Suicide in India

We read with interest the editorial on suicide prevention from a life course perspective (Gunnell & Lewis, 2005). It offers a broad conceptual overview of the issues related to suicide.

Recent reports from Vellore suggest that suicide rates in India are grossly underreported (Joseph *et al*, 2003; Aaron *et al*, 2004; Abraham *et al*, 2005; Prasad *et al*, 2005). The average annual suicide rate was 95 per 100 000 for the years 1994–99. The rates in adolescent males and females and those over 55 years were 148, 58 and 189 per 100 000 respectively. Data from India on the contribution of mental illness to suicide rates are limited. A study from Chennai reported a higher risk of mental disorder among people who die by suicide compared with controls (Vijaykumar & Rajkumar, 1999). However, other evidence suggests that chronic stress and precipitating life events rather than severe mental disorders are the major risk factors for suicide. Recent adverse life events, interpersonal stress and relationship difficulties, severe financial distress, the use of alcohol and issues related to gender have all been associated with suicide (Prasad *et al*, 2005). The depiction of suicide in the mass media is also contributory. Last but not least is the fact that many people seem to accept suicide as an option when faced with extreme mental distress.

Although psychiatric disorders are often associated with suicide in the West and medical models are employed, in developing countries social, economic and cultural factors must be considered when attempting to explain the persistently high rates, the impulsive and stress-related deaths and the apparent widespread 'acceptability' of such an option in society. Considering suicide as a single phenomenon or even as a single final pathway might be simplistic. Many diverse approaches, tailored to regional factors, will have to be implemented simultaneously to produce any global reduction in suicide rates.

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Metabolic syndrome and schizophrenia

In his recent editorial Dr Thakore (2005) rightly highlights the importance of the association of the metabolic syndrome and one of its consequences, type 2 diabetes, with schizophrenia. Despite acknowledging that antipsychotic drugs can induce substantial weight gain, he avoids ascribing the metabolic disturbances in schizophrenia to drug-induced obesity. His suggestion that untreated schizophrenia is itself associated with metabolic disturbance is based on a study of 19 people who had substantially greater deposits of intra-abdominal fat than a control group (Ryan *et al*, 2004). This contrasts with other studies showing that 40 antipsychotic-naïve patients with schizophrenia had no elevation in intra-abdominal fat compared with controls (Zhang *et al*, 2004) and that 50 did not differ from a control group in terms of body mass index, fasting plasma glucose or insulin (Arranz *et al*, 2004). In attempting to explain discrepancies in terms of methodological differences, Dr Thakore is wrong to state that the control group of Zhang *et al* consisted of 'elderly men'; controls were well matched for age and gender with the patient group.

These larger studies also show that antipsychotic drug treatment is associated with increased intra-abdominal fat (Zhang *et al*, 2004) and insulin resistance (Arranz *et al*, 2004), despite negative findings from Ryan *et al* (2004). The risk of diabetes in schizophrenia is higher in patients receiving olanzapine rather than conventional antipsychotics (Koro *et al*, 2002); olanzapine is particularly liable to induce weight gain. These and other studies indicate that antipsychotic drug treatment can result in metabolic morbidity. It would thus be misleading, if not dangerous, to imply that obesity resulting from treatment with some antipsychotic drugs is not associated with the development of the metabolic syndrome and type 2 diabetes.

Dr Thakore listed criteria for the metabolic syndrome; these have now been superseded by a more clinically accessible and less stringent definition. The core criterion is central (abdominal) obesity, defined by waist circumference, plus two of four risk factors from elevated triglycerides, reduced high-density lipoprotein cholesterol, raised blood pressure and raised fasting plasma glucose (International Diabetes Federation, 2005).

Declaration of interest

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Author's reply: Critical methodological differences between the studies of Zhang *et al* (2004) and Ryan *et al* (2004) might explain why the two fail to agree. Zhang *et al* reported a standard deviation of 50 for the age of the controls, indicating that some were elderly; moreover the groups were not matched for gender. This is important as elderly males have higher amounts of intra-abdominal fat (IAF). Lifestyle parameters such as diet, exercise,

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smoking and alcohol intake were not measured or indeed compared between the two groups. Furthermore, we are not given any indication as to how an individual was selected for scanning, as not all of the controls and patients recruited had a magnetic resonance imaging (MRI) scan. The authors did not use the same scanning techniques as Seidell *et al* (1990), who were among the first to describe the single-slice technique for estimating IAF area. There were large differences in terms of inversion and repetition times. Moreover, the most critical aspect of using a single scan to estimate IAF is to ensure that the scan is taken at the level of L4/L5 vertebra, which is best located by a radiological lateral scout and not palpation as performed by Zhang *et al*. Furthermore, MRI is not a 'precise and reliable means of determining the two fat measures with better resolution than computed tomography', as it can erroneously estimate the amount of IAF by 20%.

From a statistical perspective, a one-way ANOVA should have been used to compare any differences between the three groups, as the use of multiple *t*-tests might have led to a type 1 error. A 'non-fasting glucose' level is not a standardised measure and is therefore meaningless. The actual values for fasting glucose decreased in both male and female patients, and fasting insulin levels decreased in females following treatment. Therefore, what Zhang *et al* show is that treatment with these two antipsychotics improves the metabolic profile of their patients despite an alleged increase in IAF.

Koro *et al* (2002) claim that olanzapine is associated with a higher risk of developing type 2 diabetes than risperidone, but this is difficult to interpret because Table 1 in their paper clearly indicates that the number of new cases of diabetes is greater in patients on risperidone (5.1%) than olanzapine (2.0%). There is little doubt that antipsychotics contribute to the development of type 2 diabetes in patients with schizophrenia. What is questionable is the magnitude of this effect. To date, the attributable risk for such an effect ranges between 2.03% for clozapine, 0.8% for quetiapine, 0.63% for olanzapine and 0.05% for risperidone (Leslie & Rosenheck, 2004).

Despite the evidence presented the debate still centres on the diabetogenic effects of certain atypical antipsychotics. The purpose of the editorial was to put these issues into perspective to ensure that patients with schizophrenia, irrespective of their prescribed medication, would be offered screening for both diabetes and the metabolic syndrome.

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CBT for treatment-resistant schizophrenia

We read with great interest the report on the randomised controlled trial (RCT) comparing cognitive–behavioural therapy (CBT) with supportive counselling for refractory psychotic symptoms of treatment-resistant schizophrenia (Valmaggia *et al*, 2005). It has a very convincing design but a few points need further discussion.

The sample size was calculated *a priori*, but an adequate number of patients could not be recruited. The small sample size led to a lack of statistical power, a limitation mentioned by the authors. However, this applied only to one intervention, supportive counselling, whereas there was an adequate estimated sample in the CBT group. Out of 62 participants randomised, post-treatment assessment was possible for 50 and follow-up was completed by 42. Although sample attrition is understandable in this kind of study the withdrawal rate is relatively high. More people in the CBT group refused assessment post-treatment compared with those who received supportive counselling. The reason for this needs to be explained. Loss of data by the assessor, leading to exclusion from the intention-to-treat analysis was greater for the group who received supportive counselling; this group already had fewer participants and the loss of data might have influenced the result.

The treatment groups were not comparable at the beginning of the study for one illness variable. The supportive counselling group reported significantly more emotional distress related to auditory hallucinations. This is important because there was no difference between the groups post-treatment and at follow-up assessment. In addition, the changes in negative symptoms reportedly favoured supportive counselling.

Valmaggia *et al* stated that 'a larger percentage of participants in the cognitive–behavioural condition showed a 20% reduction in symptoms on the positive sub-scale of the PANSS' (Positive and Negative Syndrome Scale); however, comparative figures for both treatments and statistical significance would have illustrated this better.

Previous RCTs of the effect on symptoms of CBT compared with other psychological interventions showed a number needed to treat (NNT) of 5 (National Institute for Clinical Excellence, 2003). In the index study, the NNT was 3 but the confidence intervals were large in the two areas where a significant difference was measured for CBT.

Valmaggia *et al* stated that CBT for refractory psychotic symptoms of schizophrenia should be available in in-patient facilities. However, the evidence from their study is not unequivocal. Although the literature suggests benefits from psychological intervention in this group of patients, more robust evidence is still required to confidently recommend one particular type of therapy over others.

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Personality subtypes and cognitive impairment in anorexia nervosa

I read with interest the article by Drs Thompson-Brenner and Westen (2005) about personality subtypes in eating disorders. Subnutrition from any cause is known to impair cognitive function and several workers have identified this in connection with anorexia nervosa (Macdonald, 1995).

The authors give no data on body mass index or weight. However, 38% of their sample had met criteria for anorexia nervosa at some point, 56% were fasting 4 days a week and half were exercising excessively.

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