

BOWEL FUNCTION, FOOD INTAKE AND THE MENSTRUAL CYCLE

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INTRODUCTION

Menstruation occurs in regular cycles from menarche to the menopause and is an external, visible sign of a multitude of internal, invisible processes involved with the potential for the human female to reproduce. The inter-related ovarian, pituitary and uterine cycles are associated with widespread biological, psychological and behavioural changes. In recent years attention has turned to the possibility of cyclical changes in food intake and bowel function following women's complaints of adverse changes, particularly premenstrually.

The pattern of change in bowel habit most frequently described by women is premenstrual constipation and/or diarrhoea with the bleed. The explanations proposed are that the raised concentration of progesterone in the luteal phase reduces gastrointestinal motility, and the release of uterine prostaglandins at the start of menses stimulates the muscles of the gut to contract, thus increasing motility. Such cyclical changes, if present, have important practical implications.

The menstrual cycle would appear to modify food intake, for women have reported an increase in appetite and/or a craving for sweet food premenstrually. A relationship between the cycling of the ovarian hormones and patterns of food consumption has been demonstrated in several animals. As for bowel habit, if a cyclical change in food intake does occur, this could be of significant practical importance. It would also raise the possibility of a causal relationship between cyclical food intake and cyclical bowel habit.

The purpose of this review is to evaluate the current evidence available on menstrual cycle related changes in food intake and bowel function, looking critically at the study methods employed. The likely underlying hormonal involvement is considered and a link between cyclical bowel habit and food intake is proposed. The significance of the findings is discussed.

MENSTRUAL CYCLE ENDOCRINOLOGY

Each menstrual cycle lasts approximately 28 days and reflects a complex interaction of hormones which regulate an ovarian cycle leading to the release of a mature oocyte into one of the oviducts, and a uterine cycle that prepares the womb for pregnancy.

For descriptive purposes the menstrual cycle may be divided into the follicular phase, characterized by follicular development and eventual rupture (ovulation), and the luteal phase which culminates in the onset of menstruation. During the last phase of follicular growth there is an exponential rise in the secretion of oestradiol in the peripheral circulation reaching a peak, on average, 1 d prior to ovulation. When the concentration of plasma oestradiol reaches a threshold level for at least 50 h there is an increase in the pulsatile release of luteinizing hormone (LH). The resultant surge in the concentration of LH initiates the processes which lead to ovulation. The surge of LH begins 32 ± 6 h before ovulation and peaks about 15 h later. During the peri-ovulatory period, the concentration of follicle-stimulating hormone changes in a similar manner to that of LH. As the corpus luteum forms it produces increasing amounts of progesterone, as well as more oestradiol. The concentration of progesterone shows a small increase on the day of the LH surge, followed by a substantial increase 24 h later. The maximum value is observed around the day of LH peak + 6 d. The peri-ovulatory changes in the secretion of ovarian steroids lead to a rise in basal body temperature. Levels of progesterone and oestradiol fall towards the end of the luteal phase leading to an increase in the secretion of follicle-stimulating hormone.

BOWEL FUNCTION AND THE MENSTRUAL CYCLE

SUBJECTIVE VIEW

In the 1950s, when interest was focused on the premenstrual syndrome, the first reports of changes in bowel habit around menstruation were made (Ferguson & Vermillion, 1957). Since then, further surveys have confirmed the findings of women's awareness of altered bowel function with the menstrual cycle (Sutherland & Stewart, 1965; Rees & Rhodes, 1976; Hinds *et al.* 1989; Whitehead *et al.* 1990). In all these surveys the majority of women describe constipation premenstrually and/or diarrhoea during menses. The reported incidence of constipation premenstrually ranges from 16% to 32% and that of diarrhoea at menses from 19% to as high as 72% of the population studied. The diarrhoea would appear to have a distinct start on the first day of menses and last for 24–48 h. The duration of premenstrual constipation is possibly longer, 2–7 d.

There are also young women with severe chronic constipation who report that their only 'normal' or spontaneous bowel actions occur during menses (Rees & Rhodes, 1976; Preston & Lennard-Jones, 1986) or that they find defecation easier at the time of menstruation (Read *et al.* 1986; Waldron *et al.* 1988).

OBJECTIVE EVIDENCE

Decreased luteal phase gut motility

Pregnancy and the progesterone hypothesis. Gastrointestinal dysfunction has frequently been described during pregnancy. Pregnant women have been noted to complain of heartburn, bloating, abdominal distension and constipation (Winship, 1975). Obviously there are mechanical alterations occurring with enlargement of the uterus, but it is now believed that the gastrointestinal problems are in part a reflection of a generalized relaxation of smooth muscles affecting also the uterus, ureters and blood vessels under the influence of the ovarian hormones, especially progesterone. Serum progesterone levels rise markedly throughout pregnancy. It has been speculated that if the relationship between gastrointestinal hypomotility and serum progesterone levels in pregnancy could be clarified, this might also explain the perceived constipation of the luteal phase of the menstrual cycle.

The subjective complaints of pregnant women have been mostly supported by objective measurement. Lower oesophageal sphincter pressure and symptomatic oesophageal reflux have been recorded with increased frequency in pregnancy (Evans & Bonslog, 1950; Nagler & Spiro, 1961; Lind *et al.* 1968; Van Thiel *et al.* 1977; Fisher *et al.* 1978*a*). Also, in women who experienced heartburn there was clear evidence of impaired gastric emptying (Davison *et al.* 1970).

A reduction in gastric tone and motility would be expected to slow gastric emptying, and slowing emptying contributes to heartburn. Generally, though, no delay in gastric emptying has been shown with pregnancy (Davison *et al.* 1970; Hunt & Murray, 1978). Certainly the small intestine is affected by pregnancy. Prolonged transit times in pregnant women compared to non-pregnant controls (Parry *et al.* 1971), and in the third (Wald *et al.* 1982; Lawson *et al.* 1985) and second trimester compared to *post partum*, have been reported. The increment in transit time is greatest as the serum progesterone levels increase from < 1 to 80 ng/ml. There is no further increase from 80 to 230 ng/ml. Thus the major increase in transit time occurs over the rise in progesterone also experienced during the menstrual cycle i.e. < 1 ng/ml during the follicular phase to 20 ng/ml during the plateau of the luteal phase. Small intestinal transit time was found not to be prolonged during the

first trimester. First trimester and postpartum progesterone levels were similar to those observed in the follicular phase of the menstrual cycle (Lawson *et al.* 1985). The studies of Wald *et al.* (1982) and Lawson *et al.* (1985) measured oro-caecal transit time. It is possible, therefore, that delayed gastric emptying was involved. However, this is thought unlikely due to the use of an isotonic liquid test meal specifically designed to minimize any role of delayed gastric emptying (Wald *et al.* 1982) and the lack of evidence of delayed gastric emptying in pregnancy.

The gall bladder appears equally affected by pregnancy; decreased rates of emptying, increased volume and stasis have all been recorded (Gerdes & Boyden, 1938, Levyn *et al.* 1933; Braverman *et al.* 1980; Kern *et al.* 1981; Everson *et al.* 1982; Ylöstalo *et al.* 1982). It is interesting that the major increase in gall bladder volume was found to occur as progesterone levels increased from < 1 to 80 ng/ml (Everson *et al.* 1982) as with the small intestine transit rate increase observed by Lawson *et al.* (1985).

Although constipation is a very common complaint of pregnancy only one study found considered the effect on the colon. An increased colonic absorptive capacity for water and sodium, which is suggestive of diminished colonic motility during pregnancy, has been described (Parry *et al.* 1970).

Testing the progesterone hypothesis. By studying contraceptive steroids in non-pregnant volunteers, the effects of progesterone can be assessed without the compounding feature of an enlarging uterus and the disruption of normal anatomic relationships. Hutson *et al.* (1989) concluded that sex hormones have an inhibitory effect on gastric emptying when they found slower emptying of liquids in postmenopausal women taking oestrogen and progesterone, and premenopausal women, when compared with men. Gender-related differences in gastric emptying, suggestive of a female sex hormone inhibitory effect, have also been shown to exist (Datz *et al.* 1987).

Lower oesophageal sphincter pressure was reduced in the phase of the pill cycle with the progestagen dimethisterone and ethinyl oestradiol, and returned to normal in the ethinyl oestradiol-only phase and menses when no medication was taken (Van Thiel *et al.* 1976). This phenomenon would indicate that it is progesterone, as opposed to oestrogen, which exerts the smooth muscle relaxation effect. Strangely, contraceptive steroids appear to have little effect on gall bladder contraction (Kern *et al.* 1982; Everson *et al.* 1983).

In vivo studies in non-pregnant animals have confirmed that progesterone has gastrointestinal relaxation properties. Lower oesophageal sphincter pressure was reduced with progesterone administration in oestrogen primed opossums (Schulze & Christensen, 1977). There was reduced small intestine motility in normal female rats as compared with castrated females (Scott & De Flora, 1983), and chronic treatment with oestradiol and progesterone in male rats significantly slowed whole gastrointestinal transit in comparison with controls (Ganiban *et al.* 1985). These studies failed to differentiate between the possible effects of oestrogen and progesterone. However, Ryan & Bhojwani (1985, 1986), when studying colonic transit, demonstrated that it is progesterone which has the major effect. After ovariectomy in rats, colonic transit increased. The administration of oestradiol and progesterone, but especially progesterone, then inhibited transit. Further, decreased colonic transit was found during pro-oestrus/oestrus in rats when oestrogen and progesterone are elevated as compared to metoestrus/dioestrus when oestrogen alone is elevated.

In vitro studies add additional weight to the theory of a progesterone effect. Reduced contractile activity of rat oesophageal tissue after progesterone (Bruce & Behsudi, 1979) and decreased opossum lower oesophageal muscle responses to gastrin and acetylcholine after 17β -oestradiol and/or progesterone (Fisher *et al.* 1978*b*) have been demonstrated. Progesterone decreased the propagation velocity of gastric slow waves, which can be

expected to exert some effect on the gastric emptying of solids (Milenov, 1976), and reduced the contractile activity of gastroduodenal junctional tissue and antral gastric smooth muscle of the rat (Bruce *et al.* 1978; Bruce & Behsudi, 1979). Progesterone treatment *in vivo*, before *in vitro* study, produced blood levels of progesterone within the range of levels attained during the peak of the female oestrous cycle, implying that physiological concentrations of progesterone may significantly influence gastrointestinal function (Bruce & Behsudi, 1979).

The only study with excised human tissue also demonstrated inhibition of stomach and colonic smooth muscle activity after progesterone administration (Kumar, 1962). Inhibition of dog colonic (Gill *et al.* 1985) and guineapig gall bladder smooth muscle (Ryan & Pellecchia, 1982) motility has also been shown. The work of Kumar (1962) failed to show any effect after oestrogen administration. Other work, however, indicates that oestrogen may even stimulate small intestine and colonic tissues (Bruce & Behsudi, 1980, 1981; Ryan & Bhojwani, 1986).

Progesterone inhibition of motilin. It is not known whether the gastrointestinal muscle relaxing effects of progesterone are direct or mediated *via* the action of a local hormone. One interesting proposal is that progesterone causes inhibition of the stimulatory peptide motilin. Intravenous infusions of motilin, adjusted to produce a rise in plasma concentration which lies within the physiological range, have been shown greatly to accentuate gastric emptying (Christofides *et al.* 1979); other actions include stimulation of the electrical and mechanical activities of the small bowel (Vantrappen *et al.* 1979) and colon (Rennie *et al.* 1980) and emptying of the gall bladder (Adrian *et al.* 1980; Itoh & Takahashi, 1981).

Mean plasma motilin concentrations are significantly reduced during the second and third trimesters of pregnancy, returning to normal *post partum*. This change is possibly progesterone mediated and may in part be responsible for pregnancy associated gastrointestinal hypomotility (Christofides *et al.* 1982).

Luteal phase evidence. There is now some evidence that the effect of progesterone as a smooth muscle relaxant responsible for intestinal hypomotility is not limited to the pregnant state; the increase that occurs during the luteal phase of the menstrual cycle seems sufficient to produce changes. Studies reporting changes in bowel function associated with constipation-type symptoms in the luteal phase are shown in Table 1. The picture, however, is not clearcut and much conflicting information exists.

The few studies conducted on oesophageal contractions, lower oesophageal sphincter pressure (Nelson *et al.* 1984) and gall bladder emptying (Nilsson & Stattin, 1967; Everson *et al.* 1982) have failed to show any relationship to the menstrual cycle.

The evidence for an effect on gastric emptying rates is confused. Gill *et al.* (1987) demonstrated decreased solid emptying rates on luteal days 18–20 when compared with follicular days 8–10. The impairment was correlated with elevated serum progesterone levels, yet when liquid emptying was studied no phase difference was found. In an earlier study of similar design, concerned with the same cycle days and monitoring also solids and liquids, Horowitz *et al.* (1985) concluded that there was no phase change. In the earliest study of gastric emptying through the menstrual cycle, MacDonald (1956) found that the rate was highest at the time ovulation. These results, however, should be interpreted with caution. Every cycle was reduced to a scale of 28 units. No confirmation is given that account was taken of the fact that luteal phase length is relatively consistent, at ~ 14 d, and it is the follicular phase that is usually responsible for the variation between individuals. If phase length was considered even, data could have been placed in the wrong phase when standardized. Notivol *et al.* (1984) found faster emptying rates during days 8–18 in comparison with days 19–28 and 1–7. This could possibly be attributed to a progesterone

Table 1. *Reported constipation-type changes in bowel function associated with the luteal phase*

Source	Subjects		Bowel function parameter measured				Changes in bowel function
	No.	Age (years), range or mean and SD	Transit time	Stool frequency	Stool weight	Faecal form	
Wald <i>et al.</i> (1981)	15	24–39	Orocaecal	—	—	—	Significantly longer transit time
Davies <i>et al.</i> (1986)	17 non-luteal 6 luteal phase	5–71	Whole gut	✓	Wet	✓	Significantly longer transit time, harder more well formed stools in luteal phase
McBurney (1991)	10	24 ± 0.7	Orocaecal	—	Wet and dry	—	Significantly lower stool wet and dry weights.

effect if the majority of women were studied toward day 8 as opposed to day 18 or their cycles were longer than 28 d, such that progesterone levels were lower during the period day 8–18 than the other two phases. Progesterone levels are likely to have been high over days 19–28 and could still have been significant, although falling markedly, over the bleed. No assessment of hormonal status was made, and no finer details given as to when measurements were taken in the individual women, to enable more concise conclusions to be drawn. Again, in a very similar study covering the same cycle days, Jonderko (1989) found a tendency towards faster emptying during days 8–18, although the results were not significant statistically.

Considering effects further down the gastrointestinal tract, oro-caecal transit time was longer in luteal days 18–20 than in follicular days 8–10 (Wald *et al.* 1981) and whole gut transit time was found to be longer in women in the luteal phase than in those in other menstrual phases or who were postmenopausal (Davies *et al.* 1986). Certain stool features too have been reported to show cyclical changes, with stools from luteal phase women being harder and more well formed than those from women in other endocrinological states (Davies *et al.* 1986) and stool weights being significantly less over days 18–21, possibly because of the coexisting reduced starch malabsorption, than in days 8–11 (McBurney, 1991).

Other workers, however, have produced contradictory results and some (e.g. Hinds *et al.* 1989; Kamm *et al.* 1989; Turnbull *et al.* 1989) have drawn the conclusion that either the magnitude of the hormonal change is insufficient to influence motor activity, or the effect is so small that it is not clinically significant and the methods used were not sensitive enough to detect these minimal differences in the number of women studied. No luteal change in oro-caecal (Turnbull *et al.* 1989; McBurney, 1991), colonic (Hinds *et al.* 1989) or whole gut (Wyman *et al.* 1978; Marlett *et al.* 1981; Bisdee *et al.* 1989; Kamm *et al.* 1989; Vlitos, 1994) transit time has been detected by certain centres. With regard to stools collected, no luteal change in stool form (Kamm *et al.* 1989; Vlitos, 1994), weight (Wyman *et al.* 1978; Davies *et al.* 1986; Bisdee *et al.* 1989; Kamm *et al.* 1989; Vlitos, 1994), water content, volume or size (Wyman *et al.* 1978) was reported.

Table 2. *Reported diarrhoea – type changes in bowel function associated with the bleed*

Source	Subjects		Bowel function parameter measured				Changes in bowel function
	No.	Age range (years)	Transit time (whole gut)	Stool frequency	Stool wet weight	Faecal form	
McCance & Pickles (1960)	98	—	—	✓	—	—	Maximum stool frequency on day 1
Heitkemper <i>et al.</i> (1988)	34	19–37	—	✓	—	✓	Significantly looser stools
Kamm <i>et al.</i> (1989)	18	22–47	✓	✓	✓	✓	No significant changes, but tendency for looser stools
Davies <i>et al.</i> (1993)	25	22–37	—	✓	—	✓	Significant increase in stool frequency and significantly looser stools compared with luteal phase
Jackson <i>et al.</i> (1994)	20	18–39	—	✓	—	✓	Significantly looser stools compared with luteal phase
Vlitos (1994)	22	19–40	✓	✓	✓	✓	Significantly shorter transit time on days 1 and 2 compared with luteal phase

Frequency of defecation, in all studies to date, has been found to exhibit no luteal decrease (Wyman *et al.* 1978; Davies *et al.* 1986; Heitkemper *et al.* 1988; Kamm *et al.* 1989; Jackson *et al.* 1994). This is perhaps not surprising as frequency shows little correlation with other parameters of bowel function, and it remains possible that other features such as gut transit time experience a progesterone induced change without a corresponding alteration in defecation frequency.

Summary. There is clearly some evidence of reduced motility in the luteal phase, resulting in altered stool properties. This complements many women’s experiences of constipation premenstrually. It is probable that these changes are mediated *via* elevated serum progesterone levels, which appear to have gastrointestinal smooth muscle relaxing properties, although the mechanism of action has not been elucidated. Certainly animal work has shown progesterone to be a significant gastrointestinal tissue relaxant, even at levels experienced normally by female animals, and during human pregnancy rising plasma concentrations have been correlated with gut hypomotility and resultant symptoms. Results from studies during the menstrual cycle, though, are not conclusive. Many workers have been unable to detect luteal phase changes, and have suggested that the levels of progesterone circulating over this period are insufficient to produce a measurable and clinically significant effect.

Increased bleed gut motility

The evidence. In recent years, interest in the possibility of increased motility of the gastrointestinal tract during menses has grown. Studies reporting changes in bowel function associated with diarrhoea-type symptoms during the bleed are shown in Table 2.

Intra-gastric pressure amplitudes and frequencies have been found to be higher at menses (days 1–2) than during the follicular phase (days 7–9) (Heitkemper *et al.* 1990). Leeds *et al.* (1982) and Davies *et al.* (1986) made similar observations of the retention of marker pellets on the day prior to menstruation, with the release of large numbers on the first and/or second day of the bleed, in individuals in their studies on diet and whole gut transit time.

When McCance & Pickles (1960) re-examined data on bowel actions, collected in 1937 but originally unpublished, defecation frequency was found to be maximized on the first day of menses. Davies *et al.* (1993) confirmed that bleed (days 1–4) defecation frequency is significantly greater than premenstrual (days –1 to –4). The stools produced at menses also appear looser in form than those produced premenstrually (Davies *et al.* 1993) and during the other menstrual phases (Heitkemper *et al.* 1988; Kamm *et al.* 1989; Jackson *et al.* 1994). Half of the constipated patients studied by Turnbull *et al.* (1989) noted an improvement in their symptoms, comprising a softening of their stools, during menstruation. The change lasted 2–7 d.

The work reviewed so far helps to substantiate, or possibly explain, the reports from women of diarrhoea during their bleed. However, as with the proposed constipating effects of progesterone premenstrually, the evidence is not all confirmatory. A number of investigators have failed to find menses-related effects on bowel function. Lack of a bleed change in whole gut transit time (Wyman *et al.* 1978), defecation frequency (Wyman *et al.* 1978; Heitkemper *et al.* 1988; Kamm *et al.* 1989; Heitkemper & Jarrett, 1992; Jackson *et al.* 1994; Vlitos, 1994), stool weight (Wyman *et al.* 1978; Vlitos, 1994), form (Heitkemper & Jarrett, 1992; Vlitos, 1994), water content, volume and size (Wyman *et al.* 1978) have all been reported.

It is possible that subject expectation, in part, explains the conflicting results. The weight of evidence for a bleed effect is found in studies of faecal form where the subjects have not been blind to the field of investigation and have rated the stools themselves (Heitkemper *et al.* 1988; Kamm *et al.* 1989; Davies *et al.* 1993; Jackson *et al.* 1994). However, a further consideration is the length of the study period. In all studies in which significant bleed changes were detected, the period of study was, at maximum, the first 4 d of the bleed (note that the design of the study of Turnbull *et al.* (1989) was a little different in that no set time period was analysed and the observations of women during their bleed are simply stated). All the studies cited in which no bleed associated alterations have been found, present a 5–8 d bleed mean (except Heitkemper *et al.* (1988) who analysed a 2 d frequency mean and found no significant bleed effect, perhaps because frequency shows little correlation with other features of bowel habit and therefore has different determinants). If symptoms are experienced for a very limited period, i.e. just the first day or two, then a longer bleed mean may hide any change, particularly if there is a compensatory change in the remainder of the bleed. In support of this theory, Vlitos (1994) could detect no significant bleed change when considering a 5 d mean; yet when only looking at the first 2 d, mean transit time was found to be significantly shorter by a mean of 10%.

The prostaglandin hypothesis. The menses-related changes in gastrointestinal function have been ascribed to elevations in tissue prostaglandin levels (Ylikorkala & Dawood, 1978). The onset of menstruation is associated with a rise of uterine prostanoids, e.g. PGE₂ and particularly PGF_{2α} (Rees *et al.* 1984), which evoke a cluster of changes including uterine cramping. These uterine prostanoids, and especially PGF_{2α}, have been shown to have powerful stimulatory effects on both the motor and secretory activity of the gut (Bennet, 1972; Thor *et al.* 1985). There is therefore the suggestion that these uterine prostanoids might exert gastrointestinal actions during menstruation, if they escape local degradation. Prostaglandins are rapidly metabolized, and it is not yet known whether those released in the uterus are carried by the circulation to adjacent gastrointestinal smooth muscle in sufficient quantities to stimulate contractions.

The experiences of dysmenorrhoeic women support the idea of prostaglandin involvement in gastrointestinal dysfunction at menses. It is suggested that dysmenorrhoea is due to the hyperactivity of the myometrium and/or reduced blood flow to the endometrium, and may be due to excessive prostaglandin production (Gonzalez, 1980). More dysmenorrhoeic than

non-dysmenorrhoeic women have a history of menses-related gastrointestinal symptoms (Chan & Hill, 1978; Shaver *et al.* 1987; Heitkemper *et al.* 1988).

There is also evidence for exacerbation of the irritable bowel syndrome (IBS) during menses. IBS patients are significantly more likely to report a bleed-related worsening of bowel symptoms than non-IBS controls (Whitehead *et al.* 1990; Heitkemper & Jarrett, 1992). The former authors showed differences in group reports that cannot be explained by a neurotic tendency, or changes in affect or behaviour within the menstrual cycle; and therefore concluded that the differences have a physiological basis and are a reflection of the hyperactivity of colonic smooth muscle in IBS sufferers in response to a variety of stimuli, including perhaps to the prostaglandins released during menstruation.

FOOD INTAKE AND THE MENSTRUAL CYCLE

SUBJECTIVE VIEW

Work as early as the 1950s records objectively hypoglycaemia in the premenstruum manifested clinically by increased appetite and/or a craving for sweets (Morton, 1950). Later surveys have consistently reported subjective, qualitative perceptions of desires for sweets and chocolates, and increased appetite during the premenstrual phase (Morton *et al.* 1953; Ferguson & Vermillion, 1957; Friedman & Jaffe, 1985). In the largest survey of 384 women, enlisted through multiple personal contacts, 58% indicated increased appetite and 61% an increased desire for sweets premenstrually on a questionnaire dealing with 147 items (Friedman & Jaffe, 1985). In a slightly smaller survey of 249 inmates of prison or reformatory, 23% acknowledged appetite increases and 37% a craving for sweets premenstrually on a self-rating scale of 21 items (Morton *et al.* 1953). In the light of such survey results, food cravings and changes in food intake are often considered to be symptoms of the premenstrual syndrome (Abraham, 1984; Bancroft & Bäckström, 1985).

OBJECTIVE EVIDENCE

Energy intake

There is a strong body of evidence suggesting that energy intake increases significantly during the luteal phase of the cycle (Table 3). However, assessment of energy intake at specified times in the menstrual cycle has been problematic.

One fundamental difficulty to be overcome when studying the menstrual cycle is the variation that occurs in cycle length, both between different women and within an individual. Pliner & Fleming (1983), in one of the earliest studies, measured food intake *via* 24 h recall on one mid-follicular and one mid-luteal day and found that energy intake was significantly greater in the luteal phase. In order to determine the correct dates for the two laboratory sessions, the experimenter contacted each subject by telephone several times per week after initial recruitment in order to ascertain the date of onset of menstruation. The post-ovulatory, luteal phase was assumed to extend over 14 d. The length of the follicular phase was determined by subtracting 14 d from the subject's estimate of her cycle length.

The majority of investigators have employed the crude division of the menstrual cycle into the 10 d before and the 10 d after onset of menses, leaving a varying number of disregarded days from the centre of the cycle. With the exception of Lyons *et al.* (1989), all found energy intake to be significantly greater during the luteal than the follicular phase (Dalvit, 1981; Manocha *et al.* 1986; Oram, 1987; Lissner *et al.* 1988; Tarasuk & Beaton, 1991). The observed mean difference in intake between the phases ranged from 87 to

Table 3. Reported significant increases in energy intake during the luteal phase

Source	Country	Subjects		Dietary assessment
		No.	Age (years), range or mean	
Dalvit (1981)	USA	8	18–22	24 h recall
Pliner & Fleming (1983)	Canada	34	18–41	24 h recall
Manocha <i>et al.</i> (1986)	India	11	22–30	Food diary with estimated weights
Gallant <i>et al.</i> (1987)	USA	10	18–45	Food diary with estimated weights
Oram (1987)	UK	6	18–22	Food diary with weighed intakes
Lissner <i>et al.</i> (1988)	USA	23	22–41	Food diary with weighed intakes and bomb calorimetry of food samples
Bäumli (1989)	Germany	8	32.7	Food diary with estimated weights
Gong <i>et al.</i> (1989)	USA	7	24–43	Taped record of food weights
Tarasuk & Beaton (1991)	Canada	14	20–47	Food diary with estimated weights
Davies <i>et al.</i> (1993)	UK	25	22–37	Food diary with estimated weights
Johnson <i>et al.</i> (1994)	USA	26	22–39	Food diary with estimated weights
Martini <i>et al.</i> (1994)	USA	18	20–34	Food diary with estimated weights
Barr <i>et al.</i> (1995)	Canada	29	20–40	Food diary with estimated/weighed intakes

674 kcal/d. Expressed as the percentage increase over mean intake during the follicular phase, recorded luteal phase increases ranged from 4% to 35%.

Gallant *et al.* (1987) found that mean energy intake was higher during the 3 d premenstrual interval than during the 3 d following the cessation of menstruation. Oram (1987) recorded a difference of 803 kcal/d in energy intake between the luteal days –10 to –1 and the post-bleed follicular days 5–14. This was a greater difference than that measured between the luteal days and bleed days 1–10. Martini *et al.* (1994) likewise found a significant increase in energy intake in the mid-luteal phase, 7–9 days after ovulation, when compared with the mid-follicular phase 7–9 d after menstrual onset.

Two studies have been conducted which quantify more precisely the relationship between the menstrual cycle and energy intake, by examining food consumption over a larger number of phases (Gong *et al.* 1989; Lyons *et al.* 1989). This was achieved by measuring a second marker of cycle phase, the time of ovulation, by either daily body temperature or urine LH. Both studies divided the cycle into the intervals (a) menses (day 1–4), (b) post-menses (day 5 to ovulatory), and (c) ovulatory (2 d at ovulation \pm 1). Gong *et al.* considered a fourth period, the luteal (ovulatory to –1), and Lyons *et al.* split the luteal phase into two intervals, the post-ovulatory (4 d post-ovulatory) and pre-menses (post-ovulatory to –1). Both groups found that energy intake appeared lowest at ovulation. A significant difference was found between intake at ovulation and the post-ovulatory and premenstrual phases (Lyons *et al.*), and likewise the luteal phase (Gong *et al.*). Lyons *et al.* also found ovulatory intake significantly lower than during the bleed; however, Gong *et al.* were unable to confirm this. Energy intake was higher in the luteal phase than in the bleed or post-menses phase, but was only significantly higher than the latter (Gong *et al.*). The greatest difference in energy intake was found between the ovulatory and luteal phase (274 kcal/d; Gong *et al.*), or more specifically between the ovulatory and post-ovulatory 4 d (324 kcal/d; Lyons *et al.*). To emphasize the magnitude of the ovulatory reduction in food intake Lyons *et al.* stressed that it was 2-fold greater than the increase of 152 kcal/d observed at weekends. Weekend energy increases have been reported by many nutritionists and have led to the widely accepted recommendation that food intake studies include at least one weekend day. It would appear that the phase of the menstrual cycle may be more

important and should be considered when food intake in women of reproductive age is assessed.

Instead of dividing the cycle into intervals, an alternative method for analysing periodic changes has been to utilize data from the whole cycle and fit it to specific mathematical functions. Baüml (1989) has shown that food intake follows a basic sine wave that reaches its maximum ~ 4 d before menses. Tarasuk & Beaton (1991) found that a sinusoidal regression curve was able to explain 14% of the variance in energy intake across 14 premenstrual and 14 post-menstrual d. The assumption of a simple sine wave, reaching its maximum ~ 4 d before menses, is consistent with the quadratic function fitted by Abraham *et al.* (1981) and Johnson *et al.* (1994) and with data from the studies by phase: a higher energy intake in the 10 d pre-menses in comparison to 10 d post-onset (Dalvit, 1981; Manocha *et al.* 1986; Oram, 1987; Lissner *et al.* 1988; Tarasuk & Beaton, 1991); higher energy consumption before menses as opposed to after menses (Oram, 1987; Gong *et al.* 1989); and higher energy intake at menses as compared with ovulation (Lyons *et al.* 1989).

Lissner *et al.* (1988) described two, rather than one, peaks of energy intake. The major peak was in the luteal phase, with a minor one mid-way through the follicular phase. The application of a mathematical modeling technique known as spectral analysis to these data revealed two periodic effects per cycle. The first corresponds to the sine curve reported by others. The second fluctuation in energy intake, Lissner *et al.* suggested, might simply represent a rebound or resonance following the first, larger fluctuation. That is, as a consequence of their depressed intake during the menstrual interval subjects might have overcompensated, producing a second peak.

It is important to bear in mind that time series analyses are often blunted by the need to pool data in one way or another. Even the fitting of sine curves imposes a model that has a regular, evenly spaced pattern of change in the pre- and post-menstrual periods. It is quite conceivable that the true patterns of deviation in energy intake are not equidistant in timing.

Certainly the body of evidence to date suggests cyclical changes in energy intake with a higher consumption in the luteal phase, confirming women's described perceptions of increased appetite during the premenstruum. A limited number of studies have, however, produced conflicting results and the possible reasons for this should be considered.

Lacey *et al.* (1978) studied a group of regularly menstruating teenagers, for 8 weeks, using subject-kept records with household measures. They found a mean increase of 3.4% in energy intake during the 4 d pre-menses and a mean decrease of 3.7% during menstruation as compared with 21 mid-cycle d, but the differences were not significant. In the women studied by Davies *et al.* (1993), energy intake was also greater in the last 4 luteal d than the bleed, but the difference failed to reach significance.

One probable reason for these outcomes was the failure to document ovulation, leading to an underestimate of the effect because of the inclusion of abnormal cycles. A unique finding in the study conducted by Barr *et al.* (1995) was that follicular and luteal intakes did not differ when cycles were anovulatory, or when records were kept before the onset of the temperature rise in cycles with a short luteal phase. However, in ovulatory cycles a highly significant increase in the energy consumed was found in the luteal phase, even with only 3 d of diet records. Their data suggest that up to 30% of cycles in women 20–40 years old may be anovulatory or have a short luteal phase, which would make the timing of data collection critical. Vollman (1977) reported that during the year of menarche 56% of menstrual cycles showed monophasic basal body temperature charts (i.e. were anovulatory). This percentage decreased as women approached their mid-twenties. The school girls of Lacey *et al.* (1978) would not have been many years post-menarche and therefore the number with anovulatory cycles is likely to have been particularly high.

It seems probable that abnormalities may have occurred in the menstrual cycle of some of the subjects studied by Fong & Kretsch (1993) because they did not detect a significant increase in body temperature during the luteal phase, which would have confirmed ovulation (Prior *et al.* 1990). Their subjects were monitored within a metabolic ward and this environmental change may have caused a degree of stress strong enough to disturb ovulation in some of the volunteers, as stress has been found to affect the menstrual cycle adversely (Nagata *et al.* 1986; Schweiger *et al.* 1988). Equally, as the authors themselves note, the small number of participants (9 women) and the large number of cycle portions studied (4 phases, as for Gong *et al.* 1989) would have decreased the study's power to detect differences. Similarly, Piers *et al.* (1995) found no luteal change in food intake, but they were studying only 13 women. They did confirm that the women were ovulating, as demonstrated by an increased serum concentration of progesterone in the luteal phase; however, they chose to study early luteal phase days (i.e. days 16–20), close to expected ovulation, when progesterone concentrations would probably not have been at their greatest. Maximum values have been observed around the day of LH peak +6 days (Collins, 1985).

The failure to confirm ovulation and hence the probable inclusion of an unknown number of anovulatory cycles within the data pool may also, in part, explain the variation in the magnitude of recorded luteal phase increases. Tarasuk & Beaton (1991) considered this large degree of spread in mean phase differences and suggested that it might be an age effect. Studies with a young population, of small age range, show a greater luteal increase in energy intake than those with an older, wider age range. Changes in menstrual cycle length with age are well documented. Cycle length (and follicular phase length) has been shown to decrease with age (Treloar *et al.* 1967; Chiazze *et al.* 1968; Sherman & Korenman, 1975). There were no age-related changes in luteal length. Tarasuk & Beaton therefore noted that it would not be surprising if the food intake effects also changed with age, or at least if the specific timing of these effects in relation to the onset of menses changed.

Barr *et al.* (1995) stated that ovulation must be documented and suggested serial measurement of urinary LH excretion, measurement of serum progesterone concentrations, or quantitative analysis of basal body temperature, the latter being a useful non-invasive method for free-living subjects. Vlitos (1994) used serial measurements of urinary LH, a progesterone metabolite and an oestrogen metabolite to confirm ovulation and the timing of the luteal phase in free-living women. Data from twenty-two women who ovulated, and experienced a luteal rise in progesterone, were analysed. No increase in energy intake was found in either the 5 d preceding the onset of menstruation or within the days of peak progesterone concentration (day of LH peak +4 to +8 confirmed *via* the progesterone metabolite analysis). One reason for the negative results might be a lack of sensitivity of records with estimated weights, yet others have used diaries or 24-h recall with estimated weights and found cyclical changes (Dalvit, 1981; Manocha *et al.* 1986; Gallant *et al.* 1987; Bäuml, 1989; Johnson *et al.* 1994). Another possible explanation is that subjects studied by others may have changed their intake according to an anticipated stereotypic pattern (Abraham *et al.* 1981; Pliner & Fleming, 1983; Gallant *et al.* 1987; Lissner *et al.* 1988; Bäuml, 1989; Johnson *et al.* 1994) as they were apparently not unaware of the study aims. However, studies where subjects were blind to the field of investigation have found significant changes (Dalvit, 1981; Manocha *et al.* 1986; Oram, 1987; Gong *et al.* 1989; Lyons *et al.* 1989; Tarasuk & Beaton, 1991; Martini *et al.* 1994; Barr *et al.* 1995). Furthermore, Barr *et al.* (1995) studied women who recorded their intakes during the latter part of the anovulatory cycle or before the onset of the temperature rise (a marker of luteal phase) in cycles with a short luteal phase; they found that these women did not increase their intake, although subject to the possible bias of women's expectations

because anovulatory and short luteal phase cycles are not obviously different to women. It is also a possibility that the diet analysts in certain previous studies have unknowingly influenced the results towards an expected outcome when estimating weights where the investigator has been aware of cycle phase (Abraham *et al.* 1981; Gallant *et al.* 1987; Tarasuk & Beaton, 1991; Johnson *et al.* 1994; Martini *et al.* 1994). However, cyclical change has been detected in double or investigator-blind studies where such bias could not operate (Dalvit, 1981; Pliner & Fleming, 1983; Manocha *et al.* 1986; Bäuml, 1989). The reason for the lack of cyclical intake change in the subjects of Vlitos (1994) is thus at present unexplainable.

Energy expenditure

In the study of basal metabolic rate (BMR) over the menstrual cycle, Solomon *et al.* (1982) found significant sine curves for five of their six subjects and reported an average difference of 359 kcal/d between the peri-ovulatory phase (lowest point) and the days just before menses (highest point). The reported pattern of BMR, which decreased at menstruation, fell to its lowest approximately 1 week prior to ovulation, remained low during the peri-ovulatory phase, and then increased until subsequent menstruation, is consistent with the patterns of energy intake described. Webb (1986) found that eight out of ten women studied showed a significant rise of 8–16% in BMR during the 14-d luteal phase as compared with the follicular phase, and Bisdee *et al.* (1989) found that metabolic rate during sleep showed cyclical changes being lowest in the late follicular phase and highest in the late luteal phase (an increase of 6%). Energy expenditure over 24 h showed the same changes, but these were not significant. Again these results complement those of energy intake. The significant increase in energy intake during the luteal phase is likely to be a response to the increased metabolic rate that occurs at this phase; however, an increase in food intake could also lead to an elevation in BMR. The data of Bisdee *et al.* (1989) suggest that the small changes in total energy expenditure, amounting to only about 2.5%, experienced by women on a constant diet are dependent on larger alterations in resting metabolic rate rather than on any change in either diet or exercise induced changes in energy balance.

Progesterone, administered to both normal and ovariectomized women, has been shown to be hyperthermic (Barton & Wiesner, 1945; Kappas & Palmer, 1965) and this has led Solomon *et al.* (1982) and Webb (1986) to conclude that the raised luteal BMR was mediated in part by progesterone. In humans, a small but significant relationship was uncovered between individual increases in sleeping metabolic rate and the subjects' falling ratio in urinary E₁-3-G: Pd-3-G indicating a possible oestrogen antagonizing action (Bisdee *et al.* 1989).

Macronutrient intake

Having confirmed that women's perception of an increased premenstrual appetite does coexist with a measurably increased energy intake, the question arises as to whether the anecdotal observations of cravings for sweets and/or chocolates signify altered food selection, and hence macronutrient consumption.

Carbohydrate. The most studied macronutrient in relation to the menstrual cycle is, as expected, carbohydrate (CHO). The data of Dalvit-McPhillips (1983) would appear to support the concept of a specific appetite for CHO. She reported that mean protein and fat intakes were almost identical post-menses and pre-menses (50 g protein and 62 g fat during both phases), but that mean CHO intake almost doubled between phases (133 g post-menses and 257 g pre-menses). These macronutrient data, however, are difficult to

interpret, for as noted by Barr *et al.* (1995) they do not correspond to the mean energy intakes reported previously for the same women (Dalvit, 1981). The mean reported energy intakes in the earlier paper were 637 kJ (152 kcal) higher during both phases than energy intakes computed from reported grams of CHO, protein and fat in the latter paper. One might suspect that alcohol intake could explain the difference, but this is unlikely because the data presented for individual subjects in the two articles seem discrepant.

Lyons *et al.* (1989) found that CHO intake was significantly greater in both the post-ovulatory 4 d and premenstrually than at ovulation (270 g and 252 g respectively compared with 235 g). Likewise, Johnson *et al.* (1994) found luteal CHO intake greater than that in the follicular phase (227 v. 200 g). Carbohydrate intakes as a percentage of energy, however, were almost identical across the cycle phases. Knowing that energy intake varies cyclically, these data emphasize the importance of considering the relative percentage contribution that the macronutrient makes to energy intake. Other investigators have failed to find any change in CHO intake in absolute terms (Abraham *et al.* 1981; Gallant *et al.* 1987; Tarasuk & Beaton, 1991; Davies *et al.* 1993) or as a percentage of energy (Johnson *et al.* 1994; Martini *et al.* 1994; Barr *et al.* 1995).

With regard to specific CHO components, a trend has been observed for sucrose intake to be higher in the luteal phase than during the bleed, follicular phase or at ovulation, but this was not significant (Gong *et al.* 1989). Conversely, mean dietary fibre intake was 4 g lower during the last 4 luteal d ($P < 0.01$) than the first 4 of the bleed (Davies *et al.* 1993). This is noted to have been due to a change in the type of CHO eaten. Premenstrually females opted for sweets, cakes and chocolate, whereas during menstruation complex CHO such as bread were preferred. Unfortunately no additional detail is given.

Studies of taste perception have shown that women judge sugar solutions as less pleasant during the mid-luteal phase than other phases (Wright & Crow, 1973; Aaron, 1975; Pliner & Fleming, 1983). This finding would perhaps suggest that women would choose more savoury foods premenstrually and conflicts with the trend observed in sucrose consumption and reports of sweet cravings. In a detailed study of cravings, desire for (a) higher sugar, (b) high starch and (c) low CHO foods was apparently not affected by the phase of the menstrual cycle that the subject was experiencing. There was, however, a greater preference for chocolate foods (in comparison with otherwise identical non-chocolate alternatives) during the bleed (Tomelleri & Grunewald, 1987). Fong & Kretsch (1993) found that mean CHO intake marginally significantly ($P < 0.06$) increased during the bleed, compared with the peri-ovulatory period, owing to an increase in sweet consumption, mostly chocolate-coated: 68 g/d candy (menses); 42 g (follicular); 49 g (peri-ovulatory); 55 g (luteal). The consumption of sugar-containing carbonated drinks was greatest during the luteal phase (417 g/d) and was significantly different from the other three phases: 370 (menses); 335 (follicular); 359 (peri-ovulatory).

In summary, measurement of CHO intake fails to demonstrate clearly that a woman's craving for sweets and/or chocolate results in a significant increase in total CHO intake premenstrually, relative to the other macronutrients. As suggested by Barr *et al.* (1995), it is possible that the commonly held notion of CHO cravings represents appetite, rather than a specific desire for CHO. Alternatively, women may react to such cravings by increasing their total energy intake rather than by altering the pattern of macronutrients consumed. There is, however, some tentative evidence of altered food selection resulting in a higher sugar intake premenstrually or menstrually, compared with the rest of the cycle, and a corresponding decrease in fibre intake. In particular, the consumption of chocolate and fibre seems worthy of further investigation.

Fat and protein. Significant changes in fat and protein intake have been demonstrated. Fat intake (g/d) was significantly higher in the luteal phase than in the 10 d post onset of

menses (79.8 v. 72.9; Tarasuk & Beaton, 1991) or the 3 d postmenses (81 v. 61; Gallant *et al.* 1987). There has also been a reported trend for a higher intake (g/d) in the luteal phase as compared with the bleed (69 v. 60; Davies *et al.* 1993) and ovulation (89/88 v. 78; Lyons *et al.* 1989). Protein intake has likewise been found to be significantly higher (g/d) in the luteal phase than at ovulation (73/72 v. 63; Lyons *et al.* 1989) or the 3 d postmenses (76 v. 52; Gallant *et al.* 1987). All the results indicate that changes in absolute fat and protein intake contribute to an increased total energy intake in the luteal phase, as was the case for CHO. In the studies where fat and protein intake was controlled for energy intake, no difference as a proportion of energy is generally seen (Lyons *et al.* 1989; Martini *et al.* 1994; Barr *et al.* 1995). Gallant *et al.* (1987) detected a significant phase change in the absolute intake of protein and fat but not in CHO. This is possibly suggestive of a change in the relative contribution to energy of each macronutrient. However this is unlikely, for although not reaching significance, there was a trend for CHO intake also to be higher premenstrually, echoing the pattern of fat and protein. Abraham *et al.* (1981) reported a similar pattern of intake for each macronutrient, although no details are given.

Only two studies have controlled for energy and found evidence of a disproportionate increase in the luteal phase in one macronutrient. Without controlling for energy, Tarasuk & Beaton (1991) found that the cyclical patterns for fat and protein intake were similar to each other and to that for energy intake. They appeared to differ in magnitude of effect rather than form. However, when the nutrients per 1000 kcal for fat and protein were examined, there was a sharp differentiation of patterns between them. They concluded that, whereas the absolute intake reflects changes in total food (energy) intake, there was evidence of independent effects relating to food selection and hence macronutrient density. Given that the pattern of fat/1000 kcal was of the same form as that for total energy, they suggested that factors influencing the selection of fat-rich foods may be important in influencing total energy intake. Johnson *et al.* (1994) found no cyclical change in protein intake in either absolute or percentage terms but, in agreement with Tarasuk & Beaton (1991), found the percentage of energy from fat significantly higher in the luteal phase than in the follicular or peri-menstrual phases. On balance, however, the overwhelming body of evidence suggests that if specific food selection does occur during the menstrual cycle, it is of insufficient strength to alter the macronutrient composition of the diet.

Micronutrient intake

Little work has been published on micronutrient intake through the menstrual cycle. Fong & Kretsch (1993) reported that, with the exception of ascorbic acid, no statistically significant differences in average intakes of minerals and water-soluble vitamins were found during the menstrual cycle, nor were significant differences found for any of the dietary indices studied when these dietary intake variables were expressed on a per 1000 kcal or a per 100 g basis. Ascorbic acid intake (mg/d) was significantly lower (154) during the peri-ovulatory phase than during the luteal (199) or the menses (191) phases. Snack items were examined to help explain the significant difference. The decrease in peri-ovulatory ascorbic acid was perhaps due to a decrease (non-significant) in citrus fruit consumed during this period. Martini *et al.* (1994) were unable to confirm a significantly reduced vitamin C intake in the mid-follicular phase. They concluded that menstrual cycle phase did not significantly affect intakes of a large number of micronutrients. Only intakes of vitamin D, riboflavin, potassium, phosphorus and magnesium were greater in the luteal phase than in the follicular. These nutrients fit the profile of nutrients commonly found in dairy products. However, no significant difference was observed in the number of daily servings from the dairy food group for different menstrual phases (2.3 servings/d in follicular phase v. 2.5 in the mid-luteal). This suggests that observed nutrient differences are due to altered intakes

from several food groups and not just the dairy group, or that the servings were larger in the luteal phase. Unfortunately no detail is given as to the definition of a serving.

Ovarian hormone influences

Animal models. The food intake of several mammalian species shows systematic variation across the ovarian cycle and has been studied in relation to the ovarian hormones, oestrogen and progesterone.

Interaction between feeding and ovarian hormones has been well documented in the oestrous cycle of rats. Food intake is low around the time of ovulation, when oestrogen levels are high relative to progesterone, and high during periods of the cycle with low oestrogen levels (Tarttelin & Gorski, 1971; Blaustein & Wade, 1976; Kanarek & Beck, 1980). Body weight undergoes corresponding changes, with weight loss while endogenous oestradiol is high and weight gain when oestradiol is low (Tarttelin & Gorski, 1971; Kanarek & Beck, 1980). After ovariectomy, when blood levels of oestrogens and progesterone are reduced, food consumption and body weight increase. Treatment of ovariectomized animals with exogenous oestrogens, to give plasma concentrations within the range of those found at oestrus, then restores the depression in food intake and body weight decreases (Wade & Zucker, 1969*a*; Bell & Zucker, 1971; Tarttelin & Gorski, 1971; Blaustein & Wade, 1976; Landau & Zucker, 1976).

When ovariectomized animals are given progesterone, no change in feeding is observed (Hervey & Hervey, 1966). This suggests that oestrogens are the principal steroid hormones to influence eating behaviour in the rat, exerting an inhibitory effect on food consumption. Progesterone is thought to have a secondary role as an inhibitor of the oestrogenic effect (Wade, 1972). If intact females are treated with progesterone then the oestrous cycle ceases and intake remains at a high level.

Wade & Zucker (1970) showed that implantation of crystals of oestradiol benzoate in the ventromedial hypothalamus (VMH) of rats depressed food intake, and Stumpf (1970) found evidence for oestrogen receptors in several parts of the hypothalamus, including the VMH. These receptors in the VMH have been assumed to be the way in which oestrogens influence intake, but Wade & Gray (1979) have presented evidence for effects of oestrogens on the metabolism of liver, muscle and adipose tissue being involved in intake depression at oestrus. For example, oestrogen treatment has marked effects on liver metabolism (Matute & Kalkhoff, 1973) and adipose tissue (Wade & Gray, 1978), both tissues which are strongly implicated in the control of food intake. It is likely, therefore, that the effects of oestrogens on intake are mediated through several mechanisms.

There appears also to be an effect of sex hormones on dietary selection and taste perception. Zucker (1969) found female rats to exhibit a higher preference for saccharin and a greater aversion to quinine than males. When oestrogen levels are low, responsiveness to sweet and bitter tastes is reduced (Valenstein *et al.* 1967; Wade & Zucker, 1969*a, b*). Administration of oestrogen and progesterone to ovariectomized animals restores female taste preference (Zucker, 1969).

Research on non-human primates also suggests an inverse correlation between oestradiol levels and food intake. In baboons and rhesus monkeys food intake is lowest for a period of 3–4 days coinciding with the mid-cycle surge in blood oestrogen that occurs at normal ovulation and is highest during the post-ovulatory luteal phase, a time of lowered oestrogen and increased progesterone (Gilbert & Gillman, 1956; Czaja, 1975; Czaja & Goy, 1975; Rosenblatt *et al.* 1980; Bielert & Busse, 1983; Kemnitz *et al.* 1984). Gilbert & Gillman (1956) speculated that progesterone was acting as an appetite stimulant during the luteal phase. They found that exogenous progesterone administered at midcycle increased food intake. Others, however, (Hess & Resko, 1973; Czaja, 1975; Czaja & Goy, 1975) saw

oestrogens as the main appetite affecting hormones, with progesterone having a secondary effect inhibiting their actions. Hess & Resko (1973), for example, demonstrated that raising serum progesterone levels during the peri-ovulatory and follicular stages with exogenous progesterone resulted in lowered endogenous oestradiol levels and prevented the pre-ovulatory oestradiol surge.

The question of whether progesterone acts as an antagonist to oestradiol remains, however. Czaja (1978) could find no such progesterone antagonism. In monkeys pretreated with progesterone, oestradiol successfully lowered food intake, progesterone had no effect. Also, female monkeys chronically stimulated with oestradiol showed no change in food intake when progesterone was given. Ovarian hormones obviously affect food intake in primates, but it is not clear exactly how they do so.

Human menstrual cycle. The consensus of opinion is that oestrogens are the principal steroid hormones to influence food intake in the rat and certain other mammals, exerting an inhibitory effect on food consumption. Dalvit (1981) concluded that oestrogen may also be acting as an appetite suppressing hormone in human females, resulting in reduced energy intakes in the follicular phase. Lyons *et al.* (1989) suggested that the reduction in food intake they detected at ovulation coincides with the expected peak in circulating oestrogen levels, and is consistent with the hypothesis in animals that oestrogen is an appetite suppressant. What these two studies have failed to do is to consider the second, albeit smaller, peak in serum oestrogen that occurs during the luteal phase in human females, when the indications are that energy intake reaches its maximum level. Presumably in humans the effects of ovarian hormones on food intake are more complicated.

Bancroft & Bäckström (1985) speculated that progesterone exerts most effect, increasing food intake. One flaw with all the food intake studies to date is that corresponding steroid hormone measurements have not been made. Lissner *et al.* (1988), for illustrative purposes only, related group mean daily energy intakes (standardized to a 28 unit cycle) to a reference 28 d pattern of ovarian hormones. They concluded that the progesterone stimulatory theory is consistent with their luteal data. In particular, the mid-luteal peak in energy intake occurred at the same time in the menstrual cycle that progesterone generally reaches maximum levels. The second peak in intake, observed in the follicular phase, they suggested might have been a resonance or regulatory response to the first. Certainly their results offer no support for an inhibitory role of oestradiol. If any relationship does exist between daily energy intake and daily oestradiol it is more likely to have been a positive one because the two variables tended to fluctuate in parallel. They further pointed out that the possibility of the cyclical pattern in food intakes reflecting hormonal fluctuations is indirectly strengthened by the fact that the energy intake patterns were independent of illness and menstrual symptoms; otherwise a plausible alternative conclusion would have been that subjects were eating less once menstruation had started because they were experiencing discomfort.

In summary, there appear clear cyclical changes in energy intake with the menstrual cycle in humans. How, and to what extent, these relate to the cycling levels of ovarian hormones is not clear. Oestradiol may depress food consumption and this is observed as reduced energy intake around ovulation. The second luteal oestradiol peak may fail to suppress intake, and indeed energy intake reaches its peak owing to either the antagonizing or directly stimulating effects of progesterone. Whether ovarian hormones directly affect appetite and intake, or whether changes in intake are secondary to changes in BMR, is also not entirely certain.

CYCLICAL FIBRE INTAKE AND BOWEL COMPLAINTS

The increased energy intake, possible modification of food selection and women's awareness of a desire for sweet foods premenstrually raise the possibility that consumption of non-starch polysaccharides (NSP) is altered cyclically (either in absolute terms or with regard to density i.e. NSP g/1000 kcals) and has some bearing on the changes in bowel habit reported with the luteal phase and bleed.

NSP intake is known to have a marked effect on gut transit time and stool consistency and weight. Meta-analysis of eleven studies in which daily faecal weight was measured accurately in twenty-six groups of people ($n = 206$) on controlled diets of known NSP content shows a significant correlation between NSP intake and mean daily stool weight ($r = 0.84$) (Cummings *et al.* 1992). All sources of NSP lead to an increase in faecal output and therefore in the components which make up faeces such as fat, nitrogen, water, minerals and trace elements. Faecal results from nearly 150 individual dietary studies of assorted fibre sources published between 1932 and 1992 have been compiled (Cummings, 1992).

No one simple hypothesis can explain the way in which NSP affects bowel habit. There are probably at least four distinct effects of NSP by which it brings about changes in faecal weight and texture. Firstly, non-fermented NSP, such as that found in coarse wheat bran, are able to exert a physical effect on intestinal bulk because they retain their cellular structure and thus their capacity to hold water. Secondly, most forms of dietary NSP are extensively degraded by microflora. The result of this is to stimulate microbial growth and a greater excretion of microbial products in faeces, again increasing faecal mass. Thirdly, increasing bulk in the large intestine stimulates colonic propulsion. As transit time falls the efficiency with which bacteria grow improves. Shortened transit time also leads to reduced water absorption by the colon and therefore the easier passage of bulkier and soft stools. Fourthly, fermentation of polysaccharides releases gases which may be trapped in the colonic contents, contributing to their bulk and plasticity. There may yet be other mechanisms of action, for example, the work of Tomlin & Read (1988) with polyvinyl tubing chips suggests an irritation by coarse lignified particles.

The NSP content of the diet has not previously been recorded through the menstrual cycle or diets have been adapted. Recently work was published which reported that dietary fibre intake was significantly higher during the bleed than the 4 d prior to onset (Davies *et al.* 1993). Mean (\pm SD) fibre intake premenstrually was 17.7 ± 6.0 g compared with 21.9 ± 5.1 g during the bleed. Of the twenty-five subjects studied, twenty-one demonstrated an increase in fibre consumption during menses. Interestingly, no significant change in energy intake was detected over the same period, although the tendency was for intake to be higher premenstrually. Mean daily stool frequency was significantly higher during the bleed (1.9 ± 0.6 v. 1.5 ± 3.0) and mean faecal form score was reduced (5.7 ± 1.9 v. 4.5 ± 1.5 on an 8 point scale) indicating that stools were less well formed during menses. The increase in fibre intake correlated with the decrease in faecal form ($r = -0.53$; $P < 0.01$). The results certainly suggest that NSP intake may indeed play a role in menstrual changes in bowel habit. The study indicated the necessity to measure NSP intake carefully through the menstrual cycle, and to take any cyclical changes into consideration when assessing the role of sex hormones on gut function. Further, careful documentation of cyclical changes in NSP consumption would appear necessary to aid effective treatment of cyclical bowel complaints. If women reduce their complex polysaccharide intake premenstrually, awareness of this and efforts to boost consumption may alleviate any symptoms of constipation.

To date only one other study is known to have investigated the intake of NSP through the menstrual cycle. Vlitos (1994) measured NSP intake in 22 free-living women (in whom

ovulation was confirmed) using food diaries with household measures. No significant change in NSP intake was found in any of the phases studied (oestrogenic, progestogenic, premenstrual and menses). Equally, no change was detected in bowel habit.

Further study is indicated on cyclical changes in fibre intake, particularly in women who complain of constipation premenstrually. NSP may also prove an aid to stabilizing any bleed associated diarrhoea.

PRACTICAL CONSIDERATIONS

CYCLICAL ENERGY INTAKE

The question that has often been raised when studying food intake in menstruating women is whether cycle-phase differences exist, and if so, are they of practical importance. In reviewing the literature to date, there has accumulated convincing evidence that energy intakes differ between menstrual cycle phases, at least when the cycle is normally ovulatory. As stated by Barr *et al.* (1995), although it may be possible (but not desirable) to ignore these changes when conducting cross-sectional studies, failure to consider menstrual cycle phase could have important implications for longitudinal studies, especially if small numbers of subjects are involved.

CYCLICAL BOWEL HABITS

Evidence to date suggests menstrual-related changes in bowel habit, in particular faster transit time during the bleed resulting in a looser stool. Hence, when conducting studies to monitor the effect of diet on bowel habit in women, cycle phase must be taken into account.

CONCLUSIONS

Large proportions of women have reported changes in bowel habit with their menstrual cycle, typically constipation premenstrually and/or diarrhoea with the bleed. Some supporting objective evidence has been obtained to confirm these women's views; however, there have equally been workers unable to detect any cyclical alterations. The reasons for this conflicting picture are not altogether clear, and until more is known, caution should be exercised when using women of reproductive age in studies concerned with dietary treatments aimed at influencing bowel function.

Dietary surveys measuring food intake confirm women's impressions of an increased appetite premenstrually, with a significantly higher energy consumption in the luteal phase of ovulatory cycles. Expressed as the percentage rise over mean follicular intake, the recorded luteal phase increase has been as high as 35%. Energy intake possibly mimics a simple sine wave, being lowest around ovulation and highest premenstrually. There is evidence to suggest that these alternations in energy intake are in response to complementary changes in BMR.

Detailed study of cravings for sweet foods and changes in CHO consumption have produced conflicting results. It appears that the overall percentage contribution to energy of the macronutrients is unaffected by cycle phase; however, there is tentative evidence for an alteration in the types of CHO consumed prior to, and possibly during, menses. Preliminary work has also indicated cyclical changes in micronutrient intake. In interpreting the results to date, however, caution should still be exercised. Few detailed studies have been undertaken with the necessary measurement of circulating ovarian hormone concentrations and confirmation of cycle normality. There is, however, a strong enough

case to recommend that menstrual cycle phase is considered when studying food intake in menstruating women. Cyclical intake in relation to bowel habit is an area which should be investigated further.

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