

Road Traffic Noise, Noise Sensitivity, Noise Annoyance, Psychological and Physical Health and Mortality

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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-1990s. 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 and noise annoyance have moderating effects on road traffic noise and psychological ill-health and secondly if noise sensitivity and noise annoyance predict 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) and phase 4 (OR=1.78 95%CI 1.26, 2.52). There was weak evidence that noise sensitivity moderated the association of road traffic noise exposure with psychological ill-health. Noise annoyance predicted psychological ill-health at phase 4 in the sample in which baseline cases of psychological ill-health were included (OR= 2.08 95%CI 1.00, 4.31). Neither noise sensitivity nor noise annoyance moderated the effects of road traffic noise on ischaemic heart disease morbidity or mortality. Noise annoyance did not moderate the effects of road traffic noise on 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, 3, 4]. There is accumulating evidence that transport noise is related to an increased risk of depression, hypertension, stroke, 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].

There is general consistency across studies, that noise sensitivity has a moderating effect on another self-report variable, noise annoyance [12, 13, 14]. Annoyance expresses mild anger, partly as a result of noise interference into everyday activities, coupled feelings of invasion of privacy and lack of control. It tends to be seen as a state or immediate response to noise but people's annoyance responses tend to be stable over time suggesting a personality-based consistency to responding. 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]. Annoyance as a mediator presupposes that high levels of emotional response associated with annoyance may be an outward manifestation of underlying physiological arousal. High levels of annoyance could represent a stress response to noise but this has not consistently been demonstrated. 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 sensitivity 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 and noise annoyance might moderate these associations; 3) the longitudinal association between road traffic noise and psychological ill-health and 4) whether noise sensitivity and noise annoyance 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 and finally whether noise annoyance 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 and noise annoyance 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 and noise annoyance 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; vii) there will be no direct association of noise annoyance with ischaemic heart disease or mortality; viii) that noise annoyance 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]. This paper has elaborated on the meaning of the noise sensitivity results in the discussion and added new results on noise annoyance.

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 12m 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 'Leq referring to the period from 6.00 - to 22.00 and at a distance of 10m 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].

Noise annoyance

Self-reported road traffic noise annoyance was measured by a single question administered at Phase 2 baseline: 'Does traffic noise at home annoy you?' with five ordered levels of response from 'never' to 'always'. For the purpose of analysis 'never', 'seldom' and 'sometimes' was classified as low annoyance and 'often' and 'always' as high annoyance.

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' [23]. 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, noise sensitivity and noise annoyance 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, noise annoyance 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 define statistical significance.

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% to 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 61-65 dBA 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.

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) (Figure 1, TW3= high noise sensitivity). Noise sensitivity was associated with a lower risk of IHD mortality even after full adjustment (Table 2). Noise annoyance was not associated with IHD mortality (Table 2). 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).

Table 2 Ischaemic Heart Disease Mortality and Morbidity by Noise Annoyance and Noise Sensitivity Ischaemic Heart Disease Mortality

Noise Annoyance	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)	Hazard Ratio (95% CI)
	Model 1	Model 2	Model 3	Model 4
	N=2346	N=2336	N=1983	N=1900
Never/Seldom/ Sometimes				
Often/Always	1.20 (0.73, 1.97)	0.93 (0.56, 1.54)	0.95 (0.56, 1.61)	0.86 (0.49, 1.50)
Noise Sensitivity (Tertiles)				
	N=2334	N=2328	N=1987	N=1883
1 st Tertile				
2 nd Tertile	0.87 (0.69, 1.09)	0.91 (0.72, 1.15)	0.91 (0.71, 1.17)	0.85 (0.66, 1.10)
3 rd Tertile	0.74* (0.58, 0.94)	0.74* (0.58, 0.95)	0.74* (0.56, 0.97)	0.71* (0.54, 0.94)
Ischaemic Heart Disease Morbidity				
Noise Annoyance				
	N=2346	N=2336	N=1983	N=1900
Never/Seldom/ Sometimes				
Often/Always	1.46* (1.03, 2.06)	1.11 (0.78, 1.59)	1.19 (0.82, 1.74)	1.12 (0.75, 1.68)
Noise Sensitivity (Tertiles)				
	N=2334	N=2328	N=1987	N=1883
1 st Tertile				
2 nd Tertile	0.97 (0.82, 1.17)	1.03 (0.86, 1.23)	1.02 (0.84, 1.25)	0.96 (0.78, 1.17)
3 rd Tertile	0.96 (0.80, 1.15)	0.94 (0.78, 1.13)	1.02 (0.84, 1.25)	0.95 (0.77, 1.16)

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, BMI, Alcohol consumption, Physical activity at leisure and Cholesterol

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, Bedroom orientation and Noise

*** $p \leq 0.001$, ** $p \leq 0.01$, * $p \leq 0.05$

There was no interaction between noise and annoyance and either IHD mortality or morbidity

Table 3 Ischaemic Heart Disease Mortality and Morbidity testing for interactions with noise annoyance

IHD Mortality		Hazard Ratio (95% CI)	Hazard Ratio (95% CI) (With Interaction)
		N=1900	N=1900
Noise	1		
	2	0.84 (0.56, 1.25)	0.76 (0.50, 1.17)
	3	1.01 (0.71, 1.42)	1.00 (0.70, 1.43)
	4	1.02 (0.66, 1.58)	1.18 (0.76, 1.83)
Noise Annoyance (NA)	Never/Seldom/Sometimes		
	Often/Always	0.86 (0.49, 1.50)	0.92 (0.42, 2.04)
Noise, NA Interaction	2, Often/Always		3.41 (0.80, 14.59)
	3, Often/Always		0.99 (0.27, 3.66)
	4, Often/Always		0.19 (0.02, 1.66)
IHD Morbidity			
		N=1900	N=1900
Noise	1		
	2	0.98 (0.73, 1.32)	0.93 (0.68, 1.27)
	3	0.90 (0.69, 1.18)	0.86 (0.65, 1.14)
	4	0.96 (0.68, 1.34)	1.00 (0.70, 1.42)
Noise Annoyance (NA)	Never/Seldom/Sometimes		
	Often/Always	1.12 (0.75, 1.68)	0.90 (0.47, 1.70)
Noise, NA Interaction	2, Often/Always		2.54 (0.75, 8.59)
	3, Often/Always		1.74 (0.67, 4.52)
	4, Often/Always		0.86 (0.28, 2.61)

Model: Adjusted for Age, Marital status, Social class, Employment, Smoking status, BMI, Alcohol consumption, Physical activity at leisure, Cholesterol, Noise at work and Bedroom orientation.

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

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 4). This association was maintained in the final model after adjustment for health behaviours, noise at work, and bedroom orientation (OR= 1.81 95%CI 1.07, 3.05). This was not statistically significant at phase 3.

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 5). 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.

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 5). 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).

High annoyance did not predict psychological ill-health in the sample with cases of psychological ill-health at baseline excluded although there was an association between annoyance and psychological ill-health at Phase 4 which was not statistically significant; this association was not found with Phase 3 psychological ill-health (Table 6). In the sample which included GHQ cases at baseline noise annoyance did predict psychological ill-health at Phase 4 (OR= 2.08 95%CI 1.00, 4.31). There was no interaction between noise and noise exposure and annoyance with psychological ill-health at either Phase 3 or Phase 4 (Table 6).

Table 6 Road traffic noise, noise annoyance and psychological ill-health GHQ: Phase 3- Noise Annoyance (No GHQ at Baseline)

		Odds Ratio (95% CI)	Odds Ratio (95% CI) (With Interaction)
GHQ Phase 3		N=1207	N=1201
Noise	1		
	2	1.56 (0.92, 2.62)	1.51 (0.89, 2.56)
	3	1.20 (0.74, 1.95)	1.25 (0.77, 2.03)
	4	1.46 (0.76, 2.80)	1.47 (0.75, 2.87)
Noise Annoyance (NA)	Never/Seldom/Sometimes		
	Often/Always	0.68 (0.19, 2.40)	0.85 (0.10, 7.37)
Noise, NA Interaction	2, Often/Always		2.50 (0.09, 70.92)
	3, Often/Always		Empty
	4, Often/Always		0.77 (0.03, 17.29)
GHQ: Phase 4		N=1051	N=1051
Noise	1		
	2	1.78* (1.05, 3.02)	1.85* (1.08, 3.15)
	3	0.82 (0.49, 1.37)	0.81 (0.48, 1.37)
	4	1.22 (0.63, 2.38)	1.36 (0.69, 2.69)
Noise Annoyance (NA)	Never/Seldom/Sometimes		
	Often/Always	2.43 (0.96, 6.13)	4.07 (0.99, 16.68)
Noise, NA Interaction	2, Often/Always		0.30 (0.02, 5.41)
	3, Often/Always		0.80 (0.08, 7.90)
	4, Often/Always		0.22 (0.01, 3.41)

Model: Adjusted for Age, Marital status, Social class, Employment, Smoking status, Alcohol consumption, Noise at work, Bedroom orientation and Physical activity at leisure.

Noise: 1 = 51-55dBA; 2 = 56-60dBA; 3 = 61-65dBA; 4 = 66-70dBA

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

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] and a recent meta-analysis has found an association between transportation noise and anxiety [34]. 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. Noise annoyance did not predict IHD mortality or morbidity and there was no interaction between noise exposure, noise annoyance and either IHD mortality or morbidity. There was a non-significant association between noise annoyance and psychological ill-health at phase 4 but this was not demonstrated at phase 3. However, noise annoyance did predict psychological ill-health at phase 4 in the sample in which baseline cases of psychological ill-health were included.

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 [35]. 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 [36].

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 [37]. 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 [38] 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 [39] 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.

The lack of an interaction between noise and annoyance in the association with IHD is not in keeping with noise annoyance being a possible moderating factor of the relationship between road traffic noise exposure and ischaemic heart disease [15]; although effect modification of noise on hypertension was only demonstrated for aircraft noise and not road traffic noise in the HYENA study [15]. Finding no direct relationship between noise annoyance and IHD outcomes also does not support noise annoyance as a mediating factor between noise exposure and IHD [40]. However, in previous analyses in this cohort, in samples containing men from both areas of Caerphilly and Speedwell, an association between annoyance and incident IHD was only found in those participants without pre-existing chronic disease, where it was surmised that the lack of association in those with pre-existing chronic disease might be due to recall bias [41]. In general, a single question on noise annoyance was possibly not a strong enough outcome measure to test this hypothesis and a frequency measure of noise annoyance is not the same as the standardised ICBEN degree of annoyance measure used in many studies in this research field. Moreover, the ICBEN measure was developed after this cohort study was undertaken.

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 [23, 39, 42, 43].

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, 39, 44, 45]. 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. [36] have found higher correlations with introversion/extraversion than with neuroticism [46]. 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 [46]? Although, inevitably, much of the predictive power of noise sensitivity for common mental disorders derives from its association with trait anxiety [45] 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 [43, 48, 49]. 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 [48]. 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' [50]. 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 [51]. 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 [52]. A strong association has been found between Idiopathic Environmental Intolerance and mood, anxiety and somatoform disorders across the lifecourse [53] and equally between MCS and major depressive disorder, and generalised anxiety disorder and severe psychological distress [54].

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 [55]. However, noise sensitivity is not always accompanied by other environmental sensitivities. Baliatsas et al, [43] found noise sensitivity associated with environmental sensitivity in 9–50% of highly noise sensitive people in their general practice community sample. Heinonen-Guzejev and colleagues found noise sensitivity could be distinguished from MCS on the basis

of factor analysis in the Finnish twin cohort study [56]. 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 [36, 56]. 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 [36].

Noise sensitivity has been associated with uncomfortable loudness levels in laboratory studies but has not been associated with especially sensitive hearing thresholds [57, 58]. 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, 59]. 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 [60]. 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' [59]. Noise sensitivity has also been associated with larger grey matter volume in several brain areas: bilaterally in the temporal poles and the hippocampus, left sided Heschl's sulcus and the right anterior insula. Some of these brain areas may have relevance to the processing of sound by the auditory cortex [61]. Further research in these disciplines may well be productive.

Noise annoyance did predict psychological health but the effect was only statistically significant in participants who scored above the case threshold on the GHQ at baseline, that is had existing psychological ill-health. This may be because existing psychological ill-health tends to lead to increasing annoyance while at the same time independently predicting future psychological ill-health. People who are already ill tend to report being more highly annoyed by noise than people who are not ill [62, 63].

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. Annoyance is a weak predictor of future psychological ill-health but does not appear to moderate the effects of road traffic noise on psychological ill-health. 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.

List Of 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

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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>)

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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Tables

Due to technical limitations, table 1,4,5 is only available as a download in the Supplemental Files section.

Figures

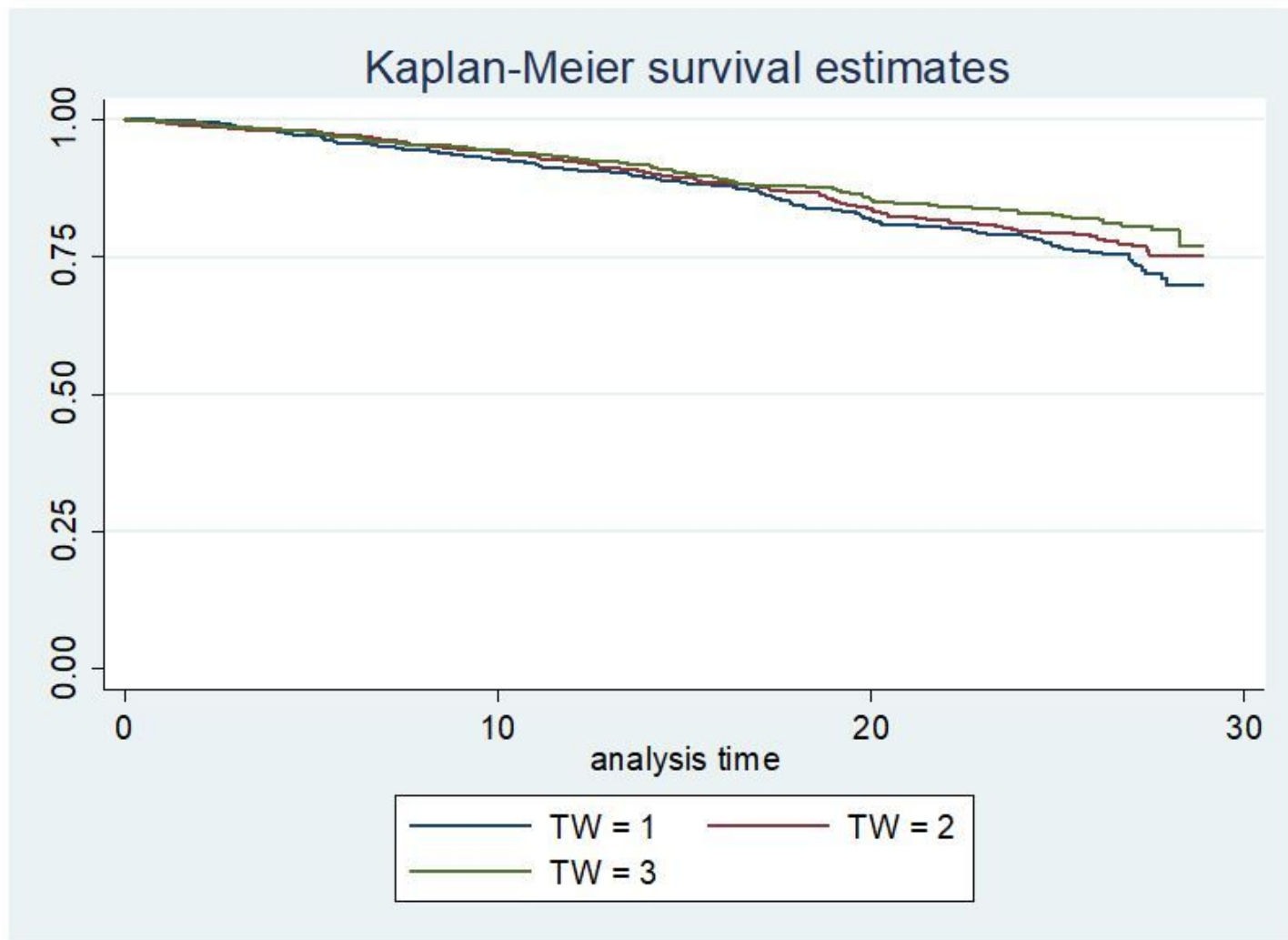


Figure 1

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

Supplementary Files

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