Sleep Disruption and Health

January 21, 2019

Abstract

A robust medical literature has linked sleep disruption to inflammatory and immune system responses that could, in theory, lead to a variety of health complications. However, limitations on both the scope and duration of laboratory studies make it difficult to establish longer-term causal links, and potential reverse causality may significantly weaken causal inference with observational data. Using a survey of Dutch adults, we contribute to the effort to investigate the causal relationship between self-reported sleep disruption and health by using individual-specific exposure to neighbour noise as an instrument for sleep disruption. We argue that neighbor noise is a relatively ex-ante exogenous shock, and we provide quantitative evidence that it fulfills the relevance, exogeneity, and exclusion restrictions for validity as an instrument. Consistent with theory, we find statistically and economically significant causal effects of sleep disruption on cardio-vascular problems, auto-immune diseases such as arthritis and lung disease, and headache. The results survive a battery of robustness checks and shed light on the health-related importance of both sleep quality and noise-related public policies.

Key words: sleep disruption, health, noise exposure, instrumental variable estimation

JEL classifications: I10, R23, D00

1 Introduction

While the healing power of sleep has been well recognized since ancient times (i.e. Shakespeare's "Nature's soft nurse[1]"), scientific research on the importance and function of sleep has progressed significantly in the last two decades. The 2017 Nobel prize for Medicine highlighted progress in understanding the biological basis of circadian rhythms, and other recent medical research has linked sleep disruption to disorders of the immune, metabolic, inflammatory, and mood regulation systems (e.g. Spiegel et al., 1999; Lange et al., 2006; Gangwisch, 2014; Morgan and Tsai, 2015; Shukla and Basheer, 2016). However, while animal and laboratory studies strongly point to the critical importance of sleep on health, robust empirical evidence on the magnitude of the impact of relatively common occurances of sleep disruption in otherwise healthy individuals is more elusive. Many observational studies find strong correlations between sleep disruption and a variety of health problems, including cardiovascular disease, obesity, diabetes, depression, and respiratory illness (e.g. Covassin and Singh, 2016; Palmer and Alfano, 2016; Knutson et al., 2006; Zee and Turek, 2006), but as many of these health conditions themselves both create and exacerbate sleep difficulties, there is a reverse causality problem in interpreting these observational correlations as causal effects (Zee and Turek, 2006).

A much more limited set of studies address the problem of causal inference by exploiting 'natural experiments' (for example, Nissenbaum et al., 2012; Gibson and Shrader, 2015) to study the cognitive, productivity, and general self-reported health effects of exogenous shifts in sleep patterns, and this paper contributes to this small but growing body of literature on the non-experimental, but causally identified, health effects of sleep disruption. In particular we analyze the link between sleep disruption and a set of specific, non-cognitive health outcomes, including cardio-vascular problems, cholesterol, blood pressure, asthma, lung disease, bone & joint problems, diabetes, fatigue, headache, alzheimers, depression, and cancer using (plausibly exogenous) exposure to neighbor noise as an instrument. Our analysis exploits a large high quality longitudinal survey of Dutch adults that provides extensive individual health information and allows us to control for a broad array of physical, socio-economic, psychometric, demographic, and residential characteristics. We demonstrate that reported sleep disruption is indeed highly correlated with many undesirable health outcomes. We then isolate the causal impact of sleep using an instrumental variables estimator, with the identifying assumption being that disturbance from neighbour noise is exogenous to health outcomes and affects health only through sleep disruption (after controlling for our physical, socio-economic, demographic, dwelling and neighbourhood characteristics). We explore and interrogate this assumption via a variety of robustness tests, including controlling for both moving house and a psychometric measure of individual sensitivity to disturbance.

[1] King Henry IV part II, 3.1.7.

We find (reduced form) associations between sleep disruption and *all* of our measured health outcomes. However the instrumental variables estimates (that address the problem of reverse causality) find statistically and qualitatively significant causal effects of sleep disruption for only on a subset of these disorders, specifically cardio-vascular, lung, bone & joint diseases (such as arthritis), and headache, all of which have been theoretically linked to sleep duration in the medical literature. Our IV estimator no longer finds any causal impact of sleep disruption on high cholesterol, blood pressure, asthma, diabetes, fatigue, Alzheimers, depression, or cancer. Among these null results, diabetes, high blood pressure (hypertension), asthma, and Alzheimers have been either theoretically or empirically linked to sleep duration in the medical literature and thus are of particular interest and we consider several possible explanations for our findings. Finally, we do not find any significant causal effect of sleep disruption on health outcomes not theoretically linked to sleep, like cancer.

The rest of the paper is organised as follows. Section 2 briefly reviews the science of sleep and health. Section 3 describes the data used in the analysis and outlines the instrumental variables estimation strategy. Section 4 presents our main results, followed by a series of robustness checks. Section 5 concludes with a discussion of policy implications and suggestions for future research.

2 Sleep and Health

At a biological level, scientists have found theoretical and experimental evidence that sleep duration could interact powerfully with both inflammatory processes and the immune system. Thus biological channels have been hypothesized linking sleep quality to cardiovascular, respiratory, metabolic, and auto-immune illnesses, and even alzheimers disease (Spiegel et al., 1999; Shearer et al., 2001; Spiegel et al., 2005; Lange et al., 2006; Gangwisch, 2014; Morgan and Tsai, 2015; Shukla and Basheer, 2016). Laboratory experiments that subject small samples of healthy individuals to sleep deprivation also find adverse effects on cognitive ability, decision-making, and risk-related behaviours (Banks and Dinges, 2007; McKenna et al., 2007). Thus both theory and laboratory findings suggest powerful potential biological mechanisms through which sleep disruption could affect numerous health and behavioural outcomes.

At the same time, numerous observational studies provide empirical evidence of strong *associations* between sleep disturbance and health outcomes, such as cardiovascular disease, respiratory disorders, and even diabetes (Zee and Turek, 2006). For example, Knutson et al. (2006) finds that sleep deficit and glycemic control are correlated in a cross sectional study of 161 volunteers with type-2 diabetes. Gangwisch et al. (2013) finds high blood pressure much more prevalent among women in the Nurses Health Studies (NHS-1 and NHS-2) who sleep fewer than five hours a night, compared to those with seven hours. However, it is difficult to conclude from these

and other observational studies that sleep disruption *caused* particular health outcomes; a plausible alternative explanation is that health problems themselves caused sleep problems, or that an underlying unobserved health condition led to both sleep disruption and disease simultaneously.

A few studies have attempted to get around this endogeneity problem by exploiting arguably exogenous sources of variation in sleep duration to identify the causal effects of sleep quality on health and cognitive outcomes. A popular approach has been to examine the effects of daylight savings time on cognitive ability, with the ensuing one hour of sleep deprivation (in April) or extension (in October) being linked to financial market fluctuations (Kamstra et al., 2000), traffic accidents (Ferguson et al., 1995; Lambe and Cummings, 2000; Varughese and Allen, 2001; Sood and Ghosh, 2007; Harrison, 2013), workplace injuries (Barnes and Wagner, 2009), and overall life satisfaction (Kountouris and Remoundou, 2014). In a clever twist on this theme, Gibson and Shrader (2015) provides causal estimates of the effects of sleep duration on wages using sunset time as an instrument. However, studies that link exogenous variation in sleep quality to non-cognitive-related health outcomes are much scarcer. To date, the only such study *to our knowledge* is that of Nissenbaum et al. (2012), who exploit variation in household distances to industrial wind turbines as an instrument to document the negative general self-reported health effects of noise-related sleep disruption.

This paper adds to this very small literature on the causal effects of sleep disruption on non-cognitive-related health outcomes by analyzing a large longitudinal survey in the Netherlands and exploiting the potential source of exogenous sleep disruption that occurs from the reported presence of noisy neighours. Noisy neighbours present a unique "shock" to an individual's ability to get a good night's sleep; noisy neighbours may come and go, and unlike other sources of environmental noise (such as street or airplane noise), neighbour noise is generally not an *ex-ante* observable characteristic of housing. Furthermore, in many cases the monetary and social costs of relocation are sufficiently high that moving house is not an immediately available option, and thus exposure to noisy neighbours may endure over a significant time frame.

This identification strategy thus rests on three primary assumptions: first, that neighbour noise is a first-order determinant of sleep quality (relevance); second, conditional on controlling for physical, socioeconomic, demographic, dwelling, and neighbourhood characteristics, that there is little or no health-related selection associated with reporting noisy neighbours (exogeneity); and third, that neighbour noise affects health primarily through its effects on sleep quality (the exclusion restriction). The first assumption is relatively uncontroversial; the auditory system is the only sensory system in humans remaining active while asleep (Velluti, 1997) and there is ample evidence that noise has a significant impact on sleep quality (Evans et al., 1998, 2001; Ising et al., 2000; Zaharna et al., 2010). We furthermore empirically confirm this association in our sample of Dutch adults. To address the second assumption we argue that loud neighbours are an *ex-ante* unobservable characteristic of housing. We cite

evidence that moving is very costly in the Netherlands, and we demonstrate in our sample that moving house is uncorrelated with health outcomes. We also explore whether individuals who are easily disturbed generally are more likely to both report noise annoyance and to (independent of sleep disruption) suffer poor health outcomes. Finally, as a robustness exercise, we introduce a second instrument (street noise) and report overidentification tests.

3 Data and Method

3.1 Data

Data for the analysis come from the Longitudinal Internet Survey for the Social Sciences (LISS) panel administered by CentERdata (Tilburg University, The Netherlands). The LISS is a high quality, internet-based annual longitudinal survey from 2008-2013 of over 8000 individuals identified using a true probability sample drawn from the Dutch population, with an enrollment rate of 48% of the total initial sample. Scherpenzeel (2009) evaluates the sampling method and finds that the LISS sample compares favorably to high-standard traditional surveys.[2]

We focus on respondents over 17 years of age in 2007 when the initial survey started, ending up with a total sample size of 5440. The LISS is an ongoing annual survey with multiple waves of question 'modules' sent to participants throughout the year. The primary modules used for this analysis was the Health module, collected in November and December in each wave, and the Housing module, collected in June and July for each wave. Although the LISS survey is longitudinal, not all respondents answered all modules in all years, and for some modules respondents answered only once or twice during the entire survey period. To get rid of transitory shock in health or environment from a specific year, we take average across the seven survey waves from 2007 to 2013. It is also more appropriate for the relationship under this study, as we are primarily interested in the effects of sleep disruption on long-term health problems that may take many years to develop.

In the Health module of the LISS respondents were asked both general and specific questions about their health, including whether they suffered from sleep disturbance (from any cause). Respondents were also asked factual health questions by having them select from a list of possible health problems in response to "Do you regularly suffer from the following diseases/problems;" "Are you currently taking medicine at least once a week?" 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

[2]For details, check Scherpenzeel and Das (2010) or visit www.lissdata.nl.

any of these questions. Specifically, health problems were coded as relating to the cardio-vascular system; joints & bones (including arthritis and skeletal problems), cancer, lung disease (including bronchitis), asthma, diabetes, blood pressure (hypertension), cholesterol, fatigue, headache (including migraine), depression, and alzheimer disease. Unfortunately the Health module was not run in 2014, so our data extends through 2013.

Our instrumental variable, collected from the Housing module, is the binary responses 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. In an auxilliary analysis we also collect information on noise from the street; *Street Noise* takes the value 1 if respondents indicated 'noise annoyance caused by factories, traffic or other street sounds,' at any time during the survey period, and 0 otherwise. Further control variables are drawn across the survey. The Personality module of the LISS survey asks respondents to rank from 1 (very inaccurate) to 5 (very accurate) whether statements about personality characteristics describes them. We use their response to 'Am easily disturbed' to explore to what extent variation in individual sensitivity might explain observed correlations throughout the analysis. In addition to the key variables of interest, the LISS also provides a large amount of data on physical, socio-economic, demographic, housing and neighbourhood characteristics that we use as control variables in the analysis. These include information on monthly household income, education level (from primary to university level), marital status, labor market status, number of hours worked gender, age, whether the respondent has ever smoked, whether they consume more than one alcoholic drink per day, body mass index (BMI), number of children in the household, and whether the respondent is religious. We also include an extensive set of variables describing the respondents' neighborhood and dwelling, including whether the neighborhood is very urban, moderately urban (as the reference category), 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 CENTeR Data allows us to construct an annual indicator of whether a respondent has moved residence during the sample period.

Table 1 presents summary statistics for all variables used in the analysis. The upper panel displays summary statistics for continuous variables. On average, surveyed individuals report a health level of 3.1 out of 5, which indicates 'good' health level. The average age is 49.5 years old, with an average BMI of 25.7. Individuals work about 32.3 hours per week, with a monthly household income of 2968.8 Euros. The lower panel presents frequencies for dichotomous variables. Around 30% experience sleep disruption, and as many as 33.5% report experience of neighbour noise. Notably, exposure to street noise (which is *ex ante* more observable than neighbour

noise) is relatively low, at 19.5%. The most frequent health problem is fatigue (75.9%), followed by bone & joint problems (63.9%).

3.2 Estimation strategy

We start with the reduced form association between sleep disruption and health outcomes as specified below:

$$health_i = \beta_1 sleep_i + X_i'\beta_X + \xi_i, \tag{1}$$

where $health_i$ is a dummy variable indicating having a specific health problem, such as a cardio-vascular condition, high cholesterol, high blood pressure, asthma, lung disease, disease of the joints & bones, diabetes, stroke, fatigue, headache, alzheimers, depression, or cancer. $Sleep_i$ is a dummy variable equal to 1 if an individual i reports sleep disruption and otherwise equals 0. X_i' is a vector of control variables of individual characteristics, which include ever-smoked indicator, alcohol consumption, BMI, age and its squared form, gender, marital status, educational level, household income, labor market status, number of kids, and religion. We also control for dwelling characteristics (air quality, a set of dummy variables if the dwelling is noisy, small, dark, cold, leaky, damp, or rotten), and neighbourhood characteristics (crime rate and urban/rural area).

As discussed above, we cannot interpret $\hat{\beta}_1$ as the *causal* effect of sleep on health due to potential endogeneity, including reverse causality (that ill health leads to sleep disruption) and simultaneity (some underlying unobservable condition that leads to both ill health and sleep disruption). We adopt instead an instrumental variable estimation to examine the causal impact of sleep disruption on health outcomes. Specifically, we exploit reported exposure to neighbourhood noise as an instrument for sleep disruption; in other words, we model sleep disruption as a function of exposure to neighbour noise, and then examine whether this measure of noise-induced sleep disruption is associated with health outcomes. By exploiting variation in sleep disruption only related to neighbour noise, we effectively remove the potential reverse causality and simultaneity between health and sleep, and are thus able to recover causal estimates.

The internal validity of this instrumental variables approach requires three conditions: relevance - that neighbour noise significantly and adversely affects the sleep quality; exogeneity - that those with ill health do not report more noisy neighbours or self-select into noisy dwellings; and exclusion - that the mechanism through which noisy neighbours affect health outcomes is through their effect on sleep disruption. We investigate all three of these assumptions, showing that first, there is ample evidence of noise-moderated sleep disruption in the Dutch sample. Second, the presence of noisy neighbours is largely an *ex-ante* unobservable characteristic of housing (exogenous to health outcomes). The noise incidents attributed to the neighbor households are not

easily anticipated before moving in due to the relative low frequency of occurrence. Housing environment in the Netherlands is generally quiet with legal regulations on noise nuisance from businesses, trains and other kinds of traffic (Hammer et al., 2017). The Netherlands is a country dominated by single-family houses which are distant between each other to reduce noise nuisance, not only in countryside but also in medium-sized cities (Elsinga and Wassenberg, 2014). Even in dense major cities like Amsterdam or Rotterdam, the traditional local architecture style—such as the Canal House—properly reduces noise influence by elevated first floor and the slim and deep floor plan. In addition, the noise incidents caused by neighbors is expected stable over time because of the low residential mobility in the Dutch neighborhoods due to expensive relocation cost and high ownership rate (Helderman et al., 2004; Van Ommeren and Van Leuvensteijn, 2005). Even if moving across neighborhood occurs, we find that that moving house is uncorrelated with health outcomes. Third, variation in personal sensitivity cannot explain the observed correlation between noise, sleep and health; and that, controlling for individual physical, socio-economic, dwelling, and neighbourhood characteristics, noisy neighbours are unlikely to affect health outcomes other than through the channel of sleep disruption. Sleep disturbance indeed attributes to the most annoying forms of neighborhood in the Netherlands by World Health Organization (WHO, 2009).

A more technical issue arises from the fact that the dependent variable, $health$, the explanatory variable of interest, $sleep$, and the instrumental variable, $noise$ are all binary dummy variables. Although a common approach to modelling binary health outcomes is to use nonlinear probit/logit models that constrain the predicted values to lie between 0 and 1 and yield coefficient estimates with convenient odds-ratio interpretations, introducing an instrumental variable derived from a similarly nonlinear first stage regression in two-stage least squares (or alternatively using two-stage residual inclusion, or 2SRI) can generate biased estimates under a wide range of distributional scenarios (Basu et al., 2017) and is generally econometrically controversial, as is illustrated by a contentious debate in the literature (see, for example, Smith and Blundell, 1986; Blundell and Smith, 1989; Blundell and Powell, 2003, 2004; Terza et al., 2008; Bhattacharya et al., 2006).

Indeed, due to the econometric complexity and uncertainty surrounding the correct functional form for binary instrumental variables estimators, Angrist and Pischke (2008) suggest using linear probability models (LPM) within a two-stage least squares (2SLS) approach that produces consistent estimates, easy-to-compute marginal effects, and unbiased standard errors. In their more formal investigation of the statistical properties of binary instrumental variables estimators, Basu et al. (2017) conclude with the same advice; unlike the nonlinear approaches, they show that the use of linear probability models in a 2SLS framework with a binary outcome and a binary instrument produces consistent estimates of the local average treatment effect (LATE) across the entire range of rarity for either outcome or treatment.

Thus, following Angrist and Pischke (2008) and Basu et al. (2017) we adopt a LPM 2SLS estimation frame-

work to generate our instrumental variables estimates. The first stage regression is estimated as:

$$sleep_i = \alpha_1 noise_i + X_i'\alpha_X + \mu_i, \tag{2}$$

where $noise_i$ is a dummy variable which indicates individual i reports noise annoyance caused by neighbours and other variables are as described above. In the second stage estimation, we use instrumented sleep disruption from the first stage to recover the causal relationship between sleep and health outcomes:

$$health_i = \gamma_1 \hat{sleep}_i + X_i'\gamma_X + \epsilon_i, \tag{3}$$

The LPM 2SLS instrumental variables estimator addresses one major problem of inference related to *incidence* in observational studies of sleep and health - reverse causality - effectively. Nevertheless, the nature of the estimator (instrumental variables) combined with the nature of the survey data (cross sectional and primarily binary in nature) combine to preclude the approach from accurately estimating the *magnitude* of an effect in a clinically meaninful way. This point can perhaps be seen most clearly by thinking of the IV estimate as the ratio of two marginal effects - the estimated effect of noise on health (dh/dn) divided by the estimated effect of noise on sleep (ds/dn). Within the linear probability framework the estimate for dh/dn gives us an estimate of the increase in the probability of reporting a health problem if neighbour noise is reported. Dividing dh/dn by ds/dn (the increase in the probability of reporting sleep disruption if neighbour noise is reported) gives us the causal estimate of the likelihood that health problems (of unknown differing severity) will be reported if sleep disturbance (of unknown differing magnitudes) is reported, among the subset of the population who report neighbour noise (of unknown frequency or loudness). This *incidence* estimate is purged of reverse causality and, if it is valid and statistically significant, tells us that sleep disturbance can have a *causal* effect on health.

However, the potential heterogeneity and uncertainty about the degree and extent of neighbour noise, sleep disruption, and health outcomes implies that we should not interpret the coefficient estimates as giving us meaningful information about the *magnitude* of the effect on health for a given increase in the severity of sleep disruption. Thus the IVE estimates help us to estimate whether observed associations could be at least partially driven by causal mechanisms, but, at least with the binary data available, do not allow us to accurately estimate the magnitude of any causal effect. Furthermore, the subset of the population that report neighbour noise, and then do or do not report sleep disruption, may be different in some ways from the general population, and if so, this heterogeneity implies a difference between the population average effect and local average effects (which is what IVE estimates). We further consider these issues of interpretation below in section 4.2 when we discuss the results.

4 Results

4.1 Correlations between sleep and health: reduced form estimates

In Tables 2a-2c we present the baseline correlations (reduced-form results) between sleep disruption and health outcomes, controlling for our full set of socio-economic, physical, and dwelling/neighbourhood variables. For each health outcome, we consider two alternative specifications, (a) and (b). In specification (a) we present the baseline regression 1. In specification (b) we explore the extent to which including additional controls for individual sensitivity to disturbance (*Easily Disturbed*) and moving house during the sample period (*Ever Moved*) in the reduced form equation moderates the coefficient estimate on $sleep$. If the observed correlation between health outcomes and sleep disturbance from specification (a) is driven either by unobserved heterogeneity in sensitivity (for example if poor health is correlated with increased sensitivity and increased sensitivity leads to poor sleep), or by self-selection via moving home, then controlling for our measures of sensitivity to disturbance and home-moving should eliminate or significantly reduce the magnitude and/or statistical significance of the coefficients on $sleep$ in specification (b).

In specification (a) of the reduced form estimates we find large and statistically significant correlations between sleep disruption and *all* of the health outcomes, whether they have been linked in the literature to sleep disruption (such as auto-immune and cardio-vascular disorders), or not (such as cancer). As discussed above we cannot interpret these estimates as *causal* effects of sleep on health, as it is just as possible that poor health leads to disrupted sleep (direct reverse causality).

In specification (b) we explore the extent to which individual sensitivity and self-selection could influence the relationship. We find that individuals who are more easily disturbed are indeed more likely to experience ill health across most of health conditions (with the exception of asthma and diabetes), though again the direction of causality is not known. Having moved house is weakly correlated with less fatigue but not with other health outcomes. More importantly, across all augmented (b) specifications, the inclusion of the additional controls for sensitivity and moving have only small effects on the magnitude and statistical significance of the coefficient estimates on *sleep disturbance*. We interpret the relative stability of the coefficient on *sleep disturbance* between specifications (a) and (b) as strongly suggestive evidence that sensitivity and selection are unlikely to be first-order drivers of the observed correlations between sleep disturbance and health outcomes.

4.2 The causal effects of sleep on health: instrumental variables estimates

A first-order threat to identification in the reduced form estimates presented in section 4.1 is the possibility of reverse causality - that poor health outcomes could cause sleep disruption. To address this possibility we exploit a plausibly exogenous source of variation in sleep disruption: exposure to noisy neighbours. The intuition is that noisy neighbours are an *ex-ante* unobservable characteristic of housing, and/or may arise (unexpectedly) with the arrival of new inhabitants. As long as moving is sufficiently costly and individuals' propensity to report having noisy neighbours does not depend on health, variation across individuals in their reported exposure to noisy neighbours is thus plausibly exogenous with respect to health outcomes.

While these two key identifying assumptions - that moving is sufficiently costly and that health does not drive sensitivity to noise - cannot be definitively proven within the constraints of our analysis, there is good suggestive and quantitative evidence that these potential sources of selection are not first-order drivers of the observed relationships. First, as anyone who has moved house can confirm, relocation does indeed carry significant costs both in time, money, and social terms. Furthermore evidence suggests that moving home is especially costly in the Netherlands; a UNHSP report rated property transfer taxes the Netherlands to be 'High' (Un-Habitat, 2013), and a report from the government indicates that almost three quarters of Dutch rental properties are highly rationed social housing that can have waiting lists that approach 7 years (Government of the Netherlands, 2016). Properties (including rental homes) in the Netherlands are mostly offered unfitted and unfurnished - they lack not only furniture, but also basics like carpets, light fittings, and major appliances. 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.

We address the second concern, that being more easily disturbed could be associated both with poor health outcomes as well as the likelihood of reporting noisy neighbours, by exploring to what extent including self-reported sensitivitiy to disturbance (*Easily Disturbed*) changes the results of the analysis; when we control for *Easily Disturbed* we thus compare individuals who are equally sensitive. However, as sensitivity to disturbance could be either a cause or a *result* of poor health, *Easily Disturbed* itself might arguably be an endogenous variable, and thus, as in Tables 2a-2c, we present the results both without this variable (specification a) and including it (specification b), and focus on the question of coefficient stability between the two specifications. In particular, if the coefficient estimates on $sleepdisruption$ remain relatively stable and robust when both including and excluding *Easily Disturbed* as a control, we argue that it is unlikely that sensitivity to disturbance is a first-order determinant driving the results.

Table 3 presents estimates of the effect of reported neighbor noise on sleep disruption - the first-stage results of the IV estimation. As expected, exposure to neighbourhood noise is positively correlated with sleep disruption. In column (1) we explore the unconditional bivariate relationship and find that, compared to those with no reports of neighbour noise, individuals in the environment with noise from neighbourhood are about 11 percentage points more likely to experience sleep disruption. In column (2) we additionally control for socio-economic, physical, and dwelling/neighbourhood characteristics and the coefficient estimate and statistical significance remain robust. In column (3) we also include *Easily Disturbed* and *Ever Moved*; we are careful not to attribute a causal interpretation as, as discussed above, these latter variables may be endogenous, but significantly we show that even when controlling for these variables, the effect of neighbour noise on sleep disruption remains robust and highly statistically significant with a p-value less than 0.001. Furthermore the F-statistics of all three specifications are above the threshold of 10 suggested by Staiger and Stock (1997) as a test for weak instruments.

With strong first-stage results, we proceed to the second stage using neighbour noise as the instrument for sleep disruption. Results are presented in Table 4 and include the full set of socio-economic, physical and dwelling/neighbourhood controls (not presented to save space but available upon request). In specification (a) we include both *Easily Disturbed* and *Ever Moved*, but because both of these variables could potentially be endogeneous to the analysis, in specification (b) both variables are excluded, and a point of interest will be comparing how robust the esimates and statistical significance of *sleep disruption* are between the two specifications.

The results presented in Table 4 find statistically significant *causal* effects of sleep disruption on cardiovascular disease, bone & joint problems, lung disease, and headache. To the best of our knowledge, despite the potential theoretical biological channels via auto-immune responses, no existing literature has estimated empirical relationships between sleep deprivation and lung disease, bone & joint diseases, or headache with which to compare our results. However the empirical relationship between cardio-vascular disease and sleep has been studied and found to be qualitatively large; for example Chien et al. (2010) compares Americans with insomnia complaints to those without and finds a relative risk ratio of 1.78 of having cardiovascular disease. Ayas et al. (2003) assesses the relationship between self-reported sleep duration and incident coronary heart disease in the Nurse's Health Study, finding conditional relative risk ratios of up to 1.45.

However, as discussed above in section 3.2, it is difficult to directly interpret the *magnitude* of the LPM coefficient estimates from Table 4 in clinically meaninful terms that are comparable to the existing medical literature. A concrete example may be instructive here. In the case of cardio-vascular disease, the (unreported but available upon request) marginal increase in the likelihood of reporting a health problem if a noise problem is reported is 0.032 (roughly we can think of this as 3.2%, although since this is a LPM we need to keep in mind the range that the computation is allowed is greater than the 0-1 bounds we would naturally be considering). At

the same time, from Table 3 regression (14), the increase in the likelihood of reporting sleep disruption if noise is reported is 0.074. The ratio of these, 0.032/0.074, gives us our IVE estimate from Table 4 column (16b), 0.44. Now consider how the estimate would adjust if the denominator (the likelihood of reporting sleep problems given a noise report) decreased - in other words, if hearing a noise was less likely to cause sleep problems - but the numerator (the likelihood of cardio-vascular problem given a noise report) stayed constant. In that case the IVE would find a bigger impact per sleep incident caused by noise, and the IVE estimate would increase. However, the likelihood that neighbour noise translates into sleep disruption will likely vary quite a bit across different circumstances and different people (remember, we also do not know the extent of either), so interpreting the IVE estimate's magnitude (from one dataset) as having clinically meaningful information for assigning risk for cardio-vascular disease, is, in our opinion, a case of data-overreach and over-interpretation.

Combined with the discussion in section 3.2, the example also highlights the importance of the exclusion restriction for understanding and interpreting the results. The IV estimator attributes all the (conditional) relationship between neighbour noise incidence and cardio-vascular incidence to sleep disturbance. However, if the reporting of neighbour noise incidence is causally related to cardio-vascular incidence by some other channel (not controlled for) other than sleep disturbance, this will result in an over-estimate of the *magnitude* of the coefficient. Thus as long as we are confident that our set of control variables is sufficient to ensure that sleep disturbance is the primary, first-order causal channel through which noise and health outcomes are related, the IV estimator will effectively eliminate the problem of reverse-causality and give us a valid indicator of whether a causal channel exists from sleep disturbance to health. However, the fact that we have binary indicator variables combined with the possibility of (untestable but hopefully second-order) violations of the exclusion restriction, together imply that we should not over-interpret the clinical significance of the *magnitudes* of the IV coefficient estimates. In other words, our analysis contributes to the literature by addressing reverse causality concerns to generate causal *incidence* estimates of sleep disturbance on health, but assessing the *extent* of the effects of differing degrees of sleep disruption on health outcomes is left to future research.

The null results presented in Table 4 are just as interesting as the positive results. While the correlational (reduced form) estimates presented in Tables 2a-2c found associations between sleep disruption and *all* of our measured health outcomes, the instrumental variables estimates no longer find any causal impact of sleep disruption on high cholesterol, blood pressure, asthma, diabetes, fatigue, Alzheimers, depression, or cancer. Among these null results, diabetes, high blood pressure (hypertension), asthma, and Alzheimers have been either theoretically or empirically linked to sleep deprivation in the medical literature and thus are of particular interest.

There are several possible explanations for the null results in the IV estimates for conditions that have been linked to sleep disruption in the existing literature. On one hand, the LISS survey only asks very general questions

about health outcomes, depending on yes-no self-reports, and thus our method and data may not be accurate enough to measure the extent of disease, or pick up some kinds of relationships in the existing sample size. For example, for Alzheimers disease we have very few observations (20) and the self-reports of these respondents may not be reliable. In the case of high blood pressure (hypertension), it has been estimated that as many of 17% of adults with hypertension may not have been diagnosed (Mozaffarian et al., 2016), and as we do not observe high blood pressure in these undiagnosed individuals, measurement error could potentially impede inference.

On the other hand, as the medical literature itself has emphasized, observed associations do not prove a causal link and are thus suggestive at best. In the case of diabetes, both theoretical biological channels and controlled laboratory studies have linked sleep disruption to decreased glucose tolerance and insulin sensitivity (Spiegel et al., 2005). Nevertheless, epidemiological evidence that has been based on observational data is limited, and possibly susceptible to reverse causality (Spiegel et al., 2005; Meisinger et al., 2005; Knutson et al., 2006). Likewise, while there are theoretical biological channels that connect sleep to asthma, the direction of causality is controversial (Majde and Krueger, 2005; Bender and Leung, 2005), or linking sleep disruption to intermediate outcomes, such as obesity (Patel and Hu, 2008), that themselves are associated with asthma, but not to asthma itself. In the case of hypertension, the existing evidence shows a strong association with sleep deprivation; not only are there plausible theoretical biological channels and observational studies linking sleep duration with blood pressure, but, in addition, experimental sleep extension has been shown to significantly reduce blood pressure in subjects who have been previously diagnosed with hypertension (Gangwisch, 2014). Nevertheless, evidence that extended sleep in people with hypertension lowers blood pressure is not yet prima facie evidence that shorter sleep duration originally *caused* the hypertension, and Gangwisch (2014) themselves label the relationship an "observed association". Palagini et al. (2013) also summarise that the "causal direction cannot be determined and the strength of these associations may vary by gender, age and type of sleep stage that has been lost". So the case is certainly not yet closed.

Thus overall, consistent with the existing theoretical and empirical clinical literature, we find evidence for a statistically significant causal effect of sleep disruption on cardio-vascular problems. We also find evidence of a causal link from sleep disruption to bone & joint problems, lung disease, and headache; while the existing literature provides plausible theoretical biological channels for these latter health outcomes, there are no comparable clinical/empirical studies and as a result we consider these results to be more suggestive in nature. At the same time, for other health outcomes (especially diabetes, hypertension and asthma) the existing literature has provided either theoretical or empirical evidence (or both) of a link with sleep, while our IVE estimates find no such relationship. While our null results are not supportive of a causal interpretation of previously observed associations in the existing literature, as discussed there are several alternative possible explanations for these

differences as well, so we raise the question of these null results primarily as an indication that further research in these areas would be especially useful.

4.3 Robustness Tests

4.3.1 Further robustness testing: the exclusion restriction

We have provided evidence to argue that neighbour noise fulfills both the relevance and exogeneity conditions necessary to constitute a valid instrument for health outcomes, but our ability to address the exclusion restriction has been necessarily more circumstantial. In particular, while we control for a large set of physical, dwelling, neighbourhood, and socio-economic characteristics, and have presented evidence that it is unlikely to be the case that neighbour noise leads to poor health outcomes via moving house, or that reported neighbor noise and health are linked via individual heterogeneity in sensitivity to disturbance, it is nevertheless impossible to completely rule out all alternative mechanisms other than sleep disruption through which neighbor noise and health could be causally linked. Thus to explore the exclusion restriction further, in Table 5 we present the analysis using *two* noise-related instruments: neighbour *and* street noise. Using two instruments allows us to formally statistically test the exclusion restriction by conducting Anderson-Rubin over-identification tests (although we note these are weak tests). On the other hand, street noise is arguably less convincing as a valid instrumental variable for sleep disruption; busy streets are both clearly observable, potentially inducing a greater degree of health-related selection, and may generate higher levels of localized air pollution, potentially further violating the exclusion restriction.

In the event, the two-instrument IVE results presented in Table 5 reveal a very similar pattern to those found in Table 4. More importantly, in all cases we fail to reject the hypothesis that the instruments are excludable, confirming the statistical validity of the exclusion restriction. Although technically above the critical 5% threshold, we note that the over-identification test for cardio-vascular problems, with p-values under 0.10, is somewhat weaker; this could be consistent with either street or neighbour noise having a weak but direct effect on cardio-vascular health independent of its effect on sleep (and thus leading to IVE estimates of the causal impact of sleep that are somewhat overstated, as discussed above in section 4.2) and leave this question for further research.

4.3.2 Further robustness testing: controlling for possible lifestyle confounders

As described above, the results presented in Table 4 control for a host of dwelling, neighbourhood, physical, and socio-economic characteristics. However there may still be unobservable omitted variables associated with

lifestyle that are correlated with neighbour noise, reported sleep disruption, and health outcomes - in particular, a concern may be that younger people may have a higher propensity for lifestyles that include partying and/or recreational drug use, and may be more likely to live near other young people who are disproportionately noisy (for the same reasons). At the same time, although we control for age (and age-squared), this demographic group may be either more or less likely to suffer particular health outcomes (perhaps related to these lifestyle choices) and this relationship may confound the estimates.

In order to address these potential concerns we carry two additional robustness checks. In Table 6 we exclude all individuals under 30 years old from the analysis. Then in Table 7 we include the full sample, but control explicitly for the self-reported frequency of taking recreational drugs ($RecDrugFreq$), which include sedatives, soft drugs (such as hashish and marijuna), ecstasy, hallucinogens (such as LSD and magic mushroom), and hard drugs (such as stimulants, cocaine, and heroin). In both Tables 6 and 7 the magnitudes of the coefficients and pattern of statistical significance remain highly consistent with the baseline results from Table 4. Thus we conclude that it is highly unlikely that the Table 4 baseline results are being driven by unobservable lifestyle confounders.

5 Discussion

While a considerable body of medical research has documented potential biological channels through which sleep disruption may influence myriad health outcomes, empirical studies of the causal effects of sleep on health in the general population are challenging and rare. Laboratory studies are necessarily limited in scope and duration, and population-wide observational studies have traditionally been unable to identify causality due to the potential for poor health to lead to sleep disruption (i.e. reverse causality).

In this paper we investigate the causal impact of sleep disruption on health using data on a wide variety of health outcomes from a survey of over 5,000 Dutch adults and a novel estimation strategy. In particular, controlling for a broad array of physical, socio-economic, psychometric, demographic, dwelling, and neighbourhood characteristics, we instrument for sleep disruption using self-reported noise from neighbours. We document a highly significant correlation between noisy neighbours and sleep disruption, and provide evidence that our instrumental variables estimates are unlikely to be driven by either selection due to moving homes or individual-level variation in sensitivity to disturbance. However, while our empirical strategy plausibly addresses the main problem of previous observational studies - that of reverse causality - the binary-IV estimator is necessarily limited in its ability to estimate the magnitude of any causal effects identified. Thus we consider the results to provide estimates indicative of causal *incidence*, but not causal *extent*, of the relationship between sleep disruption and

health outcomes.

In the reduced form we find strong associations between sleep disruption and *all* of the health outcomes surveyed. However, these observational associations may be driven by reverse causality if poor health leads to sleep disruption, which seems likely. When we instead estimate *causal* incidence using our instrumental variables strategy that plausibly addresses the problem of reverse causality, we find significant effects of sleep disruption only on cardio-vascular disease, lung disease, bone & joint problems (including arthritis), and headache. All of these health problems have been linked to sleep duration via theoretical potential biological mechanisms in the medical literature, although to our knowledge only cardio-vascular disease has been empirically linked to sleep duration in population studies.

At the same time, unlike the reduced form correlations, our IV approach *fails* to find any causal incidence between sleep disruption and cholesterol, blood pressure (hypertension), asthma, diabetes, fatigue, Alzheimers, depression, or cancer. Among these null results, diabetes, hypertension, asthma, and Alzheimers have been either theoretically or empirically linked to sleep deprivation in the medical literature, so our finding of no causal relationship contributes some cautionary evidence in these cases. Nevertheless, there are multiple possible explanations for the null results, including the possibility that our data is inadequate, or that the population affected by reported neighbour noise may be different from the general population, so more research with a focus on addressing potential sources of reverse causality for these health outcomes would be especially fruitful.

The overall pattern of results is robust to changes in the sample population (i.e. excluding those under 30 years of age) and to additionally controlling for recreational drug use. Formal (albeit weak) over-identification tests provide further confidence that sleep disruption is the most likely, primary first-order mechanism through which reported neighbour noise and health problems are related. However, as it is impossible to fully exclude the possibility that additional mechanisms may play a role for some health outcomes, this possibility of (presumably second-order) violations of the exclusion restriction further justifies our interpretation of the results as indicative of incidence more than extent of the relationships.

Finally, in addition to providing evidence consistent with a causal link between sleep disturbance and health effects associated with cardio-vascular disease, auto-immune disorders (such as arthritis), lung disease, and headache, the paper also highlights some hidden costs of noise pollution. Traditionally neighbour noise has been viewed more of a local nuisance than as a public health issue (Hammer et al., 2017), but as evidence of the health costs of noise mounts the question of noise control should become more of a priority for policy makers and urban planners.

References

Angrist, J. D. and J.-S. Pischke (2008). *Mostly harmless econometrics: An empiricist's companion*. Princeton university press.

Ayas, N. T., D. P. White, J. E. Manson, M. J. Stampfer, F. E. Speizer, A. Malhotra, and F. B. Hu (2003). A prospective study of sleep duration and coronary heart disease in women. *Archives of internal medicine 163*(2), 205–209.

Banks, S. and D. F. Dinges (2007). Behavioral and physiological consequences of sleep restriction. *J Clin Sleep Med 3*(5), 519–528.

Barnes, C. M. and D. T. Wagner (2009). Changing to daylight saving time cuts into sleep and increases workplace injuries. *Journal of applied psychology 94*(5), 1305.

Basu, A., N. Coe, and C. G. Chapman (2017). Comparing 2sls vs 2sri for binary outcomes and binary exposures. Technical report, National Bureau of Economic Research.

Bender, B. G. and D. Y. Leung (2005). Sleep disorders in patients with asthma, atopic dermatitis, and allergic rhinitis. *Journal of allergy and clinical immunology 116*(6), 1200.

Bhattacharya, J., D. Goldman, and D. McCaffrey (2006). Estimating probit models with self-selected treatments. *Statistics in medicine 25*(3), 389–413.

Blundell, R. and J. L. Powell (2003). Endogeneity in nonparametric and semiparametric regression models. *Econometric society monographs 36*, 312–357.

Blundell, R. W. and J. L. Powell (2004). Endogeneity in semiparametric binary response models. *The Review of Economic Studies 71*(3), 655–679.

Blundell, R. W. and R. J. Smith (1989). Estimation in a class of simultaneous equation limited dependent variable models. *The Review of Economic Studies 56*(1), 37–57.

Chien, K.-L., P.-C. Chen, H.-C. Hsu, T.-C. Su, F.-C. Sung, M.-F. Chen, and Y.-T. Lee (2010). Habitual sleep duration and insomnia and the risk of cardiovascular events and all-cause death: report from a community-based cohort. *Sleep 33*(2), 177–184.

Covassin, N. and P. Singh (2016). Sleep duration and cardiovascular disease risk: Epidemiologic and experimental evidence. *Sleep Medicine Clinics*.

Elsinga, M. and F. Wassenberg (2014). Social housing in the netherlands. *Social Housing in Europe*, 21–40.

Evans, G. W., M. Bullinger, and S. Hygge (1998). Chronic noise exposure and physiological response: A prospective study of children living under environmental stress. *Psychological science 9*(1), 75–77.

Evans, G. W., P. Lercher, M. Meis, H. Ising, and W. W. Kofler (2001). Community noise exposure and stress in children. *The Journal of the Acoustical Society of America 109*(3), 1023–1027.

Ferguson, S. A., D. F. Preusser, A. K. Lund, P. L. Zador, and R. G. Ulmer (1995). Daylight saving time and motor vehicle crashes: the reduction in pedestrian and vehicle occupant fatalities. *American Journal of Public Health 85*(1), 92–95.

Gangwisch, J. E. (2014). A review of evidence for the link between sleep duration and hypertension. *American journal of hypertension 27*(10), 1235–1242.

Gangwisch, J. E., D. Feskanich, D. Malaspina, S. Shen, and J. P. Forman (2013). Sleep duration and risk for hypertension in women: results from the nurses health study. *American journal of hypertension 26*(7), 903–911.

Gibson, M. and J. Shrader (2015). Time use and productivity: The wage returns to sleep.

Government of the Netherlands (2016). Government of the Netherlands website. Accessed: 2016-02-25.

Hammer, M. S., Y. Fan, S. S. Hammer, T. K. Swinburn, M. Weber, D. Weinhold, and R. L. Neitzel (2017). Applying a novel environmental health framework theory (i-act) to noise pollution policies in the united states, united kingdom, and the netherlands. *Journal of Environmental Planning and Management*, 1–22.

Harrison, Y. (2013). The impact of daylight saving time on sleep and related behaviours. *Sleep medicine reviews 17*(4), 285–292.

Helderman, A. C., C. H. Mulder, and M. Ham (2004). The changing effect of home ownership on residential mobility in the netherlands, 1980–98. *Housing Studies 19*(4), 601–616.

Ising, H., C. Braun, et al. (2000). Acute and chronic endocrine effects of noise: review of the research conducted at the institute for water, soil and air hygiene. *Noise and health 2*(7), 7.

Kamstra, M. J., L. A. Kramer, and M. D. Levi (2000). Losing sleep at the market: The daylight saving anomaly. *The American Economic Review 90*(4), 1005–1011.

Knutson, K. L., A. M. Ryden, B. A. Mander, and E. Van Cauter (2006). Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. *Archives of internal medicine 166*(16), 1768–1774.

Kountouris, Y. and K. Remoundou (2014). About time: Daylight saving time transition and individual well-being. *Economics Letters 122*(1), 100–103.

Lambe, M. and P. Cummings (2000). The shift to and from daylight savings time and motor vehicle crashes. *Accident Analysis & Prevention 32*(4), 609–611.

Lange, T., S. Dimitrov, H.-L. Fehm, J. Westermann, and J. Born (2006). Shift of monocyte function toward cellular immunity during sleep. *Archives of internal medicine 166*(16), 1695–1700.

Majde, J. A. and J. M. Krueger (2005). Links between the innate immune system and sleep. *Journal of Allergy and Clinical Immunology 116*(6), 1188–1198.

McKenna, B. S., D. L. Dickinson, H. J. Orff, and S. Drummond (2007). The effects of one night of sleep deprivation on known-risk and ambiguous-risk decisions. *Journal of sleep research 16*(3), 245–252.

Meisinger, C., M. Heier, H. Loewel, et al. (2005). Sleep disturbance as a predictor of type 2 diabetes mellitus in men and women from the general population. *Diabetologia 48*(2), 235–241.

Morgan, D. and S. C. Tsai (2015). Sleep and the endocrine system. *Critical care clinics 31*(3), 403–418.

Mozaffarian, D., E. J. Benjamin, A. S. Go, D. K. Arnett, M. J. Blaha, M. Cushman, S. R. Das, S. de Ferranti, J.-P. Després, H. J. Fullerton, et al. (2016). Heart disease and stroke statistics2016 update: a report from the american heart association. *Circulation 133*(4), e38–e360.

Nissenbaum, M. A., J. J. Aramini, C. D. Hanning, et al. (2012). Effects of industrial wind turbine noise on sleep and health. *Noise and Health 14*(60), 237.

Palagini, L., R. Maria Bruno, A. Gemignani, C. Baglioni, L. Ghiadoni, and D. Riemann (2013). Sleep loss and hypertension: a systematic review. *Current pharmaceutical design 19*(13), 2409–2419.

Palmer, C. A. and C. A. Alfano (2016). Sleep and emotion regulation: An organizing, integrative review. *Sleep Medicine Reviews*.

Patel, S. R. and F. B. Hu (2008). Short sleep duration and weight gain: a systematic review. *Obesity 16*(3), 643–653.

Praag, B. V. and B. Baarsma (2005). Using Happiness Surveys to Value Intangibles: The Case of Airport Noise*. *The Economic Journal 115*(2000), 224–246.

Scherpenzeel, A. C. and M. Das (2010). True Longitudinal and Probability-Based Internet Panels: Evidence from the Netherlands. In M. Das, P. Ester, and L. Kaczmirek (Eds.), *Social and Behavioral Research and the Internet: Advances in Applied Methods and Research Strategies*, pp. 77–104. Boca Raton: Taylor & Francis.

Shearer, W. T., J. M. Reuben, J. M. Mullington, N. J. Price, B.-N. Lee, E. Smith, M. P. Szuba, H. P. Van Dongen, and D. F. Dinges (2001). Soluble tnf-α receptor 1 and il-6 plasma levels in humans subjected to the sleep deprivation model of spaceflight. *Journal of Allergy and Clinical Immunology 107*(1), 165–170.

Shukla, C. and R. Basheer (2016). Metabolic signals in sleep regulation: recent insights. *Nature and Science of Sleep 8*, 9.

Smith, R. J. and R. W. Blundell (1986). An exogeneity test for a simultaneous equation tobit model with an application to labor supply. *Econometrica: Journal of the Econometric Society*, 679–685.

Sood, N. and A. Ghosh (2007). The short and long run effects of daylight saving time on fatal automobile crashes. *The BE Journal of Economic Analysis & Policy 7*(1).

Spiegel, K., K. Knutson, R. Leproult, E. Tasali, and E. Van Cauter (2005). Sleep loss: a novel risk factor for insulin resistance and type 2 diabetes. *Journal of applied physiology 99*(5), 2008–2019.

Spiegel, K., R. Leproult, and E. Van Cauter (1999). Impact of sleep debt on metabolic and endocrine function. *The Lancet 354*(9188), 1435–1439.

Staiger, D. and J. H. Stock (1997). Instrumental variables regressions with weak instruments. *Econometrica* (3), 557–586.

Terza, J. V., A. Basu, and P. J. Rathouz (2008). Two-stage residual inclusion estimation: addressing endogeneity in health econometric modeling. *Journal of health economics 27*(3), 531–543.

Un-Habitat (2013). *Property Tax Regimes in Europe*.

Van Ommeren, J. and M. Van Leuvensteijn (2005). New evidence of the effect of transaction costs on residential mobility. *Journal of regional Science 45*(4), 681–702.

Varughese, J. and R. P. Allen (2001). Fatal accidents following changes in daylight savings time: the american experience. *Sleep medicine 2*(1), 31–36.

Velluti, R. A. (1997). Interactions between sleep and sensory physiology. *Journal of Sleep Research*, 61–77.

WHO (2009). *Night Noise Guidelines for Europe*.

Zaharna, M., C. Guilleminault, et al. (2010). Sleep, noise and health: review. *Noise and Health 12*(47), 64.

Zee, P. C. and F. W. Turek (2006). Sleep and health: everywhere and in both directions. *Archives of internal medicine 166*(16), 1686–1688.

Tables

Table 1: Summary Statistics

Continuous Variables					
Variable	Obs	Mean	St. Dev.	Min	Max
Health Level	5104	3.12	0.64	1	5
Easily Disturbed	5104	2.7	0.85	1	5
BMI	5104	25.7	4.3	11	50
Age	5104	49.5	13.1	19.5	88.5
HH number of kids	5104	0.88	1.02	0	6
HH Income	5104	2,968.8	2,863	250	126,111
Hours	5104	32.3	12.9	0	106
Rec Drugs (freq.)	570	1.14	2.20	0.03	31

Dichotomous Variables

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

Variable	Obs	Frequency	Variable	Obs	Frequency
Sleep Disruption	5104	0.302	Unemployed	5104	0.091
Neighbour Noise	5104	0.335	Housewife	5104	0.397
Street Noise	5104	0.195	Student	5104	0.073
Bad Air	5104	0.103	Retired	5104	0.327
Cardiovascular	5104	0.147	Primary Education	5104	0.015
Cholesterol	5102	0.150	Secondary Education	5104	0.163
Blood Pressure	5102	0.238	Post-Secondary Education	5104	0.683
Asthma	5099	0.057	Tertiary Education	5104	0.175
Lung Disease	5104	0.137	Religious	5104	0.453
Bones&Joints	5104	0.639	Crime in Area	5104	0.164
Diabetes	5102	0.074	Urban Area	5104	0.593
Fatigue	5104	0.759	Rural Area	5104	0.247
Headache	5104	0.272	Ever Moved	5195	0.229
Alzheimer	5049	0.004	Dwelling dark	5104	0.040
Depression	5104	0.083	Dwelling cold	5104	0.060
Cancer	5049	0.042	Dwelling leaky	5104	0.039
Ever Smoked	5104	0.661	Dwelling damp	5104	0.081
Daily Drinker	5104	0.251	Dwelling rotten	5104	0.033
Male	5104	0.506	Dwelling small	5104	0.136
Married	5440	0.776	Rec Drugs	4589	0.124

Table 2a: Correlation between sleep disruption and health outcomes: reduced form regressions

	(1) cardio-vascular		(2) cholesterol		(3) blood pressure		(4) asthma	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.090***	0.080***	0.076***	0.067***	0.099***	0.090***	0.023**	0.022**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.003)	(0.006)
Smoker	0.014	0.014	0.032**	0.032**	0.022	0.022	-0.002	-0.002
	(0.160)	(0.154)	(0.002)	(0.002)	(0.053)	(0.051)	(0.786)	(0.794)
BMI	0.005***	0.005***	0.009***	0.009***	0.019***	0.019***	0.003***	0.003***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.001)	(0.001)
Age	-0.012***	-0.013***	0.001	0.000	0.002	0.002	-0.001	-0.001
	(0.000)	(0.000)	(0.791)	(0.874)	(0.427)	(0.521)	(0.480)	(0.635)
Age^2	0.018***	0.018***	0.007*	0.007*	0.007*	0.008*	0.002	0.001
	(0.000)	(0.000)	(0.027)	(0.025)	(0.019)	(0.016)	(0.379)	(0.481)
Male	0.038**	0.049***	0.070***	0.080***	0.015	0.025	-0.023**	-0.023**
	(0.002)	(0.000)	(0.000)	(0.000)	(0.259)	(0.070)	(0.006)	(0.009)
Married	0.031*	0.029*	0.027*	0.025*	0.014	0.013	0.005	0.005
	(0.010)	(0.017)	(0.032)	(0.047)	(0.282)	(0.352)	(0.541)	(0.554)
University	-0.010	-0.006	-0.017	-0.014	-0.016	-0.013	0.018	0.018
	(0.475)	(0.649)	(0.212)	(0.302)	(0.281)	(0.387)	(0.060)	(0.062)
HH Income	-0.038***	-0.037**	-0.027*	-0.025*	0.004	0.005	-0.006	-0.006
	(0.001)	(0.001)	(0.027)	(0.037)	(0.781)	(0.696)	(0.413)	(0.421)
Easily Disturbed		0.029***		0.026***		0.027***		0.002
		(0.000)		(0.000)		(0.000)		(0.629)
Ever moved		-0.022		-0.015		-0.018		0.009
		(0.055)		(0.205)		(0.155)		(0.310)
N	5104	5104	5102	5102	5102	5102	5099	5099
R^2	0.115	0.120	0.163	0.166	0.209	0.212	0.015	0.015

robust *p*-values in parentheses

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

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

Table 2b: Correlation between sleep disruption and health outcomes: reduced form regressions (cont.)

	(5) lung disease		(6) bone & joint		(7) diabetes		(8) fatigue	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.116***	0.107***	0.170***	0.147***	0.020*	0.017*	0.121***	0.103***
	(0.000)	(0.000)	(0.000)	(0.000)	(0.019)	(0.048)	(0.000)	(0.000)
Smoker	0.030**	0.031**	0.054***	0.054***	0.018*	0.018*	0.037**	0.038**
	(0.002)	(0.002)	(0.000)	(0.000)	(0.012)	(0.012)	(0.004)	(0.003)
BMI	0.008***	0.009***	0.010***	0.010***	0.011***	0.011***	0.004**	0.004**
	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.000)	(0.004)	(0.003)
Age	-0.011***	-0.011***	0.008*	0.010**	-0.003	-0.004	-0.018***	-0.019***
	(0.000)	(0.000)	(0.022)	(0.005)	(0.106)	(0.084)	(0.000)	(0.000)
Age^2	0.013***	0.013***	-0.002	-0.004	0.006**	0.007**	0.013***	0.013***
	(0.000)	(0.000)	(0.585)	(0.314)	(0.008)	(0.006)	(0.000)	(0.000)
Male	-0.015	-0.005	-0.073***	-0.052**	0.022*	0.026**	-0.057***	-0.038*
	(0.220)	(0.656)	(0.000)	(0.002)	(0.012)	(0.005)	(0.000)	(0.012)
Married	0.026*	0.024	0.037*	0.032	0.012	0.011	0.013	0.010
	(0.037)	(0.054)	(0.029)	(0.055)	(0.171)	(0.195)	(0.370)	(0.524)
University	0.009	0.012	-0.007	-0.002	-0.006	-0.005	0.004	0.010
	(0.486)	(0.368)	(0.726)	(0.931)	(0.511)	(0.600)	(0.814)	(0.542)
HH Income	-0.046***	-0.044***	-0.028	-0.025	-0.016	-0.015	-0.040**	-0.037**
	(0.000)	(0.000)	(0.079)	(0.126)	(0.065)	(0.074)	(0.004)	(0.008)
Easily Disturbed		0.027***		0.063***		0.009		0.053***
		(0.000)		(0.000)		(0.057)		(0.000)
Ever moved		-0.007		0.025		-0.011		-0.036*
		(0.560)		(0.135)		(0.211)		(0.015)
N	5104	5104	5104	5104	5102	5102	5104	5104
R^2	0.083	0.086	0.104	0.115	0.092	0.093	0.066	0.076

robust p-values in parentheses

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

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

Table 2c: Correlation between sleep disruption and health outcomes: reduced form regressions (cont.)

	(9) headache		(10) Alzheimers		(11) depression		(12) cancer	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.202***	0.179***	0.009***	0.009***	0.106***	0.090***	0.034***	0.031***
	(0.000)	(0.000)	(0.000)	(0.001)	(0.000)	(0.000)	(0.000)	(0.000)
Smoker	0.009	0.009	-0.001	-0.001	0.009	0.010	0.002	0.003
	(0.520)	(0.484)	(0.681)	(0.689)	(0.248)	(0.223)	(0.660)	(0.647)
BMI	0.004**	0.004**	0.000	0.000	0.003**	0.003**	-0.000	-0.000
	(0.008)	(0.007)	(0.523)	(0.522)	(0.001)	(0.001)	(0.425)	(0.411)
Age	-0.003	-0.002	-0.000	-0.000	0.005*	0.006**	-0.004*	-0.004
	(0.275)	(0.621)	(0.496)	(0.510)	(0.020)	(0.006)	(0.031)	(0.059)
Age^2	0.000	-0.002	0.001	0.001	-0.005*	-0.006**	0.007**	0.006**
	(0.997)	(0.614)	(0.356)	(0.374)	(0.026)	(0.009)	(0.002)	(0.003)
Male	-0.158***	-0.137***	0.001	0.002	-0.026**	-0.011	0.001	0.003
	(0.000)	(0.000)	(0.499)	(0.293)	(0.009)	(0.277)	(0.839)	(0.644)
Married	0.039*	0.034*	0.007***	0.007***	0.009	0.006	0.009	0.008
	(0.015)	(0.032)	(0.000)	(0.000)	(0.350)	(0.540)	(0.209)	(0.233)
University	-0.008	-0.003	-0.000	0.000	-0.000	0.003	-0.001	-0.001
	(0.624)	(0.851)	(0.943)	(0.975)	(0.974)	(0.754)	(0.930)	(0.950)
HH Income	-0.044**	-0.040**	-0.006*	-0.006*	-0.050***	-0.048***	-0.010	-0.009
	(0.005)	(0.009)	(0.017)	(0.019)	(0.000)	(0.000)	(0.203)	(0.221)
Easily Disturbed		0.062***		0.002		0.043***		0.006
		(0.000)		(0.097)		(0.000)		(0.088)
Ever moved		0.023		-0.001		0.009		0.009
		(0.148)		(0.654)		(0.361)		(0.197)
N	5104	5104	5049	5049	5104	5104	5049	5049
R^2	0.129	0.142	0.015	0.016	0.067	0.082	0.049	0.050

robust *p*-values in parentheses

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

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

Table 3: Instrumental variables estimation: first stage regressions

	(13) Sleep Disruption	(14) Sleep Disruption	(15) Sleep Disruption
Neighbour Noise	0.111***	0.074***	0.058***
	(0.000)	(0.000)	(0.000)
Ever Smoked		0.033*	0.033*
		(0.018)	(0.015)
BMI		0.007***	0.007***
		(0.000)	(0.000)
Age		0.007*	0.011**
		(0.040)	(0.002)
Age^2		-0.003	-0.006
		(0.410)	(0.087)
Male		-0.117***	-0.078***
		(0.000)	(0.000)
Married		-0.009	-0.017
		(0.574)	(0.287)
University		-0.020	-0.011
		(0.262)	(0.528)
Household Income		-0.068***	-0.061***
		(0.000)	(0.000)
Easily Disturbed			0.102***
			(0.000)
Ever moved			0.052**
			(0.001)
N	5102	5102	5102
R^2	0.013	0.079	0.113
$F-statistic$	67.44***	14.48***	20.24***

robust p-values in parentheses

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

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

Table 4: Instrumental Variables Estimation

	(16) cardio-vascular		(17) cholesterol		(18) blood pressure		(19) asthma	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.484*	0.444**	0.036	0.085	0.059	0.108	0.197	0.166
	(0.019)	(0.006)	(0.849)	(0.570)	(0.773)	(0.504)	(0.154)	(0.121)
Easily Disturbed	-0.013		0.030		0.030		-0.016	
	(0.572)		(0.157)		(0.177)		(0.270)	
Ever moved	-0.044*		-0.014		-0.016		-0.000	
	(0.011)		(0.393)		(0.333)		(0.971)	
N	5104	5104	5102	5102	5102	5102	5099	5099
	(20) lung disease		(21) bone & joint		(22) diabetes		(23) fatigue	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.433*	0.410**	1.402***	1.262***	-0.071	-0.040	-0.089	0.035
	(0.029)	(0.008)	(0.000)	(0.000)	(0.592)	(0.699)	(0.697)	(0.845)
Easily Disturbed	-0.007		-0.068		0.018		0.073**	
	(0.731)		(0.111)		(0.219)		(0.003)	
Ever moved	-0.025		-0.043		-0.006		-0.026	
	(0.146)		(0.202)		(0.600)		(0.189)	
N	5104	5104	5104	5104	5102	5102	5104	5104
	(24) headache		(25) Alzheimer		(26) depression		(27) cancer	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.766**	0.764***	0.022	0.023	0.162	0.228	-0.096	-0.055
	(0.005)	(0.000)	(0.445)	(0.333)	(0.275)	(0.056)	(0.373)	(0.510)
Easily Disturbed	0.001		0.001		0.035*		0.019	
	(0.979)		(0.828)		(0.030)		(0.114)	
Ever moved	-0.009		-0.002		0.005		0.016	
	(0.712)		(0.457)		(0.688)		(0.081)	
N	5104	5104	5049	5049	5104	5104	5049	5049

robust p-values in parentheses

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

Included in regression but not shown: Dwelling characteristics, neighbourhood characteristics, BMI, Age, Age^2, Male, Married, HH income, educational level, labor market status alcohol consumption, number of children, religious status.

Table 5: Robustness: Use Neighbour Noise and Street Noise as Instruments

	(28) cardio-vascular		(29) cholesterol		(30) blood pressure		(31) asthma	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.279	0.290*	0.050	0.084	0.170	0.180	0.146	0.134
	(0.072)	(0.023)	(0.744)	(0.505)	(0.314)	(0.196)	(0.185)	(0.139)
Easily Disturbed	0.009		0.028		0.019		-0.011	
	(0.620)		(0.105)		(0.323)		(0.354)	
Ever moved	-0.033*		-0.014		-0.023		0.002	
	(0.024)		(0.328)		(0.155)		(0.829)	
N	5104	5104	5102	5102	5102	5102	5099	5099
over-id test	3.388	3.622	0.016	0.000	0.865	0.684	0.435	0.348
$p-value$	0.066	0.057	0.901	0.992	0.352	0.408	0.509	0.555

	(32) lung disease		(33) bone & joint		(34) diabetes		(35) fatigue	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.371*	0.363**	1.235***	1.155***	-0.101	-0.069	0.051	0.112
	(0.020)	(0.005)	(0.000)	(0.000)	(0.349)	(0.431)	(0.778)	(0.454)
Easily Disturbed	-0.001		-0.051		0.021		0.058**	
	(0.961)		(0.127)		(0.082)		(0.004)	
Ever moved	-0.021		-0.034		-0.004		-0.034	
	(0.163)		(0.243)		(0.688)		(0.062)	
N	5104	5104	5104	5104	5102	5102	5104	5104
over-id test	0.316	0.341	0.657	0.557	0.157	0.289	1.208	0.712
$p-value$	0.574	0.559	0.418	0.456	0.692	0.591	0.272	0.399

	(36) headache		(37) Alzheimer		(38) depression		(39) cancer	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.712***	0.722***	0.047	0.041	0.112	0.180	-0.023	-0.005
	(0.001)	(0.000)	(0.150)	(0.121)	(0.343)	(0.071)	(0.795)	(0.946)
Easily Disturbed	0.006		-0.002		0.041**		0.011	
	(0.791)		(0.588)		(0.002)		(0.259)	
Ever moved	-0.006		-0.003		0.008		0.012	
	(0.787)		(0.235)		(0.497)		(0.147)	
N	5104	5104	5049	5049	5104	5104	5049	5049
over-id test	0.123	0.139	1.747	1.742	0.339	0.607	1.357	1.234
$p-value$	0.726	0.710	0.186	0.187	0.561	0.436	0.244	0.267

robust *p*-values in parentheses

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

Included in regression but not shown: Dwelling characteristics, neighbourhood characteristics, BMI, Age, Age^2, Male, Married, HH income, educational level, labor market status alcohol consumption, number of children, religious status.

Table 6: Robustness - Exclude Individuals under 30 Years Old

	(40) cardio-vascular		(41) cholesterol		(42) blood pressure		(43) asthma	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.439*	0.422**	0.053	0.098	0.046	0.095	0.211	0.182
	(0.020)	(0.006)	(0.772)	(0.515)	(0.816)	(0.555)	(0.101)	(0.080)
Easily Disturbed	-0.005		0.030		0.033		-0.017	
	(0.798)		(0.133)		(0.120)		(0.209)	
Ever moved	-0.038*		-0.015		-0.018		-0.001	
	(0.026)		(0.349)		(0.295)		(0.909)	
N	4606	4606	4604	4604	4604	4604	4603	4603
	(44) lung disease		(45) bone & joint		(46) diabetes		(47) fatigue	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.432*	0.418**	1.330***	1.227***	-0.083	-0.053	-0.059	0.054
	(0.019)	(0.006)	(0.000)	(0.000)	(0.517)	(0.615)	(0.786)	(0.760)
Easily Disturbed	-0.005		-0.057		0.019		0.074**	
	(0.786)		(0.132)		(0.177)		(0.001)	
Ever moved	-0.021		-0.050		-0.004		-0.029	
	(0.218)		(0.119)		(0.752)		(0.148)	
N	4606	4606	4606	4606	4604	4604	4606	4606
	(48) headache		(49) Alzheimer		(50) depression		(51) cancer	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.763**	0.765***	0.025	0.024	0.182	0.237*	-0.056	-0.030
	(0.002)	(0.000)	(0.331)	(0.258)	(0.187)	(0.041)	(0.570)	(0.714)
Easily Disturbed	0.002		0.000		0.034*		0.014	
	(0.934)		(0.961)		(0.025)		(0.193)	
Ever moved	-0.010		-0.002		0.005		0.016	
	(0.672)		(0.312)		(0.716)		(0.080)	
N	4606	4606	4536	4536	4606	4606	4536	4536

robust p-values in parentheses

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

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

Table 7: Robustness: Control for Drug Taking Frequency

	(52) cardio-vascular		(53) cholesterol		(54) blood pressure		(55) asthma	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.476*	0.441**	-0.030	0.026	0.017	0.076	0.271	0.230*
	(0.021)	(0.008)	(0.874)	(0.868)	(0.936)	(0.649)	(0.060)	(0.044)
Easily Disturbed	-0.014		0.034		0.037		-0.024	
	(0.518)		(0.105)		(0.097)		(0.125)	
Ever moved	-0.046**		-0.012		-0.021		-0.003	
	(0.008)		(0.464)		(0.234)		(0.800)	
N	4589	4589	4589	4589	4589	4589	4588	4588
	(56) lung disease		(57) bone & joint		(58) diabetes		(59) fatigue	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.534*	0.492**	1.244***	1.150***	-0.143	-0.099	1.014***	1.020***
	(0.011)	(0.003)	(0.000)	(0.000)	(0.290)	(0.358)	(0.001)	(0.000)
Easily Disturbed	-0.020		-0.050		0.026		0.006	
	(0.380)		(0.193)		(0.086)		(0.852)	
Ever moved	-0.035		-0.035		-0.004		-0.019	
	(0.055)		(0.269)		(0.755)		(0.468)	
N	4589	4589	4589	4589	4589	4589	4589	4589
	(60) headache		(61) Alzheimer		(62) depression		(63) cancer	
	(a)	(b)	(a)	(b)	(a)	(b)	(a)	(b)
Sleep Disruption	0.916**	0.882***	0.010	0.013	0.201	0.236*	-0.112	-0.075
	(0.002)	(0.000)	(0.718)	(0.571)	(0.163)	(0.046)	(0.315)	(0.396)
Easily Disturbed	-0.018		0.002		0.020		0.020	
	(0.575)		(0.542)		(0.209)		(0.111)	
Ever moved	-0.011		-0.001		0.006		0.012	
	(0.658)		(0.560)		(0.651)		(0.190)	
N	4589	4589	4553	4553	4589	4589	4553	4553

robust p-values in parentheses

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

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