

## ***Empirical Support for an Evolutionary Model of Premenstrual Syndrome***

### **Abstract**

Premenstrual Syndrome (PMS) is very old, common, and panhuman. Definitions and diagnostic methods vary greatly, and proximate etiological research has thus far been unfruitful (Connolly, 2001). Reiber (2007) recently published an evolutionary model in which PMS is hypothesized to be a byproduct of adaptations that maximize chances of mating and fertilization during the follicular phase of the cycle. This suggests that how a woman feels during the follicular phase of the cycle should be predictable by evolutionarily relevant variables related to reproduction, such as age, parity, and resources.

Two hundred fifteen participants completed self-report biographical and medical questionnaires at screening, then prospectively rated 17 symptoms of PMS on a daily basis throughout the cycle. For 14 of the 17 symptoms recorded, mean follicular-week symptom severity was statistically significantly predicted by age, parity, and resources. Total symptom load during the follicular week was significantly negatively correlated with age and resources, and positively correlated with parity. Women meeting diagnostic criteria for PMS had significantly lower total symptom loads (that is, they felt better) during the follicular week of the cycle than did women without PMS. This suggests that women have PMS *not* because something negative happens with the approach of the premenstruum, but rather because evolutionarily adaptive positive states are fostered during the follicular phase of the cycle for some women. This conceptual shift may lead to improvement in definitional and diagnostic approaches, more fruitful proximate research, and better clinical treatment.

## Introduction

There are two types of explanation for characteristics of biological organisms: proximate, or mechanistic, explanations that answer the “how” questions; and ultimate, or evolutionary, explanations that answer the “why” questions. Historically, investigations of Premenstrual Syndrome (PMS) have tackled proximate questions, including biological, endocrinological and psychological mechanisms that underlie the syndrome. This report presents empirical tests of an ultimate, or evolutionary, explanation concerning why PMS exists and the implications of that model, and suggests how we might begin to synthesize this type of approach with more proximate psychological models.

### *Premenstrual Syndrome*

PMS is very old, common, and panhuman. It has been recognized throughout history (see Adams, 1939, for references to Hippocrates and Pliny; Prichard, 1837: 156-157; see Rubinow & Schmidt, 1995, for citations from von Feuchtersleben, 1847). Frank formally identified *premenstrual tension* as a medical syndrome in 1931, and Dalton and Greene coined the term *Premenstrual Syndrome* and elaborated diagnostic criteria in 1953. The true prevalence of PMS is difficult to determine because of self-treatment and differences in availability and access to medical care, definitions and diagnostic criteria, and cultural practices. Prevalence estimates (Table 1) range from 7.7 (Gaulrapp, Backe, & Steck, 1995) to 90% (Riley, 1986) of women affected. The World Health Organization (WHO) has concluded that "...a large proportion of women in third world nations in different world regions *do* experience the same kinds of physical

and emotional symptoms as women in Western industrialized nations...data show clearly that the expression of symptoms is *not* a Western phenomenon" (Ericksen, 1987: 186; my italics; Table 1).

PMS is characterized by cyclic symptom fluctuation: symptoms appear premenstrually, abate with the onset of menses, and reappear premenstrually in the next cycle. There are various clinical definitions of PMS; all require symptom-free days early in the cycle. A popular physician's guide (Severino & Moline, 1989: 142-161) requires, over two or more cycles of prospective daily ratings, marked premenstrual worsening of at least five symptoms that are not exacerbations of other disorders. If at least one of the five symptoms is dysphoric, and life activities or relationships are markedly affected, Premenstrual Dysphoric Disorder (PMDD, Diagnostic and Statistical Manual of Mental Disorders (DSM-IV, American Psychological Association, 1994) may be diagnosed. The definitions of Dalton and Greene (1953; Dalton, 1986) and the International Classification of Diseases (ICD-10, Premenstrual Tension, PMT; WHO, 1992) are more inclusive, and accept any number and type of symptoms that warrant presenting to a physician. In the face of these (and other) definitions, it is not clear whether PMS belongs in the realm of "psychological / emotional / mental" disorders (e.g., through DSM-IV criteria), or "physical" disorders (e.g., ICD-10 categorization), or if the distinction is even meaningful with respect to this "disorder".

Table 1. Studies of PMS around the world, with prevalence rates (where estimated)

Country	Reference	Prevalence Rate (%)	
		Affected	Incapacitated
People's Republic of China	Mao & Chang 1985		
	Rossignol, Zhang, Chen & Xiang 1989		
	Chang, Holroyd & Chau 1995		
	Zhao, Wang & Qu 1998	30.4%	
Taiwan	Hsaio, Liu, Chen & Hsieh 2002	73%	
Japan	Takeda, Tasaka, Sakata & Murata 2006	95%	5.3%
India	Banerjee, Roy & Takkar 2000		
South Africa	Gunston 1986		
Nigeria	Adenaike & Obidoye 1987		
	Toriola 1989		
	Fakeye & Adeyoke 1994		20%
Zimbabwe	McMaster, Cormie & Pitts 1997		
Morocco	Montero, Bernis, Lovkid, Hilali & Baali 1999	51.2%	
Italy	Monagle, Dan, Krogh, Jossa, Farinero & Trevisan 1993		
Spain	de la Gandara Martin & de Diego Herrero 1996	30.5%	3-10%
France	Serfaty & Magneron 1997	35%	
Iceland	Sveinsdottir 1998		
Finland	Kantero & Widholm 1971	68%	
Sweden	Andersch, Wendestam, Hahn & Ohman 1986		
	Hammarback, Damber & Backstrom 1989	61-85%	
Switzerland	Flug, Largo & Prader 1985		
United Kingdom	Dalton & Greene 1953		
	Magos, Brincat & Studd 1986		
	van den Akker & Steptoe 1989		
	van den Akker, Eves, Service & Lennon 1995		
Bulgaria	Kolarov, Sirakov, Burdarova, Zhekova & Borisov 1996		
Poland	Skierska, Leszczynska-Bystrzanowska & Gajewski 1996	40.5%	
	Ciszek-Doniec, Poreba, Zogala & Olszowka 1999		
Germany	Gaulrapp, Backe & Steck 1995	7.7%	
Canada	Fradkin & Firestone 1986		
	Youdale & Freeman 1987		
Mexico	Marvan & Diaz Eroza 1995		
Australia	Beaumont, Abraham, Argall & Simson 1978		
	Hart, Coleman & Russell 1987		
	Campbell, Peterkin, O'Grady & Sanson-Fisher 1997	85%	11-32%
	Treloar, Heath & Martin 2002		
United States	Price, DiMarzio & Gardner 1986	70-90%	20-40%
	Boyle, Berkowitz & Kelsey 1987	39-81%	
	Johnson, McChesney & Bean 1988	87%	3.2%
	ACOG 1989	20-40%	
	Severino & Moline 1990	30%	

Diagnostic methods, including *which* symptoms “count” and *how* they are counted, also vary greatly. There are currently over 65 different daily symptom rating instruments (Budeiri, Li Wan Po, & Dornan, 1994) and >150 symptoms associated with PMS (Dalton, 1986). Thus, diagnosis is instrument-specific. Additionally, women with no symptoms in common can be diagnosed with the same syndrome. A recent multidisciplinary expert panel (Halbreich, Backstrom, Eriksson, O’Brien, Calil, Ceskova, Dennerstein, Douki, Freeman, Genazzani, Heuser, Kadri, Rapkin, Steiner, Wittchen, & Yonkers, 2007) has (again) called for the establishment of a uniform definition, measurement tools, standards and practices.

The obvious link between PMS and the menstrual cycle has directed etiological research toward a search for underlying proximate (mechanistic) causes. However, no clear results have emerged from investigations of endocrinological, genetic, or other biochemical etiologies of PMS (Connolly, 2001). In addition, no treatments derived from such proximate research programs have been any more effective than placebo (Tucker & Whalen, 1991). Clare (1985: 478) and Connolly (2001) conclude that any single, proximate, causal factor is unlikely to sufficiently explain either individual symptoms or the entire syndrome. Tucker & Whalen (1991: 311) attribute the poor state of understanding in PMS research to "inadequate theoretical development" in the field.

### *Evolutionary model of PMS*

Addressing this shortfall, Reiber (2008) recently published an evolutionary model aimed at explaining *why* the wellbeing of women fluctuates across the cycle. In this model, PMS is hypothesized to be a byproduct of adaptations that maximize chances of mating and fertilization. During the fertile (follicular) phase of the menstrual cycle,

women ornament more (Haselton, Mortezaie, Pillsworth, Blecke-Rechek, & Frederick, 2007) and wear sexier/bolder clothing (Grammer, Renninger, & Fischer, 2004), go to clubs more (Haselton & Gangestad, 2006), are more attentive to “maleness” (Macrae, Alnwick, Milne, & Schloerscheidt, 2002), increase ranging activities including locomotion and volunteering for more social activities (Fessler, 2003), and flirt more with men not their mates (Haselton & Gangestad, 2006; Gangestad, Thornhill, & Garver, 2002).

Women’s facial appearance (Roberts, Havlicek, Flegr, Hruskova, Little, Jones, Perrett, & Petrie, 2004) and body odors (Doty, Ford, Preti, & Huggins, 1975; Singh & Bronstad, 2001; Thornhill, Gangestad, Miller, Scheyd, McCullough, & Franklin, 2003) are also more attractive to men, and their sexual desire is higher (Haselton & Gangestad, 2006; Gangestad, Thornhill, & Garver, 2002; Bullivant, Sellergren, Stern, Spencer, Jacob, Mennella, & McClintock, 2004) during the fertile phase of the menstrual cycle. When these adaptive states abate -- during the premenstruum—women are less social, less active, feel less sexy and less flirtatious and desirous; they feel relatively worse, and men find them less attractive. Taken together, this defines a state of suboptimal general wellbeing, and may be subjectively experienced as symptoms. In its extreme, clinical form, this lower state is PMS. Thus, while PMS is *not* adaptive in and of itself, it results from the cessation of evolutionarily adaptive, heightened, positive states that are fostered to increase the chances of mating and fertilization during the fertile phase of the menstrual cycle.

This suggests that how a woman feels during the follicular phase of the cycle should be predictable by evolutionarily relevant variables related to reproduction, such as age, parity, and resources. These variables may also therefore be indirectly related to PMS, since the better a woman feels during the follicular phase of her cycle, the

larger or more noticeable may be the change in her state of wellbeing between the follicular and luteal phases of her cycle. This report presents empirical tests of these arguments.

## **Methods**

### *Participants*

The use of human subjects in this research was approved by the appropriate Psychosocial Institutional Review Board (IRB). Participants were contacted through professional, educational, and social organizations using flyers recruiting for a women's health study. All women participated in informed consent procedures as required by the IRB. Women were paid \$25 upon completion of the study. Two hundred eighteen (218) women participated in the study. One subject identified herself as homosexual; she was eliminated from analyses as a potential outlier. Two other women were eliminated because their completed screening questionnaires later revealed conflicts with exclusionary criteria. This left a final sample of 215 women.

Participants were required to be regularly menstruating, but were *not* required to have PMS. To rule out potential confounds to diagnosis of PMS, women were excluded if they: 1) were pregnant or lactating, 2) had begun to experience any symptoms of menopause, 3) had failed to have three regular menstrual cycles immediately prior to the study, 4) had undergone any surgery to their ovaries, and/or 5) had been diagnosed with clinical depression, thyroid disease, hypoglycemia, or diabetes. Because oral contraceptives (OCs) have been argued to both alleviate and exacerbate PMS (Severino & Moline, 1989: 181-184), and clinical trials of OCs as a treatment for PMS have failed (Connolly, 2001; Joffe, Cohen, & Harlow, 2003), women using them were

*not* excluded from the study.

### *Instruments and data collection*

Participants completed self-report biographical and medical questionnaires at screening. Basic descriptive information included age, number and age(s) of children, and resource level (annual household income in US \$). Medical questions included menstrual and gynecological histories, histories of surgeries, medications, contraceptive choices, psychological disorders, depression, diabetes, thyroid disease, hypoglycemia, cancers, and other medical problems. Women also gave *a priori* estimations of the extent and severity of their PMS.

To avoid potential biases associated with retrospective symptom reporting, a standard prospective Daily Symptom Checklist adapted from Severino & Moline (1989, based on Endicott, Nee, Cohen, & Halbreich, 1986) was used. Each day, women marked the severity of each of 17 symptoms on a scale of 1 to 6, from none to extreme. Symptoms included: decreased sociality; decreased interest in activities; decreased ability to get work done; bloating; mood swings; feeling blue, sad, or low; feeling anxious or nervous; feeling irritable or angry; headaches; aberrant sleep; decreased concentration; breast pain; food cravings; decreased energy level; general pain; feeling cheated; and feeling unhappy.

Three guidelines for using the daily records were given. First, women began daily symptom ratings on the first day of the calendar month immediately following screening. Because women's cycles are not synchronized with calendar months, this would distribute any bias or potential confounds related to instrument familiarity or boredom evenly across the cycles of the participant pool rather than aggregating it at

the end of the cycles when PMS might be present. Second, while women were allowed to use marks from previous days as an experiential guide when evaluating each new day, they were not allowed to alter marks from previous days. Finally, women were instructed to complete the chart at the same time each day to prevent fluctuations within days from confounding change across days.

*The independent variables: age, parity, and resources*

Age, parity, and resources are the three independent variables that were evaluated. Age was recorded in years. Parity was recorded as a raw count of living children only (no stillbirths or abortions). All offspring were assumed to be equally evolutionarily valuable. Total annual household income (in US\$) was used as a crude proxy for resources; non-monetary types of support were not evaluated. No attempt was made to evaluate the women's actual control over or access to household resources, the conversion of resources into children, consumption by children, or the nature of the distribution of resources between mother and child(ren) or between siblings. These are conservative assumptions that should bias outcomes against the expected relationships.

*The dependent variables*

The basic concept underlying PMS is symptom change between the follicular and premenstrual weeks of the cycle. The first day of menstrual bleeding was assigned day 1 of the cycle. For each woman, the mean rating for each symptom was calculated over two periods: days 5 through 11 of the menstrual cycle, the follicular week, which is typically symptom-free; and the last seven days of the cycle, the luteal week, during

which symptoms are predicted to be present if a woman has PMS. Change between the weeks was then calculated, and diagnostic criteria (Severino & Moline, 1989) were applied to each woman's symptom change data to generate and assign a dichotomous diagnostic result: PMS (PMS+) or no PMS (PMS-). For a woman to receive a diagnosis of PMS, at least five symptoms had to worsen by at least two degrees between the follicular and luteal phases of the cycle.

Two aggregate measures of total symptom load were also computed for each woman, by summing the mean ratings of all 17 symptoms during each of the two target weeks. High scores on weekly symptom load indicate a greater number and severity of symptoms (hence, feeling worse); low scores indicate few or only very mild symptoms (hence, feeling better). Finally, women were categorized into two groups based on the pattern of change in total symptom load between the two target weeks; those having a higher total symptom load during the luteal phase of the cycle than during the follicular phase were designated "sufferers" (even though they may not qualify for a *clinical* diagnosis of PMS), and those having a lower total symptom load during the luteal phase of the cycle than during the follicular phase were designated "improvers" across the cycle. This distinction is important for two reasons: 1) clinical definitions are not widely agreed upon and are not currently defined by discrete, biologically meaningful states; thus, this comparison allows a wider view of symptom change than simply following clinical diagnostic criteria; and 2) it recognizes that in addition to those with PMS and those whose symptoms worsen but who fail to meet diagnostic criteria, there is another category: women whose symptoms change in the opposite direction—that is, who actually improve across the cycle.

### *Analyses*

All analyses were carried out in Systat version 10.2 for Windows. Because age, parity, and resources might be expected to be highly correlated, multicollinearity was ruled out by computing the correlations between each pair of independent variables. All were less than 0.6. All regression models used the same independent variables: age, parity, and resources. Multiple regression models were constructed to evaluate the predictive power of the independent variables (age, parity, and resources) on mean symptom ratings during the target weeks (follicular and luteal). Two-group t-tests were used to compare women who met the criteria for diagnosis (PMS+) to those who did not (PMS-), and those who worsen across the cycle (“sufferers”) to those who improve across the cycle (“improvers”). Finally, median splits along the independent variables were generated to compare women at the two ends of each continuum. Chi-square analyses were then computed to show the percent of women above and below the median value who were PMS+ vs. PMS-, and who were sufferers vs. improvers. For all analyses, alpha was set at 0.05. To be conservative, analyses were run both on the overall sample as well on the subset of participants not using OCs. Results did not differ between the two samples in terms of either statistical or clinical significance. Thus, results presented here include women using OCs.

## **Results**

Participant characteristics are presented in Table 2. Participants were predominantly non-Hispanic Caucasian Americans of western European descent. Education levels and occupations ranged widely; amongst the participants were unemployed high school dropouts as well as a Ph.D.-level psychologist who manages a

major corporation. Forty-five women (20.93%) reported current use of OCs.

Table 2. Participant characteristics ( $n=215$ )

variable	min.	max.	mean	std. dev.	median
Age (yrs.)	17	50	33.2	8.4	35
Parity	0	6	1.5	1.3	2
Resources (x10,000 US \$)	0	7	3.9	2.2	4
Education (yrs.)	11	20	14.7	1.9	15
Cycle length (days)	15	37	28.1	2.7	28
Age at menarche (yrs.)	10	17	12.7	1.2	13
Self-rating of PMS severity (0-5, none-extreme)	0	5	2.4	1.2	2

On baseline questionnaires, 93% (200) of the women reported *a priori* that they regularly suffer from PMS. Just under 52% reported symptom severity between nonexistent and mild PMS, and about 48% reported moderate to extreme PMS. However, only 12.09% (26 women) actually qualified for a diagnosis of PMS. Moreover, of the 26 women who qualified for a diagnosis, only 6 rated their symptoms as severe or extreme before prospectively tracking them; the other 20 rated their symptoms between nonexistent and moderate.

Table 3 shows the results of multiple regression models using age, parity, and resources to predict mean follicular-week severity for each of the 17 symptoms. Fourteen of the seventeen models are statistically significant and in the directions predicted for each of the three independent variables. Total symptom load during the follicular week shows the same pattern. It is significantly negatively correlated with age

( $p=0.019$ ) and resources ( $p=0.003$ ), and positively correlated with parity ( $p=0.184$ );  $F_{\text{model}}=6.42$ , adjusted multiple  $r^2=0.07$ ,  $p<0.0001$ . This indicates that women who are older, have fewer children, and more resources feel better during the follicular week than do those who are younger, have more children, and fewer resources. No significance was found for similar models examining symptoms and symptom load during the premenstrual week.

*Table 3.* Results of multiple regression models demonstrating the relationship between age, parity, and resources, and mean symptom severity during the follicular week of the menstrual cycle

Follicular Week Symptom Mean	Direction of relationship			Model Adjusted Multiple $r^2$	Model F-ratio
	Age (yrs)	Parity	Resources (\$)		
decreased sociality	- ***	+	- *	0.036	3.66 ***
decreased interest in activities	-	+	-	0.048	4.599 ***
decreased work	- **	+	- **	0.041	4.081 ***
bloating	-	+	- *	0.003	1.21
mood swings	- *	+	- **	0.015	2.075 *
blue, sad, low feelings	- **	+	- **	0.046	4.441 ***
anxious, nervous	+	- *	- **	0.023	2.692 **
irritable, angry	- **	+ *	- **	0.039	3.884 ***
headaches	- ***	+ ***	- ***	0.059	5.488 ***
decreased sleep	- *	+	- **	0.024	2.791 **
decreased concentration	- ***	+	- ***	0.084	7.511 ***
breast pain	-	+ **	- **	0.026	2.893 **
food cravings	-	+ *	- ***	0.027	3.01 **
decreased energy	- **	+	- ***	0.084	7.561 ***
general pain	-	+	- ***	0.023	2.709 **
feel cheated	- ***	+ ***	- ***	0.059	5.448 ***
unhappiness	- **	+	+	0.004	1.297

\* $0.10 \geq p > 0.050$ ; \*\* $0.050 \geq p > 0.010$ ; \*\*\* $0.010 \geq p$

Table 4 compares women who meet diagnostic criteria for PMS (PMS+) to those who do not (PMS-). While the PMS+ women have significantly higher total symptom loads during the luteal week (by definition, of course), they also have significantly lower total symptom loads (that is, they feel better) than PMS- women during the follicular week of the cycle (also see Figure 1, Panel A). As predicted, PMS+ women are also significantly older and have more resources than PMS- women. However, contrary to expectations, PMS+ women have more children than PMS- women.

*Table 4.* Comparison between women meeting diagnostic criteria (PMS+) and women not meeting diagnostic criteria (PMS-)

	PMS + (n=22)		PMS – (n=193)		<i>t</i>
	Mean	SD	Mean	SD	
Luteal Week Total Symptom Score	54.63	10.59	34.36	10.78	-8.49 ***
Follicular Week Total Symptom Score	25.56	8.47	29.26	9.52	1.92 **
Age (yrs.)	35.18	7.1	33.05	8.61	-1.31 *
Parity	1.91	.97	1.55	1.37	-1.55 *
Resources (x10,000 US \$)	5.05	1.96	3.78	2.25	-2.81 ***

\* $0.10 \geq p > 0.050$ ; \*\* $0.050 \geq p > 0.010$ ; \*\*\* $0.010 \geq p$

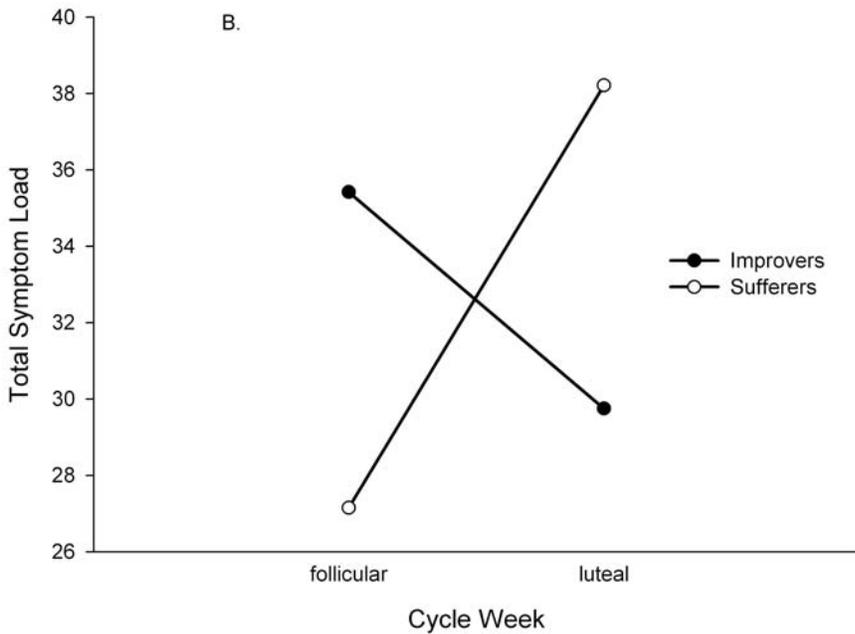
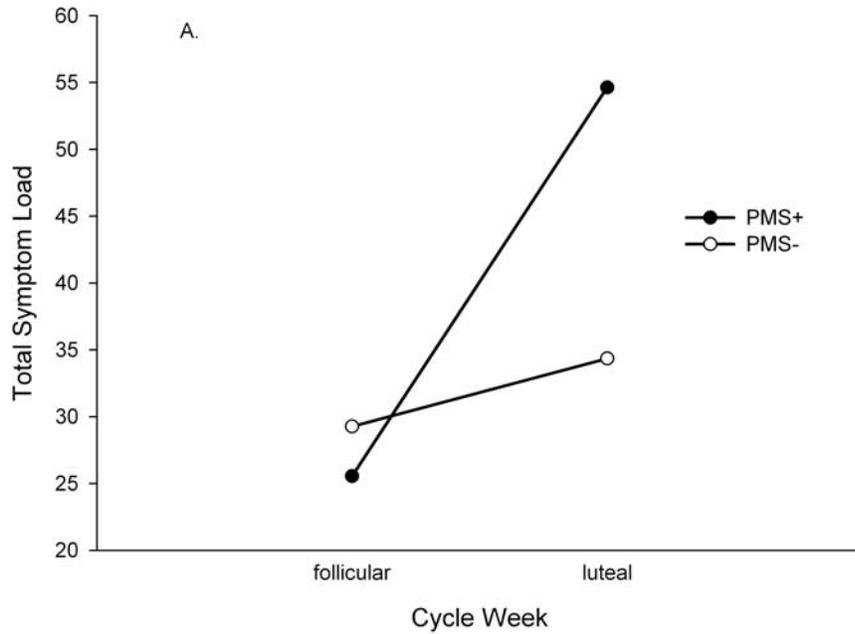


Figure 1. Mean total symptom load during the follicular and luteal weeks of the cycle, for PMS+ vs. PMS- women (panel A) and improvers vs. sufferers (panel B) (Panel A, PMS+ vs PMS-: Repeated measures ANOVA  $F_{\text{week} \times \text{group}} = 166.64$ ,  $p < 0.0001$ ; Panel B, Improvers vs Sufferers: Repeated measures ANOVA  $F_{\text{week} \times \text{group}} = 122.75$ ,  $p < 0.0001$ .)

Similarly, Table 5 compares sufferers (whose symptoms worsen across the cycle) to improvers (whose symptoms improve). While the sufferers have significantly higher total symptom loads during the luteal week (of course), they also have significantly lower total symptom loads (that is, they feel better) than improvers during the follicular week of the cycle (also see Figure 1, Panel B). As predicted, sufferers are also significantly older and have more resources than improvers. Again, contrary to expectations, sufferers have more children than improvers, although not significantly.

*Table 5.* Comparison between women whose symptoms worsen across the cycle and women whose symptoms improve across the cycle

	sufferers (n=170)		improvers (n=45)		<i>t</i>
	Mean	SD	Mean	SD	
Luteal Week Total Symptom Score	38.21	12.53	29.75	9.20	-5.05 ***
Follicular Week Total Symptom Score	27.15	8.21	35.41	11.02	4.70 ***
Age (yrs.)	33.84	8.1	31.11	9.56	-1.75 **
Parity	1.63	1.28	1.44	1.55	-0.74
Resources (x10,000 US \$)	4.03	2.2	3.47	2.42	-1.41 *

\* $0.10 \geq p > 0.050$ ; \*\* $0.050 \geq p > 0.010$ ; \*\*\* $0.010 \geq p$

Median splits of the data indicate that of those who are older than the median age, 14.41% are PMS+, while only 5.77% of those who are younger are PMS+ ( $\chi^2=4.37$ , 1 df,  $p=0.04$ ). Of those who are wealthier than the median, 14.52% are PMS+, while only 4.4% of those who are poorer are ( $\chi^2=5.85$ , 1 df,  $p=0.02$ ). And of those who have more children than the median, 13.33% are PMS+, while only 6.32% of

those with fewer children are ( $\chi^2=2.84$ , 1 df,  $p=0.09$ ). Figure 2 (panel A) shows these results. At the other end of the spectrum of cyclic symptom change are those women who improve across the cycle. Of those who are younger than the median age, 26.92% improve across the cycle compared to 15.32% of those who are older than the median age ( $\chi^2=4.37$ , 1 df,  $p=0.04$ ). Of those who are poorer than the median, 25.27% improve across the cycle compared to 17.74% of those who are wealthier than the median ( $\chi^2=1.80$ , 1 df,  $p=0.18$ ). And of those who have fewer children than the median, 27.37% improve across the cycle compared to 15.83% of those who have more children than the median ( $\chi^2=4.26$ , 1 df,  $p=0.04$ ). Panel B of Figure 2 illustrates these comparisons.

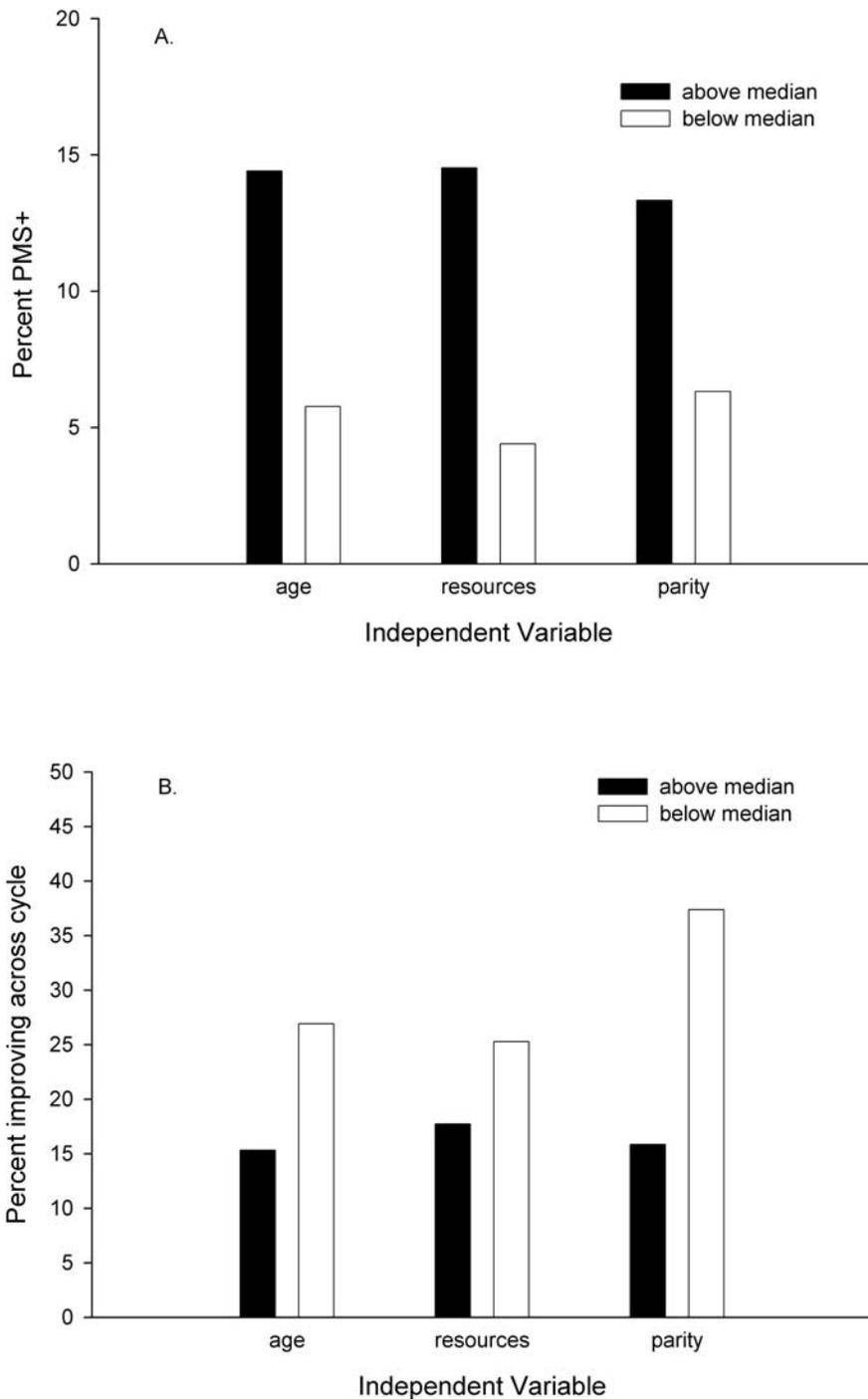


Figure 2. Percent of participants meeting clinical diagnostic criteria for PMS (panel A) and improving across the cycle (panel B), by age, resources, and parity (Panel A:  $\chi^2_{age}=4.37$ , 1 df,  $p=0.04$ ;  $\chi^2_{resources}=5.85$ , 1 df,  $p=0.02$ ;  $\chi^2_{parity}=2.84$ , 1 df,  $p=0.09$ ; Panel B:  $\chi^2_{age}=4.37$ , 1 df,  $p=0.04$ ;  $\chi^2_{resources}=1.80$ , 1 df,  $p=0.18$ ;  $\chi^2_{parity}=4.26$ , 1 df,  $p=0.04$ .)

## Discussion

The discrepancy between women's self-reports of symptoms and their diagnostic outcomes is striking. Many women who report suffering do not qualify for a diagnosis under currently accepted diagnostic standards. Moreover, the disjunction is not simply a matter of degree. It is *not* the case that women who fail to meet diagnostic criteria have symptoms that are just too mild. On the contrary, many women reporting severe problems also fail to meet the criteria for a diagnosis. This suggests that the widely-accepted diagnostic criteria do not reflect women's experiences, and points to a need for the re-evaluation of diagnostic definitions and methods (see Halbreich, Backstrom, Eriksson, O'Brien, Calil, Ceskova, Dennerstein, Douki, Freeman, Genazzani, Heuser, Kadri, Rapkin, Steiner, Wittchen, & Yonkers, 2007).

While PMS is *not* adaptive in and of itself, these findings suggest that it results from the cessation of evolutionarily adaptive, heightened, positive states during the fertile (follicular) phase of the menstrual cycle. These heightened follicular states, reflected by lower symptom severity and lower overall symptom load, were found to be predictable by age, parity, and resources. Older age, lower parity, and higher resources were predictive of feeling better during the follicular week of the cycle. This is consistent with the proposed evolutionary function of heightened states of wellbeing during the follicular phase of the cycle: increased mate attraction and fertilization (Haselton, Mortezaie, Pillsworth, Blecke-Rechek, & Frederick, 2007; Grammer, Renninger, & Fischer, 2004; Haselton & Gangestad, 2006; Macrae, Alnwick, Milne, & Schloerscheidt, 2002; Fessler, 2003; Gangestad, Thornhill, & Garver, 2002; Roberts, Havlicek, Flegr, Hruskova, Little, Jones, Perrett, & Petrie, 2004; Doty, Ford, Preti, &

Huggins, 1975; Singh & Bronstad, 2001; Thornhill, Gangestad, Miller, Scheyd, McCullough, & Franklin, 2003; Bullivant, Sellergren, Stern, Spencer, Jacob, Mennella, & McClintock, 2004). Who is evolutionarily expected to be attempting to increase mating and fertilization?: those who are running out of time for reproduction (i.e., those who are older), have few(er) children, and have ample resources to support an (additional) offspring.

Those who were not evolutionarily expected to be attempting to increase mating and fertilization during the follicular phase of the cycle—younger women, with higher parity, and fewer resources—followed an inverse trajectory across the cycle (“improvers”). They were found to feel better during the luteal, and worse during the follicular, phase of the cycle. Instead of attempting to initiate reproduction, their heightened state of wellbeing during the luteal phase of the cycle may serve to retain mates, garner resources, and/or improve condition prior to initiating reproduction.

These insights suggest a paradigm shift in the field of PMS research. Traditionally, PMS has been thought of as “something gone wrong” during the premenstruum, because of the focus on symptoms and the temporal connection between PMS and menstruation. However, this evolutionary approach indicates that women’s wellbeing is expected to fluctuate across the cycle, and lower states of wellbeing (and clinical PMS, in the extreme) are only one part of that larger cycle. PMS is being driven by women’s state of wellbeing during the follicular phase of the cycle. Notice, too, that the “improver” pattern of change could be interpreted as “pseudo-PMS” during the follicular phase of the cycle! (Note that others (e.g., Schnall, Abrahamson, & Laird (2002)) have similarly found women experiencing improvements across the cycle as well as worsening.) In either case, the critical question seems to be: at what point in

a women's cycle will heightened wellbeing be most evolutionarily advantageous? For those in condition to initiate reproduction, heightened wellbeing is expected to be fostered during the follicular phase to increase chances of mating and fertilization. For those not in condition to initiate reproduction, heightened wellbeing might be better fostered during the luteal phase to retain mates, garner resources, and/or improve condition in the face of impending menstruation.

### *Synthesizing ultimate and proximate models of PMS*

The vast majority of PMS research has focused on proximate explanations, and is neither mutually exclusive of, nor discounted by, this evolutionary model. In fact, all evolutionary explanations require supporting proximate mechanisms-- the endocrinology, biophysiology, psychology, cognition— through which they are actuated. The evolutionary model has the potential to inform the continuing search to understand how PMS works by providing clues for how to refine the proximate models in more potentially fruitful ways. It is possible that this new framework will unmask relationships that were previously obscured by limited (and variable) definitions and diagnostic methods, and lack of a theoretical framework.

Consider the relationship between PMS and sex hormones. Although the role of sex hormones in PMS has been investigated to no avail (Connolly, 2001), the evolutionary framework suggests that such proximate endocrinological causes should be re-evaluated with respect to fluctuation across the entire cycle, with particular attention to the follicular phase, instead of the luteal and menstrual phases, of the cycle. This research is currently underway, examining women's daily estradiol and progesterone levels in relation to symptoms.

Various proximate psychological theories of PMS also make sense in conjunction with this evolutionary framework. For example, Schnall, Abrahamson, & Laird (2002) found that women who were responsive to bodily changes across the cycle were more likely to report mood changes, both positive and negative. Moreover, reminders of cycle stage prevented PMS-like mood changes in women responsive to their bodily cues. Similarly, Blake *et al.* (1998) argue that negative interpretations of physiological changes result in the experience of PMS.

What these (and other psychological) models suggest is that psychology and physiology track one another, that some cyclic fluctuations can be personally attributed either to body or to emotions/mind, and that conscious awareness can alter perception and/or attribution, and possibly mitigate patterns. This is expected within an evolutionary framework, since it is the confluence of anatomy, physiology, psychology, and behavior that generates ultimate effects. Lions that think (or feel) like lambs are unlikely to be highly successful.

How can the evolutionary model enhance these proximate psychological models? The evolutionary framework suggests that, in addition to psychological perception of changing physical states, psychological states themselves change across the menstrual cycle. More research is needed to parse out these two different psychological tracks, since one can be successfully altered to reduce symptoms (Schnall, Abrahamson, & Laird, 2002) but the other may not respond to these same interventions.

#### *Future directions*

Additional research should also investigate the cross-cultural epidemiology and

reproductive ecology of PMS in light of these findings. For instance, in natural fertility populations, women's reproductive lives are largely governed by the balance between energetic, physiological and ecological parameters (see Ellison, 2001a, 2001b; Vitzthum, 2001). Thus these women might not be expected to experience PMS since they may not be selected to enhance mating and fertilization during the follicular phase of the cycle.

The evolutionary model suggests predictable changes in women's experiences throughout the lifecourse as well. Prospective longitudinal studies should investigate changes in patterns of women's cyclic well-being as they pass through major life stages. For instance, this model predicts that patterns of cyclic change would shift as women mature, obtain mates, have children, garner and lose resources, and separate from mates.

This evolutionary approach suggests changes in how we think about PMS. Instead of investigating it as an isolated disorder in its own right, it should be placed within the larger context of fluctuations across the cycle in the physical and psychosocial well-being of women. Rather than thinking of PMS as something gone wrong with physiology and/or endocrinology during the premenstruum, it would be productive, as this model suggests, to shift the etiological focus to proximate underpinnings of adaptive socio-psycho-behavioral phenomena during the follicular phase of the cycle. From the vantage point of this evolutionary model, hypotheses can be generated and tested to elucidate etiological mechanisms, epidemiology, and clinical management practices.

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