Health status as a potential effect modifier of the relation between noise annoyance and incidence of ischaemic heart disease

W Babisch, H Ising, J E J Gallacher

Aims: Traffic noise is a psychosocial stressor. Epidemiological studies suggest chronic noise stress to be a risk factor for cardiovascular disorders.

Methods: In a prospective cohort study, the association between annoyance and disturbances due to road traffic noise and the incidence of ischaemic heart disease (IHD) was studied in 3950 middle aged men.

Results: Depending on the questionnaire item, non-significant odds ratios for IHD incidence ranging from 0.9 to 1.4 were found for the highly noise annoyed/disturbed subjects when compared with the less annoyed/disturbed subjects, over the six year follow up period. However, this relation was strongly modified by the prevalence of pre-existing chronic diseases. In subjects free of any chronic disease at the beginning of the follow up, significant odds ratios between 1.7 and 3.0 were seen. In the subgroup with chronic diseases no such noise effects were seen. This surprising result of no effect in the group of people with a potential risk, due to pre-existing health problems, may be because of the dilution of the true effect due to recall bias.

Conclusions: Annoyance and disturbance due to road traffic noise is associated with a higher incidence of IHD. Prevalence of disease can be an important effect modifier of the relation between noise annoyance and health outcomes.

Main messages

- Noise annoyance due to exposure to road traffic may be a risk factor for the incidence of ischaemic heart disease. The prevalence of pre-existing chronic diseases modifies this association.

Policy implications

- Chronic environmental noise stress increases the risk of ischaemic heart disease.
example, stress hormones) > biological risk factors (for example, blood pressure, blood lipids) > disease (for example, myocardial infarction).16

METHODS

Two cohorts of 2512 (Caerphilly, South Wales) and 2348 (Speedwell, England) middle aged men in the United Kingdom were recruited to study the predictive power of already known and new risk factors for IHD. In both studies, investigators followed identical protocols with respect to noise, medical, sociodemographic, and biochemical measurements, and statistical data analyses. The response rates for phase 1 were 89% (Caerphilly) and 92% (Speedwell), which were extremely high and gave little concern about selection bias. The first follow up investigations (phase 2) were conducted after approximately four years. The second follow up investigations (phase 3) were conducted after approximately 10 years. Inasmuch as a detailed noise questionnaire was first administered during the second phase of the study, the follow up analyses presented here refer to the reconstructed cohorts of phase 2 and the observation period from phase 2 to phase 3.

The reconstructed cohort of phase 2 of the Caerphilly sample consisted of 1951 men of the original cohort who were seen again at the clinic (5.3% had died), plus 447 men of the same age range who had moved into the area since the original cohort was identified. Altogether these were 2398 men, aged 47–67 years. The Speedwell reconstructed cohort consisted of the 2055 men of the original cohort who were seen again at the phase 2 clinics (4.5% had died), aged 48–66 years. No new subjects were recruited in Speedwell. The statistical noise analyses were carried out on a pooled sample of 3997 men aged 47–67 years, who had filled in the noise questionnaire during phase 2. The average follow up interval for these men was 67 months (SD 6 months).

Long term and short term noise measurements were carried out in the streets where subjects lived and noise maps of the areas were established. The subjects were grouped according to 5 dB(A) categories of the outdoor A-weighted average sound pressure level, from 6 to 22 hours (Leq, 6–22 h). Because of the high correlation between day and night noise levels in the communities (correlation coefficient r = 0.94, mean difference 8 dB(A)), this noise level is used as an indicator for the overall traffic noise exposure of the streets in the study. In personal interviews annoyance and disturbance of traffic noise at home were assessed on a five grade scale (never, seldom, sometimes, often, always). The questionnaire items referred to annoyance (global item), disturbance of concentration, conversation, or listening to radio/TV and relaxation, being nervous or tense, disturbance in going to sleep, and waking up at night.14 The construction of the noise questionnaire was closely related to the recommendations given in the literature.40–42

The incidence of ischaemic heart disease was defined when a major IHD event occurred between the follow up phases. These events could either be IHD death (coded ICD 410–414 on death certificate), definite clinical non-fatal myocardial infarction (MI) meeting the WHO criteria regarding clinical history, electrocardiogram (ECG) and enzyme changes (via written documentation in hospital or general practitioner records), or ECG defined MI that met WHO criteria (major 1–1–any, or selected moderate 1–2–1 to 1–2–5, or 1–2–7) Q waves when there were no Q waves (1–1–any, 1–2–any, or 1–3–any) for the reference phase, and no left bundle branch block (7–1) on any ECG prior to the qualifying ECG.43

All statistical analyses on the relation between traffic noise and IHD incidence were controlled (model adjusted) for potential confounding factors—that is, age, social class, marital status, smoking, body mass index, family history of myocardial infarction, employment status, subjective noise sensitivity based on a single item, cohort (Caerphilly or Speedwell), prevalence of IHD (clinically assessed by ECG (Minnesota coding) or chest pain interview), and the self reported lifetime prevalence of illnesses including chronic diseases (general health interview). The latter included heart attack or coronary thrombosis, stroke, yellow jaundice or liver disease, kidney disease, gout, thyroid problems, high blood pressure, blood clots or phlebitis (inflammation in the legs), and diabetes. A new variable—pre-existing disease—was defined which was coded “1” when either prevalence of IHD or lifetime prevalence of any of the chronic diseases during phase 2 was true. Coding “0” referred to a strictly “disease free” group of subjects where prevalent diseases could not confound the association between noise annoyance and IHD incidence. Different mechanisms could be considered about how pre-existing disease could affect the relation between noise and IHD incidence. One is that pre-existing disease could cause psychological strain that affects the subjective ratings of noise of the individuals. Another is that pre-existing disease may be an additional stressor that could increase the physiological sensitivity of the individuals towards other stressors such as noise (see the discussion).

With regard to possible effect modification of pre-existing disease—the primary issue of this article—the analyses on the relation between noise and IHD incidence were stratified according to the presence of pre-existing disease. Separate analyses were carried out for subjects with and without disease prevalence (separate models). Relative risks are given with reference to the lowest noise exposure category in each subgroup (odds ratio 1).

The multiple logistic regression technique (SPSS 9.0) was applied to calculate estimates of the relative risk (odds ratio) and the 95% confidence intervals, and to control for confounding factors, based on cumulative incidence cases during the follow up period. No person-years were assessed, because the actual occurrence of the event during the six years follow up was unknown.

RESULTS

Death certificates were available for all men who died before the final examination occurred, with the exception of one from the Caerphilly cohort and two from the Speedwell cohort. In Caerphilly 94% and in Speedwell 87% of the 10 year follow up survivors were seen at the clinic again. In addition, 2% and 3%, respectively, had postal cardiovascular death certificates. The clinical diagnoses, offical admission for chest pain, and records from all local hospitals were then searched for the clinical diagnoses. The six year cumulative incidence of major IHD was 161 (of 2398) and 191 (of 2055) subjects in the Caerphilly and Speedwell cohorts, respectively. The average annual incidence rate was 1.38% for both cohorts. A total of 3997 men filled in the noise questionnaire (90%). Due to missing values, adjusted analyses in the study refer to the pooled sample of 3950 men for whom complete information on all the considered variables was given. The average age of the men in the pooled sample was 57.3 years (SD 4.5).

Table 1 shows the odds ratios of the relation between control variables and IHD incidence. The results are given for the total sample and the subsamples of subjects with and without pre-existing disease, prior to follow up. The following factors were significantly associated with IHD risk: smoking, family history of IHD, age, body mass index, unemployment, area, IHD prevalence, and pre-existing disease. These effects are very similar in men with and without pre-existing disease.

Table 2 gives the percentages of men in each disturbance/ annoyance category for each questionnaire item. Depending on the item, between 3% and 4% of the men were highly
annoyed/disturbed by traffic noise (categories 4 + 5 = “often” + “always”) and a further 10–20% were moderately annoyed/disturbed by traffic noise (category 3 = “sometimes”) annoyed/disturbed by traffic noise.

Table 3 gives the prevalence of pre-existing disease in each objective (sound level) and subjective (annoyance/disturbance) exposure category. Regarding the sound level, no associations between the outdoor traffic noise level and disease prevalence can be seen. However, regarding annoyance/disturbance ratings, significant trends towards higher prevalences in higher annoyed/disturbed subjects were found.

Table 4 gives the percentages of highly annoyed/disturbed subjects according to traffic noise level for the total sample and the two strata of subjects with and without pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoyed men increases with higher traffic noise in both strata. Men with pre-existing disease. The proportion of highly annoy...
research has been carried out on the relation between sound level and noise annoyance including modifying factors. Annoyance is understood as a mediator in the relation between sound and somatic health. However, little information is available on the relation between noise annoyance and health. Studies suggest that the relation between self-reported subjective health and psychological factors interacts with the prevalence of chronic conditions and the subjects' disabilities. Studies on work stress showed that subjective appraisal of stressors by persons with increased blood pressure may be an invalid measure of objectively verifiable stressors. The "response bias model" and the "adaptation level theory" were used to explain differences in reported annoyance under steady state conditions except for changing noise conditions. According to these concepts, individual minimum and maximum points on an annoyance scale may be dependent on the level of noise exposure itself and one's optimal level of stimulation. The latter differs from one person to another as a matter of experience and the individual's ability and costs to adapt. Non-noise factors, such as physical health, may affect the individual's expectation level and response scale.

The results of this prospective study on the impact of road traffic noise on the incidence of IHD gave only marginal support to the hypothesis that subjects with health problems may be a risk group for adverse health effects of environmental noise, when the objective sound level is considered. Higher odds ratios in higher noise (sound) exposed subjects were only seen in the subgroup of subjects with pre-existing diseases, if at all. A simple explanation is that these people have less reserve to cope with the additional noise stress. Another explanation is that the noise further increases psychophysiological arousal, which may be already higher in these people with health problems. In noise regulations, ill people (hospital areas) are often considered as a potential risk group for higher susceptibility towards noise stress. As far as ratings of annoyance/disturbance are concerned, subjects with pre-existing disease in the present study were more often highly annoyed/disturbed by traffic noise than subjects without such health problems, even for low sound exposure levels (table 4). Conceptually, pre-existing disease acted as a moderator of the relation between sound and annoyance. Furthermore, pre-existing disease acted as an effect modifier on this relation. Positive trends of increasing

**Table 3 Prevalence of pre-existing diseases in different traffic noise exposed subjects**

<table>
<thead>
<tr>
<th>Exposure variable</th>
<th>Exposure category</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>Group</th>
<th>Trend</th>
</tr>
</thead>
<tbody>
<tr>
<td>Traffic noise level (dB(A))</td>
<td>51–55</td>
<td>56–60</td>
<td>61–65</td>
<td>66–70</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Traffic noise level</td>
<td>38.3%</td>
<td>37.0%</td>
<td>41.4%</td>
<td>37.7%</td>
<td>0.561</td>
<td>0.622</td>
<td></td>
</tr>
<tr>
<td>Adjusted traffic noise level*</td>
<td>38.7%</td>
<td>37.3%</td>
<td>38.2%</td>
<td>36.4%</td>
<td>0.905</td>
<td>0.518</td>
<td></td>
</tr>
</tbody>
</table>

**Table 4 Percentages of "highly" annoyed/disturbed ("often" + "always") subjects in each sound level category stratified by the prevalence of pre-existing diseases (PD)**

<table>
<thead>
<tr>
<th></th>
<th></th>
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<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Annoyance</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>2.1%</td>
<td>6.1%</td>
<td>8.8%</td>
<td>10.3%</td>
<td>0.000</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>1.7%</td>
<td>4.6%</td>
<td>5.3%</td>
<td>10.1%</td>
<td>0.000</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>Concentration</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>4.2%</td>
<td>4.2%</td>
<td>5.3%</td>
<td>5.0%</td>
<td>0.694</td>
<td>0.290</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>3.0%</td>
<td>3.8%</td>
<td>3.8%</td>
<td>3.2%</td>
<td>0.880</td>
<td>0.611</td>
<td></td>
</tr>
<tr>
<td>Conversation, radio, TV</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>6.0%</td>
<td>5.0%</td>
<td>7.5%</td>
<td>7.9%</td>
<td>0.688</td>
<td>0.356</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>3.1%</td>
<td>3.7%</td>
<td>7.1%</td>
<td>5.0%</td>
<td>0.092</td>
<td>0.052</td>
<td></td>
</tr>
<tr>
<td>Relaxation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>2.8%</td>
<td>3.4%</td>
<td>5.7%</td>
<td>3.7%</td>
<td>0.092</td>
<td>0.052</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>4.1%</td>
<td>4.3%</td>
<td>9.1%</td>
<td>7.0%</td>
<td>0.021</td>
<td>0.008</td>
<td></td>
</tr>
<tr>
<td>Nervous or tense</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>3.0%</td>
<td>2.6%</td>
<td>3.5%</td>
<td>6.0%</td>
<td>0.045</td>
<td>0.023</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>3.6%</td>
<td>4.3%</td>
<td>4.8%</td>
<td>12.3%</td>
<td>0.000</td>
<td>0.000</td>
<td></td>
</tr>
<tr>
<td>Falling asleep</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>2.4%</td>
<td>2.6%</td>
<td>2.9%</td>
<td>3.6%</td>
<td>0.640</td>
<td>0.217</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>1.8%</td>
<td>2.1%</td>
<td>2.6%</td>
<td>1.6%</td>
<td>0.818</td>
<td>0.731</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>3.4%</td>
<td>3.6%</td>
<td>3.2%</td>
<td>7.0%</td>
<td>0.274</td>
<td>0.186</td>
<td></td>
</tr>
<tr>
<td>Waking up</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>– PD</td>
<td>2.9%</td>
<td>2.6%</td>
<td>4.4%</td>
<td>4.6%</td>
<td>0.134</td>
<td>0.032</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>2.6%</td>
<td>1.7%</td>
<td>3.4%</td>
<td>2.7%</td>
<td>0.690</td>
<td>0.743</td>
<td></td>
</tr>
<tr>
<td>+ PD</td>
<td>3.3%</td>
<td>4.3%</td>
<td>5.9%</td>
<td>7.9%</td>
<td>0.063</td>
<td>0.007</td>
<td></td>
</tr>
</tbody>
</table>

*PD, +PD: subjects with and without pre-existing disease.
disturbances with increasing sound level were only seen in subjects with pre-existing disease and not in healthy subjects. The percentages of highly disturbed subjects were independent of the sound level in men without pre-existing disease (table 4). On the other hand, annoyance (not disturbance) due to traffic noise showed the usual trend of an increase with increasing sound level in both subgroups. This suggests that noise disturbance and noise annoyance refer to different dimensions of reported noise stress in this study. The global item of noise annoyance comprises attributes of nuisance and disturbance. With regard to IHD incidence, the healthy subjects' abilities of coping with the physical noise stress may be more effective. No increase in IHD risk was found in men with no pre-existing disease with increasing sound level (table 5). On the other hand, men with pre-existing disease were more often affected by traffic noise when considering disturbed activities (table 4), and showed a slight, but non-significant tendency towards a higher IHD risk with increasing sound level (table 5). Contrary to expectation regarding the subjective perception of the noise, these men were not at higher risk. On the other hand, there was a strong increase in IHD risk in men without pre-existing disease with increasing annoyance/disturbance ratings.

The high response rates obtained in all phases of the study and the comprehensive assessment of IHD occurrence during the follow up, using all sources of information raise little concern about possible selection bias. However, recall bias could be an explanation of the interaction phenomenon. IHD incidence, IHD prevalence, and the pre-existing health condition were assessed on the basis of clinical measurements and standardised interviews by physicians who knew nothing about the noise exposure of the study subjects. The noise interview was made after the clinical interviews. This means that diagnostic bias and over-reporting of disease history because of the noise was not a problem in the study. However, due to the presence of a chronic disease, over-reporting of exposure (annoyance/disturbance) could be the case. Subjects with manifest health problems may be more likely to give exaggerated answers about their annoyance/disturbance by traffic noise in the interview although not virtually being stressed by the noise. They may tend to blame their environment for their health difficulties, hoping that the result of the study might influence future noise policy. Subjects who believe that the noise contributes to the health problem may be more dissatisfied and annoyed with noise. In such cases, subjects who gave high annoyance ratings may have shown weaker physiological reactions than those with the same annoyance ratings who did not give such distorted answers. This dilutes the statistical noise effect. However, it may also be possible that some subjects played down the noise because they thought that it was of minor importance compared with their health problem, although they may have perceived the noise as a physiological stressor. Particularly with regard to sleep disturbances it has been shown that habituation does not take place when physiological reactions are considered. In such cases, subjects who gave low annoyance ratings may have shown higher physiological reactions than those with the same annoyance ratings who did not give such distorted answers. This again dilutes the statistical noise effect. All in all, these are sources of exposure misclassification that possibly could make the interaction phenomenon non-significant.

### Table 5

<table>
<thead>
<tr>
<th>Exposure variable</th>
<th>Noise exposure category</th>
<th>Traffic noise level (dB(A))</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>51–55</td>
<td>56–60</td>
<td>61–65</td>
<td>66–70</td>
<td></td>
</tr>
<tr>
<td>Traffic noise level</td>
<td>1.00</td>
<td>0.71 (0.46 to 1.11)</td>
<td>0.68 (0.44 to 1.03)</td>
<td>1.07 (0.70 to 1.65)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>1.00</td>
<td>0.78 (0.42 to 1.47)</td>
<td>0.97 (0.55 to 1.72)</td>
<td>1.03 (0.53 to 1.94)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+PD</td>
<td>1.00</td>
<td>0.65 (0.35 to 1.23)</td>
<td>0.44 (0.23 to 0.84)</td>
<td>1.08 (0.60 to 1.95)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adjusted traffic noise level*</td>
<td>1.00</td>
<td>0.69 (0.42 to 1.12)</td>
<td>0.64 (0.37 to 1.09)</td>
<td>1.31 (0.78 to 2.21)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PD</td>
<td>1.00</td>
<td>0.71 (0.35 to 1.43)</td>
<td>0.89 (0.44 to 1.80)</td>
<td>0.84 (0.36 to 1.99)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>+PD</td>
<td>1.00</td>
<td>0.70 (0.35 to 1.38)</td>
<td>0.43 (0.18 to 1.00)</td>
<td>1.82 (0.92 to 3.58)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Results are given for the total sample (n = 3950) and the stratified subsamples of subjects with (n = 1519) and without (n = 2431) pre-existing diseases (PD).

*PD: subjects with and without pre-existing disease.

Adjusted for room orientation and window opening habits.
could lead to an underestimation of the true effect of the noise disturbance/annoyance on IHD incidence in the total sample. Consequently, in the subsample free of pre-existing diseases where this source of exposure misclassification was not present, the subjects that were highly annoyed/disturbed by traffic noise had a markedly and sometimes a significantly higher risk of IHD incidence, depending on the item used. It should be noted that these results refer to prospective findings obtained from subjects who developed IHD during follow up. All noise measurements and interviews were taken before the follow up.

Two conclusions can be drawn from the study: Firstly, noise annoyance/disturbance is associated with IHD incidence (shown in men without pre-existing disease) and may be a risk factor. Secondly, annoyance/disturbance ratings from subjects with health problems (men with pre-existing disease) may be influenced by other factors, and must be viewed with caution with regard to recall bias. Prevalence of a disease is an important effect modifier in the relation between noise and cardiovascular health outcome in epidemiological studies. Recall bias, in particular, may be an issue when exposure and/or outcome are assessed on a subjective basis. In social surveys, approximately 40–66% of the variation of community reaction to noise has been explained by the sound level and other known factors. Non-physical characteristics of the noise source are of great importance in the perception of noise annoyance. From the present study it can be seen that the prevalence of diseases may be a factor to be considered in social surveys and epidemiological studies, to improve the prediction of community responses to noise and to assess health effects of chronic noise stress. Noise annoyance is a subjective indicator of exposure that may be subject to recall bias for the reasons discussed. The subjective assessments of exposure may cause problems in observational studies. The interaction phenomenon was not much an issue when objective indicators of exposure were used. Certainly, there may be other effect modifiers to be considered. The focus here was on one which we thought would be of particular importance.

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