In reality, it is highly unlikely that there has been a true rise and fall in homicide among mentally ill people in England and Wales over the past 50 years. These figures are entirely based on statistics which reflect the workings of the Criminal Justice system (a charge to which I plead guilty).[2] They merely reflect changes in processing defendants by the courts. The probable culprit for declining diminished responsibility was declining enthusiasm for treating personality disordered and sexually deviant killers under the Mental Health Act legal category 'Psychopathic Disorder'. The authors did not provide statistics on other forms of manslaughter. These have increased in recent years, suggesting that defence lawyers have become more successful in putting forward alternative defences to murder than diminished responsibility.

I agree with the authors that sociological and legal factors (mainly the latter) have effects on rates of homicide due to mental disorder. But it is the overall base rate of homicide in the population that matters and with which these figures must be compared. This differs markedly between different countries. In those where it is very high, such as South America and Sub-Saharan Africa, mental disorder is almost irrelevant as an epidemiological risk factor. The authors refer to a small number of studies suggesting a correlation between rates of homicide among the mentally ill and rates among the rest of the population. It may well be that the 'laws'[2] they refer to are too rigid. For example, it makes sense that a country that allows handgun ownership is more likely to have killers with schizophrenia who use a handgun, and at a rate higher than in countries where handguns are banned, although the evidence for this remains thin on the ground. But from the public health perspective does it matter? Handguns are the key risk factor, not schizophrenia.

England and Wales have a low but steadily rising rate of homicide. It is unrealistic to propose mental health services as a public health intervention, but will be popular with politicians. Social geographers have demonstrated that social exclusion and growing social inequalities are the strongest correlates with this phenomenon affecting young men in England and Wales.[3]

1 Large M, Smith G, Swinson N, Shaw J, Nielson O. Homicide due to mental disorder in England and Wales over 50 years. *Br J Psychiatry* 2008; **193**: 130–3.

2 Coid J. The epidemiology of abnormal homicide and murder followed by suicide. *Psychol Med* 1983; **13**: 855–60.

3 Shaw M, Tunstall H, Dorling D. Increasing inequalities in risk of murder in Britain. Trends in the demographic and spatial distribution of murder, 1981–2000. *Health Place* 2005; **11**: 45–54.

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Authors' reply: We welcome interest in our study of homicide in England and Wales. However, we disagree with Coid's assertion that the conclusions are illogical because the same social factors that were associated with the increase in homicides by the mentally ill up to the 1970s were present when those homicides declined. There are several possible reasons for decline in homicide by the mentally ill, including the availability of treatment. Coid's assertion that a fall in homicide due to better treatment must mean that the earlier rise was due to deteriorating mental health services is a similar oversimplification.

There has been no change in the law regarding diminished responsibility since 1957. Coid's explanation that the decline in homicide by the mentally ill since the late 1970s was due to a change in the threshold for the verdict of diminished responsibility is not supported by any data. Moreover, a change in threshold for diminished responsibility would not explain the decline in the verdicts of 'not guilty due to mental illness', 'permanently unfit for trial' and 'infanticide'. We also defend the use of legal outcomes to define cases. Given the careful attention paid to homicide matters by the courts, their verdicts are likely to be reasonably sensitive and highly specific.[1]

Vinkers *et al* report 8 years of data from The Netherlands, without showing that rates of homicide by the mentally ill have declined over a longer period. However, a lack of a decline in The Netherlands might not be unexpected, as we have found that 40% of homicides in psychotic illness occur before treatment,[1] that delay in the initial treatment of schizophrenia is associated with a greater proportion of homicides during the first episode of psychosis[2] and that jurisdictions with mental health laws that require a patient to be dangerous before they can receive involuntary psychiatric treatment, such as The Netherlands, have longer delays in the treatment of early psychosis.[3]

We look forward to a challenge to our findings based on data rather than opinion and speculation.

1 Nielssen O, Large M. Rates of homicide during the first episode of psychosis and after treatment: a systematic review and meta-analysis. *Schizophr Bull* 2008; doi: 10.1093/schbul/sbn144 (Epub ahead of print).

2 Large M, Nielssen O. Evidence for a relationship between the duration of untreated psychosis and the proportion of psychotic homicides prior to treatment. *Soc Psychiatry Psychiatr Epidemiol* 2008; **43**: 37–44.

3 Large M, Nielssen O, Ryan C, Hayes R. Mental health laws that require dangerousness for involuntary treatment may delay the initial treatment of schizophrenia. *Soc Psychiatry Psychiatr Epidemiol* 2008; **43**: 251–6.

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Antipsychotics and risk of diabetes in schizophrenia

Smith *et al* state that there is increasing concern among clinicians about the association between second-generation antipsychotics and diabetes.[1]

It is interesting then that while commenting on the lack of systematic reviews and meta-analyses that support this concern, the authors go on to investigate not the relationship between starting antipsychotics and developing diabetes, but the relative risk of developing diabetes between groups of patients commenced on first-generation and second-generation antipsychotics. It is questionable whether this meta-analysis addresses, in any clinically meaningful way, the risk of developing diabetes after starting an antipsychotic, whether second or first generation. This would appear to be more usefully addressed by looking at the absolute risk.

The authors report on the difficulties in finding high-quality trials to include in their study. This is illustrated by the inclusion of only 11 trials out of an identified 1974. Smith *et al* then go on to outline their own criteria for a study to be considered of 'high quality'. These criteria include a prospective design and at least 1 year of follow-up recorded. It is of note then that of the 11 studies eventually included in the analysis, only 3 were prospective. Furthermore, of these 3 prospective trials, none was longer than 3 months. All trials included in the review could, therefore, be classified as low quality. The test for heterogeneity between studies, applied by the authors, further illustrates the highly significant methodological heterogeneity between studies.

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We would suggest that given the overall poor quality of studies found in the review there seems to be no rationale for going on to conduct a meta-analysis. One common pitfall of any meta-analysis is that if you put only poor-quality data in, you will get poor-quality data out. Consequently, this meta-analysis would seem to add little to the current evidence base with regard to antipsychotics and diabetes, except, perhaps, the confirmation that the studies on this subject are heterogeneous and generally of poor quality.

If one does want to consider whether a significant relationship exists between antipsychotic use and diabetes, or a metabolic syndrome, then the CATIE study[2] would seem to provide reasonably robust evidence that such a relationship does exist. This large, randomised, prospective study, carried out over a period of 18 months, has data collected at baseline and following the introduction of antipsychotic, and demonstrates clinically and statistically significant adverse changes in blood glucose, weight and cholesterol. This is particularly the case for those patients commenced on olanzapine.

Declaration of interest

R.P. has received speakers' honoraria from Janssen-Cilag, Eli Lilly and Wyeth Pharmaceuticals.

1 Smith M, Hopkins D, Peveler RC, Holt RI, Woodward M, Ismail K. First- *v.* second-generation antipsychotics and risk for diabetes in schizophrenia: systematic review and meta-analysis. *Br J Psychiatry* 2008; **192**: 406–11.

2 Lieberman JA, Stroup TS, McEvoy JP, Swartz MS, Rosenheck RA, Perkins DO, et al. Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *N Engl J Med* 2005; **353**: 1209–23.

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Authors' reply: We acknowledge Smith & Porter's interest in the reasons for why we did not focus on the relationship between merely starting any antipsychotic and developing diabetes, but instead reviewed the evidence for an association between diabetes and type of antipsychotic medication. There has been increasing concern that second-generation antipsychotics may be more diabetogenic than first-generation antipsychotics in patients with schizophrenia. Despite this concern, there is a lack of good evidence to support this apparent phenomenon and so it was essential to carry out our systematic review prior to developing guidelines for diabetes screening and management.

We agree with Smith & Porter that our paper has found strong heterogeneity between studies which is clearly an important finding from our study. It is only by undertaking systematic reviews that one can determine that heterogeneity exists. Therefore, without our systematic review this would not have been clear. Our meta-analysis uses random effects methodology, which means we have analysed the average effect over the studies. This is a meaningful concept in the presence of heterogeneity. As for looking at absolute risks, the heterogeneity between studies is so great as to make even random effects pooling absurd. This is why pooled analyses virtually always pool relative risks rather than risk differences.

Smith & Porter have highlighted our conclusions that methodological limitations were found in most studies. As current evidence is poor, it should not be used alone in making clinical decisions concerning diabetes screening and management for patients with schizophrenia. Regardless of whether first- or second-generation antipsychotics are prescribed, routine screening for diabetes in all patients with schizophrenia should be undertaken.

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Pharmacology and human morality

Maybe I am missing something but what is new in the proposition Spence has outlined?[1] When a Yanomani tribesman snorts a powerful concoction of hallucinogens he does so as part of a ritual that includes the shamanistic healing of others in the tribe and maintaining tribal cohesion through tradition. When a footballer plays on despite injury, with pain relieved by analgesia, he does this in part for his team and fans. When a Peruvian highlander chews coca leaves so that he can work longer hours he does so to keep his family fed; and the same applies to the kratom user in the Far East. When millions of soldiers took amphetamines to enable them to fight for longer hours, thereby exposing themselves to ever greater dangers, they did so to win what they believed to be just wars. When a mother solicits fertility treatment so as to produce a child that will not only add to the family, but also potentially save the life of another sibling, the use of these potentially dangerous drugs is largely driven by the mother's need to save the other child. When groups of men gather every afternoon in the Yemen and chew qat, this is a social activity enhanced by the use of qat. In the Middle East, coffee shops have always served this purpose, providing socially stimulating conversation, and do so in Europe to this day. Tobacco has had a similar use in many countries and alcohol has done much the same, despite the harm associated with the use of both of these substances. Psychiatrists, on a small scale, have started to use what some term empathogens (i.e. MDMA) so that they can better understand and help their patients (although the less charitable question their motives).

I think we would be splitting hairs to argue that taking a drug to achieve a moral end is fundamentally different from achieving a moral end through use of a drug; they exist on a continuum. Drugs simply allow us to explore and alter our behaviour and thoughts. How we use this allowance is up to us.

1 Spence SA. Can pharmacology help enhance human morality? *Br J Psychiatry* 2008; **193**: 179–80.

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In a recent editorial, Spence stated that the pharmacological interventions currently available in psychiatry also improve moral behaviour.[1] He subsequently argued that there is no fundamental difference with moral enhancement therapy, medication specifically developed to increase moral behaviour. Spence gave the example of a patient who continues to take antipsychotic medication because he knows he can be violent when unwell and he wants to prevent risks to others.

Spence asserted that whether an intervention assists in 'moral enhancement' or not crucially depends upon the goals of the