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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G. Katz, R. Durst, Y. C. Barel, H. Y. Knobler The Jerusalem Mental Health Center, Kfar Shaul Hospital, affiliated with the Hebrew University, Hadassah Medical School, Jerusalem, 91060, Israel

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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B. Panagiotis, G. Maria, L. Aris Department of Psychiatry, University of Ioannina Medical School, Kommenou 5 Arta, GR-47100, Greece

Definitions of depression

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

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