patients with acute psychotic states. These studies have been extensively reviewed by Meltzer (1976). The source of this increase is thought to be skeletal muscle since it is the skeletal muscle isoenzyme that is found in these patients. However, recently some uncertainty has arisen owing to the finding that the brain-type isoenzyme of CPK is highly unstable (Nealon and Henderson, 1975; Cho and Meltzer, 1979).

We have measured the CPK isoenzyme patterns of three groups of psychiatric patients using a technique thought to preserve brain-type CPK isoenzyme activity (Cho and Meltzer, 1979). The blood samples were promptly brought to 4°C and were transported to the laboratory in ice. Most assays were performed immediately, but if this was impossible samples were deep frozen for a few days at most. Total CPK activity was determined by the method of Rosalki (1967) and CPK isoenzymes were separated and measured by a sensitive fluorescent technique based on the method of Somer and Konttinen (1972).

The subjects were 30 female inpatients. All had been admitted to hospital within the previous two days. Using Spitzer's research diagnostic criteria (Spitzer *et al*, 1975) 10 patients had definite schizophrenia, 10 had definite manic disorder and the remaining 10 had various neurotic disorders. All the subjects were between 20 and 55 years old. Those who had received a recent muscle injury or intramuscular injection were excluded as were any who had been forcibly restrained. None of the patients had thyroid, muscle or cardiovascular disease and none abused alcohol. Patients taking drugs known to affect CPK activity were also excluded.

We found that in all three groups of patients the CPK isoenzyme pattern was the same as in normal healthy individuals with no brain-type CPK isoenzyme being present. It therefore seems probable that in patients with acute psychotic states serum CPK activity does indeed originate from skeletal muscle.

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References

CHO, H. WON. & MELTZER, H. Y. (1979) Factors affecting stability of isoenzymes of creatine phosphokinase. *American Journal of Clinical Pathology*, **71**, 75–82.

MELTZER, H. Y. (1976) Neuromuscular dysfunction in schizophrenia. *Schizophrenia Bulletin*, **2**, 106–35.

NEALON, D. A. & HENDERSON, A. R. (1975) Lability of human creatine kinase isoenzymes at 37C: A complication of electrophoretic separation. *Journal of Clinical Pathology*, **28**, 834–6.

ROSALKI, S. B. (1967) An improved procedure of serum creatine phosphokinase determination. *Journal of Laboratory and Clinical Medicine*, **69**, 696–705.

SOMER, H. & KONTTINEN, A. (1972) Determination of serum creatine kinase isoenzymes by fluorescence technique. *Clinica Chimica Acta*, **40**, 133–8.

SPITZER, R. L., ENDICOTT, J. & ROBINS, E. (1975) Research diagnostic criteria for a selected group of functional disorders. *Instrument Number 58*. 2nd ed. New York State Psychiatric Institute.

PREMENSTRUAL SYNDROME

DEAR SIR,

I find Dr Katharina Dalton's objections (*Journal*, August 1980, **137**, 199) to Dr Gwyneth Sampson's double-blind trial of progesterone in the premenstrual syndrome (*Journal*, September 1979, **135**, 209–15) very difficult to understand. The Moos Menstrual Distress Questionnaire, as its title and Dr Dalton suggest, does indeed measure affective and somatic discomfort and behavioural change during the menstruum, but it was also expressly designed to measure, retrospectively, such changes in other phases of the cycle as well (Moos, 1969). Its ambition in this respect renders it somewhat unwieldy to use (Clare, 1977; Rouse, 1978) and many prefer to use Form T, which is composed of the same 47 items but which allows the subject to rate herself daily throughout a cycle. Dr Sampson appears to have used the daily self-rating form in her study. In the circumstances, Dr Dalton's objection that the MDQ 'only measures menstrual distress' is utterly mistaken and can have no bearing on Dr Sampson's results.

Dr Dalton queries the definition of the premenstrual syndrome used by Dr Sampson. In doing so, she provides her own, whereby *only* women who have symptoms premenstrually and *at no other time* in the cycle qualify as sufferers. The problem with this definition is that it presupposes that symptoms such as anxiety, depression, irritability, headache, backache and tension are relatively uncommon. The fact is, however, that many of the symptoms which make up the premenstrual syndrome are not uncommon and occur intermittently in women of childbearing years (Banks and Beresford, 1979; Ingham and Miller, 1979). In such cases, only the evidence of a premen-

strual exacerbation of symptoms occurring at other times may indicate a premenstrual component. Dr Dalton insists that these women are not premenstrual sufferers but it is not at all clear on what grounds, clinical, hormonal or therapeutic, she advances this distinction. She must surely know too that symptom ratings in the various cycle ratings repeatedly show high and significant correlations from phase to phase, (Moos, 1969; Halbreich and Kas, 1977; Taylor, 1979). There are significant correlations too between ratings of dysmenorrhoea and premenstrual affective symptoms, premenstrual somatic symptoms and premenstrual pain.

Dr Dalton herself is clearly aware of the difficulty, for elsewhere she has drawn our attention to those 'unlucky sufferers' whose symptoms 'start at ovulation, increase in severity during the premenstruum and resolve gradually during menstruation leaving only a few days in which good health is enjoyed' (Dalton, 1975). Indeed, one of the earliest papers on the subject, by herself and Dr Raymond Greene, (Greene and Dalton, 1953), so defined the syndrome as to include just such cases. Reproving Dr Sampson for using a definition which Dr Dalton herself provided over 20 years ago seems, if I may be permitted an indelicacy, a little below the belt.

However, Dr Dalton does raise an interesting point when she doubts the wisdom of equating reporting with complaining. She is right to be cautious. A recent study of over 500 women attending GPs found that 95 per cent reported some kind of somatic, affective or behavioural change premenstrually (Clare, in preparation). Such changes appear ubiquitous and merely eliciting their presence tells us nothing. Nor, however, can one simply rely on the fact that women present for treatment, without knowing more than is usually provided about how they come to identify themselves as 'ill' and how they differ, if they do differ, from women who do not come forward.

My one reservation concerning Dr Sampson's work relates to her use of sine curves in analysing the data from the diaries. Such a method presupposes a symmetry within the menstrual cycle such that the portion of the cycle in which the woman scores highly on individual symptoms is equal to the portion of the cycle in which she scores low, which is not necessarily so. It also presupposes a dip in symptom scores with a minimum score at some point in the cycle. A more appropriate approach, and one that fits the reality of symptom variation throughout the premenstrual sufferer's cycle, involves fitting polynomials to the scores obtained on individual symptoms or factors and examining for significance of the resulting fit using the F-test, as in fitting a regression line.

I doubt, however, if my reservation affects Dr Sampson's overall result. Dr Dalton may insist that 'progesterone is the specific treatment for premenstrual syndrome' but the fact remains that in the twenty years since this claim was first made, not a single properly controlled trial has shown it to be significantly superior to placebo nor to the many other treatments, such as pyridoxine, diuretics, monoamine oxidase inhibitors and bromocriptine, on whose behalf others argue as enthusiastically as Dr Dalton argues for progesterone.

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References

BANKS, M. H. & BERESFORD, S. A. A. (1979) The influence of menstrual cycle phase upon symptom recording using data from health diaries. *Journal of Psychosomatic Research*, **23**, 307–13.

CLARE, A. W. (1977) Psychological problems of women complaining of premenstrual symptoms. *Current Medical Research and Opinion*, **4 (4)**, 23–8.

DALTON, K. (1975) Why don't doctors treat the premenstrual syndrome? *MIMS Magazine*, **No. 15**, 89.

GREENE, R. & DALTON, K. (1953) The premenstrual syndrome. *British Medical Journal*, *i*, 1007–16.

HALBREICH, U. & KAS, D. (1977) Variation in the Taylor MAS in women with premenstrual syndrome. *Journal of Psychosomatic Research*, **2**, 391–3.

INGHAM, J. G. & MILLER, P. McC. (1979) Symptom prevalence and severity in a general practice population. *Journal of Epidemiology and Community Health*, **33**, 191–8.

MOOS, R. H. (1969) Typology of menstrual cycle symptoms. *American Journal of Obstetrics and Gynaecology*, **103**, 390–402.

ROUSE, P. (1978) Premenstrual tension: a study using the Moos Menstrual Questionnaire.

TAYLOR, J. W. (1979) The timing of menstruation-related symptoms assessed by a daily symptom rating scale. *Acta Psychiatrica Scandinavica*, **60**, 87–105.

PSYCHIATRIC DISTURBANCE IN MENTALLY HANDICAPPED PATIENTS

DEAR SIR,

There are considerable objections to the study described by Craft and Schiff (*Journal*, September 1980, **137**, 250–5) of fluphenazine in mentally handicapped hospital patients.

Firstly, the 'behavioural disturbance ratings' described amount to no more than ordinary scales peculiar to each clinician and patient. Their summation, and the use of parametric statistics in their interpretation, is spurious, even had they been shown to be reliable (which they were not).