British Journal of Psychiatry (1993), **162**, 481–486

The Premenstrual Syndrome: A Twin Study

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Data are presented from 157 monozygotic and 143 dizygotic female twin pairs of reproductive age who completed a self-report questionnaire on the premenstrual syndrome (PMS). For global PMS scores, the correlation coefficient for the monozygotic twins (0.55) was double that of the dizygotic ones (0.28). The results are consistent with the hypothesis that aggregation for global PMS scores is determined by genetic factors acting additively over a number of loci. Two alternative explanations are suggested: that monozygotic twins share greater environmental similarities than dizygotic ones; or that PMS scores may be a reflection of some other underlying characteristic (such as neuroticism), which is itself genetically determined.

Despite over 60 years having elapsed since Frank (1931) first coined the term 'premenstrual tension', there is continuing disagreement and controversy over the definition of this syndrome. Its existence as a discrete entity has frequently been questioned. The premenstrual syndrome (PMS) has been relegated to an appendix in DSM–III–R (American Psychiatric Association, 1987) which comprises "categories needing further study". This is perhaps a reflection of Drummond & Tonks' (1985) observation that "pinning it [PMS] down has similarities to picking up a globule of mercury; the moment you seem to have it, it escapes between your fingers".

Given this lack of consensus regarding definition, it is not surprising that there is disagreement regarding how the syndrome should be assessed, and inconsistent and contradictory findings with respect to aetiology.

At least 150 symptoms have been proposed to comprise the syndrome, and quite different symptom clusters have been identified by different researchers (Rubinow & Roy-Byrne, 1984). There is disagreement as to whether physical symptoms should be included; whether only symptoms which completely disappear for the remainder of the cycle should be considered; whether their severity or simply presence should be assessed; whether functional impairment should be a criterion; and whether positive premenstrual changes should be taken into account. It seems likely that PMS comprises a number of different overlapping syndromes rather than a single discrete entity (Rubinow *et al*, 1984).

In the literature to date, two different approaches have been taken to the assessment of PMS. The first uses retrospective questionnaires which ask women to report their experiences on either their last cycle or a 'typical' cycle. The second uses prospective daily ratings (via menstrual diaries) of symptoms experienced throughout one or more cycles. The latter are costly and generate mountainous amounts of data requiring complex mathematical analytic procedures, such as sine-wave fitting (Sampson & Jenner, 1977). Data gathered from retrospective questionnaires are potentially contaminated by retrospective falsification and over-reporting.

Some studies using both techniques have reported poor agreement between them (Rubinow & Roy-Byrne, 1984; Christensen & Oei, 1989). This has usually been interpreted as indicating that the prospective approach is the more valid and reliable. Hart *et al* (1987) have presented a defence of the retrospective approach based upon the large variation which frequently occurs between cycles in the same woman and the influence of extraneous life events upon symptoms. Halbreich *et al* (1982) and Schilling (1981) also question the superiority of the prospective approach, citing studies in which prospective and retrospective methods yield results which are in close agreement.

From a practical point of view, prospective daily ratings on multiple symptoms over several cycles would exceed the resources of most researchers if large samples are involved.

With the possible exception of alterations in β-endorphin (Chuong *et al*, 1985; Facchinetti *et al*, 1987), no consistent biological marker of PMS has emerged in replicated studies. In addition, some findings suggest that symptom levels in PMS sufferers may relate more to where the women *believe* they are in a cycle rather than where they actually are (Ruble, 1977). Such observations provide pointers towards a predominantly psychological aetiology in which genetic factors may play a less direct role.

An alternative model postulates that the psychological symptoms of PMS arise from neurophysiological alterations caused by the central action of gonadal steroids, or (unknown) central neurotransmitters, or neuromodulator substances. At least three

https://doi.org/10.1192/bjp.162.4.481 Published online by Cambridge University Press

independent lines of evidence suggest that alterations in brain function occur in synchrony with the menstrual cycle, and that such alterations are biologically, not psychologically, mediated. Firstly, Altemus *et al* (1989) have documented alterations in response to dichotic auditory stimuli premenstrually in comparison with the follicular phase. Alterations were noted in women with and without PMS; however, the former had more marked changes. Kirsch & Geer (1988) found similar trends using a different neurophysiological measure, namely skin conductance changes to an auditory stimulus. Van den Akker & Steptoe (1989) and Collins *et al* (1985) have reviewed other studies of neurophysiological alterations in menstruating women which have yielded both positive and negative results in relation to cycle phase.

Secondly, Becker *et al* (1982) have documented a number of studies (as well as their own) showing consistent cycle-dependent changes in women's electroencephalographic recordings, which are most prominent in the α range.

Thirdly, in non-human primates, Hausfater & Skoblick (1985) have demonstrated increased feeding, social withdrawal and possible aggression in perimenstrual baboons in the wild. Both Sassenrath *et al* (1973) and Rowell (1963) also reported marked increased aggression in captive Rhesus monkeys perimenstrually.

The predominantly biological mechanisms inherent in this type of model would potentially be more susceptible to genetic influences.

In an extensive review, Drummond & Tonks (1985) evaluated 19 aetiological theories of PMS. Rubinow & Roy-Byrne (1984) provide a similar comprehensive review of possible aetiologies. Curiously, neither paper makes any mention of a possible genetic contribution. DSM-III-R in its description of "late luteal phase dysphoric disorder", under the heading "familial pattern", simply states "no information". Literature on the genetics of PMS is indeed meagre and unsatisfying. Chern *et al* (1980) have reviewed this literature, albeit incompletely.

Kantero & Widholm (1971) were the first to explore empirically the notion that PMS 'runs in families'. In a study of 5000 menstruating girls and their mothers, they demonstrated highly significant correlations between mother and daughter on a variety of menstrual variables, including 'premenstrual tension' (correlation 0.24, $P<0.001$). The authors did not state how the syndrome was assessed. Clearly, the findings shed no light on the relative contributions of genetic as opposed to environmental influences.

In 1987, van den Akker *et al* studied 462 female twin pairs using a retrospective PMS questionnaire. The authors concluded that "premenstrual tension, as reflected in symptom reporting based on the most recent premenstrual week, is found to be largely heritable in the present study". They appropriately cautioned that retrospective questionnaires may overestimate symptoms in relation to daily prospective ratings, and state "it is possible that monozygotic twins have a tendency to report symptoms of discomfort in a similar manner, because of the intimacy of the relationship, compared to dizygotic twins".

Dalton *et al* (1987) reported on 15 monozygotic and 16 dizygotic twin pairs, one member of whom had sought treatment at a PMS clinic. PMS diagnosis was "confirmed by a menstrual chart" (no criteria specified). Fourteen of the 15 monozygotic pairs were concordant for PMS, whereas only 7 of the 16 dizygotic pairs were concordant. The concordance for monozygotic twins was significantly higher than that for a group of (non-twin) sibling pairs.

In short, the available evidence suggests a possible genetic component; however, the methodological shortcomings of these studies render it difficult to judge their generalisability.

Method

Questionnaires were mailed to 200 monozygotic and 200 dizygotic female twin pairs enrolled in the voluntary Australian National Health & Medical Research Council Twin Registry. In cases where only one member of a pair responded, a second set of questionnaires was forwarded to the non-responding twin.

Inclusion criteria were only that the twins be aged between 25 and 40 years and not currently pregnant.

The Australian Twin Registry determines zygosity by questions on similarity during childhood and on confusion in identifying the twins in childhood. These have been validated against blood markers (Sarna *et al*, 1978).

Information was elicited on basic sociodemographic characteristics, use of contraceptive pills, regularity of cycle, and dysmenorrhoea. In addition, each woman completed the 36-item retrospective, self-report PMS questionnaire designed and published by Steiner *et al* (1980). The decision to use this particular questionnaire was based on five considerations.

(a) The questionnaire has its origins in an item analysis of Moo's Menstrual Distress Questionnaire (MDQ) in which emphasis was placed on selecting the subset of MDQ items which PMS sufferers identified as having the greatest *change* between the follicular and premenstrual phases. The latter is defined in the questionnaire as the five days preceding the onset of menstrual flow. Thus, the questionnaire is specifically geared to detect changes from background levels. It tends to emphasise psychological rather than physical changes.

(b) In addition to global PMS scores, the questionnaire provides 10 subscale scores along dimensions which are widely accepted in the literature as being characteristic of

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PMS: irritability, tension, efficiency, dysphoria, motor coordination, cognitive function, appetite change, sexual drive and activity, physical symptoms, and social impairment.

(c) Responses to each item are simply yes/no, which avoids the ambiguity inherent in more graduated responses, namely that many minor complaints result in the same score as a few major ones.

(d) Women are asked to rate their 'usual' experiences which, to some extent, avoids reports based on atypical cycles which may contaminate the data if a specific cycle is designated.

(e) Prospective daily symptom ratings (over two cycles) on a sample of this size (600) would generate over 1.5 million items of data. The collection and analysis of such an expanse of data lay beyond the resources of the present study. While acknowledging that retrospective questionnaires may suffer from some retrospective falsification and over-reporting, there is no obvious reason why zygosity should influence the extent of this, and hence the parameters of interest in the present study should not be substantially affected.

Data analysis

Scores on the PMS subscales (and total scores) can be regarded as parametric data. Testing of the genetic hypothesis involves ascertaining whether the Pearson correlation coefficients for the monozygotic (MZ) twin pairs are significantly higher than those for the dizygotic (DZ) ones. The hypothesis is directional and hence one-tailed tests are appropriate.

Fisher's Z scores were used to determine the significance of differences in correlation coefficients between independent samples of monozygotic and dizygotic twin pairs (Ferguson, 1971). For a given correlation r,

$$Z = 1/2 \log_e [(1+r)/(1-r)] \quad (1)$$

Z has an approximate normal distribution with variance $1/(n\text{-}3)$, where n is the number of pairs. The 95% confidence interval for Z goes from $Z-1.96/(n\text{-}3)$ to $Z+1.96/(n\text{-}3)$, and this can be converted into a 95% confidence interval for r by inverting equation (1). Let Z_m and Z_d be the transformed correlation coefficients for n_m monozygotic and n_d dizygotic pairs, respectively. Then under the null hypothesis that $r_m = r_d$,

$$Z = (Z_m - Z_d)/\sqrt{[1/(n_m-3)+1/(n_d-3)]}$$

Z again has an approximately normal distribution (mean 0, variance 1).

Statistical inference for differences in means was carried out by t-tests and for differences from unity in the ratio of variances by F tests. Differences in the distribution of categorical variables were assessed by standard contingency table analyses.

Results

Completed questionnaires were received from 157 MZ pairs and 143 DZ pairs, whose characteristics are presented in Table 1. This represents a response rate of approximately 75% for each zygosity. Apart from their being voluntarily registered twins, this was essentially an unselected community sample of 600 women aged between 25 and 40 years.

Table 1
Sample characteristics

	Monozygotic twins (157 pairs)	Dizygotic twins (143 pairs)
Mean (s.d.) age: years	34 (3.8)	33 (4.4)
Marital status: %		
single	1	4
married	91	88
separated/divorced	8	8
Social class[1]: %		
professional or semiprofessional	39	42
skilled or semiskilled	36	40
unskilled	25	18
Gravidity: %		
0	8	13
1	13	13
2	34	38
>2	45	36
Parity: %		
0	9	16
1	15	14
2	44	41
>2	32	29
% taking contraceptive pill	23	31
Cycle regularity: %		
very regular	47	58
fairly regular	40	33
fairly irregular	7	5
very irregular	6	4

1. Classification derived from Congalton (1969).

A fundamental assumption of the twin method is that the MZ and DZ groups have been sampled from the same population. Inspection of Table 1 reveals a marked similarity between the two groups on sociodemographic characteristics. Several of the minor differences, including the one-year difference in mean age, attain *statistical* significance (because of the large sample size), but are insignificant in any practical sense.

Using t-tests, there were no significant differences between the MZ and DZ group on any of the PMS subscale or global mean scores. Likewise, the variances of these scores were virtually identical. Frequency distributions of subscale and total scores tended to be positively skewed, with similar skewness parameters in the MZ and DZ groups (i.e. there was a tendency for more women to have low PMS scores and a minority to have very high ones).

Within the MZ and DZ groups, the twin and co-twin had virtually identical means, variances, and skewness. Ferguson (1971) points out that parametric correlation coefficients are unaffected by departures of the distribution from normality, provided the two are the 'same shape'. Hence, transformations aimed at normalising the distributions were deemed unnecessary.

Table 2 presents the Pearson correlation coefficients within the MZ and DZ groups for each of the 10 PMS subscale scores and the global score. Correlations for MZ twins exceed those for DZ twins on all subscales, and the

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Table 2
Comparison of Pearson correlations for PMS subscales and global scores

	Monozygotic twins: r_m (95% confidence level)	Dizygotic twins: r_m (95% confidence level)	Z	P (one-tailed)
Irritability	0.41 (0.40–0.42)	0.34 (0.32–0.35)	0.74	NS
Tension	0.43 (0.42–0.45)	0.15 (0.13–0.16)	2.70	<0.005
Efficiency	0.26 (0.24–0.28)	0.21 (0.19–0.22)	0.47	NS
Dysphoria	0.36 (0.35–0.38)	0.29 (0.28–0.30)	0.73	NS
Motor coordination	0.37 (0.36–0.39)	0.10 (0.08–0.12)	2.50	<0.01
Cognitive function	0.37 (0.35–0.38)	0.06 (0.05–0.08)	2.75	<0.005
Appetite change	0.29 (0.28–0.30)	0.24 (0.23–0.25)	0.48	NS
Sexual drive/activity	0.24 (0.22–0.25)	0.04 (0.02–0.05)	1.74	NS
Physical symptoms	0.33 (0.31–0.34)	0.24 (0.23–0.26)	0.79	NS
Social impairment	0.26 (0.24–0.27)	0.10 (0.08–0.11)	1.40	NS
Global PMS score	0.55 (0.54–0.56)	0.28 (0.26–0.30)	2.80	<0.005

difference is statistically significant for three of the subscales (tension, motor coordination and cognitive function) and also for the global score.

If PMS scores are strongly age dependent, this may spuriously inflate the correlation coefficients for *both* MZ and DZ twins, since the members of each pair are matched for age. In fact, all subscale scores, as well as total score, are significantly positively correlated with age, suggesting older women have higher levels of PMS. However, the correlation coefficients are quite small, ranging from 0.07 to 0.15. They attain significance only because of the large sample size and, at most, approximately 2% of the variance in scores can be accounted for by age. It seems unlikely therefore that age effects are exerting any significant influence on the results.

Discussion

Fundamental to the interpretation of these results are two questions.

(a) To what extent do these data reflect a genetic influence in women's self-report responses to Steiner *et al*'s questionnaire?
(b) To what extent do subscale and global scores on the questionnaire provide a reliable and valid measure of PMS symptoms?

Genetic influence on questionnaire responses

The correlation for DZ twins in the present study (0.28) is similar to that obtained by Kantero & Widholm (1971) from the 5000 mother–daughter pairs (0.24), both types of pairs sharing (on average) half their genes.

Correlations for MZ twins exceed those for DZ twins on every subscale; on global scores the correlation for MZ twins is double that for DZ twins, the difference being highly significant ($P=0.002$). These results are consistent with the hypothesis that familial aggregation for global PMS scores is determined by genetic factors acting additively over a number of loci.

As shown in Table 1, there are no apparent differences between the MZ and DZ groups which could account for the higher correlations in the former. The only alternative to a genetic explanation would be to postulate that MZ twins, owing to their knowledge of being 'identical', spending more time together and experiencing greater similarities in environment, appearance, etc., have a greater tendency to introject similar stereotypic (or other) beliefs about PMS, which leads to greater similarity in their responses. Morris-Yates *et al* (1990) have examined in detail the possible influences of varying environmental similarity in twin studies. They conclude that, although MZ twins share a more similar environment, this is *elicited* by the twins rather than being *imposed* upon them by others. The eliciting of similar environments is probably itself genetically determined and "therefore, for the purposes of most analyses, the equal environments assumption is valid" (Morris-Yates *et al*, 1990).

In the present study, there is no significant difference in total variance of subscale or total scores between the MZ and DZ groups (71.0 and 74.0 respectively for total scores). As discussed by Jardine *et al* (1984), if DZ twins have less similar environments, their total variance should be greater than that for MZ twins. Thus, in the present results, differential environmental effects do not appear to have much influence.

As pointed out by Warner & Bancroft (1990), the effect of the oral contraceptive pill (OCP) upon PMS is controversial, both exacerbation and therapeutic effect having been reported. Of the DZ group, 31% were taking OCPs as opposed to 23% of the MZ group; however, concordance rates for the use of OCPs were similar (72% and 69% respectively). Moreover, for all 600 women, the mean PMS score

https://doi.org/10.1192/bjp.162.4.481 Published online by Cambridge University Press

for OCP users was 59.7 as compared with 58.2 for non-users, a difference of no significance. Thus, it is highly unlikely that the large differences in the correlation coefficients between the MZ and DZ groups are attributable to OCP.

In summary, given the magnitude and consistency of the differences between the correlations for MZ and DZ twins, it seems plausible to conclude that a substantial genetic factor is influencing these women's responses to the questionnaire.

Do questionnaire responses reflect PMS?

The issue here is whether the questionnaire is reliable, and whether it constitutes a valid measure of PMS.

Test–retest reliability of the instrument has not been established. If the present sample is regarded as 600 independent subjects, Cronbach's α coefficient for the global PMS score is 0.94. This represents the mean of all possible split-half reliabilities and suggests a high level of internal consistency.

As regards validity, the questionnaire items are a subset of those used in Moo's Menstrual Distress Questionnaire, which has been well validated. However, one cannot necessarily claim the validity of the original instrument for this subset of items.

As with all symptom self-report questionnaires, it is possible that some of the variance may be accounted for by personality or other factors unrelated to PMS *per se*. For example, a tendency to over-report symptoms, owing either to a lower threshold for experiencing discomfort or a tendency to label 'normal' physiological changes as abnormal. In addition, there may be a component of 'suggestibility' stemming from the items themselves, resulting in some women having a bias to respond positively. Any or all such characteristics may have genetic determinants. For example, Jardine *et al* (1984) have demonstrated significant genetic influences upon the trait of 'neuroticism'. Taylor (1979) has presented evidence that neuroticism may be an important determinant of women's experiences and self-reports of their menstrual cycle. It is likely that such traits would exert an influence on *any* PMS self-reports, regardless of whether they were collected retrospectively or prospectively.

Conclusions

The present results suggest that genetic factors have a substantial effect upon women's scores on Steiner *et al*'s questionnaire. The most parsimonious explanation of these observations is that they reflect a genetic influence on PMS. However, the possibility that PMS scores are substantially influenced by other (genetically determined) factors, such as neuroticism, cannot be excluded. The magnitude of the latter effect, as well as the influence of environmental factors during development, upon PMS require further study.

Acknowledgement

The author expresses his appreciation to Dr John Hopper, Director, Australian NH&MRC Twin Registry for his helpful comments upon this paper.

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