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Fluoxetine and suicidal behaviour

SIR: Power & Cowen (*Journal*, December 1992, **161,** 735–741) have performed a comprehensive overview of available data. However, it is of concern that they believe many clinicians are still disquietened despite the balance of evidence suggesting there is no association between fluoxetine and suicidal behaviour. The quote from an American psychiatrist merely highlights the media involvement and the fact that medical care in the USA may be compromised by threats of litigation.

Both the Committee on Safety of Medicines (CSM) and the Food and Drug Administration (FDA) have issued statements on fluoxetine. In September 1991 the FDA reviewed all the data and concluded that there was "no credible evidence" to link fluoxetine to suicidal or aggressive behaviour. A recent CSM Current Problems (1992) and a Manufacturing Chemists Association (MCA)/CSM paper (Price *et al*, 1992) also lend extra weight to this, stating "there is little to support the suggestion that fluoxetine induces suicidal or aggressive behaviour".

Drs Power and Cowen rightly concentrate on the scientific aspects of the controversy. They remark on the fact that development of suicidal ideation in patients taking antidepressants is not new. In the Damluji and Ferguson (1988) case studies two of four patients on desipramine who were subsequently switched to fluoxetine made a complete recovery with no recurrence of suicidal ideation.

The hypothesis of a rare idiosyncratic reaction, perhaps in the context of induced akathisia, is proposed. The data available to support this hypothesis are far from established. A recent paper by Baldessarini *et al* (1992) failed to show a dopamine-inhibiting effect of fluoxetine either with acute or repeated doses. The suggestion is also made that, of the selective serotonin (5-hydroxytryptamine, 5-HT) reuptake inhibitors (SSRIs), fluoxetine is most activating. Although data varies (and most work has been done at 40 mg) it is, in fact, not the case. Kerr & Hindmarch (1991), in their review of the cognitive and behavioural effects of the 5-HT reuptake inhibitors, conclude that these drugs are essentially neutral.

We will continue to be vigilant in our collection of safety data for fluoxetine. As the SSRIs in general become more commonly used, it may be that specific effects become more clearly delineated and clinicians will learn to manage these as they have those of the older tricyclic antidepressants which are arguably more toxic to patients.

BALDESSARINI, R. J., MARSH, E. R. & KULA, N. S. (1992) Interactions of fluoxetine with metabolism of dopamine and serotonin in rat brain regions. *Brain Research*, **579,** 152–156.

COMMITTEE ON SAFETY OF MEDICINES (1992) Safety of fluoxetine (Prozac): Comparison with fluvoxamine (Faverin). *Current Problems* (June).

DAMLUJI, N. F. & FERGUSON, J. M. (1988) Paradoxical worsening of depressive symptomatology caused by antidepressants. *Journal of Clinical Psychopharmacology*, **8,** 347–349.

KERR, J. S., HINDMARCH, I. & SHERWOOD, N. (1991) The comparative psychopharmacology of 5-HT reuptake inhibitors. *Human Psychopharmacology*, **6,** 313–317.

PRICE, J., *et al* (1992) Safety of fluoxetine: comparison with fluvoxamine. *Pharmacoepidemiology and Drug Safety*, **1,** 111–117.

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Periodic psychosis associated with the menstrual cycle and childbirth

SIR: I agree with Crammer (*Journal*, December 1992, **161,** 859) that the relationship between mental states and monthly cycles is complex, and add that a better understanding of this relationship may help untangle the biological basis of certain mental disorders. However, I am sorry that the relationship between the mental state and the menstrual cycle was not more convincing in my case report (*Journal*, September 1992, **161,** 402–404). The graph presents an accurate record of the timing of the patient's mental state and menses, as far as it is possible to demonstrate such things pictorially. As stated in the text, three of six psychotic episodes began in the week prior to menses, and two in the fortnight before.

Sometime after writing the report, my patient moved and was unfortunately lost to follow-up. It later transpired that she did not take any medication after losing contact. During this period she had a stable relationship and became pregnant, sustaining the longest period of good health that she had enjoyed for a number of years. Both she and her relatives were so struck by the disappearance of her regular relapses that they believed she was now free from her illness. After a full-term and uncomplicated delivery she returned home. However, three days later she disclosed to a midwife ideas about killing the baby. On the fourth post-partum day she was admitted to a local psychiatric unit, perplexed, anxious, and experiencing accusatory auditory

hallucinations and persecutory delusions. Five days later she was overtalkative, very active, and irritable, and ten days later she had mild pressure of speech. She made a good recovery and was discharged on lithium.

It appears that one of Crammer's criteria for linking the menstrual cycle and the mental state has been fulfilled. While amenorrhoeic due to pregnancy, the patient remained well. Three days after birth she relapsed with a similar clinical picture to those episodes linked to the menstrual cycle.

I have now reported the case of a woman with a premenstrual psychotic illness who subsequently suffered from a puerperal relapse. This is complementary to those cases of puerperal psychosis and subsequent premenstrual relapse (Brockington *et al*, 1988) and adds substantially to the hypothesis that these phenomena have the same aetiology. One factor common to both the premenstrual and the postnatal period is the change in the sex hormones – and it has been argued that there is an interaction between changes in the levels of the sex hormones and dopamine receptor sensitivity (Wieck *et al*, 1992). Relapse of a puerperal psychosis has also been noted after the removal of a hydatidiform mole, when levels of gonadotrophins, progesterone and also oestrogen would be expected to fall (Hopker & Brockington, 1991). The circumstantial evidence that puerperal psychotic illness is secondary to falling levels of hormones is considerable. It does, however, remain true that there are a number of different hormones and neurotransmitters that may be responsible for this intriguing interaction.

BROCKINGTON, I. F., KELLY, A., HALL, P., *et al* (1988) Premenstrual relapse of puerperal psychosis. *Journal of Affective Disorders*, **14**, 287–292.

HOPKER, S. W. & BROCKINGTON, I. F. (1991) Psychosis following hydatidiform mole in a patient with recurrent puerperal psychosis. *British Journal of Psychiatry*, **158**, 122–123.

WIECK, A., KUMAR, R., HIRST, A. D., *et al* (1992) Increased sensitivity of dopamine receptors and the recurrence of affective psychosis after childbirth. *British Medical Journal*, **303**, 613–616.

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Gender differences in schizophrenia

SIR: Lewis (*Journal*, October 1992, **161**, 445–450) comments that gender differences in schizophrenia are established findings. Iacono & Beiser (1992) noted the conventional belief is that the incidence of schizophrenia is the same for both sexes. They commented that surveys over the past ten years have provided inconsistent results. They referred to studies in Asia, Europe, and North America, suggesting that an excess of schizophrenia does occur among males.

A prospective assessment of gender differences in psychiatric illness among a sample of 70 patients admitted to a psychiatric unit, in South Africa, showed the following results. The incidence of schizophrenia was more common among men than women ($P=0.003$). The majority of patients in the 10–30 year age group were men, while women patients predominated in the 30–50 year age group ($P=0.029$). This study showed that the men presented earlier with mental illness, this tending to be schizophrenia.

Dr Lewis comments on the biological basis for the male predominance. Though sociocultural factors relating to traditional societies may be unique in Africa, it would appear that results from South Africa are consistent with those from the rest of the world.

IACONO, W. G. & BEISER, M. (1992) Are males more likely than females to develop schizophrenia? *American Journal of Psychiatry*, **149**, 1070–1074.

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Carbamazepine and episodic dyscontrol

SIR: Sugarman (*Journal*, November 1992, **161**, 721) reports three individuals with outbursts of violence who responded well to carbamazepine. Interestingly, all had olfactory hallucinations and reported *déjà vu* experiences. These features point towards a diagnosis of temporal lobe epilepsy despite the absence of electroencephalogram (EEG) changes in two of the cases. Therefore, it is not surprising that the response to anticonvulsants was favourable.

I would agree the episodic dyscontrol may be "best understood as paroxysmal violence, due to epilepsy-like dysfunction of limbic structures in the temporal lobe", but this would appear to be a rather unfashionable view. I think it is worth noting that Maletsky's cases (1973) often had long histories of violent behaviour and, despite the 'out of character' quality of the episodes, I suspect many psychiatrists would have diagnosed them as suffering from an antisocial personality disorder.

The DSM–III–R criteria for intermittent explosive disorder have been designed specifically to exclude