Correspondence

EDITED BY LOUISE HOWARD

Do SSRIs affect personality traits?

Sir: Ekselius & von Knorring (1999) concluded that significant effects on personality traits are seen in depressed patients treated with selective serotonin reuptake inhibitors (SSRIs). It is possible that modulation of neurotransmitter systems affects personality traits. However, it is far-fetched to derive such conclusions from this study. First, personality assessment by a single self-rating is inadequate. Second, while in a state of depression severe enough to warrant antidepressant medication is not the best time to assess personality. Third, all the items in the scale used by Ekselius & von Knorring to assess personality are affected in depression. Ideally, this issue can be studied only in subjects who have no mental illness but do have personality problems. They should have a comprehensive assessment of personality and mood at baseline and at follow-up. There should also be control groups on placebo and/or on other antidepressant drugs.

Ekselius, L. & von Knorring, L. (1999) Changes in personality traits during treatment with sertraline or citalopram. *British Journal of Psychiatry*, **174**, 444–448.

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Authors' reply: It is easy to agree that, ideally, the effect of SSRIs on personality traits should be studied in subjects who have personality problems but no mental illness. However, conclusions can also be derived from other sources. We had three good reasons to believe that the results of our study could be interpreted as an effect of SSRIs on personality traits. First, in an earlier study by Perris *et al* (1979), in-patients suffering from depressive syndromes completed the Karolinska Scales of Personality (KSP) (Schalling *et al*, 1987) during depression and when recovered after treatment with tricyclic antidepressants or electroconvulsive therapy. Twelve out of 15 scales remained stable. The only significant changes concerned components of anxiety and social desirability. Thus, it is not true that all items in the scales are affected in depression. Instead, the KSP scales are fairly independent of the state of the subject. Second, in our study (Ekselius & von Knorring, 1999), changes in the depressive symptomatology, assessed by means of the Montgomery–Åsberg Depression Rating Scale (Montgomery & Åsberg, 1979), explained no more than 0–8.4% of the changes seen in any separate personality scale. Third, the results are in line with those of Knutson *et al* (1998) who demonstrated, in healthy volunteers, that relative to placebo, SSRI administration changed personality traits and reduced focal indices of hostility.

Ekselius, L. & von Knorring, L. (1999) Changes in personality traits during treatment with sertraline or citalopram. *British Journal of Psychiatry*, **174**, 444–448.

Knutson, B., Wolkowitz, O. M., Cole, S. W., et al (1998) Selective alteration of personality and social behavior by serotonergic intervention. *American Journal of Psychiatry*, **155**, 373–379.

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Lipid supplementation in schizophrenia

Sir: We read with interest the excellent editorial by Walker *et al* (1999). However, the analogy of adrenoleukodystrophy (ALD) and schizophrenia is questionable. We would like to focus on some aspects that could be misinterpreted.

First, in ALD, prior to suggesting a dietary treatment, the causal relationship between accumulation of very long chain fatty acids (VLCFAs) and clinical symptoms had been clearly defined. ALD leads to large zones of demyelination through the accumulation of VLCFAs with a chain length of 24 or more predominantly in the adrenal cortex and the white matter of the central nervous system. Based on this pathogenetic mechanism, attempts were made to lower the levels of VLCFAs in patient tissues. In contrast, in the studies of cerebral lipid metabolism in patients with schizophrenia, there is a plethora of sometimes contradictory findings and no cause of the disorder relating to lipid metabolism has as yet been firmly established. Therefore, Walker *et al*'s suggestion that supplementation with polyunsaturated fatty acids (PUFAs) will "help to relieve schizophrenia", seems overly enthusiastic.

The second problem relates to fatty acid therapy itself. In ALD, dietary therapy is based on the observation that monounsaturated fatty acids, such as oleic acid, can interfere with the elongation of saturated fatty acids, possibly by competing for a microsomal elongating enzyme system. Erucic acid (22:1 n-9), a component of rapeseed oil, has an even greater effect. A 4:1 mixture of glyceryl trioleate and trierucate referred to as Lorenzo oil reduces the plasma levels of VLCFAs of ALD patients. Most interestingly, however, Lorenzo oil therapy results at the same time in a marked reduction in plasma levels of n-3 and n-6 PUFAs (Moser *et al*, 1992). Thus, the dietary therapy in ALD results in exactly that change in lipids that is now suggested to represent a causal factor for schizophrenia. It is this change which, according to Walker *et al*, should be reversed by supplementation.

Third, the clinical efficacy of Lorenzo oil does not warrant high-flown expectations for PUFA supplementation in schizophrenia. Lorenzo oil does not have a significant effect on the rate of progression of childhood ALD (Moser *et al*, 1992) and it was questioned whether it was taken up at all in brain tissue (Poulos *et al*, 1994). Furthermore, an unanticipated effect on platelet count limited its use (Zinkham *et al*, 1993).

The editorial conveys optimism about the potential clinical efficacy of PUFA supplementation in schizophrenia. The analogy