score dropped to 9 points. Soon after, he was able to fly back home escorted by his family.

Case 2: Mrs L., a 43-year-old woman with no previous psychiatric history was admitted 72 hours following an eastbound flight to Israel (−10 hours). The patient exhibited severe jet-lag manifestations culminating in florid psychotic state with elevated manic affect. Her BPRS score reached 33 points.

Treatment solely with melatonin (6 mg at bedtime) was initiated. This resulted in a good night's sleep and restoration of day/night schedule. After four nights of melatonin administration, the patient's delusional ideations resolved, and her BPRS score dropped to 8 points. She was able to fly back home unescorted. From follow-up inquiries, we learned that she did not need psychiatric treatment following her westbound return flight.

The clinical implication of this report is that apart from triggering psychotic relapse, jet-lag and changes in circadian rhythm may be associated with *de novo* psychotic breakdown. Other psychological factors, such as unfamiliar surroundings, strange language, cultural clash, and religious excitement may also play a role.

Melatonin is proposed as an agent which induces sleep and overcomes jet-lag manifestations (Arendt & Deacon, 1997; Brzezinski, 1997). Case 1 illustrates that the combined use of antipsychotic medications and melatonin served to reduce signs of psychosis and restored circadian rhythm. Case 2 suggests that melatonin may suffice in jet-lag-associated psychosis.

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Development of obsessive and depressive symptoms during risperidone treatment

Sir: Risperidone, like clozapine, has been associated with the induction or exacerbation of obsessive–compulsive symptoms, which has been hypothesised to be related to its 5-HT_2 antagonistic action (Eales & Layeni, 1994; Kopala & Honer, 1994; Remington & Adams, 1994; Alzaid & Jones, 1997). It could also be speculated that risperidone's antiserotonergic properties could lead to obsessive and depressive symptoms, as the following case demonstrates.

A 29-year-old man with ICD–10 paranoid schizophrenia was placed on risperidone monotherapy (4 mg/day). Within one month he developed ICD–10 major depression and obsessions (repetitive cursing thoughts with religious and sexual content). The depression interfered seriously with everyday activities. After starting fluoxetine (20 mg/day) his obsessions resolved within two weeks and the depression resolved within three weeks. Over the fourth week he developed akathisia, and fluoxetine was discontinued. The akathisia resolved but within the next four weeks the depressive and obsessive symptoms relapsed. Risperidone was decreased to 2 mg/day but the symptoms did not resolve (although he experienced a reduction in frequency and intensity) and the treatment was stopped. The patient was put on pimozide without re-emergence of these symptoms. He had no prior history of obsessive or depressive symptoms. There was no evidence of an organic aetiology.

Unlike many traditional antipsychotics, risperidone is a more potent antagonist of serotonin (5-HT_2) than of dopamine (D_2) receptors and this action has been postulated to contribute to its atypical effects and to produce or unmask obsessive–compulsive symptoms (Eales & Layeni, 1994; Kopala & Honer, 1994; Remington & Adams, 1994; Alzaid & Jones, 1997). There have not been any reported cases of depression or combination of depressive and obsessive symptoms in the literature up to now. The emergence of these symptoms during the course of treatment with risperidone, the positive effects of fluoxetine, the re-emergence of these symptoms after discontinuation of fluoxetine and their resolution when risperidone was discontinued, together provide strong evidence that risperidone was a causative factor.

The combination of a serotonin receptor blocker (risperidone) and a serotonin reuptake inhibitor (fluoxetine) appears to be antagonistic. It is well known that fluoxetine is useful for the treatment of depression and obsessive–compulsive symptoms. Our data suggest that serotonin blockade may underlie the development of depressive and obsessive symptoms in the course of risperidone treatment. The success of fluoxetine in treating these symptoms supports this conclusion further, as do reports involving obsessive symptoms managed with fluvoxamine (Remington & Adams, 1994) or with discontinuation of risperidone (Kopal & Honer, 1994).

However, fluoxetine caused akathisia, although it did not exacerbate the psychotic symptoms in this patient. Clinicians need to be aware of fluoxetine's potential to activate psychotic processes or cause extrapyramidal side-effects (Lindenmayer *et al*, 1990).

These topics are complicated because of the different actions of risperidone and fluoxetine on different parts of the central nervous system and on different types of receptors (5-HT_1, 5-HT_2, D_1, D_2, etc.) (Eales & Layeni, 1994; Kopala & Honer, 1994), the interactions of these two agents, as well as antidepressant effects of risperidone (Dwight *et al*, 1994).

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Definitions of depression

Sir: The naturalistic follow-up study by Ramana *et al* (1999) emphasised the

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continuing confusion that exists in the definition of recovery, remission, relapse and recurrence of depression, in spite of the pains that the authors took to use contemporary definitions and guideline recommendations for continuation and maintenance antidepressant treatment.

The definition adopted by the authors was derived from Frank *et al* (1991) where remission begins when a patient does not have any of the Research Diagnostic Criteria (RDC) symptoms of major depression. If remission is maintained for eight weeks, the patient is considered recovered. Return of symptoms of major/minor depression during the eight weeks after losing symptoms heralds a relapse, whereas if this occurs after an eight-week symptom-free interval the individual is considered to have had a recurrence. The pivotal importance given to a two-month symptom-free interval in differentiating a relapse from a recovery is embodied in DSM–IV (American Psychiatric Association, 1994) as well, whereas ICD–10 (World Health Organization, 1992) skirts the issue by using the phrase "several months" rather than commit itself to a definite time frame. However, continuation-phase treatment with antidepressants is usually recommended for four to six months after full recovery. Using the recommendation of four months of continuation treatment (Depression Guideline Panel, 1993), Ramana *et al* (1999) observed that 31 of 77 subjects who 'recovered' from depression had a return of symptoms in the continuation phase of antidepressant treatment. This implies that they had not actually recovered from the underlying pathophysiology of the episode but had only achieved symptomatic recovery. They would be then classified as having had a relapse rather than a recurrence, as would be the case if Frank *et al*'s definition (1991) were followed.

A recent randomised controlled trial by Reimherr *et al* (1998) on the optimal length of continuation therapy in depression addresses this crucial issue in the research and treatment of depressive disorders. Based on their trial, which involved prospective transfer to placebo at multiple points, the authors recommend an additional 26 weeks of fluoxetine after remission to prevent re-emergence of depressive symptoms, thereby proposing that the end of this period defines recovery from the underlying pathophysiology of an episode of depression. This suggests that two months of remission, as proposed by Frank *et al* (1991) and in DSM–IV, is inadequate to define complete recovery from an episode of depression, and warrants fresh attempts to achieve consensus definitions for remission, recovery, relapse and recurrence in major depressive disorder.

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Criteria for traumatic grief and PTSD

Sir: We were interested in Prigerson *et al*'s (1999) consensus criteria for traumatic grief. There were some concepts in the distinction from post-traumatic stress disorder (PTSD) with which we would take issue. Criterion B, in particular, appears to overlap significantly with PTSD. Avoidance of reminders is one of the key criteria in DSM–IV PTSD (American Psychiatric Association, 1994), as well as emotional numbing. In fact, most of Prigerson *et al*'s criterion B can be seen in PTSD. We also feel concerned about the two months of symptoms which the authors have used as a time scale, since clinically this overlaps with normal grief. The distinctness of this diagnosis must, therefore, be questioned. No mention is made of the adjustment reaction and the distinction from this, and in ICD–10 (World Health Organization, 1992) many atypical grief reactions are put in this section.

The authors had a 42% response rate in their study which was predominantly female, of a mean age of 61 years and Caucasian. This undermines the generalisability of the study. Furthermore, in our clinical work on PTSD, avoidance symptoms often delay presentation and this might be significant in the rest of their sample.

Post-traumatic stress disorder is a condition with a 50–95% comorbidity (Green *et al*, 1992) and it is inherently problematic to sort out comorbidity (Yehuda & Mcfarlane, 1995). Further, a recent epidemiological study found a risk of developing PTSD of 31% following unexpected death of a loved one (Breslau *et al*, 1998). A recent paper reinforced the link between grief and PTSD, showing that they appear to share common predictors (Sprang & McNeil, 1998). If this is the case, then perhaps PTSD and traumatic grief syndrome represent a spectrum of severity, or are potential alternatives, or are potentially comorbid. We also wonder whether including this as a sub-specifier in PTSD might be a better place for it, rather than as a distinct diagnosis. The work which the authors have undertaken is preliminary but we feel may assist in the better definition of PTSD and traumatic grief.

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Authors' reply: We appreciate the attention Dr Fox and colleagues have drawn to the distinction between the criteria for PTSD and those we propose for traumatic grief. As stated in our article: "we acknowledge the reaction to be a stress response syndrome

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