

Road traffic noise, noise sensitivity, psychological and physical health and mortality

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Research

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Abstract

Background:

Both physical and psychological health outcomes have been associated with exposure to environmental noise. It is not known whether all individuals are equally susceptible to these effects. Noise sensitivity has chiefly been examined in studies of annoyance where it has been shown to moderate the annoyance responses to transport-related noise. Noise sensitivity could have the same moderating effect on physical and psychological health outcomes related to environmental noise exposure but this has been little tested. Noise sensitivity which is also associated with sensitivity to chemicals, light and odours could be an indicator of a more pervasive susceptibility to ill-health related to environmental sources.

Methods:

A cohort of 2398 men between 45 and 59 years, the longitudinal Caerphilly Collaborative Heart Disease study, was established in 1984/88 and followed into the mid-1990 s. Road traffic noise maps were assessed at baseline. Baseline psychological ill-health measures were measured in phase 2 in 1984/88, at phase 3 follow up 1989/93 and phase 4 follow up in 1993/6. Ischaemic heart disease and risk factors were measured in clinic and by questionnaire at baseline and through hospital records and administrative records of deaths during follow up. This study aimed to test if noise sensitivity has a moderating effect on road traffic noise and psychological ill-health and secondly if noise sensitivity predicted physical and psychological ill- health and mortality, irrespective of exposure to road traffic noise.

Results:

Road traffic noise was associated with Phase 4 psychological ill-health but only among those exposed to 56-60dBA ($OR = 1.98$ 95%CI 1.21, 3.24). High noise sensitivity was associated with lower mortality risk ($HR = 0.71$, 95%CI 0.54–0.94). High noise sensitivity was associated longitudinally with psychological ill-health at phase 3 ($OR = 1.82$ 95%CI 1.30, 2.56). There was also weak evidence that noise sensitivity moderated the association of road traffic noise exposure with psychological ill-health.

Conclusions:

Noise sensitivity is a specific predictor of psychological ill-health and may be an indicator of current psychological ill-health as part of a wider construct of environmental susceptibility. It may increase the risk of psychological ill-health when exposed to road traffic noise.

Background

Studies have linked long term exposure to road traffic noise to increased hypertension risk, myocardial infarction, cardiovascular and stroke mortality [1, 2]. There is accumulating evidence that transport noise is related to an increased risk of depression, hypertension, cardiovascular disease and mortality [3, 4, 5, 6, 7, 8]. The stress hypothesis has been put forward as the most likely mechanism underlying the effects of environmental noise on health where chronic noise exposure of sufficient intensity leads to increased stress responses, hypertension, metabolic syndrome, diabetes mellitus and increased risk of cardiovascular disease [9].

Noise sensitivity, based on scales of self-report responses to a range of sounds, has been used to differentiate people with a strong dislike of noise from those who are indifferent to noise or who are not bothered at all by noise, so-called 'imperturbables' [10]. Does everyone respond physiologically to noise exposure in the same way – probably not? However, whether high self-reported noise sensitivity equates to high levels of physiological responsiveness to noise and subsequent greater susceptibility to disease than for those with low noise sensitivity is uncertain. Noise sensitivity has been associated with some indices of raised physiological response (e.g. tonic heart rate and defence/startle responses to noise in the laboratory) but the strengths of the associations between autonomic nervous system functioning and noise exposure tend to be weak and inconsistent [11].

In one area there is some consistency: that noise sensitivity has a moderating effect on another self-report variable, annoyance to noise, shown across a range of studies [12, 13, 14]. There has been controversy over whether high levels of annoyance might be a transitional stage on the pathway to disease with theories of this mechanism also operating through direct effects of environmental noise on physiological responses without a mediating step of cognitive appraisal [15]. The evidence is scant on the more distant link of noise sensitivity as a moderator of the effects of environmental noise on physical ill-health, for instance, cardiovascular outcomes [16] and possibly more likely, psychological ill-health.

Noise sensitivity has associations with ill-health, for instance, with the award of disability pensions in Finland [17]. From twin studies there is evidence of an underlying genetic basis to noise sensitivity which could be linked to susceptibility to ill-health [18]. Studies have repeatedly found largely cross sectional associations with both psychological ill-health [11, 19, 20] and personality traits such as neuroticism and trait anxiety [21, 22, 23]. Noise sensitivity is also associated to other environmental stimuli [24]. Could trait anxiety, or a similar concept, negative affectivity, be part of a unifying construct of fearfulness of the risks of the external world that underlies noise sensitivity and a range of environmental sensitivities? In longitudinal analyses, in a UK study of civil servants noise sensitivity predicted common mental disorder but not coronary heart disease or cardiovascular mortality except in certain subgroups, namely as a predictor of angina in lower employment grades in the UK civil service [25]. In the Caerphilly study, at earlier phases, road traffic noise was demonstrated to be related longitudinally to symptoms of anxiety but not to more general measures of common mental disorder including depression as well as anxiety [26]. Whether noise sensitivity moderates the effects of road traffic noise on psychological ill-health remains untested.

In this paper, in the Caerphilly Collaborative Heart Disease Study, a longitudinal cohort study of men, we examine: 1) the longitudinal association between road traffic noise and cardiovascular morbidity and mortality and 2) whether noise sensitivity might moderate these associations; 3) the longitudinal association between road traffic noise and psychological ill-health and 4) whether noise sensitivity might moderate these associations. We also tested if noise sensitivity, independently of noise exposure, was a longitudinal predictor of ischaemic heart disease morbidity and mortality and psychological ill-health. Our hypotheses were: i) road traffic noise exposure at baseline (phase 2) will predict ischaemic heart disease morbidity and mortality at follow-up; ii) noise sensitivity will not moderate the association between traffic noise exposure and ischaemic heart disease; iii) road traffic noise exposure will not predict psychological ill-health; iv) noise sensitivity will moderate the association of road traffic noise exposure on psychological ill-health; v) there will be no direct association of noise sensitivity with ischaemic heart disease or mortality; vi) that noise sensitivity will predict future psychological ill-health. An earlier version of this paper was published as a conference paper at the International Congress on Noise as a Biological Health Problem in 2017 [27].

Methods

Sample

The Caerphilly Collaborative Heart Disease Study [28] was set up as a cohort study of men in South Wales in the early 1980's to investigate risk factors for ischaemic heart disease (IHD). Men 45–59 years old living in Caerphilly, South Wales, UK, were eligible for inclusion. Initial screening included self-report questionnaires and clinic visits for anthropometry, blood pressure measurement and blood samples for cardiac risk factors. Initially 2512 (89%) of the eligible 2818 men were screened [29]. At phase 2, the first follow-up the cohort was enhanced with additional men who had recently moved to the area. This established a new cohort baseline for the 1984/88 population-based study comprising of 2398 men.

Traffic noise exposure

In 1984 measurement of A-weighted sound pressure level was carried out street by street to derive maps of road traffic noise [30]. On three consecutive days noise measurements were carried out continuously involving all busy roads and many side streets. Additionally, short-term measurements of L_{eq} 30 minutes were conducted during representative periods of the day (10.00-18.00hr) on all other relevant streets. Most traffic exposed dwellings were within 12 m from the street. Using the noise measurements and the maps derived therefrom participants were categorised into 5 dB groups of traffic noise emission level, in terms of L_{eq} referring to the period from 6.00 - to 22.00 and at a distance of 10 m from the street. Daytime outdoor noise level was then used as a general metric of street traffic noise. More sophisticated mapping was not available in the 1980s.

Noise sensitivity

Weinstein's 10-item self-report noise sensitivity scale, derived from his original 21 item scale, was used to measure noise sensitivity [25]. Scores were divided into tertiles of low, medium, and high sensitivity for analysis. Cronbach's alpha for this scale in the preliminary sample at baseline was 0.78 [24].

Ischaemic heart disease morbidity and mortality

Electrocardiogram (ECG) and cardiac enzyme levels were used to identify possible ischaemic heart disease events. These were obtained from hospital records and were evaluated against standard diagnostic criteria; Incident ischaemic heart disease (IHD) events were defined as: IHD death (ICD-9 codes 410–414); non-fatal myocardial infarction (MI) (a cardiac event satisfying WHO criteria); and electrocardiographic evidence of MI(major or moderate Q/QS waves, Minnesota codes 1-1-1 to 1-2-5 or 1-2-7 on any follow up ECG when there were no Q/QS waves, Minnesota codes 1-1-any, 1-2-any, or 1-3-any on the recruitment ECG) [29]. Information on deaths was obtained from notifications to the Office of National Statistics.

Psychological ill-health

The 30-item General Health Questionnaire (GHQ), which measures common mental disorder, predominantly depression and anxiety, was used to identify psychological ill-health [31]. ROC analysis was used in a subsample of 97 men to determine a threshold of 4/5 to distinguish between 'probable non-cases' and 'probable cases' [24]. Trait anxiety was measured by the Trait Scale of the State-Trait Anxiety Inventory [32]. Measurements of psychological ill-health were taken at phase 2 baseline in 1984/88, at phase 3 follow up 1989/93 and phase 4 follow up in 1993/6.

Covariates

At baseline, smoking history, alcohol history, physical activity at leisure, previous history of cardiovascular disease, bedroom orientation, noise at work and Registrar General classification of social class were obtained by questionnaire. BMI was calculated after height was measured on a Holtain stadiometer and body weight using a beam balance. Total serum cholesterol was measured using enzymatic procedures [28].

Statistical Analysis

Cox Proportional Hazard Models were used to analyze the association of road traffic noise and noise sensitivity with IHD morbidity and mortality. Stata Version 14 (StataCorp, 2015) was used to perform all data analysis. The models were initially run univariately and then run adjusted in a hierarchical fashion. Initial adjustments were for age, social class, marital status, and employment status. The final model included additional adjustment for smoking status, BMI, cholesterol, alcohol consumption, physical activity at leisure, noise at work, earlier history of IHD and bedroom orientation either facing towards or away from the road.

Logistic regression was employed to analyze the association of road traffic noise, noise sensitivity and anxiety with psychological ill-health. Initial adjustments in these models included age, social class,

marital status, employment status, smoking status, BMI, alcohol consumption, physical activity at leisure, bedroom orientation and noise at work. Noise sensitivity models were also explored with trait anxiety. Interactions between sensitivity and anxiety with road traffic noise were analyzed, but stratification was not possible because of low power. All analyses will be assessed at the 5% statistical level to be possibly statistically significant.

Missing data

The sample size of the 1984/88 population-based cohort comprised of 2398 men. The analyses represented in this paper are based on complete records due to the limitation of predictive variables to impute missing observations. The sample for the Cox-Proportional Hazards Models at phase 2 was therefore reduced as item responses ranged from 0–13.6% (cholesterol). The sample sizes for the logistic regression models based on phase 3 and phase 4 data were reduced considerably as the GHQ was poorly completed (27.9% missing at phase 3 and 37.7% missing at phase 4).

Results

IHD morbidity or mortality was not associated with road traffic noise exposure in these analyses (Table 1). In the initial model there was a suggestion of lower IHD morbidity in the 61-65dBA noise category but this was not observed in the model with full adjustment. There was no statistically significant interaction between road traffic noise exposure and noise sensitivity and either IHD morbidity or mortality.

Table 1
Road traffic noise and Ischaemic Heart Disease Mortality and Morbidity

Noise	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)
IHD mortality	Model 1	Model 2	Model 3
	N=2364	N=2353	N=1915
1			
2	0.86 (0.59, 1.26)	0.82 (0.56, 1.20)	0.83 (0.55, 1.24)
3	0.89 (0.66, 1.20)	0.89 (0.66, 1.19)	0.98 (0.70, 1.38)
4	1.20 (0.82, 1.75)	1.14 (0.77, 1.67)	1.00 (0.65, 1.54)
IHD morbidity			
1			
2	1.03 (0.78, 1.36)	0.95 (0.72, 1.25)	0.98 (0.73, 1.32)
3	0.80* (0.64, 1.00)	0.78* (0.62, 0.98)	0.90 (0.69, 1.17)
4	1.26 (0.95, 1.67)	1.25 (0.94, 1.67)	0.96 (0.69, 1.34)
Noise: 1 = 51-55dBA; 2 = 56-60dBA; 3 = 61-65dBA; 4 = 66-70dBA			
Model 1: Univariate; Model 2: Adjusted for age, marital status, social class and employment; Model 3: Adjusted for age, marital status, social class, employment, smoking status, BMI, alcohol consumption, physical activity at leisure, cholesterol, noise at work, pre-CHD history and bedroom orientation. *** p<=0.001, ** p<=0.01, *p<=0.05			

In order to test the full impact of noise sensitivity on physical health, associations with IHD mortality were examined. IHD mortality rather than all-cause mortality was selected because of previous analyses showing associations between environmental noise and IHD mortality. High noise sensitivity, somewhat unexpectedly, was found to be associated with a lower risk of IHD mortality than medium and low noise sensitivity (HR = 0.71, 95%CI 0.54–0.94) (Fig. 1, TW3 = high noise sensitivity). Noise sensitivity was replaced in the model by trait anxiety to test whether trait anxiety had a similar association with mortality as noise sensitivity. There was a similar lower risk of mortality associated with high trait anxiety (HR = 0.68, 95%CI 0.49, 0.94).

When examining incident cases of psychological ill-health a sample was selected from which GHQ cases were removed at baseline. A small significant association between road traffic noise at baseline and Phase 4 psychological ill-health was found even after initial adjustment in model 1 for sociodemographic factors but only among those exposed to 56-60dBA (OR = 1.98 95%CI 1.21, 3.24) (Table 2). This association was maintained in the final model after adjustment for health behaviors noise at work, and bedroom orientation (OR = 1.81 95%CI 1.07, 3.05). This was not statistically significant at phase 3.

Table 2
Road traffic noise and Psychological ill-health at phase 3 and phase 4

Noise	Odd Ratio (95% CI)		Odd Ratio (95% CI)
	Model 1*	Model 2	
Phase 3	N=1254	N=1250	N=1214
1			
2	1.61 (0.99, 2.62)	1.48 (0.90, 2.45)	1.52 (0.90, 2.56)
3	1.09 (0.69, 1.73)	1.10 (0.69, 1.74)	1.18 (0.73, 1.91)
4	1.21 (0.65, 2.26)	1.35 (0.71, 2.54)	1.41 (0.74, 2.68)
Phase 4			
	N=1085	N=1082	N=1056
1			
2	1.98** (1.21, 3.24)	1.88* (1.14, 3.13)	1.81* (1.07, 3.05)
3	0.82 (0.51, 1.34)	0.82 (0.50, 1.36)	0.84 (0.51, 1.39)
4	1.18 (0.63, 2.23)	1.27 (0.67, 2.42)	(0.68, 2.52)
Noise: 1 = 51-55dBA; 2 = 56-60dBA; 3 = 61-65dBA; 4 = 66-70dBA			
*Sample who were not GHQ cases at baseline			
Model 1: Univariate; Model2: Adjusted for age, marital status, social class and employment; Model 3: Adjusted for age, marital status, social class, employment, smoking status, alcohol consumption, noise at work, bedroom orientation and physical activity at leisure.			
*** p<=0.001, ** p<=0.01, *p<=0.05			

At baseline there was an interaction between road traffic noise and noise sensitivity with phase 3 psychological ill-health. The men who were highly noise sensitive in the 66-70dBA (highest) noise exposure group had a high risk of psychological distress (OR = 7.57 95%CI 1.35, 42.49) (Table 3). This interaction was still present after adjustment for the Spielberger trait anxiety scale (OR = 7.36 95%CI 1.30, 41.64). There was high variability around this estimate. This interaction was not statistically significant using phase 4 data.

Table 3
Road traffic noise, noise sensitivity and psychological ill-health GHQ: Phase 3- Weinstein Sensitivity

		Odds Ratio (95% CI)	Odds Ratio (95% CI) (With Interaction)
	N=1524	N=1524	
Noise	1		
	2	1.71* (1.11, 2.63)	1.13 (0.51, 2.51)
	3	1.22 (0.83, 1.79)	0.83 (0.40, 1.74)
	4	1.28 (0.75, 2.19)	0.40 (0.09, 1.78)
Weinstein Sensitivity (WS)	1		
(Tertiles)	2	1.58** (1.13, 2.21)	1.36 (0.91, 2.03)
	3	1.82*** (1.30, 2.56)	1.37 (0.91, 2.04)
Noise, WS Interaction	2,2		1.58 (0.55, 4.58)
	2,3		2.08 (0.68, 6.37)
	3,2		1.42 (0.53, 3.81)
	3,3		1.98 (0.75, 5.20)
	4,2		2.47 (0.43, 14.00)
	4,3		7.57* (1.35, 42.49)
Noise: 1 = 51-55dBA; 2 = 56-60dBA; 3 = 61-65dBA; 4 = 66-70dBA			
Adjusted for age, marital status, social class, employment, smoking status, alcohol consumption, noise at work, bedroom orientation and physical activity at leisure.			
*** p<=0.001, ** p<=0.01, *p<=0.05			

High and moderate levels of noise sensitivity at baseline were associated longitudinally with psychological ill-health at phase 3, (High noise sensitivity OR = 1.82 95%CI 1.30, 2.56; Moderate noise sensitivity OR = 1.58 95%CI 1.13, 2.21) (Table 3). Similarly, noise sensitivity predicted psychological ill-health at Phase 4 (High noise sensitivity OR = 1.78 95%CI 1.26, 2.52); Moderate noise sensitivity OR = 1.67 95% CI 1.19, 2.35). This association remained statistically significant after further adjustment for trait anxiety (High noise sensitivity at Phase 3 adjusted for trait anxiety OR = 1.53 95%CI 1.08, 2.18; High noise sensitivity at Phase 4 adjusted for trait anxiety OR = 1.44 95%CI 1.00, 2.07).

Discussion

Road traffic noise exposure was not associated longitudinally with IHD morbidity and mortality in these cohort analyses. There was also no consistent association with psychological ill-health, although there have been associations with depressive symptoms and insurance claims for depressive illness in other studies [8, 33]. Earlier analyses in this cohort did find a longitudinal association between road traffic noise and symptoms of anxiety [26]. As hypothesised the interaction of road traffic noise, noise sensitivity and IHD outcomes was not statistically significant. By contrast, in accordance with the hypothesis, road traffic noise and noise sensitivity did show significant interactions with psychological ill-health. Independently, noise sensitivity was associated with lower rates of mortality and, in contrast, predicted higher levels of psychological ill-health, the latter in keeping with our initial hypothesis.

Earlier analyses in this cohort, at 10 year follow up, showed an increased relative risk of IHD in relation to road traffic noise especially among those 15 years or more in the same place [34]. Exposure misclassification and the lengthening of the interval between baseline traffic noise assessment and ascertainment of health outcomes may explain the lack of association in these recent analyses. There may have been self-selection out of the noisiest areas or not into noisy areas, even at baseline, for the most noise sensitive, or this could have occurred during follow up. However, mobility of noise sensitive persons out of noisy areas has not been found in other studies [35].

Previous studies have also found that noise sensitivity did not moderate the association of road traffic noise exposure and IHD events [16]. Similarly, noise sensitivity was not a predictor of cardiovascular outcomes in an earlier study of civil servants except for participants in the lower employment grades where it predicted angina pectoris [25]. This is in contrast to a study of Finnish twins where noise sensitivity was a predictor of cardiovascular mortality in women but not in men [36]. Such a gender difference is in keeping with the results from the Caerphilly Study in men but not with the results in civil servants both men and women (Whitehall II Study), although in the latter study men and women were combined for analysis. A further explanation might lie in the outcomes chosen. In the Finnish Study noise sensitivity was a significant predictor of cardiovascular mortality (ICD codes 390–459,100–199) but not coronary heart disease mortality (ICD codes 410–414, 120–125). In the Caerphilly Study we only included ischaemic heart disease (also known as coronary heart disease ICD codes 410–414). Although this outcome measure issue would not explain the lack of results in the Whitehall Study which did include cardiovascular outcomes such as stroke morbidity and mortality.

If noise sensitivity were an independent predictor of physical ill-health it would be expected that it should be associated increased mortality rates. The fact that we found it was associated with lower mortality rates was not what we expected, although in keeping with findings from a previous study [16]. Lee et al [37] found that highly anxious young people had lower accident mortality up to the age of 25 years because they tended to avoid putting themselves in high risk situations which could have high mortality risk attached. Conversely, in older people they found high levels of anxiety were associated with higher non-accident mortality rates, perhaps as a result of prolonged physiological hyper-reactivity associated with chronic anxiety. Our cohort was middle-aged and older men, not strictly comparable with the population in Lee et al's study, nevertheless, it may be that noise sensitive people are more cautious and

less likely to take risks that could increase mortality. Noise sensitivity has been associated with phobic disorders in a sample of women [38] and fearfulness and avoidance which are part of phobic disorders might be characteristic of some people with noise sensitivity and could be associated with health-protective behaviours. Earlier analyses in this cohort found an association between noise level and noise sensitivity with less highly sensitive men living in the highest noise exposure areas so it may be that more sensitive men tend to choose to live in less noisy areas where that choice is possible [23]. It does not seem that this effect on mortality is mediated through health behaviours as our results were adjusted for smoking, leisure-time physical activity, BMI and alcohol use. Lower mortality rates were also found when noise sensitivity was replaced by trait anxiety in the models supporting an essential role for long-term anxiety in this association.

Noise sensitivity may moderate the effects of road traffic noise on psychological ill-health, although there was some inconsistency between phases 3 and 4 and the confidence intervals were wide so that our analyses may have been underpowered. Independently of road or aircraft noise exposure noise sensitivity has been shown to be strongly associated with a range of common mental disorders [21, 23, 38, 39, 40]. Most of these earlier studies have been cross sectional, this study as well as another study [25] have confirmed that noise sensitivity is associated with psychological ill-health longitudinally.

Noise sensitivity has also been associated with neuroticism [21, 23, 38, 41, 42]. Neuroticism is a construct similar to trait anxiety and has links to negative affectivity, a tendency to report life experiences and perceptions negatively. Noise sensitivity does not seem to be just trait anxiety or neuroticism and Shepherd et al. [35] have found higher correlations with introversion/extraversion than with neuroticism [43]. A question often asked in this literature is how specific is noise sensitivity to noise or much is it part of a wider range of responses to environmental stimuli driven largely by chronic anxiety [44]? Although, inevitably, much of the predictive power of noise sensitivity for common mental disorders derives from its association with trait anxiety [42] we did find that these associations still remained after adjusting for Spielberger trait anxiety. It is possible that there was still some residual confounding in the analyses of noise sensitivity and psychological ill-health adjusting for trait anxiety.

Noise sensitivity has been linked to sensitivity to other aspects of the environment such as sensitivity to chemicals, electromagnetic fields, light and odours [40, 45, 46]. Sensitivity to chemicals is often defined under the rubric of Multiple Chemical Sensitivity (MCS). MCS is a condition purported to be related to exposure to low levels of environmental chemicals which for most people would not result in health effects. In one study seventy three per cent of MCS also were noise intolerant [45]. Another name for this condition that suggests it covers a broader spectrum of exposures than just chemicals is 'Idiopathic Environmental Intolerance' (IEI). This is defined by three criteria: '1. It is an acquired disorder with multiple recurrent symptoms. 2. It is associated with diverse environmental factors tolerated by the majority of people. 3. It is not explained by any known medical or psychiatric/psychological disorder' [47]. A strong overlap has been found between Idiopathic Environmental Intolerance and Somatoform Disorders. In a study comparing Somatoform Disorders with Idiopathic Environmental Intolerance more than half of the latter group could be classified as fulfilling the criteria for Somatoform Disorders [48]. Both groups also

had higher levels of trait anxiety and somatic symptom attributions than the control group. More subjects with IEI reported allergies than the control group although these were not supported by objective changes in IGE levels. There was longitudinal stability of these conditions over a year and baseline negative affectivity and somatosensory amplification (a tendency to focus and amplify symptoms) predicted these conditions at one year follow up [49]. A strong association has been found between Idiopathic Environmental Intolerance and mood, anxiety and somatoform disorders across the lifecourse [50] and equally between MCS and major depressive disorder, and generalised anxiety disorder and severe psychological distress [51].

It has been proposed that this environmental sensitivity may relate to a hyper-responsive central nervous system with increased reactivity of the limbic system in the brain although no physiological evidence has been found to support this [52]. However, noise sensitivity is not always accompanied by other environmental sensitivities. Baliatsas et al, [40] found noise sensitivity associated with environmental sensitivity in 9–50% of highly noise sensitive people in their general practice community sample. Thus although noise sensitivity may be a symptom of IEI or even somatisation disorder in some cases it is not necessarily associated with other environmental sensitivities in all cases [35, 53]. To that extent noise sensitivity is not a single reified entity but may be a non-specific indicator of sensitivity to sounds alone or part of a wider IEI or psychiatric syndrome. Thus noise sensitivity might have a multiple origins [35].

Noise sensitivity has been associated with uncomfortable loudness levels in laboratory studies but has not been associated with especially sensitive hearing thresholds [54, 55]. Thus it does not seem to be related abnormalities in the peripheral auditory system. The associations with sympathetic nervous system activity may reflect associations with state or trait anxiety rather than being specific to noise sensitivity [11, 56]. A study using electro- and magnetoencephalography measuring mismatch negativity found that noise sensitivity categorised with the Weinstein scale was associated with altered sensory processing in the auditory cortex implying a central cortical origin for noise sensitivity [57]. This very interesting study requires replication; it is a type of neurophysiological validation of a self-report noise sensitivity scale but it does not directly link these auditory processing characteristics to vulnerability to ill-health as might be expected if noise sensitivity is related to increased susceptibility to ill-health. Intriguing exploratory EEG studies suggest that there may be a deficit in sensory gating in noise sensitive subjects leading to sensory ‘overload’ [56]. Further research in these disciplines may well be productive.

It may be difficult to generalise too far from these results as the population, although representative of the local area, was confined to middle-aged and older men living in a very specific geographical area. A strength of the study was the careful ascertainment of cardiac outcomes, the high response rate and follow up response longitudinally. The psychological ill-health outcomes would have been stronger had we had a standardised psychiatric interview instead of a questionnaire. Missing data for psychological ill-health outcomes in phase 3 and 4 was a limitation. Adjustment for room orientation could be over-adjustment as it may remove some of the variance due to noise exposure, although analyses not including adjustment for room orientation were little changed.

Conclusions

There is some evidence that noise sensitivity may be related to susceptibility to ill-health, especially psychological ill-health in relation to noise exposure. Also noise sensitivity is a risk factor for future psychological ill-health independent of noise exposure. There needs to be further understanding of the neurophysiological correlates of noise sensitivity before much more progress can be made in the associations of noise sensitivity with ill-health.

Abbreviations

IHD

Ischaemic Heart Disease

Leq

Equivalent continuous sound level in decibels

dB

Decibel

ECG

Electrocardiogram

MI

Myocardial infarction

GHQ

General Health Questionnaire

BMI

Body Mass Index

HR

Hazard Ratio

OR

Odds Ratio

ICD

International Classification of Diseases

MCS

Multiple Chemical Sensitivity

IEI

Idiopathic Environmental Intolerance

EEG

Electroencephalogram

Declarations

Competing interests

The authors declare they have no competing interests

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Authors' contributions

Conceptualization SAS; methodology, CC, MS, SAS; formal analysis: CC, MS; writing original draft SAS; writing- reviewing and editing SAS, CC, MS, JG, WB; Investigation JG, WB; data curation JG, WB. The authors read and approved the final manuscript.

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Ethics approval and consent to participate

All participants in the study gave written informed consent. The study was approved by the South Glamorgan local research ethics committee and adhered to the Declaration of Helsinki.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated or analysed in this study are available through the Data Custodian Professor Yoav Ben Shlomo at Bristol Medical School: Population Health Sciences
<https://www.bristol.ac.uk/population-health-sciences/projects/caerphilly/about>

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Figures

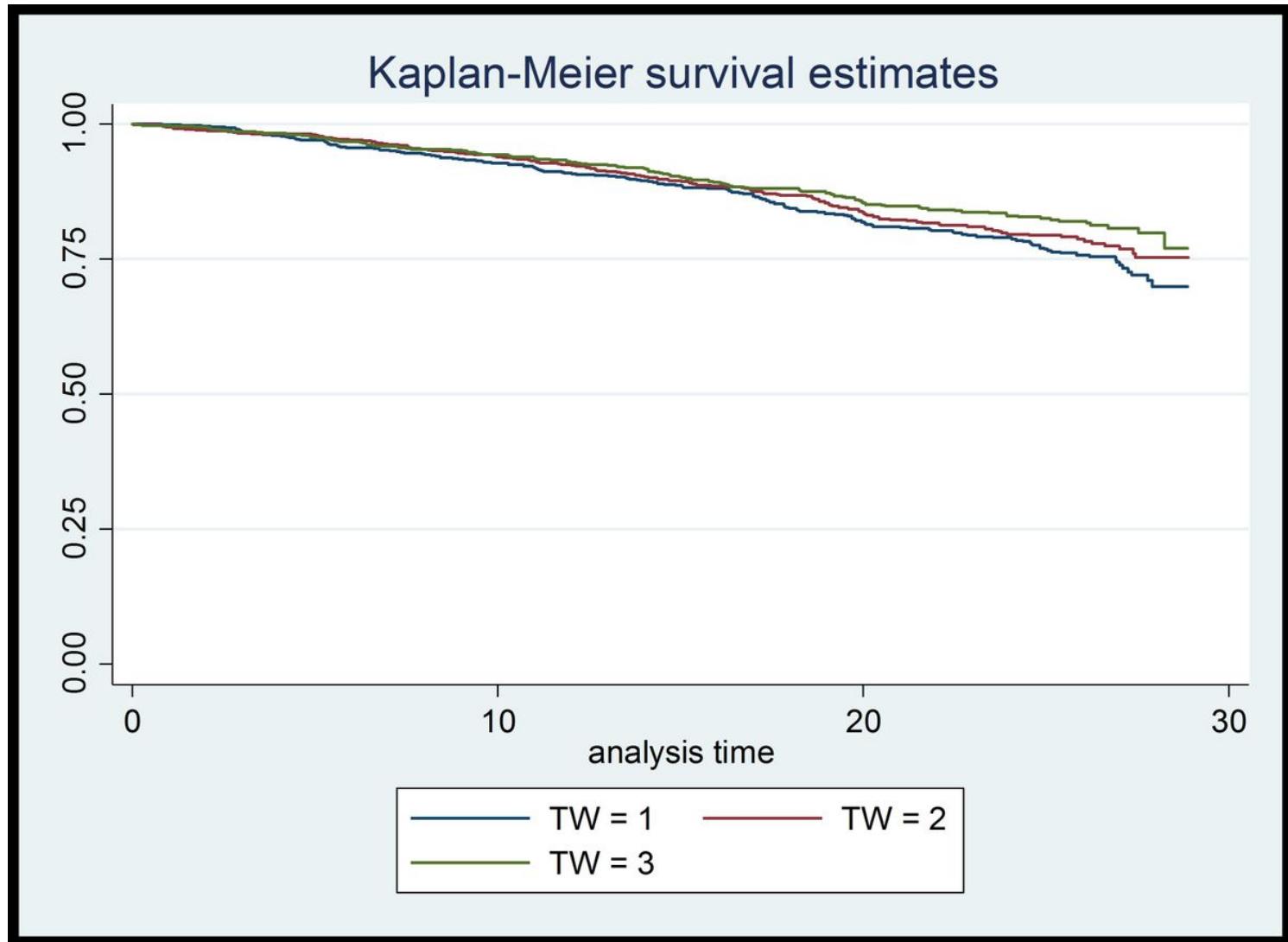


Figure 1

Survival analysis of high (TW3), medium (TW2) and low (TW1) noise sensitivity and mortality