quetiapine or ziprasidone for 12 months, weight change was greatest on olanzapine and among the lowest on ziprasidone.[3] Ziprasidone was not associated with untoward effects on any other metabolic risk factors.

A pooled analysis in 2009 of over 100 Pfizer-sponsored randomised controlled trials found no significant differences between 1605 individuals given ziprasidone and 677 given placebo in total cholesterol, fasting glucose or fasting triglycerides (details available on request).

Yood *et al*,[4] in a 55 287-member inception cohort of antipsychotic users, found 357 cases of newly treated diabetes. Ziprasidone was among the group of agents with the lowest risk of diabetes. Patients exposed to olanzapine and clozapine had an increased risk of the illness.

A consensus statement on antipsychotic drugs and obesity published by the American Diabetes Association *et al* in 2004 concluded that increased risks of obesity, dyslipidaemia and diabetes are most associated with clozapine and olanzapine; little or no significant weight gain, diabetes and dyslipidaemia was associated with aripiprazole and ziprasidone, although it should be noted that these agents had not yet been used extensively at that time.[5] Further, the panel suggested switching patients who develop worsening glycaemia or dyslipidaemia to a second-generation antipsychotic not associated with significant weight gain or diabetes (i.e. ziprasidone or aripiprazole). Standards of practice that promote agents with lower metabolic risks may be a confounding factor in naturalistic studies.

Kessing *et al* acknowledge that 'individuals at higher risk of diabetes because of a personal history of obesity or inactivity, a family history of diabetes or other risk factors may have been prescribed agents perceived to confer a lower risk of diabetes'. This channelling bias affects the generalisability of their results.

They report a low risk of diabetes for aripiprazole, but the drug did not becoming commercially available in Denmark until 2004, only 1.5 years before the end of this 10-year study. A small number of patients were exposed for a limited period of time, making the direct comparison with ziprasidone not meaningful.

With regard to Table 3, given the widely differing times of drug exposure and the ultimate position of any individual agent within a single patient's treatment regimen, conclusions about the risk of an individual agent *v*. a drug class may be inappropriate based on this study design.

We are concerned about how clinicians will interpret Kessing *et al*'s findings for ziprasidone, as the results stand in contrast to the relative risks for diabetes reported in the established literature.

1 Kessing LV, Thomsen AF, Mogensen UB, Andersen PK. Treatment with antipsychotics and the risk of diabetes in clinical practice. *Br J Psychiatry* 2010; **197**: 266–71.

2 Meyer JM, Davis VG, Goff DC, McEvoy JP, Nasrallah HA, Davis SM, et al. Change in metabolic syndrome parameters with antipsychotic treatment in the CATIE Schizophrenia Trial: prospective data from phase 1. *Schizophr Res* 2008; **101**: 273–86.

3 Kahn R, Fleischhacker WW, Karayal O, Siu C, Pappadopulos E, EUFEST Study Group (2009) EUFEST: the effects of first and second generation antipsychotics on metabolic and cardiovascular risk factors. In *American Psychiatric Association 162nd Annual Meeting, 2009 New Research Abstracts*, Abstract NR1-043. APA, 2009.

4 Yood MU, DeLorenze G, Quesenberry, CP Jr, Oliveria SA, Tsai AL, Willey VJ, et al. The incidence of diabetes in atypical antipsychotic users according to agent – results from a multisite epidemiologic study. *Pharmacoepidemiol Drug Saf* 2009; **18**: 791–9.

5 American Diabetes Association, American Psychiatric Association, American Association of Clinical Endocrinologists, North American Association for the Study of Obesity. Consensus Development Conference on Antipsychotic Drugs and Obesity and Diabetes (consensus statement). *Diabetes Care* 2004; **27**: 596–601.

Declaration of interest

D.V., D.K. and O.N.K are employed by Pfizer Inc.

Douglas Vanderburg, Pfizer Inc., 235 East 42nd Street, New York, NY 10017, USA, email: Douglas.Vanderburg@pfizer.com; **Denis Keohane**, **Onur N. Karayal**, **Elizabeth Pappadopulos**, Pfizer Inc., New York, New York, USA

doi: 10.1192/bjp.198.2.157a

Authors' reply: We thank Dr Vanderburg and colleagues for their comments on our paper. We used observational, non-randomised, routinely collected data to describe the rate of incident diabetes among patients treated with antipsychotics in clinical practice. These data reflect the way antipsychotics are handled by individual clinicians on the basis of their knowledge of effects and side-effects for the specific patient. Findings from analyses of our data cannot be used to infer causality between individual antipsychotics and diabetes and may be at odds with findings from randomised trials and other studies aimed at testing specific hypotheses. Our results on the individual antipsychotics describe the prevalence of diabetes among patients for whom the clinician decided to prescribe a given antipsychotic.

Declaration of interest

L.V.K. has been a consultant for Bristol-Myers Squibb, Eli Lilly, Lundbeck, AstraZenica, Pfizer, Wyeth, Servier and Janssen-Cilag.

Lars Vedel Kessing, Department of Psychiatry, Rigshospitalet, University Hospital of Copenhagen, 2100 Copenhagen, Denmark, email: Lars.Vedel.Kessing@regionh.dk; **Per Kragh Andersen**, Department of Biostatistics, University of Copenhagen, Copenhagen, Denmark

doi: 10.1192/bjp.198.2.158