p < 0.01$, $^{***}\ p < 0.001$

Also included but not shown: Rural, Urban, Student, Retired, Housewife, HH Income.

Table 15: Determinants of Moving Residence: Between and Within Relationship

	Cross Section		LPM Panel Fixed Effects	
	(1)	(2)	(3)	(4)
	Move Ever	Move Ever	Moved	Moved
Neighbour Noise	-0.006	-0.005	0.003	0.009
	(0.714)	(0.737)	(0.715)	(0.405)
Street Noise	0.057*	0.057*	0.002	-0.003
	(0.010)	(0.010)	(0.838)	(0.832)
Health Level	-0.001	0.002	-0.009	-0.008
	(0.890)	(0.864)	(0.076)	(0.085)
Stress Index		0.007		
		(0.373)		
Air Quality	-0.046	-0.044	-0.009	-0.002
	(0.131)	(0.147)	(0.451)	(0.897)
Dwelling Small	0.116***	0.113***	0.015	0.039
	(0.000)	(0.000)	(0.460)	(0.090)
Dwelling Dark	-0.090*	-0.092*	0.002	-0.041
	(0.030)	(0.028)	(0.950)	(0.250)
Dwelling Cold	0.168***	0.172***	-0.005	0.002
	(0.000)	(0.000)	(0.824)	(0.948)
HH Income	-0.002	-0.001	-0.037**	-0.038**
	(0.890)	(0.956)	(0.009)	(0.007)
Married	-0.059***	-0.061***	-0.033	-0.034
	(0.000)	(0.000)	(0.148)	(0.127)
University	0.052*	0.051*		
	(0.030)	(0.032)		
hh_numkids	-0.031***	-0.030***		
	(0.000)	(0.000)		
N	5147	5119	13928	13928
R^2	0.169	0.169	0.007	0.009

*R*obust p-values in parentheses

* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Included in regession but not shown: Other dwelling and neighbourhood characteristics, alcohol consumption, other educational levels, labor market and religious status, smoking history air quality, BMI, age, agesq, and gender.