Firstly, item 3 of the Hamilton scale for depression, ratings on which provide the data for the analysis, is an insensitive measure of suicidality: a rating does not entail the asking of any standard questions, and the anchor points for scoring are not well defined. Furthermore, in interviews characteristic of clinical trials clinicians noting an improvement in depression will tend, by virtue of a halo effect and the counterintuitive nature of the emergence of suicidality in such circumstances, to rate scores down.

Secondly, though Dr Beasley and colleagues' analysis shows clearly that fluoxetine reduces suicidality in depression, Teicher *et al* referred to suicidality consequent on the development of symptoms such as akathisia or dysphoria.[2] Dr Beasley and colleagues did not address the question of whether patients taking fluoxetine were more liable to develop akathisia or agitation than patients taking placebo. It seems highly likely that they were. Of these, possibly some may go on to develop suicidal ideation that is in some as yet unspecified way consequent on drug induced akathisia or dysphoria. Such a mechanism seemed to underlie the development of suicidal ideation in two patients taking fluoxetine whom we saw recently.[3]

An alternative method that can be used to determine whether antidepressants lead to the emergence of suicidality entails using a test-retest procedure. There is an ethical problem with such a method in the case of compounds that may induce suicidality. In so far as it was possible to pursue this method we did so[3]; the outcome was a strong suggestion that fluoxetine may lead to suicidality, as may other drugs active on the serotoninergic system (which includes most tricyclic antidepressants).

The significance of the emergence of suicidality during treatment with any psychotropic compounds as opposed to during the course of a depressive episode is that during treatment it can be anticipated and forestalled by warning patients.

Though it is helpful to have some of the issues surrounding the question of fluoxetine and suicidality tackled so competently,[1] it would be a pity if the rush to douse the flames of media hype surrounding this issue obscured recognition of a phenomenon that the cases originally reported by Teicher *et al*[2] helped draw attention to.

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Health of the nation

SIR,—We should like to emphasise some of the important points in Dr P G J Burney's article on a strategy for asthma,[1] points that are in danger of being lost in the epidemiological arguments.

It is essential to include asthma as a key area because it is the commonest chronic disease affecting all age groups in England, and because it is a major cause of preventable deaths and ill health; it is the only cause of preventable death with a higher standardised mortality ratio in 1987 than in 1979.

The consultative document and Dr Burney have accepted this necessity, but both imply controversy as to whether intervention reduces suffering and whether targets may be set. Repeated studies have shown that there are preventable factors in over 80% of deaths.[2] One study showed that if patients with severe asthma were admitted under a general medical firm they were 10 times more likely to be readmitted than if their initial care had been under a respiratory physician.[3] Suffering results from underuse of regular preventive treatments, and yet these reduce symptoms,[4] may reduce risk of the airway narrowing becoming fixed,[5] and are more effective than relieving bronchodilator treatments.[6] They have also been shown to reduce school absenteeism due to asthma.[7]

The current debate about treatment with β agonists and possible side effects of inhaled steroids should not be allowed to imply that there is no consensus on how to treat asthma. In perhaps no other condition has there been such clear agreement as to management of both children[8] and adults.[9,10] In adults the recommended shift away from dependence on regular treatment with β agonists, and the recommendations regarding reducing absorption of inhaled steroids, predated the subsequent controversies and are not influenced by them.

We accept the argument that in setting targets allowances must be made for the increasing prevalence of the condition. We also recognise that high rates of admission to hospital due to asthma may be a good, rather than a bad, phenomenon. Nevertheless, as 80% of deaths involve preventable factors we must set a target for their future reduction and establish now a confidential inquiry into these deaths. There should also be a target for a greater proportion of patients in hospital to be seen by or followed up by a specialist. In the community targets can be set for establishing asthma registers within general practice and for setting up systems for regular structured review of patients by properly trained health professionals. Urgent research is also needed to identify why the condition is increasing in prevalence.

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SIR,—Professor John Garrow has produced a concise and topical summary of obesity.[1] Though I fully support his suggestion that one of the three important measures to prevent obesity should be education and the early detection of obesity at primary school age, I believe that it is important that appropriate target figures for the population in question are chosen for limiting excessive weight gain in children. In the United Kingdom an average (50th centile) child will, between 7 and 12 years, gain about 14 kg (boy) to 17 kg (girl),[2] not 22 kg as Professor Garrow states. Therefore, the suggestion that if a child gains only 18 kg over this period his or her weight will normalise is clearly not correct for British children. Indeed, this rate of weight gain for a child whose weight is on the 90th centile of weight for height will virtually maintain the centile weight for height position.

Despite recommendations to the contrary[3] weight for height centile tables are rarely used to assess obesity in British paediatric practice, though simple visual comparison of the relative centile positions of a child's weight and height on charts appropriate for the local population is commonly used. Alternative techniques to assess obesity, such as estimations of skinfold thickness, require skill in their use, the application of which is limited by interobserver error. Body mass indices with exponents specific for sex and age[4] or measurement of actual body composition by means of innovative techniques with lower interobserver error, such as bioelectrical impedance,[5] may in future prove to be of value in assessing obesity in childhood populations.

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AUTHOR'S REPLY,—I am grateful to Dr Gregory for pointing out my error concerning the normal weight gain of children: the text should have read age 5 years, not 7 years. The weights of children on the 50th and 90th centiles at age 5 years are about 18 and 22 kg and at age 12 years 40 and 54 kg, respectively. Thus the overweight child is only 4 kg overweight at age 5 and, if he or she gains 18 kg (instead of 22 kg) in the next seven years, should achieve normal weight for height.

I agree that weight for height is not an ideal measure of obesity in children, especially around puberty, when weight and height increase at different rates, but body mass index serves quite well in prepubertal children and is minimum at about age 5.[1] Bioelectric impedance is easy to measure, but most of the correlation between impedance and fat free mass is derived from the measures of weight, height, and age that are used in the prediction formulas.[2] Although impedance is claimed to measure body water, it does not accurately predict changes in body water during diuresis[3] or dialysis.[4]

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SIR,—Peter Anderson concludes that a strategy to reduce disease related to alcohol should have two strands: a high risk approach directed at heavy drinkers and a population approach to reduce overall consumption.[1] We agree that it would be desirable to truncate the distribution of risk by