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LETTERS TO THE EDITOR

SSRI Discontinuation Syndrome Treated with Fluoxetine

Dear Editor

The selective serotonin reuptake inhibitors (SSRI) paroxetine, sertraline, fluvoxamine and fluoxetine may cause a discontinuation syndrome presenting with dizziness, paraesthesia, lethargy, nausea, lowered mood, anxiety, agitation, vivid dreams, insomnia, headache and irritability (Coupland *et al*., 1996; Schatzberg *et al*., 1997a). Dizziness and paraesthesia are usually its most prominent symptoms. Its hallmark features are emergence of new symptoms soon after discontinuation, generally mild and short-lived (2 weeks) symptoms rapidly reversed by reintroduction of the SSRI and minimized by slow tapering or by using long half-life SSRIs (Schatzberg *et al*., 1997a). It is more common with the short half-life SSRIs paroxetine and fluvoxamine (Lejoyeux and Ades, 1997). A similar discontinuation syndrome was also reported with the selective serotonin–norepinephrine reuptake inhibitor venlafaxine (Benazzi, 1996). SSRI discontinuation syndrome may be caused by a sudden decrease of synaptic serotonin in the face of down-regulated serotonin receptors (Coupland *et al*., 1996).

I would like to describe two patients who, unable to discontinue venlafaxine and sertraline for severe withdrawal symptoms, were able to discontinue them by adding, and later discontinuing, fluoxetine. A MEDLINE search found only one similar report about venlafaxine (Giakas and Davis, 1997).

A 74-year-old woman with recurrent major depressive disorder, in remission for months with venlafaxine, 75 mg/day, gradually tapered it (37.5 mg/day for 7 days, 18.75 mg/day for 10 days). Soon after discontinuation she had dizziness, paraesthesia, anxiety, nausea, agitation and insomnia. After three days, unable to tolerate these symptoms, she had to restart venlafaxine, 75 mg/day, and these symptoms disappeared in a day. Three weeks later venlafaxine was tapered again (37.5 mg/day for 5 days, 18.75 mg/day for 5 days), but it was associated from the start with fluoxetine, 20 mg/day. After venlafaxine discontinuation she had only mild paraesthesia and anxiety for a week. One week later fluoxetine was reduced to 10 mg/day for 5 days and then discontinued, without withdrawal symptoms during the following weeks. She took no other drugs.

A 65-year-old woman with recurrent major depressive disorder, in remission for months with sertraline, 50 mg/day, gradually tapered it (25 mg/day for 10 days, 12.5 mg/day for 10 days). Soon after discontinuation she had dizziness, nausea, agitation, irritability, insomnia and depressed mood. After a week, unable to tolerate these symptoms, she had to restart sertraline, 25 mg/day, and these symptoms disappeared in a day. One month later sertraline was tapered again (12.5 mg/day for 7 days), but it was associated from the start with fluoxetine, 5 mg/day. Sertraline was discontinued without problems. Fluoxetine was discontinued a week later without withdrawal symptoms during the following weeks. She took no other drugs.

These patients had typical SSRI discontinuation symptoms (Schatzberg *et al*., 1997a), which appeared despite slow tapering, and which did not recur, or were much less severe, when tapering was associated with fluoxetine. Later discontinuation of fluoxetine was not followed by withdrawal symptoms. They suggest that fluoxetine might be used to suppress/control SSRI discontinuation symptoms. The long half-life of fluoxetine active metabolite (4–16 days) (Schatzberg *et al*., 1997b) might reduce the risk of withdrawal symptoms (Schatzberg *et al*., 1997a). Only a few cases of fluoxetine withdrawal symptoms were reported in comparison with paroxetine, fluvoxamine and sertraline (Lejoyeux and Ades, 1997), which have a short half-life (15–66 hours) (Schatzberg *et al*., 1997b). SSRI half-life might be the critical factor

determining the risk of discontinuation syndrome in vulnerable individuals.

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CAGE

Dear Editor

Before dumping the CAGE in the bin as a result of Luteral *et al.*'s survey in this journal (1997), perhaps readers might ponder the significant excess of authors over subjects identified as having alcohol dependency syndrome. If the prevalence of a disorder is as low as this, there is no point in screening for it (Wilson and Jungner, 1968). However, the number of identified cases is so small that chance alone could have produced a strikingly low or strikingly high agreement between the clinical assessment and the screening measures used.

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