'DeLong replies'

SIR–We appreciate the letter from Drs Bates and Wilson. We are in complete agreement regarding the possibility of precipitating mania by using selective serotonin reuptake inhibitors (SSRIs) in patients with bipolar diathesis. We also think this is an important side-effect limiting SSRI treatment in some autistic children. This is one of several factors emphasizing the similarities between autism and major affective disorder/bipolar disorder.

Any difficulties caused by such escalation during treatment with SSRIs can be minimized by discussing at the outset the possibility of increased activation or agitation resulting from SSRI treatment, so the medication can be stopped promptly if such side-effects occur. In some cases, the SSRI is so beneficial that it is thought worth while to add a mood-stabilizer to the SSRI to permit the continued use of the latter. However, we would suggest that one cannot be sure that every instance of hyperactivity induced by SSRIs represents the precipitation of mania. As one of the early proponents of the concept of juvenile bipolar disorder in the US,[1] I agree with the difficulty of diagnosing mania in children.

The five children who developed bipolar disorder during follow-up had all been SSRI responders, treated for more than 3 years.

We agree that great vigilance is required when using SSRIs in the autistic spectrum group of children. We try to discuss all aspects of the problem with families before instituting such trials. The marked beneficial effect in some children, sustained over years, in our judgment, makes the effort worthwhile.

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Reference

1. DeLong GR. (1978) Lithium carbonate treatment of select behavior disorders in children suggesting manic-depressive illness. *J Pediatr* **93:** 689–94.

Erratum

'Folic acid and prevention of birth defects'

Van Dyke et al.

DMCN Vol 44: 426–29

The primary author of the above mentioned annotation wishes to point out the following corrections to the article:

p 427, para 5, line 6 should read: 'Increased folic acid intake could potentially reduce the prevalence of some types of congenital heart disease.'[34]

p 428, para 3, line 4 should read '... heart disease. Individual differences in specific enzymes involved with folic acid metabolism and associated amino acid pathways may potentially be risk factors for NTD and possibly other abnormalities. Polymorphisms of specific enzymes, particularly MTHR, have been linked to specific metabolic effects.[59]'

References:

p 426, para 5, line 9: delete ref. 7.

p 427, Table I: delete refs. 37 and 51; add ref. 56

p 427, para 1, line 9: add ref. 41.

p 427, para 8, line 9: change ref. 24 to 50

p 427, para 4, line 11: add ref. 10

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