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L-TRYPTOPHAN IN MATERNITY BLUES

DEAR SIR,

We were most interested to read Dr Harris' report (*Journal*, September 1980, **137**, 233–35) of a double-blind trial of L-tryptophan in the puerperium. The failure of exogenous L-tryptophan to affect the incidence of severity of maternity blues is probably not surprising. In a recent study (*Journal*, May 1980, **136**, 498) we showed that whether or not the blues is associated with lowered free tryptophan is strongly affected by seasonal factors. In addition, we showed that, in our subjects, failure of total tryptophan to rise after parturition was a significant indicator not only of the blues but of complaints of depression in the ensuing six months.

It is difficult to see how such a brief disturbance of tryptophan at parturition could bear a causal relationship to outcome at six months and accordingly we suggested that it may indicate an occult disturbance in tryptophan handling, perhaps related to a more generalized membrane transport disorder, which may be a predisposing factor for the development of depression. Thus we envisaged that disturbances in tryptophan dynamics during the puerperium could be biological markers of susceptibility to depression rather than primary causative factors. Dr Harris' findings support this view and in this sense disturbances in total or free tryptophan at parturition may indeed be epiphenomena as he suggests, not of the blues, but of a more fundamental disturbance which does bear a causal relationship to depressed mood.

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THE ART OF MEASURING SEROTONIN UPTAKE IN PLATELETS

DEAR SIR,

Arora and Meltzer (*Journal*, October 1980, **137**, 396–99) criticize the paper by Coppen *et al* (*Journal*, March 1980, **136**, 235–38), mainly on methodological grounds. Their main point seems to be that when 5-HT uptake is determined at concentrations greater than 1 μM of 5-HT, the effects of passive diffusion cannot be entirely corrected. According to my studies (and those of others) this is not correct—provided one uses an adequate 'blind value' to be subtracted from the measured uptake values (Lingjaerde, 1977). The easiest way to obtain this blind value is that used by Coppen *et al:* to add varying concentrations of 5-HT to samples kept in the cold. When this blind value is used, I find that the initial uptake rate at 37°C shows all the characteristics of saturable, active uptake, at least up to 8 μM of 5-HT. Thus, it is completely blocked by low concentrations of antidepressants like clomipramine, and by omitting sodium or chloride from the incubation medium.

It may be possible to obtain the same data also by extrapolating back from the linear part of the uptake vs. concentration curve, as recommended by Stahl and Meltzer (1978), Tuomisto and Tukiainen (1976) and Tuomisto *et al* (1979). However, I find this method less satisfactory, because of the uncertainty in assessment of the 'linear part' of the (hyperbolic) uptake curve.

Like Arora and Meltzer, I was surprised by Coppen *et al's* finding of reduced 5-HT uptake in plasma after adding lithium carbonate. In my own *in vitro* studies with lithium (added as chloride) I have never seen an inhibitory effect of lithium, even in high concentrations. Neither have I seen a stimulatory effect, except in the absence of K^+; lithium thus seems to have a 'potassium-like' effect on 5-HT uptake (Lingjaerde, 1977). However, 5-HT uptake decreases very rapidly with increasing pH (Lingjaerde, 1977). Could it be that the inhibitory effect of lithium carbonate found by Coppen *et al* is due simply to increased pH?

Indeed, the measurement of 5-HT uptake in platelets is blessed with many pitfalls!

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