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doi: 10.1192/pb.37.1.33a

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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doi: 10.1192/pb.37.1.34

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

part of a biopsychosocial approach rather than a treatment of last resort.

Finally, I am glad to hear about the Royal College of General Practitioners' involvement with the Royal College of Psychiatrists in coming up with a collaborative framework. I welcome the Bailey *et al* article and the joint collaboration and would hope more joint work is carried out in the future between primary and secondary care teams.

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doi: 10.1192/pb.37.1.34a

Authors' response: Dr Chaparala asks if we would have been better mentioning how duration of untreated psychosis affects long-term outcomes. Is not a 20-year mortality gap for men, and 15 years for women, a significant long-term outcome and an impact of untreated cardiometabolic risk deserving of some earlier intervention?

Notwithstanding incontrovertible evidence that antipsychotics cause problematic weight gain, we do not suggest antipsychotics are the sole explanation of increased cardiovascular disease, but do highlight how antecedent risks can become established in the critical early treatment phase. This is further supported by another recent systematic review observing cardiometabolic changes only after antipsychotic initiation.[1] The subsequent trajectory of weight gain, increasing metabolic disturbance and sustained heavy smoking provides a compelling link between schizophrenia and cardiovascular disease,[2] the single most important cause of premature death in this population.

Furthermore, the National Institute for Health and Clinical Excellence (NICE) are clear in their recommendations that these adverse cardiovascular risks should be identified at the earliest opportunity and managed using the appropriate NICE guidance for prevention of these conditions (the 2009 updated guidance for schizophrenia, CG82; recommendation 10.4.1.3). And yet when the recent Royal College of Psychiatrists' National Audit of Schizophrenia (NAS) examined the implementation of NICE recommendations in community settings (NAS report 2012; www.rcpsych.ac.uk/quality/NAS), it found that only 29% of people with schizophrenia across England and Wales had received an adequate assessment of cardiometabolic risk within the previous 12 months; 44% had not even been weighed.

Does this apparent lack of concern about adverse cardiometabolic consequences revealed by the NAS matter? After all, Dr Reed is reassured about antipsychotic safety by the FIN11 study of Tilhonen *et al*. However, authorities De Hert *et al*[3] have challenged this study's conclusions, listing methodological weaknesses which include

> 'incomplete reporting of data, questionable selection of drug groups and comparisons, important unmeasured risk factors, inadequate control for potentially confounding variables, exclusion of deaths occurring during hospitalization leading to exclusion of 64% of deaths on current antipsychotics from the analysis, and survivorship bias due to strong and systematic dierences in illness duration across the treatment groups.'

Dr Reed raises the issue of switching antipsychotics and how this may destabilise control of psychosis but may have missed the point of Weiden's editorial that he refers to. While indeed not advocating switching antipsychotics in someone established on treatment, Weiden highlights how two randomised studies demonstrated the positive value of switching antipsychotics to counteract rapid weight gain and metabolic change, concluding: 'Practice guidelines and public policy should recommend that clinicians consider the value of switching antipsychotics in patients with elevated metabolic risk.'[4]

Dr Chaparala suggests we are abandoning antipsychotics. No, but we are in good company in questioning the dominance of psychopharmacology.[5] Moreover, excessive reliance on antipsychotic treatment is suggested by the NAS finding of wide variation in the availability of psychological treatments across England and Wales: even in those patients whose response to antipsychotics had been unsatisfactory, 34% were not offered any form of psychological treatment despite NICE recommendations that these should be considered.

What we urge is responsible prescribing, particularly in the critical early phase of illness and sensitivity by us as doctors to how these young people may feel about the effects of our treatments. Perhaps the final word should go to the closing comment of Dr Tagore's letter: 'We must never be economical with the truth about the drugs we are all too happy to dish out.'

Declaration of interest

D.S. is current member of two Guideline Development Groups (GDG) for NICE: NICE guidance for children and young people affected by psychosis and schizophrenia, and NICE guidance for adults with psychosis and schizophrenia. The views expressed are not those of GDG, NCCMH or NICE. (The declaration applies to this letter and to the original article.)

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