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Menstrual cycle and appetite control: implications for weight regulation

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Hormonal fluctuations associated with the menstrual cycle influence appetite control and eating behaviour. Energy intake varies during the reproductive cycle in humans and animals, with a periovulatory nadir and a luteal phase peak. Patterns of macronutrient selection show less consistency but a number of studies report carbohydrate cravings in the premenstrual phase, particularly in women with premenstrual syndrome. The cyclical nature of food cravings are frequently, but not invariably, associated with depression. Fluctuations in appetite, cravings and energy intake during the menstrual cycle may occur in parallel with cyclical rhythms in serotonin, which can be accompanied by affective symptoms. The premenstrual phase can be considered as a time when women are especially vulnerable to overconsumption, food craving and depression; this is often associated with low serotonin activity.

Key words: appetite/food craving/menstrual cycle/serotonin

Introduction

In this last decade of the twentieth century, obesity is becoming an ever increasing public health problem. In turn this has stimulated the current epidemic of dieting (Blundell and Bauer, 1994). At the same time people are confronted by a vast array of food products, and food consumption is heavily promoted. Dieting is particularly common in young women. It is estimated that 40–50% of women are on a diet at any one time (Polivy and Herman, 1987). Factors which influence the ease of overconsumption and the difficulty of underconsumption are likely to be important in the maintenance of a stable body weight and in the prevention of the development of obesity.

The regular hormonal fluctuations associated with the menstrual cycle may influence appetite control and eating behaviour. In addition, particular conditions associated with the menstrual cycle, most notably premenstrual syndrome (PMS), may predispose women to changes in appetite control. It is therefore important to identify any changes in eating associated with the menstrual cycle in general, and with PMS in particular, in order to be able to deal effectively with any implications for health and well-being.

What is the premenstrual syndrome?

The premenstrual syndrome is a collection of behavioural, somatic and physical symptoms which occur in the 7–10 days prior to the onset of menstruation and which are relieved at or shortly after commencement of menstrual flow. In its most severe form this is also known as premenstrual dysphoric disorder (PMDD) (Gold, 1994)*. Although a large range of symptoms have been associated with PMS, the more common symptoms which can be said to characterize the syndrome include depression, irritability, mood swings, water retention-based symptoms such as breast tenderness and bloating, changes in appetite and food cravings. These symptoms can be measured using subjective rating scales. The distinction between PMS and PMDD relates to the severity of the symptoms. PMDD is less prevalent than PMS, affecting ~5% of women. To be clinically significant, it is generally accepted that the symptoms of PMDD that a sufferer experiences must endure consistently, at a severe level, for at least two cycles (American Psychiatric Association, 1994). For the purposes of this review, the term PMS is used to encompass mild and severe forms of premenstrual symptoms. Over the years a number of assertions have been made about the relationship between the existence of this syndrome and changes in food consumption which could influence weight regulation.

Eating behaviour and appetite control

Particular physiological and psychological characteristics of the menstrual cycle and PMS could influence the expression of appetite through a variety of mechanisms and processes which influence the control of food intake. The overall regulation of eating is complex and can best be envisaged as a psychobiological system which serves to integrate biological and environmental influences (e.g. Blundell, 1991). One important feature of the environment is, of course, the nutritional composition of the food supply. Human food intake can be assessed by means of quantitative aspects of food consumption such as the energetic value of food and its macronutrient composition (proportion of fat, protein and carbohydrate). Appetite is also represented by qualitative aspects such as food choice, food preferences and appreciation of the sensory aspects of food (taste, palatability, mouth-feel etc.). In addition, subjective phenomena such as the perception of hunger, fullness and hedonic sensations which accompany eating are also important (Hill *et al.*, 1995). Along with these characteristics of consumption are particular food cravings and urges to eat food in general or specific food products. Consequently, the phenomenon that scientists and others call

*PMDD replaces late luteal phase dysphoric disorder (LLPDD) in *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn (American Psychiatric Association, 1994). LLPDD appeared in a research appendix to the third edition of this manual.

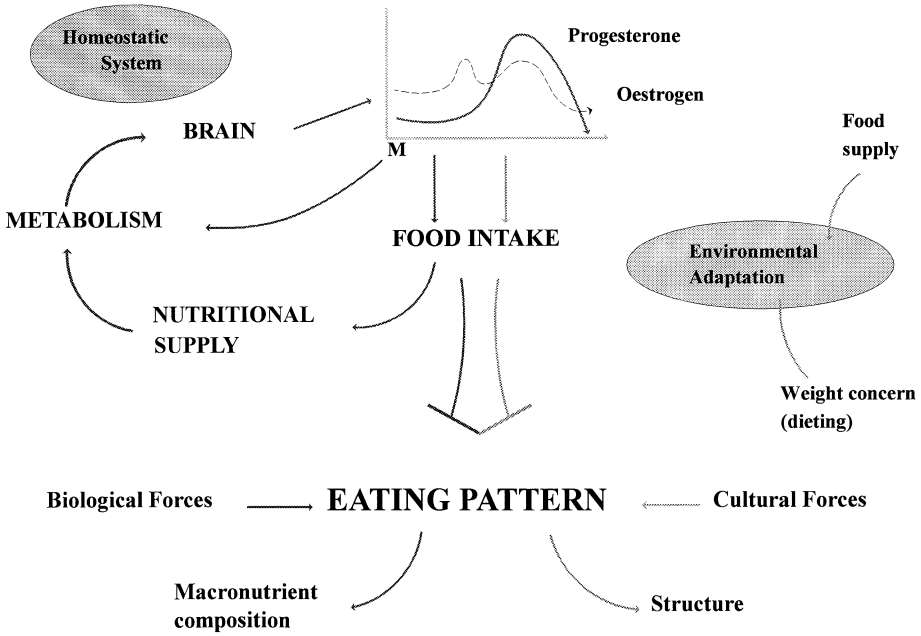

Figure 1. The fundamental elements influencing the control of food intake. The diagram illustrates how hormonal fluctuations underlying the menstrual cycle exert direct and indirect effects on the homeostatic system controlling food intake and the pattern of eating.

food intake can be analysed in terms of structured patterns of eating (meals, snacks etc.) and nutritional components (selection of fat, carbohydrate etc.). Subjective sensations (the experience of a drive to eat) accompany these patterns and may be thought of as causing these adjustments. It follows that any changes induced by the hormonal fluctuations of the menstrual cycle or by PMS may be detected in various aspects of eating, including changes in hunger, cravings for certain foods, alteration in meal size or snacking, adjustments in consumption of fat or carbohydrate and an overall change in energy intake.

The schematic diagram in Figure 1 indicates how food intake forms part of a homeostatic system whilst also being adapted to environmental demands. Two particularly important features of our current environment are the prevalence of dieting as a lifestyle for many women, and the abundance of high-fat, highly palatable foods in the food supply. The homeostatic system does not operate in symmetrical fashion. The system appears to be strongly defended against undereating but only weakly protected against overconsumption (see Blundell, 1996). Therefore, whereas dieting to lose weight is extremely difficult, overconsumption is relatively easy and can occur passively. In turn, this passive overconsumption is likely to lead to weight gain and possibly obesity (Lawton *et al.*, 1993). This means that any overconsumption (either via generally increased energy intake or involving cravings or urges for particular foods) which occurs premenstrually is likely to lead to incremental weight gain which, in turn, will engender accompanying psychological problems.

Figure 1 illustrates how hormonal changes underlying the menstrual cycle can modulate the principles of homeostatic regulation. This means that hormonal rhythms will influence the capability of people to undereat (to maintain dieting), and the ease of overconsumption. It seems appropriate to use this model to account for the relationship between hormonal rhythms, energy balance, and adjustments in various components (qualitative and quantitative) of food intake. It follows that hormonal changes could directly alter the biological drive to modulate eating or could exert an indirect influence by physiological adjustment so as to make individuals more susceptible or vulnerable to facilitating or stimulating environmental forces.

Menstrual cycle changes in food intake

Animal studies

Cyclical changes in hunger motivation or food intake have been observed in many animal species during the oestrus cycle; studies indicate a consistently lower intake around the time of ovulation. In rodents, feeding is depressed coincident with oestrus (Brobeck *et al.*, 1947; Jennings, 1969; Herberg *et al.*, 1972; Wade, 1972; Leshner and Collier, 1973; Tarttelin and Gorski, 1973). However, human and other primate menstrual cycles are distinctly different to the oestrus cycle of the rodents. Primates have a prolonged luteal phase following ovulation, which is characterised by elevated levels of circulating progesterone (Rosenblatt *et al.*, 1980).

Studies in a number of primate species (see Table I) indicate that highest food intake occurs consistently in the luteal phase with the nadir of food intake around the time of ovulation (Krohn and Zuckerman, 1938; Gilbert and Gillman, 1956; Czaja, 1975; Rosenblatt *et al.*, 1980). An ovulatory decrease in food intake has also been observed in other mammals such as pigs (Friend, 1969) goats (Forbes, 1971) and sheep (Tarttelin, 1968).

Initially, elevated food intake in the luteal phase was attributed to increased concentrations of progesterone (Gilbert and Gillman, 1956). Later, the ovulatory decrease in intake was attributed to the appetite-suppressant effects of oestrogen

Table I. Cyclical patterns of food intake in mammals

Species	Intake phase		Reference
	Highest	Lowest	
Baboon	Luteal	Follicular/ ovulatory	Gilbert and Gillman (1956)
Rhesus monkeys	Luteal	Ovulatory	Rosenblatt *et al.* (1980)
Rhesus monkeys	Luteal	Ovulatory	Czaja (1978)
Pigtail monkeys	Luteal	Ovulatory	Krohn and Zuckerman (1938)
Pig		Pre-ovulatory	Friend (1969)
Goat		Ovulatory	Forbes (1971)
Sheep		Ovulatory	Tarttelin (1968)
Guinea-pig		Ovulatory	Czaja and Goy (1975)
Rat		Oestrus	Herberg *et al.* (1972)

(Czaja, 1975, 1978), the luteal phase increase being thus due to the inhibitory action of progesterone on oestrogenic activity.

Given the relatively similar reproductive cycles of primates and humans, it is to be expected that similar fluctuations in food intake should occur during the menstrual cycle, under natural conditions.

Human studies

In humans, when food intake has been examined in relation to the menstrual cycle a distinct pattern of fluctuation has been observed. Generally, energy intake is higher in the postovulatory or premenstrual phase of the cycle than in the preovulatory or follicular phase (see Table II for review of research). Examination of these phases has usually focused on two 10 day periods either side of ovulation.

In the majority of studies luteal energy intake is significantly higher than energy intake in the follicular phase. Of 30 studies that compare cycle phases in 37 groups of women, 25 studies report significantly higher luteal energy intake than follicular intake. The remaining 12 comparisons show no significant effect, though the trend for similar groups in each study is invariably in the same direction. The two exceptions are Wurtman *et al*. (1989) and Krakow (1992) who show follicular energy intake to be non-significantly greater than luteal intake in subjects with no premenstrual symptoms and women not taking oral contraceptives respectively. Both of these studies, however, have some inherent methodological weaknesses. For example, in both studies participants were aware of the menstrual cycle focus, a factor known to influence reporting (Ruble, 1977), and in Wurtman *et al.*'s study screening for PMS was based on retrospective reports which are likely to amplify any cyclical pattern (Warner *et al.*, 1991).

In contrast, such statistically significant cycle-related trends are not observed in women using oral contraceptives (Anantharaman-Barr *et al.*, 1988; Krakow, 1992), or women with anovulatory cycles (Barr *et al.*, 1995). In both of these conditions, cyclic fluctuations in hormones are absent or minimized. In addition, eating trends across the menstrual cycle are not seen in women with highly restrained eating patterns (Schweiger *et al.*, 1992). This could be due to the effects of low energy intake on hormonal cycles, or because of a tonic inhibition of eating which obscures any underlying physiological influence.

The time of ovulation represents the nadir of food intake during the menstrual cycle (Lyons *et al.*, 1989; Gong *et al.*, 1989; Fong and Kretch, 1993; Johnson *et al.*, 1994). Moreover, it has been suggested that these changes in eating parallel changes in basal metabolic rate across the menstrual cycle (Solomon *et al.*, 1982; Webb, 1986). Specifically energy expenditure has been shown to increase in the postovulatory phase (Webb, 1986). This could be due to separate hormonal action on energy intake and expenditure respectively, or to the driving action of energy intake on some aspect of energy expenditure (such as diet-induced thermogenesis).

One potential explanation for the fluctuations in food intake is alterations to insulin sensitivity during the menstrual cycle. Marsden *et al.* (1996) assessed carbohydrate metabolism and insulin sensitivity at receptor and post-receptor level in women in the follicular or luteal phase of an ovulatory ovarian cycle, in the physiological target organ adipose tissue. They found a reduction in insulin receptor binding in the luteal phase but no change in overall insulin action on adipocyte glucose uptake and lipolysis. The effect of menstrual cycle phase, i.e. of hormone concentrations on insulin binding, is congruent with impaired insulin sensitivity in conditions where concentrations of oestrogen and progesterone are raised either artificially, as in oral contraceptive use, or naturally, as in pregnancy. However, although receptor binding is altered in the luteal phase, compensatory change in the post-binding sites allows overall insulin action in adipocytes to remain normal.

Macronutrient intake

In contrast to cyclic effects on total energy intake, reports about the patterns of macronutrient intake during the menstrual cycle are less consistent (see Table III) and sometimes have achieved celebrity status.

There has been some suggestion of significantly increased carbohydrate consumption premenstrually (Dalvit-McPhillips, 1983; Hrboticky *et al.*, 1989; Lyons *et al.*, 1989; Brzezinski *et al.*, 1990), reductions in protein and carbohydrate intake at ovulation (Lyons *et al.*, 1989), and premenstrual increases in fat intake (Anantharaman-Barr *et al.*, 1988; Tarasuk and Beaton, 1991; Johnson *et al.*, 1994) or in fat and protein intake (Gallant *et al.*, 1987). The increase in carbohydrate consumption has been referred to as carbohydrate craving and has been regarded as an index of a particular type of eating disorder (Wurtman, 1993).

There are however, inconsistencies both between and within studies. Results may differ depending on whether actual intake (in grams) of a macronutrient or the relative proportion that a macronutrient contributes to total percentage energy intake is considered. While absolute intake of a macronutrient may increase significantly from the follicular to luteal phase (e.g. Barr *et al.*, 1995), the percentage of energy intake as, e.g., fat or carbohydrate may fail to differ. The well-publicized view has been that it is carbohydrate intake that increases in the luteal phase (Wurtman *et al.*, 1989). However, there are an equal number of studies which document significant increases in fat intake at this stage of the cycle. Such results may

Table II. Cyclical patterns of energy intake in women

Study	Sample size	Days measured	Energy intake (MJ)				
			Luteal (L)	Menstrual	Follicular (F)	Ovulatory	L versus F
Abraham *et al.* (1981)	23	35 (whole cycle)	nr	nr	nr	nr	L > F*
Anantharaman-Barr *et al.* (1988)	22 – 8 oc	7 days pre/7 days post	nr		nr		ns
	– 14 noc		nr		nr		L > F*
Barr *et al.* (1995)	45 – 29 ovulatory	3×3 days (pre/ovul./post)	3.27		8.01	nr	L > F*
	– 13 anovulatory		7.91		8.21	nr	F > L ns
Brzezinski *et al.* (1990)	17 PMS – meals	1 day pre/1 day post	7.28		6.04		L > F*
	– snacks		3.86		1.80		L > F*
Dalvit (1981)	8	10 days pre/10 days post	8.12		6.02		L > F*
Dalvit-McPhillips (1983)	8	10 days pre/10 days post	7.22		5.22		L > F*
Fong and Kretch (1993)	9	1–28 (whole cycle)	10.46	10.31	9.87	9.39	ns
Gallant *et al.* (1987)	18 –9 control	2×3 days (pre/post)	8.09		6.27		L > F*
	–9 PMS		6.56		5.71		L > F ns
Giannini *et al.* (1985)	20	whole cycle	nr	nr	nr	nr	no cyclical trend
Gong *et al.* (1989)	7	1–28 (whole cycle)	8.54	7.89	7.67	7.39	L > F*
Hill and Blundell (1989)	12 – PMS	whole cycle			nr		L > F*
Hrboticky *et al.* (1985)	8	1 day pre/1 day post	7.72		7.52		L > F
Hrboticky *et al.* (1989)	10	1 day pre/1 day post	nr		nr		L > F*
Johnson *et al.* (1994)	26	1–28 (whole cycle)	7.84	7.6	7.15	7.15	L > F*
Krakow (1992)	98 – 47 noc	whole cycle	8.23	8.64	8.803		F > L ns
	– 47 oc	whole cycle	8.86	8.64	8.58		L > F ns
Lariviere *et al.* (1994)	8	2×1 days (pre/post)	8.29		7.39		L > F*
Lissner *et al.* (1988)	23	10 days pre/10 days post	9.77		9.41		L > F*
Lyons *et al.* (1989)	25	1–28 (whole cycle)	9.13	9.05	8.45	7.87	L > F*
McCoy *et al.* (1988)	12	7 days pre/7 days post	8.95		8.27		L > F*
Manocha *et al.* (1986)	11	10 days pre/10 days post	6.74		5.44		L > F*
Martini *et al.* (1994)	18	3 days pre/3 days post	7.98		7.32		L > F*
Netter *et al.* (1993)	40 – disturbed eaters	9 days pre/9 days post	nr		nr		L > F*
	– non-disturbed		nr		nr		L > F*
Oram (1987)	6	10 days pre/10 days post	11.56		8.74		L > F*
Piers *et al.* (1995)	13	5 days pre/5 days post	7.12		7.12		ns
Pliner and Fleming (1983)	34	1 days pre/1 days post	8.42		7.49		L > F*
Rogers and Jas (1994)	42 – (snacks)	25 L/17 follicular	3.35		1.46		L > F*
Schweiger *et al.* (1992)	21 – 13 unrestrained	12 days pre/12 days post	9.42		9.08		L > F*
	– 9 restrained		7.21		6.99		L > F ns
Sophos *et al.* (1987)	9	7 days pre/7 days post	7.79		7.44		L > F ns
Tarasuk and Beaton (1991)	14	10 days pre/10 days post	8.00		7.62		L > F*
Wurtman *et al.* (1989)	19 – control	1 day pre/1 day post	8.46		8.72		F > L ns
	9 – PMS	1 day pre/1 day post	10.02		7.92		L > F*

*Significant difference between luteal and follicular phases ($P > 0.05$). nr = measured but actual intake not reported; oc = oral contraceptive; noc = no oral contraceptive; PMS = premenstrual syndrome; ns = non-significant.

represent general increases in appetite rather than specific increases in intake of a particular macronutrient.

Methodological issues

All studies on the menstrual cycle are confronted by formidable methodological problems. Studies of food intake and the menstrual cycle often contain some serious methodological flaws or inconsistencies. These include techniques for recording food intake, inadequate determination of menstrual cycle phases, the failure to consider more than two (rather long) cycle phases and the frequent 'averaging' across menstrual cycles. These methodological problems will reduce the sensitivity of experiments and may jeopardize the validity of the conclusions made about the nature of fluctuations in food intake during the menstrual cycle.

One frequent problem concerns those experiments using small numbers of subjects; these studies may not have had sufficient statistical power to detect small effects in motivation or behaviour. This problem is compounded when multiple phases are compared and degrees of freedom reduced. Furthermore, few studies have taken account of menstrual cycle-related symptoms, which are most commonly reported in the premenstrual phase. These symptoms may influence behaviour in an indirect way (in contrast to direct hormonal influence). Consequently, in our view, research on the pattern of eating and motivation accompanying the menstrual cycle should be critically examined and cautiously interpreted.

Premenstrual syndrome and food intake

PMS is characterized by the dramatic occurrence of symptoms in the premenstrual phase, which are relieved following the onset of menstruation. Increases in appetite and/or food cravings are considered characteristic of PMS. A frequent dilemma which arises in studies of PMS is whether PMS is an extreme of the normal experience of the menstrual cycle or is a qualitatively different phenomenon.

Table III. Macronutrient intake (% of energy intake) in the follicular (F) and luteal (L) phases

		Carbohydrate		Fat		Protein	
Study	*n*	F	L	F	L	F	L
Abraham *et al.* (1981)	23	nr*	nr*	nr*	nr*	nr*	nr*
Barr *et al.* (1995)	29 ovulatory	57.5	56.1	28.9	31.3	12.7	12.4
	13 anovulatory	56.3	54.1	30.5	32.9	12.5	13.1
Tarasuk and Beaton (1991)	14	43.8	43.07	36.01	37.56*	14.56	14.12
Dalvit-McPhillips (1983)	8	39.9	55.8*	44.4	32.6*	15.7	11.6
Fong and Kretch (1993)	9	44.2	44.5	41.9	41.7	12.9	13.0
Gallant *et al.* (1987)	9	43.1	40.9	36.6	37.7*	13.9	15.7*
	9 PMS	34.6	40.0	48.8	43.6	15.0	16.0
Johnson *et al.* (1994)	26	47.5	47.9	35.4	37.4*	15.4	14.7
Lyons *et al.* (1989)	18	47.0	45.9	35.8	36.8	13.5	13.4
Oram (1987)	6	46.5	42.2	37.1	38.1	14.5	16.4
Schweiger *et al.* (1992)	9 unrestrained	42.0	43.0	41.0	40.0	13.0	14.0
	9 restrained	45.0	39.0	39.0	40.0	13.0	14.0
Sophos (1987)	14	47.3	46.1	36.4	33.7	15.2	15.1
Martini *et al.* (1994)	18	51.3	50.1	33.1	34.2	15.6	15.6
Hrboticky *et al.* (1989)	8	54.02	54.09	28.03	28.31	13.35	13.4
Brzezinski *et al.* (1990)	17 PMS						
	meals	28.6	32.5*	48.65	48.1*	21.1	17.01
	snacks	27.9	32.8*	52.3	50.5*	17.7	15.2

*Significant difference between luteal and follicular phases ($P > 0.05$). nr = measured but actual intake not reported; PMS = premenstrual syndrome.

In relation to food intake, the former view would imply that PMS sufferers experience similar temporal fluctuations in appetite and intake to 'normal' women but at a greater intensity. There have, however, been few objective studies of intake in PMS sufferers. Two studies report luteal intake to be significantly higher than follicular intake (Hill and Blundell, 1989; Brzezinski *et al.*, 1990), one a non-significant trend in the same direction (Gallant *et al.*, 1987) and one a non-significant reversed effect (Wurtman *et al.*, 1989). Both studies which fail to detect effects (Gallant *et al.*, 1987; Wurtman *et al.*, 1989) are based on far fewer subjects than the studies which confirm the similar pattern of increased luteal energy intake in PMS sufferers as non-symptomatic women. Giannini *et al.* (1985) reported a positive relationship between caloric intake and severity of PMS symptoms. Women who reported more severe symptoms recorded higher caloric intake. However, while caloric intake was measured by daily intake diaries, PMS symptoms were assessed by retrospective questionnaire at the end of the study, a method which tends to exaggerate symptom reporting.

More recently, Both-Orthman *et al.* (1988) found an increase in subjective ratings of appetite in the premenstrual phase as compared with the postmenstrual phase in both PMS and control subjects. Levels of reporting in the premenstrual phase were significantly higher in the PMS subjects than in the controls. Concurrent ratings of mood and depression were significantly correlated with ratings of appetite in the premenstrual phase for the PMS subjects only, suggesting a relationship between mood and appetite which distinguishes this group from the control (non-PMS) group. This coincidence of appetite increase and mood change has led to suggestions that these may be causally linked.

Concerning the intake of specific macronutrients Gallant *et al.* (1987) found that both control and PMS subjects increased carbohydrate intake postmenstrually but consumed more calories, protein, fat and vitamin B_6 premenstrually. Increases in intake for the PMS subjects were smaller than for controls. These data are in contrast to the findings of Dalvit-McPhillips (1983) which show an increase in carbohydrate intake premenstrually. The macronutrient data reported by Dalvit-McPhillips (1983) does not correspond to total energy intakes reported previously from the same women (Dalvit, 1981) and Dalvit's subjects were not reported to be PMS sufferers. Rogers *et al.* (1992) have suggested that foods which are preferentially consumed premenstrually tend to be highly palatable and with high hedonic properties. These foods tend to be high in both carbohydrate and fat. Indeed, the 12% energy increase reported by Hill and Blundell (1989) was mainly due to a rise in the number of high fat/high carbohydrate snacks consumed. It is therefore difficult to draw firm conclusions about specific changes in macronutrient consumption.

PMS, mood and food cravings

Studies of food craving during the menstrual cycle have produced a range of findings about both the pattern and nature of food cravings. The predominant trend, however, seems to be an increase in both the frequency and severity of food craving in the premenstruum. This is clearly depicted in Figure 2, based on data from 5546 women who reported symptoms in a retrospective questionnaire (Dye *et al.*, 1995). Even allowing for some elevation in reporting given the retrospective nature of the study, a clear increase in food craving in the premenstrual phase (compared with postmenstrual) is evident. This pattern is true for severe and moderate food cravings. Interestingly, for mild food cravings the opposite pattern is observed. This suggests that food cravings are more frequent and more severe premenstrually, but they can also occur in a much weaker form at other points in the menstrual cycle in the same women.

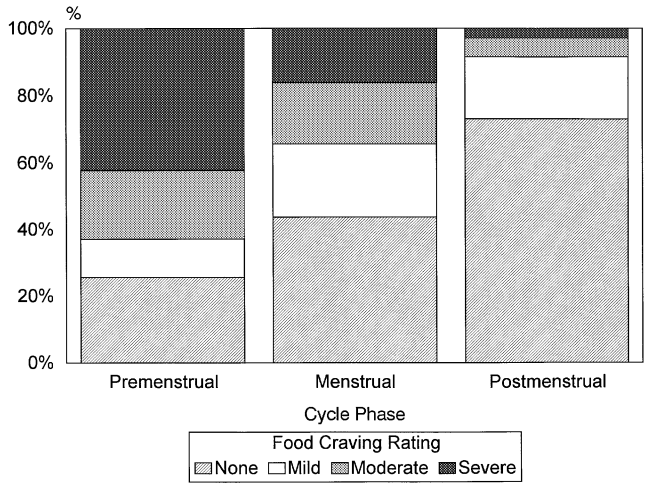

Figure 2. Percentage of subjects reporting food cravings (varying degrees of severity) at three stages of the menstrual cycle. These data show a greater frequency and intensity of cravings in the premenstrual phase. This figure is based on data from a sample of 5546 women (adapted from Dye *et al.*, 1995).

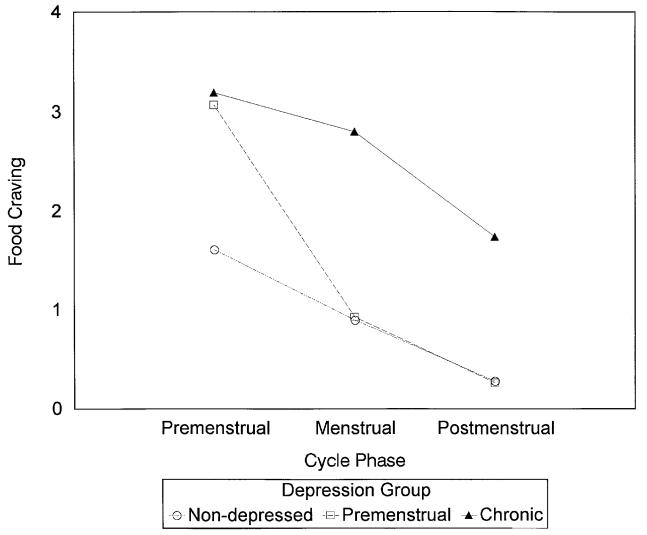

Figure 3. Relationship between the incidence of food cravings and three phases of the menstrual cycle in three groups of women. The graph indicates non-depressed women (square symbols), women with chronic depression (diamonds) and women with depression only during the premenstrual phase (circles). The vertical axis indicates the degree of reported food craving on a scale from 1 (mild) to 4 (severe). (Adapted from Dye *et al.*, 1995.)

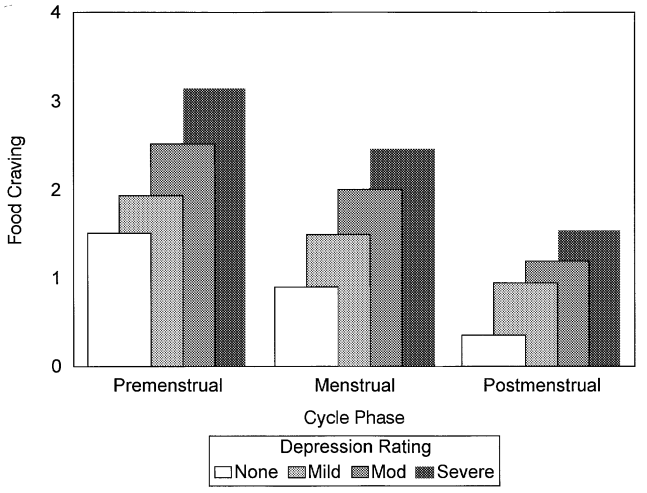

Figure 4. Relationship between the incidence of food cravings and the severity of depression in three phases of the menstrual cycle. The vertical axis indicates the severity of reported food craving on a scale from 1 (mild) to 4 (severe). Bars indicate concurrent rating of depression from none to severe during each of three menstrual cycle phase (horizontal axis). The figure is based on data from a sample of 5546 women (adapted from Dye *et al.*, 1995).

This tendency for increased food craving in the pre-menstrual phase has been observed in varied samples of women (e.g. with and without PMS and/or premenstrual depression), suggesting that the experience of PMS sufferers is more severe but not necessarily qualitatively different from that of normal women. There does not seem to be a difference in the foods which are craved (as opposed to frequency or severity) by PMS or non-PMS subjects. However, it is worth considering closely whether the craved foods belong to any particular nutrient category (e.g. carbohydrates).

This is important because of the known effects of food on mood and the well-documented incidence of mood change (particularly depression) in the premenstrual phase, especially in PMS sufferers. Consequently, a relationship between food craving (possibly for carbohydrate-rich foods) and depression could occur. The logic of this relationship is that women crave those particular foods whose consumption would ameliorate the depression. Some studies have indicated that these two symptoms may co-exist premenstrually (Cohen *et al.*, 1987; Bancroft *et al.*, 1988). A more recent study has clarified the relationship between food craving and depression.

Figure 3 is based on 919 women, selected on the basis of their clear patterns of depression rating during three menstrual cycle phases. From the figure, it is evident that depression is positively related to food craving. In women with 'chronic' depression (i.e. depression reported as severe or very severe in each cycle phase examined) ratings of food craving are elevated to a 'moderate' level at each phase. This contrasts with the low ratings of food craving observed in women who report no depression at any phase of the cycle ('non-depressed' group). This latter group, however, do show a cyclical pattern in their reports of food craving, indicating that a rhythm of craving can occur in the absence of depression, but at a lower level. The pattern of craving shown by those women who reported severe or very severe depression only in the premenstrual phase (a feature which characterizes the PMS) confirms the relationship between food craving and depression. The food cravings of this PMS group are identical to those of the 'chronic depressed' group when their depression ratings are also the same, but the level of craving falls to that of the non-depressed group when depression also falls.

The relationship between food cravings and depression in Figure 3 clearly suggests that these menstrual cycle-related symptoms are not independent. These data, however, are based on approximately one-fifth of the original data, i.e. those women with relatively easily classified temporal patterns of depression ($n = 919$). Figure 4 shows the relationship between depression rating and food craving rating during each phase

of the menstrual cycle for a large sample of unselected women ($n = 5\,546$). A positive linear relationship between depression and food craving is evident in each cycle phase. The more severe the rating of depression, the greater the degree of food craving. The occurrence of more severe food cravings in the premenstrual phase (in contrast to the menstrual and postmenstrual phases) is also clear. The interrelationship between food craving and depression is maintained at all phases of the menstrual cycle and the size of the sample suggests a robust relationship between these symptoms at all phases of the menstrual cycle.

The role of serotonin in food intake and food craving

In recent years, research has identified the neurotransmitter serotonin [also known as 5-hydroxytryptamine (5-HT)] as a particularly important component of the biological system influencing eating (Blundell, 1977). Serotonin is implicated in the effects of dietary composition on brain function (e.g. Fernstrom and Wurtman, 1971) and in the control of meal patterns and the urge to eat (Blundell, 1992). Since serotonin is also implicated in physical and psychological symptoms occurring during the menstrual cycle (and especially in PMS), a consideration of the role of serotonin may help in the understanding of appetite changes related to PMS.

Serotonin has been implicated as the mediating factor in the relationship between mood and appetite (Wurtman, 1993). This hypothesis is based on the evidence that low levels of serotonin induce dysphoric mood. It is argued that craving for particular food products (containing carbohydrates) occurs in order to raise the levels of serotonin in the brain and it has been suggested that this may be an adaptive mechanism to compensate for a relative lack of serotonin premenstrually. Therefore eating CHOs serves as a form of self-medication to raise mood. The mechanisms through which the carbohydrate content of the diet influences the uptake of tryptophan into the brain and in turn increases the synthesis of serotonin has been well described elsewhere (Fernstrom and Wurtman, 1971).

It has been demonstrated (Wurtman *et al.*, 1989) that the deliberate administration of carbohydrate can relieve premenstrual depression in PMS sufferers, defined by retrospective symptom ratings. The consumption of a carbohydrate-rich, protein-poor evening meal improved mood in PMS sufferers in the late luteal phase but had no effect on mood in the follicular phase or in non-symptomatic control subjects. This is consistent with a carbohydrate-induced increase in serotonin occurring during the premenstruum. However, as noted earlier there is little evidence for a selective craving for carbohydrate foods premenstrually or for a preferential intake of carbohydrate. Consequently, the idea that individuals attempt to medicate themselves by eating carbohydrate is not yet substantiated. However, there are other reasons to consider a role for serotonin in premenstrual food craving and increased energy intake.

Ovarian steroids may have a modulating role in the serotonergic system, affecting metabolism, activity and receptors (Rapkin, 1992; Severino, 1994). Animal studies suggest changes in serotonin levels during the oestrus cycle (Biegon *et al.*, 1980; McEwen and Parsons, 1982; Fischette *et al.*, 1984) and in response to oestradiol administration (Kato, 1960). In humans, concentration of serotonin (V_{max}) is lowest premenstrually (Taylor *et al.*, 1984; Tam *et al.*, 1985). There is evidence that serotonin levels in whole-blood, and plasma and platelet uptake and content are lower premenstrually in women with PMS (Rapkin *et al.*, 1987; Ashby *et al.*, 1988, 1992). Similar fluctuations have been observed in melatonin, which is synthesized from serotonin (Parry, 1994).

Neuroendocrine challenge tests have been used to measure changes in serotonin function. Normally, infusion of the serotonin precursor L-tryptophan (or fenfluramine) produces an increase in plasma prolactin. In women with PMS, prolactin responses to a neuroendocrine challenge test are blunted premenstrually (Halbreich, 1990; Bancroft *et al.*, 1991). The neuroendocrine response can be affected by dieting in women (Goodwin *et al.*, 1987), a factor not controlled for in these studies.

The serotonin agonist, buspirone, has a high affinity for 5-HT_{1A} receptors. Yatham *et al.* (1989) found that the prolactin response to a buspirone challenge test was greater during the luteal phase and suggested that this is due to supersensitivity of 5-HT_{1A} receptors at this time. These results were confirmed by Dinan *et al.* (1990) who excluded the possibility of variable absorption during the menstrual cycle. Buspirone has also been reported to have beneficial effects on premenstrual symptoms (David *et al.*, 1987; Rickels *et al.*, 1989), as have other serotoninergic compounds such as fluoxetine (Rickels *et al.*, 1990; Stone *et al.*, 1991; Menkes *et al.*, 1992; Wood *et al.*, 1992). D-Fenfluramine has been shown to be effective in suppressing luteal increases in appetite in PMS sufferers (Hill and Blundell, 1989; Brzezinski *et al.*, 1990). However, these studies conflict in their findings on mood symptoms.

Thus studies in women with PMS show a consistent trend toward decreased levels of serotonin premenstrually (Severino, 1994). A recent study (Ulrich *et al.*, 1994) has demonstrated that central, hypothalamic regulation of follicle stimulating hormone (FSH) and prolactin release involves serotonin, specifically 5-HT_3 receptor-mediated processes. There is also well-documented evidence of serotonin's role in the regulation of the oestrous cycle in rodents (Vitale and Chiocchio, 1993). These data, coupled with evidence of rhythmicity in peripheral serotonin, suggest that while ovarian steroids may modulate the serotonergic system, serotonin also modulates gonadotrophin secretion.

Serotonin is also implicated in the aetiology of depression, and neuroendocrine findings in women are consistent with a higher incidence of depression in women than in men (Meltzer, 1990). However, we cannot infer that peripheral serotonin levels predict central levels in humans, although such a relationship has been demonstrated in non-human primates (Raleigh and McGuire, 1980). On the basis of the finding (McGuire *et al*., 1983) that the serotonin system of the vervet monkey may be affected by environmental factors and social interaction, Rapkin (1992) has developed an appealing theory of physiological deregulation of the serotonin system. This proposes that physiological down-regulation, i.e. reduction in serotonin, may produce negative somatic and behavioural symptoms such as those which characterize PMS, whereas

women without PMS may be able to minimize deregulation via for instance, environmental interaction.

The effects of lowered serotonin are most likely to be observed in those behaviours most closely regulated by the neurotransmitter. It can be suggested that the fluctuation in appetite and energy intake observed in women during the menstrual cycle reflect cyclical rhythms in serotonin, which may be accompanied by affective symptoms in women with PMS but which are present to a lesser degree (or are better tolerated) in women without PMS. Serotonin has been shown to influence both satiation (meal size) and satiety (post-meal inhibition) (Blundell, 1992). During the premenstrual phase, serotonin activity is relatively low, and therefore there will be relatively weaker control over appetite. Indeed it can be deduced that altered activity at particular serotonin receptors (5-HT_{1B} and 5-HT_{2C}) modulates the ability to resist risk factors for overeating and a positive energy balance (Blundell, 1996). Consequently, during the premenstrual phase individuals will be more susceptible to many stimuli (internal and environmental) that facilitate eating and elicit food craving. Thus, the premenstrual phase of the menstrual cycle can be considered a time when women are especially vulnerable to overconsumption and craving, and also to depression (due to low serotonin activity).

Dieting and the menstrual cycle

Most of this discussion has been concerned with the effects of hormonal changes on food intake. It should be borne in mind, however, that food intake also affects hormones, and therefore food intake will affect the rhythm of the menstrual cycle. One major dimension here is the concept of dieting, which may involve ~40% or more of the female population. Dieting can be defined as self-induced attempts to restrict food consumption and to control the pattern of eating. Therefore dieting is likely to influence measured food consumption, food choice and other aspects of eating. Pirke (1987) and Pirke *et al.* (1989) have shown that dieting causes menstrual irregularities. The ovarian cycle was disrupted by either disturbed follicular development, i.e. the lack of development of a dominant follicle and low levels of oestrogen, or by luteal phase defects, i.e. impaired progesterone secretion by the corpus luteum. The former effect results in absolute infertility, the latter in significantly compromised fertility. In addition, episodic luteinizing hormone secretion during the follicular phase was altered by dieting. Three major factors which influence this relationship have been identified: (i) age: younger women are more susceptible to diet-induced menstrual irregularities; (ii) amount of weight loss: the greater the weight loss the higher the likelihood of menstrual irregularities; (iii) the nature of the diet: vegetarian diets affect the cycle more than a non-vegetarian diet, even when both cause the same amount of weight loss.

It follows that any studies on the relationship between hormonal state, food cravings and eating should take into account the prevalence of dieting among women in the study sample. This is particularly important in studies on PMS since it is known that a large proportion of PMS sufferers are concerned about their weight and shape and are likely to be dieting. Given the increased susceptibility to risk factors for overconsumption likely to be present just before menstruation, dieting will be even more difficult to sustain during this period and there are likely to be more frequent occurrences of counter-regulation (Polivy and Herman, 1985).

To date, however, these hypotheses remain untested because the prevalence of PMS in dieters, the ease of adherence to a diet during the premenstrual phase and the effect of dieting on premenstrual symptoms have not been studied. This is an area which merits further research.

Since this review concerns the relationship between appetite control and hormonal conditions linked with reproductive function, it is worth drawing attention to a possible role for the ob-protein ('leptin') (Halaas *et al.*, 1995). It has been proposed that leptin serves as a signal linking adipose tissue to central neural pathways. In animals and humans the plasma concentrations of leptin correlate well with body mass index (BMI) and percentage body fat. Consequently, this ob-protein could be the much sought after lipostatic factor. In addition, it has been demonstrated that injections of leptin can restore fertility in mutant mice (ob/ob) which have difficulty reproducing (Chehab *et al.*, 1996). Injections of leptin also cause an early onset of reproductive function in normal female mice (Chehab *et al.*, 1997). These effects may occur because leptin signals the amount of body fat and because reproductive function is normally switched off when levels of body fat fall too low. In humans it is known that leptin levels are very low in patients with anorexia nervosa (Grinspoon *et al.*, 1996) and that female athletes with amenorrhea fail to display a diurnal rhythm of leptin (Laughlin and Yen, 1997). Although the role of leptin in appetite control in humans remains to be verified, some interaction between leptin, serotonin and reproductive hormones could shed further light on appetite fluctuations within the menstrual cycle. However, at the moment this idea should be considered speculative.

Conclusions

It is clear that PMS is a clinical phenomenon with implications for health and well-being. However, considerable methodological problems face researchers who investigate PMS and other phenomena associated with the menstrual cycle. A wide variety of characteristics and events have been attributed to the rhythm of the menstrual cycle and to the premenstrual phase in particular. Definitive statements cannot yet be made about certain issues. However, we feel that the following general conclusions can be drawn from the published body of research; (i) Orderly fluctuations in eating and other measures of food consumption do occur during the menstrual cycle. Generally there is an increase in energy intake and appetite during the pre-menstrual phase (when compared to the ovulatory or postmenstrual phases); (ii) This increase in energy intake and appetite during the premenstrual phase also occurs in women who suffer from PMS. In these women the appetite changes may be experienced with greater intensity and given increased psychological emphasis; (iii) During the premenstrual phase food cravings are increased in frequency and intensity. Food

craving appears to be more severe in women with PMS; (iv) Food craving is positively related to depression during the menstrual cycle. Food craving is more intense when depression is severe, and depression is greater during the premenstrual phase. Food craving is linked to depression in women with PMS; (v) These changes in appetite and cravings indicate the probability of active or passive overconsumption of food during the premenstrual phase and in women with PMS. In turn, this suggests a potential for weight gain; (vi) Changes in serotonin metabolism are implicated in the vulnerability to risk factors, increased food consumption, food craving and depression during the premenstrual phase. These phenomena could be treated by behavioural, nutritional or pharmaceutical strategies designed to influence the synthesis and/or synaptic activity of serotonin.

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