Correspondence

Recovery and medical model – yes, science does matter

Authors' reply We welcome Holloway's (2008) commentary on our article 'Recovery and the medical model' (Mountain & Shah, 2008) and agree that the article raises further questions. We would, however, like to clarify that the medical model described relates to evidenced interventions practised by a range of professionals, including doctors. Doctors are certainly not the only professional group to use evidenced-based interventions. We agree that the task of answering the question 'Does recovery work?' is complex and Holloway helpfully describes some of the methodologies that may have to be deployed.

We acknowledge that by using the four elements of Resnick's work other important themes, such as meaning and self-management, were not explored. However, should Resnick's work be considered less relevant than the work of others? Of course the user perspective is central to the recovery agenda but this raises the issue of professional involvement in this agenda. In the same way that users advocate to be active participants in their care, professionals should also be actively included as equal partners to progress and mainstream recovery practice. If this is not encouraged, the view of psychiatrists as authoritarian and of professional care as 'something to get away from' could be needlessly promulgated, to the detriment of both parties and of developing recovery-oriented services. The relationship is complex, given service users' drive for self-determination, influenced by their experience of professional care and complicated by the potential power differential between the professionals and the often vulnerable individuals seeking their help. However, in working together to understand and develop recovery-based practice we must not let that dynamic persist. It is in developing humanistic skills while not foregoing our other professional skills that we find more in common with our patients than what separates us. This could be the common ground allowing all parties to use their unique sets of skills and capacities.

Holloway, F. (2008) Is there a science of recovery and does it matter? Invited commentary on... Recovery and the medical model. *Advances in Psychiatric Treatment*, **14**, 245–247.
Mountain, D. & Shah, P. (2008) Recovery and medical model. *Advances in Psychiatric Treatment*, **14**, 241–244.

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Fragile male, not fragile-X

No doubt the fragile-X chromosome plays a small part in male disadvantage, but the point made in my review (Kraemer, 2000), referred to by Branney & White (2008), is that the male is biologically less resilient. Skuse *et al*'s (1997) ingenious study of Turner syndrome shows that the X chromosomes inherited from mothers produce a different phenotype from paternally derived ones, but the bulk of male disadvantage probably derives from the Y chromosome.

Branney & White also err in summarising my argument. It is not that 'this disadvantage is immediately mitigated once an infant's sex is known' (p. 260). On the contrary it is compounded, in ways they themselves describe in their article. Prevailing assumptions about male resilience add 'social insult to biological injury' (Kraemer, 2000: p. 1612).

Branney, P. & White, A. (2008) Big boys don't cry: depression and men. *Advances in Psychiatric Treatment*, **14**, 256–262.
Kraemer, S. (2000) The fragile male. *BMJ*, **321**, 1609–1612.
Skuse, D., James, R. S., Bishop, D. V. M., *et al* (1997) Evidence from Turner's syndrome of an imprinted X-linked locus affecting cognitive function. *Nature*, **387**, 705–708.

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Smoking bans and clozapine levels

The smoking ban implemented in the UK this July (Campion *et al*, 2008) will greatly affect psychiatric in-patients, of whom as many as 74% are smokers (Meltzer *et al*, 1996). Plasma concentrations of certain psychotropics are known to be affected by smoking status. Smokers are usually prescribed higher doses than non-smokers and abrupt smoking cessation will lead to high plasma concentrations and potentially more side-effects.

Clozapine plasma concentrations can rise 1.5 times in the 2–4 weeks following smoking cessation (de Leon, 2004) and in some instances by 50–70% within 2–4 days. If baseline plasma concentrations are higher – particularly over 1 mg/litre – the plasma concentration may rise dramatically owing to