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Authors' reply: McPhedran & Baker point out an unsolved problem of Australian suicide research. There are concerns about the quality of mortality data and statistics based upon them. Therefore, they urge researchers to approach Australian firearms data with caution. The authors cite a letter to the *Medical Journal of Australia* (De Leo, 2007) which highlighted inconsistencies in Australian mortality data since the year 2001 and called for standardised certification procedures of deaths according to ICD–10 and for other improvements of death registries. However, in Austria autopsies are performed when there is any uncertainty regarding the cause of death. The autopsy rate is high, with a mean rate of 29% in 1991–2000 (Waldhoer *et al*, 2003). If the cause of death is not clear, an additional investigation by Statistics Austria takes place. Statistics Austria registers deaths as suicide if that is the most probable cause of death. The International Classification of Diseases (ICD–8, –9, –10) has been applied for many years and there are no signs of a decrease in the data quality of Statistics Austria. The work reported by Kapusta *et al* (2007) is based on these data.

Furthermore, De Leo (2007) realistically states that some underreporting is ubiquitous and has to be tolerated in suicide statistics. On the other hand, underreporting of firearm deaths seems less probable than underreporting of, for example, deaths due to poisonings (with longer survival periods), which tend to be classified as disease-related deaths.

We agree with McPhedran & Baker that Australian firearm laws should be re-evaluated on the basis of more reliable data, but as long as sufficient evidence is not available, theoretical assumptions that Australian firearm laws had no life-saving effects remain speculative. This applies also to Europe where independent scientific evaluations of firearm law are still rare.

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Cardiovascular risk with antipsychotics: case–control study or survey?

Mackin *et al* (2007) highlighted the significantly higher risk of cardiovascular and metabolic diseases in people with severe mental illness. Rates of metabolic syndrome and cardiovascular risk similar to those in schizophrenia have been reported in bipolar disorders, and atypical antipsychotics have been approved for the treatment of the latter (Fagiolini *et al*, 2005; Birkenaes *et al*, 2007). This implies that all such populations should be studied for putative long-term adverse outcomes, as in the timely study of Mackin *et al* (2007).

However, some methodological issues need clarification. Mackin *et al* state that their study is a case–control study, but by definition a case–control study starts with an outcome and investigates exposure to putative risk factors in groups with and without the outcome (Lewallen & Courtright, 1998), generating a measure of relative risk with regard to a given risk factor. Mackin *et al* started with a group with mental illness on antipsychotics and studied the prevalence of metabolic disease and cardiovascular risk compared with controls. Thus the study has really used a survey design with a control group. The use of a control group alone does not justify the label of a 'case–control study'.

As an important corollary of this distinction, the sample size is rather low for a community-based survey. Mackin *et al* mention that comparative data for physical comorbidity in people with diagnoses other than schizophrenia are sparse; unfortunately, the study fails to generate such data owing to the inadequate sample size. We feel that Mackin *et al* have gone beyond their brief to analyse the effect of individual factors such as diagnoses, type of antipsychotic and smoking; not surprisingly, they failed to emerge with convincing findings as the sample was underpowered to generate such data.

Finally, we wonder whether the inclusion of several patients with depression and anxiety is appropriate for a study on 'severe mental illness', a term traditionally reserved for psychotic and bipolar disorders. The common denominator seems to be 'treated with antipsychotics' rather than 'severe mental illness'. It is interesting to note that the type of antipsychotics had no impact on the outcome measures (except serum insulin). If replicated in a much larger community sample, this so-called negative finding could have far-reaching implications regarding choice of treatment. Another important factor in the secondary analysis could have been the duration of treatment with antipsychotics and the dosages used. In a recent study, higher doses of medication were associated with increased cardiovascular risk scores (Osborn *et al*, 2006). Including the dosage and duration of antipsychotics in the analysis could provide important insights regarding the true impact of antipsychotics on the outcome measures.

This study, like several others, reiterates that patients treated with antipsychotics are at heightened risk for cardiovascular events and metabolic syndrome. Longitudinal studies are needed to explore the relative contribution of putative aetiological factors to physical comorbidity in severe mental illness.

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