Fifty one general practitioners were questioned about their knowledge of the link between smoking and Crohn's disease when they telephoned to refer patients to hospital urgently.

Forty per cent (41/102) patients smoked (28 women, 13 men); 19 were ex-smokers. Sixty four per cent (26) of the smokers were under 42; 35 smokers began before the onset of Crohn's disease.

Thirteen patients (11 smokers, 2 non-smokers) were aware of the adverse affects of smoking on their Crohn's disease. All 13 had been informed by their hospital doctor. No ex-smokers knew of the link, and all said they had given up cigarettes for reasons unrelated to their Crohn's disease. Fifteen of the 41 smokers had never been asked to stop smoking by their hospital doctor, and 27 had never been asked to stop by their general practitioner.

Only 5 of the current or ex-smokers felt that smoking had influenced symptoms they associated with their Crohn's disease. Of these, three felt that smoking improved symptoms and two that their symptoms worsened.

Two of the 51 general practitioners questioned knew of the adverse association between smoking and Crohn's disease.

Comment

This survey indicates that many general practitioners and their patients with Crohn's disease are unaware that the incidence and course of Crohn's disease are influenced by smoking. Furthermore, some gastroenterologists do not provide their patients with this information. This is compounded by fact that most smokers do not feel that smoking influences the symptoms they associate with their Crohn's disease.

In patients with asthma, chronic obstructive airways disease, and ischaemic heart disease, advice to stop smoking is regarded as important. Because Crohn's disease runs a chronic course with clinical relapses often requiring surgery, anything to improve the course of the disease, such as stopping smoking, should be strongly encouraged.

We should do more to educate both general practitioners and our patients about these facts. Patients will then be in a better position to make an informed decision to quit smoking and to have a positive effect on their disease.

Funding: None.
Conflict of interest: None.

1 Cottone M, Rosselli M, Orlando A, Oliva L, Puleo A, Capello M, *et al.* Smoking habits and recurrence in Crohn's disease. *Gastroenterology* 1994;**106**:643-8.
2 Sutherland L, Ramcharan S, Bryant H, Fick G. Effect of cigarette smoking on recurrence of Crohn's disease. *Gastroenterology* 1990;**98**:1123-8.
3 Person PG, Ahlbom A, Hellers G. Inflammatory bowel disease and tobacco smoke. A case-control study. *Gut* 1990;**31**:1377-81.
4 Lindberg H, Jarnerot G, Huitfeldt B. Smoking in Crohn's disease: effect on localisation and clinical course. *Gut* 1992;**33**:779-82.

(Accepted 11 April 1996)

Road traffic noise and psychiatric disorder: prospective findings from the Caerphilly study

Stephen Stansfeld, John Gallacher, Wolfgang Babisch, Martin Shipley

Unit for Social and Community Psychiatry, Department of Psychiatry and Behavioural Sciences, University College Medical School, London W1N 8AA
Stephen Stansfeld, *senior lecturer*

MRC Epidemiology Unit (South Wales), Llandough Hospital, Penarth, Glamorgan
John Gallacher, *research psychologist*

Institut fur Wasser- und Lufthygiene des Bundesgesundheitsamtes, Berlin, Germany
Wolfgang Babisch, *research scientist*

Department of Epidemiology and Public Health, University College London Medical School, London WC1E 6BT
Martin Shipley, *senior lecturer*

Correspondence to:
Dr Stansfeld.

BMJ 1996;313:266–7

Although environmental noise causes annoyance, there is little evidence from studies of psychological symptoms, psychotropic drug use, mental hospital admissions, and community studies that it causes psychiatric disorder.[1] Exposure to aircraft noise was not associated with psychiatric disorder in a cross sectional survey in west London,[2] but the population exposed to noise may have been biased by prolonged noise exposure and may represent "survivors" of noise, the most vulnerable to noise having moved away or never having moved into the noisy area. The relation between traffic noise at baseline and psychiatric disorder at follow up is explored in the Caerphilly collaborative heart disease study, a population unlikely to have been selected by noise exposure.

Subjects, methods, and results

In the second phase of the Caerphilly study all men aged 50-64 years living in Caerphilly, south Wales, were invited to attend a screening clinic.[3] Follow up was carried out five years later. Street measurement of A-weighted sound pressure level (dB(A)) was used to derive traffic noise maps, and subjects were grouped into five-decibel categories of traffic noise emission level, in terms of the average sound pressure (Leq) from 6 am to 10 pm.[4]

The 30 item general health questionnaire was used to establish the presence of psychiatric disorder; it was validated against psychiatric interview in a subsample and a case threshold of 4/5 was established.[5] Depression and anxiety subscales were extracted from the general health questionnaire.

The association between noise exposure and subsequent psychiatric disorder was investigated using multiple regression. We adjusted for the possible confounding effect of baseline morbidity by including this as a covariate in the regression model. Least squares means have been presented both unadjusted and adjusted for baseline psychiatric morbidity so that the magnitude of this confounding can be examined. This sample size has a power of 90% to detect differences of 0.22 in anxiety scores, 0.15 in depression scores, and 0.45 in general health questionnaire scores between high and low noise levels.

A total of 2398 men were present in the phase 2 sample of the study, the baseline for these analyses. The initial response rate to the survey was 89%. At five year follow up, 162 men had died, 31 had moved, 337 refused to attend the clinic, and 143 either refused the questionnaire or it was omitted. Prospective data were available for 1725 men. Non-response at follow up was not associated with either noise exposure or baseline psychiatric caseness.

If traffic noise causes psychiatric disorder, a dose-response relation between traffic noise level and psychiatric disorder might be expected. We found no overall association (or linear trend) between noise level at baseline and psychiatric disorder measured prospectively (table 1), even after adjusting for confounding factors such as social class, employment status, marital status, physical illness, and baseline morbidity. However, the subjects living at the lowest noise level had the lowest level of psychiatric disorder. Adjusting for room orientation to the street and hearing threshold made little difference to these results.

Noise could be more specifically related to anxiety or depression than to the broad span of morbidity covered by the general health questionnaire. Subscales for anxiety and depression extracted from the questionnaire showed similar patterns as above, although there was a non-linear relation between noise exposure and anxiety, adjusting for baseline anxiety score (table 1).

Table 1—*Association between road traffic noise level and psychiatric disorder. Values are mean (SE) score on general health questionnaire (GHQ) unless otherwise stated*

	Road traffic noise level (dB(A))				
	51-55 dB (n = 1218)	56-60 dB (n = 153)	61-65 dB (n = 233)	66-70 dB (n = 104)	P value for tests of heterogeneity
Adjusted for age (n = 1707)	2.99 (0.14)	3.83 (0.39)	3.17 (0.32)	3.44 (0.48)	0.22
Adjusted for age, social class, employment status, marital status, physical ill health, and baseline GHQ score (n = 1590)	2.57 (0.21)	3.37 (0.39)	2.65 (0.34)	2.96 (0.46)	0.29
Psychiatric caseness (% scoring ≥5 on GHQ)	22.5	32.0	24.9	25.0	0.07
Mean (SE) anxiety score adjusted for age, social class, and noise sensitivity and anxiety at baseline (n = 1583)	4.70 (0.07)	5.20 (0.18)	4.89 (0.15)	5.02 (0.21)	0.03
Mean (SE) depression score, adjusted for age, social class, and noise sensitivity and depression at baseline (n = 1587)	1.19 (0.05)	1.39 (0.13)	1.32 (0.11)	1.21 (0.16)	0.34

Comment

Although there was little association between road traffic noise level at baseline and overall minor psychiatric disorder at follow up, there was some evidence for differences in anxiety scores. The results of this prospective study confirm the results of previous cross sectional studies and suggest that environmental noise is not an important cause of overall psychiatric disorder but nevertheless may contribute to anxiety. We cannot rule out the possibility that effect modification by unmeasured variables or response bias in the measurement of morbidity may be masking an association between noise and psychiatric disorder. The traffic noise levels in this sample are fairly typical of those in Britain but do not include the highest levels of traffic noise exposure and therefore do not preclude an association at higher levels of noise exposure. It is also possible that environmental noise might have a pathogenic effect on mental health only in concert with other stressors which have not been assessed in this study.

We thank Dr Peter Elwood for his continuing support and encouragement, Peter Sweetnam and Dan Sharp for helpful statistical advice, David Poor for data management, and Louise Price for secretarial support.

Funding: The study was supported by the Medical Research Council. Stephen Stansfeld was a Wellcome Trust training fellow during the initial period of the study.

Conflict of interest: None.

1 Stansfeld SA. Noise, noise sensitivity and psychiatric disorder: epidemiological and psychophysiological studies. *Psychol Med* 1992;suppl 22:1-44.
2 Tarnopolsky A, Morton-Williams J. *Aircraft noise and prevalence of psychiatric disorders, research report.* London: Social and Community Planning Research, 1980.
3 Caerphilly and Speedwell Collaborative Group. Caerphilly and Speedwell collaborative heart disease studies. *J Epidemiol Community Health* 1984;38:259-62.
4 Stansfeld SA, Gallacher JEJ, Sharp DS, Yarnell JWG. Social factors and minor psychiatric disorder in middle-aged men: a validation study and population survey. *Psychol Med* 1991;21:157-67.
5 Goldberg DP. *The detection of psychiatric illness by questionnaire.* London: Oxford University Press, 1972.

(Accepted 16 May 1996)

Retrospective study of influence of deprivation on uptake of cardiac rehabilitation

Jill Pell, Alastair Pell, Caroline Morrison, Oliver Blatchford, Henry Dargie

Department of Public Health, Greater Glasgow Health Board, Glasgow G1 1ET
Jill Pell, *senior registrar*
Caroline Morrison, *consultant*
Oliver Blatchford, *senior registrar*

Department of Cardiology, Stobhill Hospital, Glasgow G21 3UW
Alastair Pell, *senior registrar*

Department of Cardiology, Western Infirmary, Glasgow G11 6NT
Henry Dargie, *consultant*

Correspondence to:
Dr Pell.

BMJ 1996;313:267–8

Mortality from ischaemic heart disease is higher in Scotland than in most developed countries.[1] Comprehensive cardiac rehabilitation after myocardial infarction, incorporating exercise training and lifestyle counselling, can reduce mortality and the rate of fatal reinfarction[2] and also improve quality of life.[3] Socioeconomic deprivation is associated with both an increased risk of developing myocardial infarction and a poorer prognosis afterwards.[4] Our aim was to determine whether deprivation affected uptake of rehabilitation after myocardial infarction.

Subjects, methods and results

Scottish morbidity record (SMR1) data were used to identify all patients discharged from Glasgow hospitals from 1 June 1994 to 31 November 1994 with an International Classification of Diseases (revision 9) code of 410 (myocardial infarction). Each patient's age, sex, postcode, comorbidities, and consultant were recorded, together with whether they died before discharge. Postcodes were used to obtain the Carstairs deprivation score for the 5000 or so residents within each postcode sector.[5] This is calculated from 1991 census data on overcrowding and male unemployment in each sector and the numbers of residents who belong to a low social class and who have no access to a car. Higher scores represent a higher level of deprivation. Four of Glasgow's five main hospitals offer a cardiac rehabilitation programme, and a list of patients invited to rehabilitation was obtained from the hospitals. Information was provided on which of these patients started the programme and which completed it.

Over the six months 1120 patients had a discharge diagnosis of myocardial infarction. Their median age was 66 years (interquartile range 57-74) and 59% were men. Only 7% of patients were recorded as having coexistent peripheral arterial disease, 5% diabetes mellitus, 4% cerebrovascular disease, and 4% renal failure. Comorbidity is, however, known to be poorly recorded (J Blair, personal communication). Two hundred and thirty three patients (21%) died before discharge. The age, sex, and deprivation scores of patients with myocardial infarction were compared with those of the Glasgow population obtained from 1991 census data. Logistic regression showed that increasing deprivation score was associated with increased risk of myocardial infarction ($P<0.0001$). This remained significant after adjustment for age and sex ($P<0.0001$). The incidence of myocardial infarction in the most deprived quartile was 1.7 times that in the least deprived.

Three hundred and sixteen (36%) of the patients discharged alive were invited to rehabilitation. Of these, 188 (59%) started the programme and 109 (34%) completed it. Stepwise multiple logistic regression analysis showed that hospital ($P<0.0001$), age ($P<0.0001$), sex ($P<0.05$), and the type of consultant (cardiologist *v* general physician; $P<0.05$) were significant independent determinants of whether patients were invited to rehabilitation (table 1). Deprivation score was not a significant factor. Uptake of rehabilitation after invitation was significantly associated with the type of consultant ($P<0.05$), hospital