Nearly half the sample favoured maintenance prescription of injectable opiates. The inevitable outcome of such practices is illicit selling on of drugs, with accidental overdoses and deaths. Indeed, much prescribed oral methadone ends up on the streets and already seems to play a part in accidental deaths.

The emergence of "dual diagnosis" patients, with both mental disorder and substance misuse, highlights the importance of psychiatric input to drug services.

Although the *Health of the Nation* recommends consultation with users and carers, it does not suggest that services should be designed solely to satisfy consumers' preference or convenience.⁵ Planning of services should be based on good evidence and sound clinical practice.

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Methadone maintenance treatment

EDITOR,—Our review of methadone maintenance treatment¹ elicited a range of views.² Wolfgang Poser and colleagues correctly comment that methadone treatment protects against death due to an overdose of opiates³—an effect that we have reviewed elsewhere.⁴ The problem of deaths due to overdoses of diverted methadone, however, is considerable in Britain and Denmark⁴ and merits further work.

Diversion of methadone is an important if underrecognised problem in Britain. It indicates poor quality treatment and can probably best be addressed through policies and guidelines on the supervision of consumption of methadone, facilities for consumption off site, and attention to lack of compliance with treatment. The issue of compliance is important and poorly studied in Europe.⁴ Kim Wolff and colleagues advocate regular measurement of plasma methadone concentrations.² We do not agree that this is the most direct method of addressing diversion of methadone. The role of urine and blood analysis in addressing poor compliance is unclear, and such tests constitute a substantial cost burden for service providers. On balance, we suspect that the cost of these procedures outweighs the benefits, but further study is needed to clarify this issue.

Frederick H Meyers's comments are not consistent with the research literature, and he cites no evidence to support his opinions.² Steve Kempney² and N J Shaw² express concern about methadone withdrawal in neonates. Methadone treatment for pregnant women who are dependent on heroin has been shown to protect maternal and neonatal wellbeing.⁵ Withdrawal of neonates is often substantially complicated, however, by mothers' use of more than one drug—for example, cocaine and benzodiazepines.

On balance, methadone treatment does have drawbacks, like many interventions in medicine do. Nevertheless, overall, properly delivered methadone treatment confers important benefits

for people who are dependent on opiates and for the community.

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Environmental lead and children's intelligence

Studies included in the meta-analysis are not representative

EDITOR,—Stuart J Pocock and colleagues' meta-analysis of 26 studies of asymptomatic exposure to lead in children concludes that "the evidence strongly supports an inverse association between body lead burden and child IQ."¹ The authors then urge caution before the relation is accepted as causal. They do this on the basis of the following considerations: Are the studies representative? Is confounding adequately dealt with? Is selection bias at work? Is causality reversed: does low IQ cause ingestion of lead?

These questions have been asked and answered before. The criticism of non-representativeness could readily be applied to Pocock and colleagues' analysis, which includes five studies reporting null effects. Three (in Bucharest, Budapest, and Sofia) have not been published independently in peer reviewed journals.² Two had major methodological difficulties severe enough to disqualify them. In one study of small sample size (n=135) half of the mothers of the infants misused a potent neurotoxin, alcohol.³ Another study had serious difficulties in analysing blood lead concentrations and was forced to change the analytical method in the middle of the investigation.⁴ On the other hand, two tooth studies that showed significant effects were excluded for arbitrary reasons.

Pocock and colleagues suggest that there may be many other unpublished and undiscovered studies that find no effect of lead and mention a single case in point. A non-anecdotal way to deal with this question is to estimate the number of unpublished null studies required to vitiate the observed joint result. This technique, known as "file drawer analysis," has in fact been applied to lead.⁵ The number of unpublished null studies required is between 26 and 82.

In their discussion of confounding the authors ignore the phenomenon of shared variance and assign the decrease in effect size when confounders are controlled exclusively to the confounder. Confounders can have a direct effect on outcome; they can also operate through increasing lead concentration. Poverty affects cognition. It also results in families living in homes with higher lead

content and surfaces that have deteriorated. Lower parental intelligence and poor care taking, in addition to directly affecting the cognition of offspring, may lead to increased absorption of lead. Ignoring this property may result in a type II error.

Selection bias of subjects was specifically evaluated in the two Boston studies and shown not to be consequential.

The issue of reverse causality dissolves under close inspection. Umbilical cord blood lead concentrations, measuring the intrauterine lead burden, are associated with later IQ deficits. Subhuman primates systematically given small doses of lead show clear deficits in learning and attention. To dismiss these studies on the basis of species differences is to ignore one of Bradford Hill's canons of causal inference: biological plausibility. There is no clearer example than the coherence of these two groups of epidemiological and experimental investigations.

Modern critics of the hypothesis linking lead with cognitive deficit should be required to provide fresh reasons for their scepticism and not use worn caveats that have been amply addressed over the past decade and a half.

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Britain must replace its lead pipes to meet WHO standards for drinking water

EDITOR,—As Stuart J Pocock and Marjorie Smith have been members of official British advisory committees, their acknowledgement that there is convincing evidence that blood lead concentrations down to 10 µg/dl (0.48 µmol/l) produce adverse neurological effects in children should have important implications for public policy.¹ The United States government set a target maximum childhood blood lead concentration of 10 µg/dl in 1991, but the British "action level" has remained at 25 µg/dl (1.2 µmol/l). The British government should now lower its target to, or preferably below, 10 µg/dl and then take steps to meet that target.

The problem of raised lead concentrations in drinking water is widespread in Britain. In Blackburn, to cite an extreme example, about 20 000 households receive water containing greatly increased concentrations—some as high as 50 000 µg/l (24 mmol/l) or even 300 000 µg/l (144 mmol/l); in 1990 almost a fifth of samples analysed by British water companies had lead concentrations above 10 µg/l.² If drinking water in Britain is to meet the World Health Organisation's new maximum lead contamination limit of 10 µg/l the only practical solution will be the replacement of all lead pipes.

Too little attention is being paid in Britain to the problem of old leaded paint and the consequent contamination of household dust. The United States has a comprehensive strategy for identifying and reducing housing stock contaminated with lead. In Britain the Department of Health has