

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 study² 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.

Mike Smith, Hillmorton Hospital Canterbury District Health Board, Private Bag 4710 Christchurch, New Zealand. Email: michael.smith@cdhb.govt.nz; **Richard Porter**, Department of Psychological Medicine University of Otago, Christchurch, New Zealand.

doi: 10.1192/bjp.194.2.186a

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.

Michelle A. Smith, Department of Psychological Medicine, Institute of Psychiatry, King's College London, SE5 9RJ, UK. Email: m.smith@iop.kcl.ac.uk; **David Hopkins**, Department of Diabetic Medicine, King's College Hospital, London, UK; **Robert C. Peveler**, Clinical Neurosciences Division, University of Southampton, UK; **Richard I. G. Holt**, Endocrinology and Metabolism Sub-division, University of Southampton, UK; **Mark Woodward**, Department of Medicine, Mount Sinai Medical Center, New York, USA; **Khalida Ismail**, Department of Psychological Medicine, Institute of Psychiatry, King's College London, UK

doi: 10.1192/bjp.194.2.187

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.

Andrew Al-Adwani, Rotherham, Doncaster and South Humber Mental Health NHS Foundation Trust, Great Oaks, Ashby High Street, Ashby, North Lincolnshire DN16 2JX. Email: al-adwani@ntlworld.com

doi: 10.1192/bjp.194.2.187a

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