Table 1—*Details of fatal methadone overdoses in Manchester, 1985-94. Values are numbers of overdoses unless stated otherwise*

Variable	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	Total
Fatal overdose:											
All methadone related	0	0	4	4	2	11	12	15	20	22	90
Methadone alone	0	0	1	2	0	6	9	6	14	14	52
Other substances only	46	59	45	53	56	56	61	39	47	50	512
Deaths associated with methadone (% of all overdoses in Manchester)	0	0	8.2	7.0	3.4	16.4	16.4	27.8	29.9	30.6	15.0
Source of methadone:											
Own prescription	0	0	2	2	1	4	3	7	9	8	36
Known diversion	0	0	2	2	1	3	5	5	7	7	32
Probable diversion	0	0	0	0	0	4	4	3	4	7	22
Deaths associated with methadone:											
England and Wales*	9	14	46	32	34	60	64	115	NA	NA	
Manchester (% of total for England and Wales)	0	0	8.7	12.5	5.9	18.3	18.8	13.0	NA	NA	
Opiate users in Manchester†:											
Methadone, alone or in combination	NA	36	31	54	68	141	234	372	320	530	
All opiates	NA	503	559	587	628	737	916	1062	935	1141	

NA = not available.
*Data from the Office of Population Censuses and Surveys.[4]
†University of Manchester Drug Misuse Database (T Millar, personal communication).

Comment

Unexpected, unexplained, and unnatural deaths are reported to the coroner. The victims have a full necropsy, with toxicological examination when no clear cause of death is found. During the study toxicological samples were submitted to one of two laboratories. Until 1985 or 1986 these laboratories estimated urinary methadone concentrations using semiquantitative enzymic methods. Subsequently, they used a fully quantitative immunoassay that can be applied to any fluid or tissue. Broad screens for drugs of misuse are routine, and detection of one misused substance prompts a search for other commonly misused substances. Underascertainment of cases is likely to have been small.

Methadone is used in two main ways in opiate addiction. In the client centred approach drug misusers are weaned off all opiates to cure addiction. In contrast, the public health approach aims at reducing the risk taking behaviour associated with heroin misuse, rendering needle sharing redundant and avoiding the risks of HIV infection and viral hepatitis.[5] The public health approach has recently been adopted by Manchester Health Commission, but we understand that it was informally adopted several years ago by some of the authorities responsible for managing drug misuse locally. This adoption coincided with the rapid increase in methadone prescription (and associated deaths) that started in 1990. We are concerned that many new clients will be recruited to methadone maintenance programmes. They may themselves be at comparatively low risk of overdose, but diversion of methadone endangers others, including children. Indeed, our findings suggest that diversion accounts for most deaths from methadone. A public health approach to opiate misuse is laudable but should be tempered with caution. We hope that the resources necessary for safer dispensing of methadone will be made available.

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Patients' awareness of adverse relation between Crohn's disease and their smoking: questionnaire survey

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Smoking is an independent risk factor for clinical, surgical, and endoscopic recurrence in Crohn's disease.[1] In a 10 year follow up of 174 patients the recurrence rate was 70% in smokers and 41% in non-smokers.[2] Passive smoking increases the risk of Crohn's disease in children[3] and of having the more severe form. Ileocolonic and small bowel disease is more common in heavy smokers.[4] On current evidence, encouraging patients to stop smoking ought to be an important part of the management of Crohn's disease, but there are few or no published data describing patients' knowledge of the association between smoking and their disease. We therefore investigated patients' and general practitioners' awareness of the link between smoking and Crohn's disease to identify the standard of education in this area.

Methods and results

A total of 102 patients (43 men) with Crohn's disease (mean age 42 (range 17-84) years) under the care of two gastroenterologists completed a questionnaire either in the outpatient clinic (n = 33) or by post (n = 69; 83% response rate). This asked whether they were a smoker or an ex-smoker; if they knew of any link between smoking and Crohn's disease; if anyone had informed them of the link and if so was it their general practitioner or hospital doctor; and if they had been advised to stop smoking. It also asked if they noticed any effect of smoking on the symptoms they associated with their disease.

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.

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

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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).