Correspondence

First, do no harm

I welcomed the special article by Bailey *et al*.[1] I share the authors' concern over the 'scandal of premature mortality' and note their recommendation to urgently review antipsychotic medication when certain adverse effects are experienced (rapid early weight gain or cardiometabolic blood disturbance). The authors do not implicate any particular antipsychotics, but guidelines suggest that clozapine and olanzapine are the most likely antipsychotics to be associated with these side-effects.[2] Neither do the authors suggest what the outcome of such a review might be, although I deduce it is implicit in the recommendation that reducing the dose or switching antipsychotic would be likely possible outcomes. I do, however, have one concern with this suggestion which relates to the risk–benefit balance of antipsychotics.

Tiihonen *et al*[3] present data from a large study which examined the effects of antipsychotics on all-cause mortality, suicide and deaths from ischaemic heart disease; one strength of this study is the examination of all-cause mortality. The researchers found that in people with schizophrenia antipsychotic use is associated with a reduced risk of death (by about a third) when compared with no antipsychotic treatment (hazard ratio 0.68, 95% confidence interval 0.65–0.71); clozapine was associated with a substantially lower risk of all-cause mortality as well as suicide. No pronounced differences between antipsychotics (including clozapine and olanzapine) were noted for mortality from ischaemic heart disease.

Thus, if a patient is switched from clozapine to an alternative antipsychotic, their risk of death may in fact be increased rather than reduced. Further, switching antipsychotics (even olanzapine) does not appear to be associated with a reduction in risk of all-cause mortality or even death from ischaemic heart disease. Given that switching antipsychotic medication is associated with harm, for example by increasing risk of relapse,[4] this leads me to question the wisdom of Bailey *et al*'s recommendation to urgently review the antipsychotic prescription in the circumstances they describe.

There may be other reasons for switching antipsychotics but Tiihonen *et al*'s findings suggest that reducing the 'scandal of premature mortality' is not one of them. This raises a dilemma for practising clinicians as to how to proceed in these circumstances.

1 Bailey S, Gerada C, Lester H, Shiers D. The cardiovascular health of young people with severe mental illness: addressing an epidemic within an epidemic. *Psychiatrist* 2012; **36**: 375–8.

2 Taylor D, Paton C. *Maudsley Prescribing Guidelines in Psychiatry* (11th edn). Wiley–Blackwell, 2012.

3 Tiihonen J, Lönnqvist J, Wahlbeck K, Klaukka T, Niskanen L, Tanskanen A, et al. 11-year follow-up of mortality in patients with schizophrenia: a population-based cohort study (FIN11 study). *Lancet* 2009; **374**: 620–7.

4 Weiden P. Switching antipsychotic medications: not enough, too often, or just right. *Am J Psychiatry* 2011; **168**: 882–4.

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Cardiovascular disease and schizophrenia: do we know enough?

We find the aims of Bailey *et al*[1] laudable. However, we would like to add a note of caution. Our main concern is that many of the recommendations are not based on evidence. Bailey *et al* assume that people with schizophrenia are the same as the general population, the so-called 'ecological fallacy'. The authors describe potential differences such as the increased risk of metabolic abnormalities including diabetes which pre-date the prescription of antipsychotics. Therefore, it cannot be assumed that what is effective in the general population will be equally effective in people with schizophrenia. For example, controversy surrounds the diabetogenic effect of statins in the general population and Nielsen *et al*[2] demonstrated that lipid-lowering medication was a greater risk factor for the development of diabetes in a cohort of people with schizophrenia than was 'high-risk' antipsychotic medication. Furthermore, a Finnish cohort study[3] replicated the finding of poor outcomes for cardiovascular disorders in patients with schizophrenia and reiterated that the excess morbidity could not be explained by prescription rates of lipid-lowering drugs.

Bailey *et al* present a comprehensive overview of cardiovascular risk management and although we may be guilty of the same assumption as the authors, we would like to emphasise the importance of cardiorespiratory fitness as a modifiable risk factor. Its significance is often neglected or understated, with guidelines emphasising medical management. However, Kilbourne *et al*[4] reported that physical inactivity (hazard ratio 1.66, 95 CI 1.59–1.74) was a greater risk factor than smoking (hazard ratio 1.32, 95% CI 1.26–1.39) for cardiovascular mortality in a cohort of people with schizophrenia. The complexity of mortality risk factors in early schizophrenia is further illustrated when one examines the relationship between body mass index (BMI) and suicide in the general population. Suicide, and not cardiovascular disease, is the major mortality risk in younger people with schizophrenia. An emerging paradox is linking an inverse association between BMI and suicide risk in the general population; hence a lower BMI may reduce cardiovascular risk but increase suicide risk.[5] Whereas there is emerging evidence that patients with schizophrenia are receiving medical treatment for cardiovascular risk factors,[3] there is little evidence so far that this has reduced mortality.[1]

If the people with schizophrenia are seen as a high cardiovascular risk population with attendant early and aggressive medical intervention, the impact on core symptom outcomes needs to be studied as some of the antipsychotics with the greatest liability for metabolic side-effects are also the more effective. Clearly, more research is required to understand the relative importance of mortality risk factors in schizophrenia and their management.[5]

Declaration of interest

R.E.H. and M.B. have received research funding and hospitality from pharmaceutical companies. H.W. is an ex-Lilly employee.

1 Bailey S, Gerada C, Lester H, Shiers D. The cardiovascular health of young people with severe mental illness: addressing an epidemic within an epidemic. *Psychiatrist* 2012; **36**: 375–8.

2 Nielsen J, Skadhede S, Correll CU. Antipsychotics associated with the development of type 2 diabetes in antipsychotic-naive schizophrenia patients. *Neuropsychopharm* 2010; **35**: 1997–2004.

3 Lahti M, Tiihonen J, Wildgust H, Beary M, Hodgson R, Kajantie E, et al. Cardiovascular morbidity, mortality and pharmacotherapy in patients with schizophrenia. *Psychol Med* 2012; **42**: 2275–85.

4 Kilbourne AM, Morden NE, Austin K, Ilgen M, McCarthy JF, Dalack G, et al. Excess heart-disease-related mortality in a national study of patients with mental disorders: identifying modifiable risk factors. *Gen Hosp Psychiatry* 2009; **31**: 555–63.

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Iatrogenicity: are we largely to blame for this epidemic?

Notwithstanding the premorbid genetic and psychosocial predispositions Bailey *et al* refer to,[1] the authors also correctly highlight the incontrovertible evidence that the obesity and metabolic syndrome epidemic we are facing is largely drug induced, as highlighted by the EUFEST study.[2] Given this, we must accept that we are essentially complicit in greatly increasing our own patients' morbidity and mortality, and that this 'epidemic within an epidemic' is iatrogenic. I cannot help but wonder whether we, as clinicians, tend to ignore a side-effect which we consider to be 'benign', in relation to the perceived lack of an immediate need to address it urgently, as opposed to, for example, an acute extrapyramidal side-effect, massively raised prolactin or marked electrocardiogram changes. I wonder whether our complacency in addressing this adverse effect profile may be borne out of a sense of our own helplessness. That is to say, because there is no straightforward solution to this multifaceted problem, we choose to ignore or at least sidestep the issue. It is precisely because of the creeping, insidious nature of these obesity-related problems that we are allowing them to develop into an 'epidemic' of such proportions.

We must ask ourselves whether it is morally acceptable to treat chronic and enduring mental illness at the expense of inflicting chronic and enduring physical illnesses. As the authors allude, if we actually bothered to ask our patients, particularly the younger ones, what it is they would be most distressed by – continued mental illness or aggressive weight gain – would it really be so surprising that a sizeable proportion would prefer to remain distressed by (or learn to cope with) their psychiatric symptoms than become morbidly obese? Should this really come as a shock to us, given the strongly body-conscious world in which we live? I suspect that our priorities as psychiatrists may not be entirely aligned with those of many of our patients. Is there a doctor–patient risk–benefit analysis mismatch at play here?

But are we really improving our patients' quality of life and promoting social inclusion by treating one stigmatising condition for another, which arguably carries even greater prejudice? After all, most of the population view morbidly obese people not only as a repulsive eyesore, but tend to apportion blame. Many view obesity as a self-inflicted condition, borne purely out of laziness and gluttony, and tend to make extremely pejorative judgements.

Notwithstanding this, although antipsychotics are the only truly effective weapons in our armament against chronic psychotic disorders, it is incumbent on us to make prescribing decisions which take from the outset the potential ramifications of such physically and socially disabling adverse effects into account.

At the end of the day, if I was a patient, I would not be happy to learn that I had developed a serious, chronic physical disorder with many potential multisystem complications (such as diabetes) as a result of taking a drug which I probably was not keen to take in the first place anyway, and was never fully appraised of the risks. We must never be economical with the truth about the drugs we are all too happy to dish out.

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Physical health epidemic in mental health

We would very much welcome the focus on physical health from secondary mental health, as advocated by Bailey *et al*.[1] However, we would like to raise the following points.

The Quality Outcomes Framework[2] now includes HbA1c levels recorded in the past 15 months to identify diabetes for patients aged 40 years and over with schizophrenia, bipolar affective disorder and other psychoses (MH20). It is worth noting that the World Health Organization has included HbA1c in its diagnostic criteria for diabetes and this is also being backed up by the National Institute for Health and Clinical Excellence.[3] We think that it is important to have HbA1c levels recorded, especially in patients on antipsychotics.

The incidence of metabolic syndrome in psychiatric patients has been covered recently in this journal,[4] but Bailey *et al* could have highlighted the need for baseline physical health monitoring before commencing on antipsychotics. Moreover, there is a known higher incidence of diabetes in patients with psychosis. Therefore, psychiatrists play a major role in reminding other clinicians and reiterating in their communication to general practitioners the importance of following parameters such as weight, blood pressure and glucose levels in the early weeks, so the primary care team are aware and the patients are appropriately followed up and supported.

Bailey *et al* seem to be suggesting that antipsychotics have no role in the management of psychosis and the disorder can be treated with a multiprofessional approach. It might have been better to mention the impact of duration of untreated psychosis on the long-term patient-related outcomes,[5] and so I would have thought that antipsychotics would be the essential