AUTHORS' REPLY: Takei and Murray's calculations (as I see it) do not address the issue, which is simple.

O'Callaghan *et al* (1991) concluded that an 88% increase in the births of patients with schizophrenia in March 1958 was due to the fact that their mothers, while in the second trimester of pregnancy, had been infected with the influenza virus. Thirty-four children who later developed schizophrenia (by narrow criteria) were born to the 16 268 mothers in the NCDS who gave birth in the week 3–9 March 1958. If O'Callaghan *et al* were right, 88/188 of these 34 children (=15.9 children) developed schizophrenia because their mothers suffered from influenza in the second trimester. However, influenza infection during pregnancy was recorded in this survey, and 945 mothers are noted as affected in the second trimester. Therefore (according to O'Callaghan *et al*) it is these 945 mothers who must have given birth to 15.9 children with schizophrenia. In reality, only one case of schizophrenia was recorded as being born to these mothers, where (according to the hypothesis) there should have been 15.9.

Therefore (I concluded) O'Callaghan *et al*'s conjecture must be mistaken. For the reasons I gave in my editorial, the other evidence that is put forward in support of the view that prenatal exposure to influenza causes schizophrenia does not seem cogent. For example, O'Callaghan *et al* (1991) stated in their summary that "five months after the peak infection prevalence (following the 1957 epidemic), the number of births of individuals who later developed schizophrenia was 88% higher than the average number of such births in the corresponding periods in the two previous and the next two years" and concluded that the relationship was causal. Sham *et al* (1992) wrote that "our results indicated that 1–2% of all schizophrenic births can be explained by the number of influenza deaths in the preceding months". If these two sets of analyses are both to be interpreted in favour of a relationship, they suggest effects that differ by two orders of magnitude – but Takei and Murray are co-authors of both papers. In the face of such unreconciled inconsistencies I do not see that the theory that prenatal exposure to influenza causes schizophrenia can be taken seriously.

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Night terrors

SIR: There is apparently some diagnostic confusion regarding the classification of the episodic phenomena presented in the case report by Lillywhite *et al* (*BJP*, April 1994, **164**, 551–554). The International Classification of Sleep Disorders (ICSD, 1990) differentiates between several paroxysmal nocturnal anxiety states. A nightmare (307.47–0) is a REM-parasomnia, awakening typically occurring out of REM-sleep. A sleep terror (307.46–1) is a disorder characterised by sudden arousal from slow wave sleep (SWS). It is an unusual NREM parasomnia in adults (<1%). Nocturnal panic episodes (300) most often occur in light, stage 2 NREM sleep.

The patient's sleep recording reported by Lillywhite *et al* shows a superficial, fragmented sleep pattern, as often recorded in patients using alcohol and benzodiazepines. In our opinion, sleep preceding these events seems to deepen, and in fact shows hypersynchronous delta bursts, an EEG pattern reported in adult somnambulism often preceding arousal (Blatt *et al*, 1991). Somnambulism is a sleep NREM stage-dependent parasomnia. The neurophysiological characteristics of this first night recording thus favour the diagnosis of nocturnal panic attacks rather than sleep terrors.

A further argument favouring this interpretation is the observation that the SWS increase reported in the second sleep recording does not deteriorate the clinical picture, as would have been expected for sleep terrors. SWS increase following serotonin agonists is not surprising, as serotonin is a neuromodulator involved in NREM sleep rebound (Sallanon *et al*, 1983). Increased serotonergic drive

correlates with motor activity and behavioural activation, and has been implicated in parasomnias such as REM sleep without atonia (Jacobs & Azmitia, 1992).

It must, moreover, be emphasised that this second sleep record is not "normalised" as suggested by the authors. There is considerably less REM sleep and more awakenings out of REM sleep. These findings are in accordance with literature describing increased alertness and decreased REM sleep following 5-HT reuptake inhibitors (Gaillard, 1990).

Considering these misperceptions we do not feel that this case report offers valid arguments for treating sleep terrors and/or somnambulism with SSRIs.

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AUTHORS' REPLY: The main comment of Van Sweden *et al* seems to be that because night terrors occur from slow wave sleep (SWS), which was minimal in our patient, the diagnosis of night terrors was wrong. The main thrust of our original discussion addressed this point and we would still contend that a major reason for the paucity of SWS was probably that each episode of SWS was rapidly terminated by a terror. We examined the nature of the SWS prior to each terror and found no evidence of hypersynchronous delta bursts; the delta activity fulfilled Rechtschaffen and Kales criteria for SWS. The next comment, that the increase in SWS on treatment did not worsen the terrors, misses the point. We used the SSRI treatment not to increase slow wave sleep directly but to reduce the anxiety that gave rise to the interruption of sleep by terrors, thus permitting more continuous SWS episodes. It seems that, in humans, SSRIs in general have no effect on SWS (Kupfer *et al*, 1991; van Bemmel *et al*, 1993) which is interesting given the established finding that directly-acting serotonin agonists such as mCPP reduce this stage (Lawlor *et al*, 1991; Katsuda *et al*, 1993).

We never claimed that the second sleep recording was normalised, although the patient thought her sleep was. The second hypnogram shows the classic picture of REM delay caused by SSRIs. Naturally we would not, on the basis of a single case, claim to have found the ideal treatment for night terrors. However, we feel that in similar cases, where night terrors are similarly profoundly problematic, SSRIs might be tried.

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Selective serotonin reuptake inhibitors and mania

SIR: Peet (*BJP*, April 1994, **164**, 549–550) recommended that patients at risk for mania be treated with selective serotonin reuptake inhibitors (SSRIs) rather than tricyclic antidepressants (TCAs).

This report does not include information about concurrent antimanic drug use. TCAs have been available longer than SSRIs, with much greater concern about TCA-induced mania or rapid-cycling. Therefore bipolar patients recently treated with newer antidepressants may be more likely to receive prophylactic antimanic treatment. This would bias the rate of treatment-emergent mania.

Peet also does not distinguish between bipolar subtypes. Antidepressant-induced mania may occur less often in bipolar II than in bipolar I disorder