

While families would agree that the prime focus of the psychiatric team must be the mental health of the patient, organisations such as ours would argue that a supportive family provides a large contribution to the short- and long-term health of the patient, whether the latter eventually lives in the family home or not.

For the family to be able to support the patient, they must have the understanding and information necessary to provide appropriate care. Ideas on confidentiality need to be re-thought. On admission to hospital, the patient may say 'no' to informing the family, but during their hospital stay and before discharge, they should be asked again. They may well have changed their mind, especially if staff have helped them to understand that sharing their problems with carers makes living with their illness easier (Carstairs *et al*, 1985). When the patient is discharged, staff should be quite sure that the patient is adamant about not informing his/her family. If s/he is adamant but intends to see the family even occasionally, s/he could be told that the family must get some minimum information, essentially phone numbers for emergencies and crises. The release of further information could be negotiated with the patient.

Ways of giving information which do not breach confidentiality (Atkinson & Coia, 1995) include the use of voluntary organisations such as National Schizophrenia Fellowship (Scotland) which can provide support and general information about treatments, the pros and cons of medication and tips on how best to help the user.

Where the family members are going to give support, they need accurate and well-balanced information on both the illness and the individual patient. If they do not get this from the psychiatric team, they may look elsewhere for enlightenment. They may then get suggestions of how to proceed which are neither relevant nor helpful to that particular patient, such as stopping medication and using alternative therapies, going back to work or college or taking up activities which put the person under stress and increase the possibility of relapse.

It is necessary that all psychiatric staff understand that discharging patients without providing information to their family could be detrimental to the patient's welfare. People with severe mental illnesses are looked after in hospital by highly trained professional staff with their own professional support systems. Discharging these patients into the community where families, the

unpaid informal carers, are given no support and information, is potentially a recipe for disaster. It simply does not make sense.

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### 5-HT<sub>2</sub> neurotransmission in major depression

**Sir:** I read with interest the neuroendocrine challenge study by Sargent *et al* (1998). The conclusion that potentiation in 5-HT<sub>2</sub> neurotransmission is unlikely to be responsible for the antidepressant effect of selective serotonin reuptake inhibitors (SSRIs) is consistent with platelet studies suggesting that up-regulation of 5-HT<sub>2A</sub> receptors may be a trait phenomenon of major depression (Hrdina *et al*, 1997). However, some additional points should be made to clarify the role of 5-HT<sub>2</sub> neurotransmission in depressive disorder.

First, concurrent use of cyproheptadine, a 5-HT<sub>2</sub> antagonist, to treat sexual dysfunction resulted in a reversal of the antidepressant effect of fluoxetine (Feder, 1991), supporting the role of 5-HT<sub>2</sub> neurotransmission in the antidepressant effect of SSRIs in some depressed patients. Second, short-term lithium treatment appears to reverse the deficit state in an animal model of depression by activating post-synaptic 5-HT<sub>1C</sub> (current terminology 5-HT<sub>2C</sub>) receptor sites (Aulakh *et al*, 1994). Further, it seems to enhance cognitive and motivational process by increasing 5-HT<sub>2</sub> neurotransmission (Harrison-Read, 1998). Hence, it is possible that initial increase in 5-HT<sub>2</sub> neurotransmission may account for the improvement in some symptoms of depression and/or for the augmenting effect of lithium carbonate in the treatment of refractory depression. Third, considering the findings that neurotransmission at 5-HT<sub>2</sub> receptors may be a trait marker of major depression (Hrdina *et al*, 1997), the down-regulation of 5-HT<sub>2</sub> receptors with long-term treatment with SSRIs might be responsible for prevention of recurrences of

depression (this awaits further investigation). In sum, the participation of 5-HT<sub>2</sub> receptors in major depressive disorder appears to be multifarious and complex.

**Aulakh, C. S., Hill, J. L. & Murphy, D. L. (1994)** Lithium treatment restores clonidine's effect in an animal model of depression. *Pharmacology, Biochemistry, and Behavior*, **47**, 985–987.

**Feder, R. (1991)** Reversal of antidepressant activity of fluoxetine by cyproheptadine in three patients. *Journal of Clinical Psychiatry*, **52**, 163–164.

**Harrison-Read, P. E. (1998)** Lithium withdrawal mania supports lithium's antimanic action and suggests an animal model involving serotonin (letter). *British Journal of Psychiatry*, **172**, 96–97.

**Hrdina, P. D., Bakish, D., Ravindran, A., et al (1997)** Platelet serotonergic indices in major depression: upregulation of 5HT<sub>2A</sub> receptors unchanged by antidepressant treatment. *Psychiatry Research*, **66**, 73–85.

**Sargent, P. A., Williamson, D. J. & Cowen, P. J. (1998)** Brain 5-HT neurotransmission during paroxetine treatment. *British Journal of Psychiatry*, **172**, 49–52.

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### Active placebos in antidepressant trials

**Sir:** Moncrieff *et al* (1998) highlight a methodological flaw in many placebo-controlled studies; anticholinergic side-effects are readily identifiable so it is not possible for these studies to be blinded and they are therefore liable to observer bias. This is the rationale for a meta-analysis of studies that used an active placebo in the form of atropine. Nine studies were identified and a smaller effect size for tricyclic antidepressant (TCA) efficacy was identified, relative to analyses pooling studies that used an inert placebo. They conclude that TCAs may not be as effective as previously assumed and that active placebos are necessary for valid double-blind studies. In the discussion, Moncrieff *et al* acknowledge that the decreased efficacy of TCAs identified in their analysis may arise if atropine had antidepressant properties *per se*. This is dismissed on the evidence that a study by Gillin *et al* (1995) failed to demonstrate antidepressant efficacy of a centrally acting anticholinergic agent relative to a peripherally acting anticholinergic. However, the centrally acting anticholinergic studied was biperidan, a relatively selective M1 antagonist. In contrast, atropine is a competitive antagonist at all