**Authors' reply:** Turnbull and his colleagues raise some important points, most of which are covered in our paper. Our first paragraph discusses the fact that PD was initially designed for use in groups. Turnbull *et al* will be aware that PD has been widely used as a stand-alone intervention for groups and individuals despite it being developed as part of a CISM.

The total period of the study was 28 months, 32 subjects were debriefed by a research psychiatrist and 25 by five burns unit nurses. The nurses were involved in procedures such as changing dressings but the outcome of subjects debriefed by a burns nurse was not worse than those debriefed by a research psychiatric registrar. Their knowledge of the physical aspects were reported by some individuals as having been beneficial (as stated in our paper the majority reported the PD as useful). A private side room was identified to use for the debriefings.

Most of the patients described some pain and many were taking analgesia but individuals were only debriefed at a time when they were felt able actively to participate in the process. To wait until individuals were totally pain-free and analgesia-free would have been to wait beyond the 13-month follow-up period in some instances. With regard to the slightly increased overall dimensions of trauma and levels of distress, these are discussed within our paper. We consider our discussion fair in that we included these along with a detrimental effect of PD and chance as the four possible explanations for our results.

The 'two hour' comment is somewhat bewildering given the fact that a longer PD was more likely to be associated with poor outcome (as stated in our paper). There were several individuals without significant psychological sequelae who had little to discuss and hence their PD was brief.

Turnbull *et al* state that the phenomenon of increased symptomatology after PD is well-recognised and probably part of the natural process of adjustment. What evidence do they have for this? An alternative explanation is that the PD may cause unnecessary increased distress in some individuals.

PD is a classical example of an innovation that has come into practice without an adequate research base (McKinley, 1981). It is only after its acceptance by many that its effectiveness has begun to be scrutinised in a systematic way. Negative results may therefore by extremely threatening to some individuals. We acknowledge that our research has some shortcomings which must be taken into account when interpreting the results (as they are in our paper) but to discard our results would be unscientific. It is important to note that our main conclusion (lack of positive effect of individual PD in this population) is consistent with the results of the other two published randomised controlled trials of individual PD (Hobbs *et al*, 1996; Lee *et al*, 1996).

**Hobbs, M., Mayou, R., Harrison, B, et al (1996)** A randomised controlled trial of psychological debriefing for victims of road traffic accidents. *British Medical Journal*, **313**, 1438–1439.

**Lee, C., Slade, P. & Lygo, V. (1996)** The influence of psychological debriefing on emotional adaptation in females following early miscarriage. *British Journal of Medical Psychology*, **69**, 47–58.

**McKinley, J. B. (1981)** From "Promising Report" to "Standard Procedure": seven stages in the career of a medical innovation. *Millbank Memorial Fund Quarterly*, **59**, 374–411.

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**P. L. Jenkins** St Cadoc's Hospital, Caerleon, Gwent

**Sir:** I was most interested to read the report of Bisson *et al*, as I am a psychiatrist, and a victim of a severe burn suffered near the end of my internship. I incurred third-degree burns to the upper 35% of my body. I was awake throughout the accident and subsequent fire. I received no psychological debriefing. I spent eight months in the burn unit. I entered psychiatric residency training 20 months after the burn.

As a burn patient and, later, a consultant psychiatrist, I have observed the coping of a number of burn victims. Some specific aspects of burn injuries might contribute to the negative results reported by Bisson *et al*. One difference between burns and other major trauma is that patients rarely report severe burns to be painful at the time of occurrence. Severe pain, which makes up much of the traumatic element of burn injuries, comes later, associated with dressing changes, debridement, grafting, physiotherapy, surgery, etc. Furthermore, progressive scarring after a burn often causes more ultimate problems (e.g. disfigurement, restriction of joint mobility) than the burn itself. Scarring can take 12–18 months to mature fully. Roca *et al* (1992) have shown that adult burn survivors often develop new symptoms of psychological distress after they have left hospital. Thus, early debriefing in the hospital may be timed too soon for most patients to benefit, in that their most traumatic experiences in relation to the burn may still be months down the road.

The pre-injury psychosocial status of the patient is probably the major determinant of the psychological outcome of burn trauma (Browne *et al*, 1985). Burns are often the result of human misadventure, which can be a direct result of personality. For example, many burns occur in the context of excessive alcohol use, or as a result of reckless behaviour. A number of such burn victims will already have prior histories of immature personality functioning and poor coping with adverse life events. Psychological debriefing, on its own, will not suffice to give such patients the ego strength to deal effectively with the ongoing suffering of a burn injury.

Bisson *et al* have performed a real service by performing this study of what might otherwise be considered a 'commonsense' intervention. I commend them, and the *Journal*, for providing awareness of these provocative negative results.

**Bisson, J. I., Jenkins, P. L., Alexander, J., et al (1997)** Randomised controlled trial of psychological debriefing for victims of acute burn trauma. *British Journal of Psychiatry*, **171**, 78–81.

**Browne, G., Byrne, C., Brown, B., et al (1985)** Psychosocial adjustment of burn survivors. *Burns*, **12**, 28–35.

**Roca, R. P., Spence, R. J. & Munster, A. M. (1992)** Posttraumatic adaptation and distress among adult burn survivors. *American Journal of Psychiatry*, **149**, 1234–1238.

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**Sir:** We read with interest the study by Bisson *et al* (1997). The finding that the debriefed group did not benefit and may actually have had a poorer outcome is supported by the similar conclusion of Hobbs *et al* (1996), but we have concerns about aspects of the methodology which led to this result.

Bisson *et al* stated that they terminated recruitment "when preliminary analysis of the data revealed possible adverse consequences for the intervention group". We agree that it is unethical to continue a trial where there is clear evidence that one group is receiving a detrimental treatment. However, when performing significance tests in interim analysis it should be remembered that the more often one analyses accumulating data the greater the chance of



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eventually detecting a spuriously significant treatment difference (type I error). Accordingly, it is good practice to decide in advance precisely what would be sufficiently strong evidence of a treatment effect to merit stopping the trial, taking into account the magnitude of the difference in outcome considered clinically important, the number of interim analyses to be performed, and the level of statistical significance required (Pocock, 1983). Unfortunately, Bisson *et al* do not state whether any stopping rules were explicitly determined before the trial commenced, or whether there was any limit on the number of interim analyses, and it is therefore possible that their study was prematurely stopped at an inappropriate point.

This difficulty would be less important were it not that at the time at which the study was terminated the debriefed group not only had experienced more severe burn traumas than the controls, but in addition almost twice as many debriefed subjects had reported significant previous trauma, both of these factors increasing their risk for the development of more numerous and severe post-traumatic symptoms (McFarlane & Yehuda, 1996). It may be that had the study continued recruiting patients according to its random protocol the background differences between the two groups would have diminished and the outcome findings could have been different.

**Bisson, J. I., Jenkins, P. L., Alexander, J., *et al* (1997)** Randomised controlled trial of psychological debriefing for victims of acute burn trauma. *British Journal of Psychiatry*, **171**, 78–81.

**Hobbs, M., Mayou, R., Harrison, B., *et al* (1996)** A randomised controlled trial of psychological debriefing for victims of road traffic accidents. *British Medical Journal*, **313**, 1438–1439.

**McFarlane, A. C. & Yehuda, R. (1996)** Resilience, vulnerability, and the course of posttraumatic reactions. In *Traumatic Stress* (eds. B. A. van der Kolk, A. C. McFarlane & L. Weisaeth), pp. 155–181. London: Guilford Press.

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## Cost-effectiveness of clozapine

**Sir:** Robert & Kennedy's editorial (1997) on our paper (Aitchison & Kerwin, 1997) cannot pass without comment. This was not a clinical trial of clozapine and should not be judged as such: our study was a cost-effectiveness analysis.

However, we would like to comment on the efficacy figure that they quoted. They quote Baldessarini & Frankenburg (1991) as giving a figure of 13% of patients being better off on clozapine than on typical neuroleptics (from trials, largely double-blind), omitting a comment from the same paper that "it is increasingly apparent that . . . about a third of patients with chronic psychosis improve more in response to clozapine than other drugs". In a more recent review, Umbricht *et al* (1995) concluded that "clozapine is the first antipsychotic agent with proven superiority over conventional antipsychotics in the treatment of severely ill, chronic schizophrenic patients", showing a response rate 26–46% higher than that to chlorpromazine or haloperidol. The Cochrane Collaboration Schizophrenia Group systematic review (Wahlbeck *et al*, 1997) on clozapine currently includes 27 randomised controlled trials, and concludes that clozapine is "convincingly more effective than 'typical' neuroleptic drugs in reducing symptoms of schizophrenia, producing clinically meaningful improvements and postponing relapse".

In the UK those who are eligible for clozapine are refractory to, or intolerant of, standard neuroleptics. The response rate of such patients to standard neuroleptics is therefore very low (the argument is circular). Randomised controlled trials are hardly necessary to conclude that there is a substantial advantage of clozapine over standard neuroleptics in treatment-resistant schizophrenia in terms of efficacy.

**Aitchison, K. & Kerwin, R. W. (1997)** Cost-effectiveness of clozapine. *British Journal of Psychiatry*, **171**, 125–130.

**Baldessarini, R. J. & Frankenburg, F. R. (1991)** Clozapine. A novel antipsychotic agent. *New England Journal of Medicine*, **324**, 746–754.

**Robert, G. & Kennedy, P. (1997)** Establishing cost-effectiveness of atypical neuroleptics. *British Journal of Psychiatry*, **171**, 103–104.

**Umbricht, D. S. G., Lieberman, J. A. & Kane, J. M. (1995)** The clinical efficacy of clozapine in the treatment of schizophrenia. *Reviews in Contemporary Pharmacotherapy*, **6**, 165–186.

**Wahlbeck, K., Cheine, M., Essali, M. A., *et al* (1997)** Clozapine for schizophrenia. Clozapine versus 'typical' neuroleptic medication for schizophrenia. In *Schizophrenia Module* (eds C. E. Adams, L. Duggan, M. J. De Jesus, *et al*). The Cochrane Database of Systematic Reviews (available in the Cochrane Library). London: BMJ Publishing.

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## Cannabis and schizophrenia

**Sir:** In their recent editorial Hall & Solowij (1997) were perhaps a little too sanguine about the relationship between cannabis consumption and schizophrenia. At least six studies, several of them prospective, have found a strong relationship between levels of cannabis use before the onset of psychotic symptoms and the subsequent development of schizophrenia and other chronic psychotic illnesses (Boutros & Bowers, 1996). Moreover, those patients with schizophrenia who had used cannabis prior to the onset of their illness are distinct, both demographically and clinically, from those who had not: younger, more often male, with better premorbid functioning, and a different symptom pattern, response to neuroleptics and subsequent disease course (Longhurst *et al*, 1997). Thus, prolonged cannabis use appears to induce chronic psychosis in a group of individuals who are sufficiently different from the general mass of patients with schizophrenia to suggest that, in the absence of such use, they might not be especially vulnerable.

For many years, clinicians have been aware that the use of cannabis may lead to persistent psychosis (Glass & Bowers, 1970). The weight of both decades of clinical experience and the current literature strongly suggests that cannabis use can result in chronic psychoses, including schizophrenia.

**Boutros, N. N. & Bowers, M. B. (1996)** Chronic substance-induced psychotic disorders: the state of the literature. *Journal of Neuropsychiatry and Clinical Neurosciences*, **8**, 262–269.

**Glass, D. S. & Bowers, M. B. (1970)** Chronic psychosis associated with long-term psychotomimetic drug use. *Archives of General Psychiatry*, **23**, 997–1003.

**Hall, W. & Solowij, N. (1997)** Long-term cannabis use and mental health. *British Journal of Psychiatry*, **171**, 107–108.

**Longhurst, J. G., Boutros, N. N. & Bowers, M. B. (1997)** Cannabis-induced chronic psychosis – an under-acknowledged disorder? *Australian and New Zealand Journal of Psychiatry*, **31**, 305–306.

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**Author's reply:** It is difficult in the space limits of an editorial to do justice to the issues involved in deciding whether cannabis use can produce chronic psychoses such as schizophrenia, when informed opinion differs and the evidence is inconsistent.


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