quotation 'hypomanic patient (secreting) large amounts of tryp*tophan*' implies a verbatim report of my remarks. In fact the nearest it comes to anything I said is the reference to the case which 'secreted large amounts of tryp*tamine*, . . .' The distinction here between tryptamine and tryptophan is not without importance, for tryptophan is the precursor both of tryptamine and 5HT (via 5-hydroxytryptophan), whereas tryptamine is *not* converted to 5-hydroxyindoles (14) and consequently effects cannot be explained as due to 5HT.

The studies on cerebrospinal fluid 5HIAA (1) showed that eight depressives had a mean level of 34 ng/ml CSF and did not differ statistically from an age-matched control group. The mean figure for eight untreated manics was 32 ng/ml and again this did not differ from an age-matched control group. After treatment with amitriptyline the mean 5HIAA in the depressives dropped to 21·3 ng/ml (a change significant at the 0·02 level), whereas the mean for the manics after lithium was 30·7, which is not statistically significant. The correlations quoted by Dr. Curzon also do not reach the 5 per cent level of significance, and as only four manic patients were given the MMPI it is unwise to ignore the statistical test. This is further supported by the fact that all manic patients were given the Brief Psychiatric Rating Scale and in this larger sample no significant correlation was observed with 5HIAA.

Although only some of the evidence has been touched on, it will be apparent how selective one must be to sustain the role for 5HT which Dr. Curzon supports. I do not doubt that 5HT and catecholamines may play a part in the depressive syndrome (perhaps related to sleep or appetite), but the occurrence of disturbed sleep and appetite in the depressive syndrome does not thereby equate them with the symptom of depressed *mood*. There is a logical chain of evidence identifying the normal mechanisms of tryptamine action, its relation to elevation of mood and its relevance in affective illness; physiology has been used as a basis for investigation of pathology (8, 9). No comparable links exist, as far as I know, for 5HT and catecholamines, and their putative role in mood was derived from observations in affective illness. Further, the objections made to the catecholamine and serotonin hypotheses of affective illness (9, 13) have not yet received satisfactory answers. Conversely, the points raised by Dr. Curzon (who supports the role of 5HT in affective illness) and those made by Weil-Malherbe (15) (who is an advocate of the catecholamine hypothesis) have both received answers (10). Time, one trusts, will make us all wiser, but meanwhile it is hoped that the relatively simple chemistry involved will not deter more of your readers from appraising matters for themselves.

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References

1. Bowers, M. B., Jr., Heninger, G. R., and Gerbode, F. (1969). 'Cerebrospinal fluid 5-hydroxyindoleacetic acid and homovanillic acid in psychiatric patients.' *Int. J. Neuropharmac.*, **8**, 255–62.
2. Coppen, A., Prange, A. J., Whybrow, P. C., Noguera, R., and Paez, J. M. (1969). 'Methysergide in mania. A controlled trial.' *Lancet*, ***ii***, 338–40.
3. Crawford, T. B. B., Ashcroft, G. W., Eccleston, D., and Smith, A. N. (1965). 'Some observations on the metabolism of indoles in 2 patients with the carcinoid syndrome.' *Gastroenterology*, **48**, 745–52.
4. Curzon, G. (1970). Letter to *British Journal of Psychiatry*, **116**, 571–2.
5. Dewhurst, W. G. (1961). Amine metabolism in depressive illness. Dissertation, London University, 1961.
6. Dewhurst, W. G., and Marley, E. (1965a). 'The effects of α-methyl derivatives of noradrenalin, phenylethylamine and tryptamine on the central nervous system of the chicken.' *Brit. J. Pharmacol.*, **25**, 682–704.
7. —— and —— (1965b). 'Action of sympathomimetic and allied amines on the central nervous system of the chicken.' *Brit. J. Pharmacol.*, **25**, 705–27.
8. —— (1968a). 'New theory of cerebral amine function and its clinical application.' *Nature*, **218**, 1130–3.
9. —— (1968b). 'Cerebral amine functions in health and disease.' Ch. 14, pp. 289–317. In *Studies in Psychiatry*. Ed. Shepherd, M., and Davies, D. L. London: Oxford University Press.
10. —— (1968c). Letter to *Lancet*, ***ii***, 514–15.
11. —— (1969a). 'Methysergide in mania.' *Lancet*, ***i***, 624–5.
12. —— (1969b). 'Methysergide in mania.' *Lancet*, ***ii***, 490.
13. —— (1969c). 'Amines and abnormal mood.' *Proc. Roy. Soc. Med.*, **62**, 1102–7.
14. Udenfriend, S., Titus, E., Weissbach, H., and Peterson, R. E. (1956). 'Biogenesis and metabolism of 5-hydroxyindole compounds.' *J. Biol. Chem.*, **219**, 335–44.
15. Weil-Malherbe, H. (1968). 'Amines, alerting and affect.' Letter to *Lancet*, ***ii***, 219–20.

Dear Sir,

I regret that there was an error in my letter to the *Journal*, May 1970, page 572. The second paragraph should read '. . . the hypomanic patient (secreting)

large amounts of *tryptamine*'. The word 'tryptophan' was used in error.

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CONCURRENCE OF TURNER'S SYNDROME AND ANOREXIA NERVOSA

DEAR SIR,

We were interested to read the paper by Forssman, Mellbin and Wahinder published in the *Journal* (February 1970, pp. 221–5). We should like to describe another case of the concurrence of these two syndromes, which is of particular interest in that the patient was one of a dizygotic twin pair, the co-twin being 'normal' physically, but being important in the psychopathology of the twin with Turner's Syndrome. This case was one of a series of 17 patients (16 female and 1 male) described in a paper on Anorexia Nervosa presented at the Annual Meeting of the Australian and New Zealand College of Psychiatrists in October, 1969.

She was referred to an endocrine out-patient clinic at the age of 14 for investigation of her small size and her amenorrhoea. Physical examination suggested Turner's Syndrome to the endocrinologist, and this was confirmed on chromosomal analysis. She was, at this stage, 133 cm. (4 ft. 5 in.) and weight 35 kg., compared to her sister's height of 158 cm. (5 ft. 3 in.) and weight 50 kg. The latter had started menstruating at the age of 11 years.

The twin with Turner's Syndrome was subsequently referred to a psychiatrist because of progressive loss of weight and increasing distress at her slight size and appearance in comparison with her twin sister. Her weight by this time had dropped to 22 kg. (48 lb.)

Psychiatric examination showed a marked reactive depressive condition. This appeared to be largely the result of repeated comparisons made by friends and relatives about the difference in the physical, intellectual and emotional development of the twins; these always being unfavourable to the smaller one. She also showed a marked disturbance in her attitude to food. She had started dieting some months previously following her sister's attempts to reduce weight. The latter, however, had stopped the diet, but the patient had continued. She took very little food, and no fats or carbohydrates. Her parents had tried various techniques to persuade her to eat, including pleading, threats and ignoring her, all without effect. They stated she regarded food 'like poison' and if forced to eat became 'hysterical' or hid the food in her pockets. Like the case described by Forssman *et al.*, this patient's parental relationships appeared to have been congenial and relaxed until the development of her anorexia. Interview with the parents showed the mother to be a somewhat anxious person and concerned about her daughter's health, but the father was a warm and relaxed individual.

She was admitted to a psychiatric unit, and put on anti-depressants, phenothiazines and modified insulin. Over a period of two months she regained her previous weight of 35 kg.

She has been followed up 15 months since then, and has required one further admission for an anorexic period when her weight fell to 25 kg. She has also had treatment with oestrogens, which have promoted some breast development and withdrawal bleeds: however, these do not appear to have helped her much psychologically as yet. EEG showed that there was a mild general excess of theta activity and anteriorly fast rhythms were prominent. During overbreathing a few bursts of theta activity with phase reversals were seen in the right posterior temporal region, the record otherwise remaining relatively stable. The record was mildly abnormal, due to the presence of some slow episodes, focal in the right posterior temporal area, during overbreathing. No other abnormality was present.

Anorexia Nervosa and Turner's Syndrome are both relatively rare conditions. Although the Anorexia Nervosa in this patient appeared to be precipitated by her depression at her unfavourable comparison with her twin sister, the association of two rare conditions, the EEG abnormalities, and the relative absence of parental psychopathology, suggests an underlying genetic predisposition.

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PHENELZINE IN OBSESSIONAL NEUROSIS

DEAR SIR,

We should like to report a case of obsessional neurosis which has been treated with phenelzine. The present case shows many similarities to that reported by Annesley (1969), the main difference between the two patients being the age of presentation.

At the present time our patient, a male, is aged 26 years, and his last admission to hospital had a duration of almost two years. He has had four admissions, all fairly lengthy, to the same hospital in the past four years, and had also had five admissions to two other psychiatric hospitals in Liverpool.