LOW-FAT DIET VS. EDUCATION SUPPORT IN THE TREATMENT OF LATE LUTEAL PHASE DYSPHORIC DISORDER

by

Jane Michele Kudlas

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APPROVED:

Richard A. Winett, Chair

Thomas H. Ollendick

George A. Clum

Jack W. Finney

Janet W. Rankin

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(Abstract)

A treatment outcome study was conducted comparing a low-fat diet intervention with an education-support group and a waiting-list control group in the treatment of premenstrual tension syndrome (PMS) or Late Luteal Phase Dysphoric Disorder (LLPDD). Subjects met provisional diagnostic criteria for LLPDD and symptoms were monitored prospectively. A low-fat diet was hypothesized too be an effective intervention for reducing the severity of both physical and emotional symptoms in women suffering from LLPDD. This was based on the theory relating raised estrogen levels to premenstrual distress, and research suggesting low-fat diets reduce estrogen levels.

The hypothesis that a low-fat diet would decrease premenstrual suffering was not supported by the results of this study. However, there appeared to be an advantage to participating in a group which provided support and information on LLPDD compared to receiving no treatment. Implications for future research, treatment recommendations, and methodological issues are discussed.

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Finally, I would like to dedicate this dissertation to my three grandparents, Ruth Fowler, John Fowler, and Lawrence "Duke" Walsh, who all passed away since I entered graduate school. I only wish you were around to see me finish.

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Chapter I

Etiology and Treatment of Premenstrual Tension Syndrome

Premenstrual tension syndrome or premenstrual syndrome (PMS) has received a great deal of attention from both the popular and research literature. Despite this widespread interest, there is no agreed upon definition for PMS (Beumont, Richards, & Gelder, 1975; Rubinow & Roy-Byrne, 1984; Spitzer, Severino, Williams, & Parry, 1989). In general, this term refers to recurrent psychological and physical changes which occur approximately one week prior to menstruation, and remit a few days following the onset of menses. Common psychological symptoms include anxiety, depression, irritability, tearfulness, mood swings and confusion. Physiological symptoms frequently mentioned are abdominal discomfort or pain, bloating, breast tenderness, and fatigue.

After reviewing the literature, Rubinow and Roy-Byrne (1984) reported that there may be as many as 150 symptoms associated with PMS. Reid (1991) noted that PMS was originally called premenstrual tension by Frank in 1931 and syndrome was added to account for the different clinical presentations. For example, some women may report a single symptom, such as depression, while other women suffer from numerous symptoms such as depression, irritability, anxiety, mood swings, headaches, and bloating.

The lack of an agreed-upon definition and differing clinical presentations has led to much confusion in the research literature. Prevalence estimates differ dramatically

depending on the criteria used to define PMS. Treatment outcome studies and research on etiological theories of PMS are difficult to compare because criteria for subject participation are extremely variable. Adding to this confusion is a belief, among many professionals and lay persons, that PMS is merely a "normal" phenomenon that most women experience (Reid, 1986).

In an attempt to standardize a definition of PMS for research and clinical purposes, Late Luteal Phase Dysphoric Disorder (LLPDD) was included as a provisional category in the Diagnostic and Statistical Manual for Mental Disorders, (3rd edition revised) (American Psychiatric Association, 1987, see appendix A). This definition was designed to consider a subset of PMS conditions in which mood disturbance is a predominant feature. In order to meet criteria, symptoms must be serious enough to interfere with work, social activities, or relationships, thereby, distinguishing between those who appear to be having normal mood fluctuations prior to menses and those who are suffering severely. The diagnosis of LLPDD must also be confirmed by prospective daily ratings of symptoms, because retrospective reports tend to be inaccurate (May, 1976; Rubinow & Roy-Byrne, 1984). However, a diagnosis of LLPDD can be given "provisionally" until it is confirmed with prospective monitoring. The advisory committee which developed the criteria for LLPDD hoped this definition would promote research and lead to optimal clinical care for individuals suffering from LLPDD (Spitzer et al., 1989).

Etiological theories and treatments of PMS and LLPDD have focused on biological and psychological factors. A wide variety of treatments has been proposed including prescription progesterone, oral contraceptives, diuretics psychotropic medication, vitamins, exercise, relaxation, and diet. Many treatment studies are anecdotal, lack appropriate control groups, or use retrospective reports. Although there are a number of well-controlled studies, only a few of these have tried to meet the diagnostic criteria outlined in DSM-III-R for LLPDD (i.e., Stone, Pearlstein, & Brown, 1991).

The purpose of this dissertation study is to conduct a well controlled treatment outcome study in which subjects meet the provisional criteria for LLPDD. Using prospective measures, this study will assess the effectiveness of a low-fat diet in treating LLPDD. This treatment is based on the theory relating symptoms of LLPDD to raised levels of estrogen. A low-fat diet is postulated to lower estrogen levels, thus relieving premenstrual distress. Reducing the consumption of fat, while increasing the intake of carbohydrates, has been proposed as a "component" of treatment for PMS by a number of physicians and researchers (Harrison, 1985; Norris & Sullivan, 1983), but it has not been tested empirically. Several research studies strongly indicated a low-fat diet was effective in relieving cyclical mastopathy or severe breast tenderness (Rose et al., 1985; Boyd, et al., 1988), which is a common symptom of LLPDD. Recent evidence suggests carbohydrate consumption can immediately relieve emotional distress associated with the premenstrual phase (Wurtman, Brzezinski, Wurtman, & Laferrere, 1989). Changing

dietary practices to treat LLPDD is extremely speculative, but it appears to have a theoretical foundation and some empirical support. Moreover, dietary interventions have been recommended by self-help books (Harrison, 1985; Norris & Sullivan, 1983); thus, many women may be trying this technique despite the lack of any treatment-outcome study.

The purpose of this dissertation is to assess the effectiveness of treating LLPDD with a low-fat diet. As a prelude to this topic, a literature review on the prevalence, etiological theories, and treatments of PMS (or LLPDD) will be presented in Chapter 1. The rationale for treating LLPDD with a low-fat diet will also be presented in greater detail. In the second chapter of this dissertation, the methodology and results of a treatment-outcome study will be described. The study will compare the efficacy of a low-fat diet intervention with an education support group in the treatment of LLPDD. The final chapter will discuss the results of this study, methodological problems, recommendations for future research and clinical treatment of LLPDD. When citing research articles, this paper will use the term PMS unless the cited article specifies all or most of the criteria for LLPDD have been met.

Prevalence Estimates

The interest in premenstrual tension is not surprising, considering it has been associated with violent crimes (Dalton, 1961; Ribeiro, 1962), accidents (Dalton, 1960),

suicide attempts (MacKinnon, MacKinnon, & Thomsom, 1959; Mandell & Mandell, 1967; Wetzel & McClure, 1972), psychiatric admissions (Glass, Henninger, Lansky, & Talan, 1971), and marital discord (Shabanah, 1963, cited in Reid & Yen, 1981). Moreover, prevalence estimates suggest a large portion of the female population suffers from PMS.

As indicated above, estimates of PMS vary widely depending on how PMS is defined. Reid and Yen (1981) reviewed questionnaire reports and concluded 70% to 90% of women experience cyclic premenstrual symptoms and 20% to 40% of the female population report some degree of impairment. In a comprehensive review of the literature, Logue and Moos (1986) concluded 40% of women experience mild premenstrual symptoms, while 2% to 10% of women suffer severely. These research reviews are based primarily on retrospective studies.

Prospective studies have recently been conducted to determine the prevalence of LLPDD. Unpublished data by Haskett (1987, cited in Rivera-Tovar & Frank, 1990) suggests 3.4% of women in their reproductive years meet the strict criteria for LLPDD. This study was based on a large community sample in which prospective ratings were used. Rivera-Tovar and Frank (1990) found similar results in a college population. They reported a prevalence rate of 4.6 %. These results were based on a sample of 217 women who monitored 33 emotional and physical symptoms daily for three months. Subjects, who were between the ages of 17 and 29 (mean = 18), were blind to the intent of the study. Rivera-Tovar and Frank noted PMS was thought to be "an affliction of

women over 30" (p.1634), but results of their study contradicted this belief. They also suggested that the prevalence rate of PMS, when strict diagnostic criteria are used, is lower than previously indicated.

The conservative prevalence estimates from the two prospective studies mentioned above dispel the belief that all women suffer from LLPDD. But, these estimates also indicate about 3% to 4% of the female population have severe premenstrual distress, to the point where symptoms during their premenstrual phase interfere with social activities, relationships, or work productivity. Thus, researching the effectiveness of treatments for this disorder is of some importance. Most current treatments for LLPDD are based on physiological theories, although psychological theories and treatments have also been researched.

Etiological Theories of PMS or LLPDD

Etiological theories of PMS or LLPDD can be categorized into two basic groups: (1) physiological, and (2) psychological or psychosocial. Physiological theories have dominated most of the research, and subsequently various medications have been used to treat LLPDD or PMS. Psychological theories have also been proposed and are currently leading to various treatment approaches.

Numerous physiological theories have been proposed. Raised levels of estrogen, deficiency in progesterone, increased levels of prolactin and aldosterone, lowered levels

of beta-endorphins, glucose intolerance, and vitamin deficiencies have all been hypothesized as possible "causes" of PMS or LLPDD. Consequently, various medications or vitamins have been suggested to treat PMS or LLPDD. These theories also imply behavioral treatments, such as modifying dietary practices and increasing exercise, may also be effective in treating LLPDD or PMS.

Psychological theories suggest that factors such as negative expectations, learned helplessness, and stress influence premenstrual suffering. Based on these theories, treatment approaches such as cognitive therapy, supportive counsel, and relaxation therapy may be effective in relieving premenstrual suffering.

Physiological Theories

Theories of premenstrual tension have typically focused on hormonal or other physiological factors. Even Frank, who coined the term "premenstrual tension" postulated premenstrual distress was caused by fluid retention due to raised levels of estrogen (cited in Beumont, Richards, & Gelder, 1975). Rubinow and Roy-Byrne (1984) reviewed the major physiological theories of PMS. Most of these theories focus on circulating levels of gonadal steroids, prolactin or aldosterone (e.g. Greene & Dalton, 1953; Horrobin, Mtabaji, Karmali, Manku, & Nassar, 1976; Janowsky, Berens, & Davis, 1973). In addition, lowered beta-endorphin levels (Halbreich & Endicott, 1981), vitamin deficiencies (Abraham & Rumley, 1987), and glucose intolerance (Morton, 1950) have all been suggested as a possible etiological factors.

Raised levels of estrogen. Elevated estrogen levels, during the luteal phase, is one of the most popular etiological theories of PMS. Several studies have reported high estrogen levels in women with PMS (Abraham, Elsner, & Lucas, 1978; Backstrom & Carstensen, 1974; Backstrom, Wide, Sodergard, & Carstensen, 1976). Backstrom and Mattsson (1975) found significant correlation between estrogen levels and PMS symptoms, depression and irritability. Despite these positive results, these studies have been criticized for inadequate entry criteria, and other methodological flaws (Rubinow et al., 1984).

Confirming earlier studies, Hammarback, Damber, and Backstrom (1989) reported results which suggest raised levels of estrogen influence premenstrual distress. Eighteen women monitored premenstrual symptoms daily for two full cycles. Daily symptom ratings and hormonal levels of the two cycles were compared. Results indicated these women reported more adverse premenstrual symptomatology during a cycle with high luteal phase plasma estradiol and progesterone concentrations than in a cycle with low luteal phase hormone concentrations. Hammarback and associates (1989) concluded that luteal phase plasma concentration of estradiol and/or progesterone are of importance to PMS symptom severity.

Progesterone deficiency. Progesterone deficiency is another popular etiological theory of PMS. Rubinow and Roy-Byrne (1984) summarized the research of progesterone deficiency and reported equivocal results. Five studies found progesterone to be reduced during the luteal phase for women suffering from PMS compared to

controls (Abraham et al., 1978; Backstrom et al., 1976; Backstrom & Carstensen, 1974; Munday, Brush, & Taylor, 1981; Smith, 1975). Contrary to these findings, another study showed an increase in progesterone during the luteal phase (O'Brien, Craven, Selby, & Symonds, 1979). Three other studies found no abnormality in progesterone secretion (Andersch, Abrahamsson, Wendestam, Ohman, & Hahn, 1979; Anderson, Larsen, Steenstrup, Svendstrup, & Nielsen, 1977; Taylor, 1979).

Rubinow and associates criticized the studies for several methodological flaws. Unclear or retrospective entry criteria were used in a number of these studies, a few failed to adequately describe rating instruments, and several of the studies choose nonuniform times to take blood samples.

Prolactin. Although elevated levels of prolactin have been suggested as a possible etiological factor (Horrobin, 1971, cited in Reid & Yen, 1981), numerous studies have found no differences in prolactin levels between women suffering from PMS and controls (Andersch et al., 1979; Andersen et al., 1977; Backstrom & Aakvaag, 1981; Benedek-Jaszmann & Hearn-Sturtevant, 1976; Munday, 1977; O'Brien & Symonds, 1982; Steiner, Haskett, Carroll, Hays, & Rubin, 1984). Rubinow and Roy-Byrne (1984) criticized one study which reported higher prolactin levels in PMS sufferers compared to controls (Halbreich, Assael, Ben-David, & Bernstein, 1976) because the prolactin levels in this study were three to four times higher than the levels reported in the other studies.

Aldosterone. Aldosterone has been suggested as a etiological factor in the PMS, and elevated levels of aldosterone have been found in the premenstrual phase (Michelakis, Yoshida, & Dormois, 1975; Gray, Strausfeld, Watanabe, Sims, & Solomon, 1968; Kats, & Romfh, 1972). However, two different studies found no differences in plasma aldosterone levels in PMS sufferers compared to women who did not experience premenstrual distress (Munday, 1977; O'Brien, Craven, Selby, & Symonds, 1979). Thus, the role of aldosterone in the presentation of premenstrual symptoms is unclear.

Beta-endorphin withdrawal hypothesis. The theory that premenstrual symptoms are related to lowered levels of beta-endorphins during the late luteal phase, originally proposed by Halbreich and Endicott (1981), has received some empirical and anecdotal support. Giannini, Martin and Turner (1990) summarized the beta-endorphin hypothesis by comparing LLPDD with morphine withdrawal. They asserted that the biochemical mechanisms associated with LLPDD are similar to those associated with morphine withdrawal. When the use of morphine, an opiate, is discontinued, withdrawal symptoms such as cramping, lability, generalized discomfort, and irritability occur. Noting that beta-endorphins are opioids and that the symptoms of morphine withdrawal are typically seen in premenstrual suffers, Giannini and associates postulated that a decease in beta-endorphins is responsible for PMS or LLPDD.

Several studies have found endorphin levels decrease just prior to menses in women reporting to suffer from PMS (Chuong, Coulam, Kao, Bergstralh, & Go, 1985; Facchinetti, et al., 1987; Giannini, Sullivan, Sarachene, & Loiselle; 1988). However,

these studies lacked control groups, and sample sizes were very small. Trying to correct these methodological flaws, Giannini and colleagues (1990) compared 22 women who met the provisional criteria for LLPDD, with a non-symptomatic control group of 22 women matched for age, race and socioeconomic status. Endorphin levels were assessed at day 20 and day 1 of the menstrual cycle. Results indicated beta-endorphin levels significantly decreased for the LLPDD group but not for the control group. Unfortunately, these researchers did not have their subjects prospectively monitor their symptoms so a diagnosis of LLPDD was not confirmed. Further research is necessary to support this plausible and interesting hypothesis.

Vitamin B deficiency. In the 1940's, it was suggested that vitamin B complex deficiency led to a excess of estrogen and a subsequent increase in premenstrual symptoms (Biskind and Biskind, 1942; Biskind & Biskind, 1943). This hypothesis was based on rat studies which found that vitamin B complex impaired estrogen metabolism. However, this conceptualization was questioned when Zondek and Brezezinski (1948; cited in Reid & Yen) who found normal estrogen metabolism in women who suffered from vitamin B complex deficiency during war time.

It has also been proposed that Vitamin B6 plays a role in the biosynthesis of dopamine and serotonin. Simply stated, higher estrogen levels may lead to a deficiency in Vitamin B6, which in turn leads to reduced biosynthesis of serotonin and dopamine. The consequence is premenstrual depression. However, studies have failed to show significant change in dopaminergic function after the administration of Vitamin B6 (i.e.

MacDonald, Collins, Tobin, & Wijayaratne, 1976; Tolis, Laliberte, Guyda, & Naftolin, 1977). Thus, this etiological theory is extremely questionable (Reid and Yen, 1981).

Magnesium deficiency. A deficiency in magnesium has been suggested as an etiological factor of PMS, but empirical reports are mixed. Abraham and Lubran (1981) found that women with PMS tend to have lower magnesium levels than women who do not suffer from PMS, while Hagen, Nesheim, & Tuntland (1985) found no differences in magnesium levels between PMS sufferers and nonsufferers.

Gallant, Bowering, Short, Turkki, and Badawy (1988) compared 9 women who had been diagnosed with PMS after one month of prospective monitoring to a group of 10 control women who did not appear to suffer from PMS. Subjects monitored their dietary intake the first three days following menses, and on days 21, 22, and 23 of their cycle. Gallant and associates found average magnesium intake was significantly higher in the control women compared to those diagnosed as suffering from PMS. However, they found no significant differences in plasma magnesium levels. Results from this study suggest magnesium deficiency may play a role in premenstrual distress, but further research is need to support this hypothesis.

Hypoglycemia or glucose intolerance. Hypoglycemia or glucose intolerance has been hypothesized as an explanation for premenstrual cravings for sweets, increased appetite, dizziness, fatigue, irritability, and dysphoric mood. Although this is an intuitively appealing theory, there is little research to substantiate it.

Some studies suggest glucose tolerance is different during the premenstrual phase. A number of studies on normal women have reported oral glucose tolerance is lower during the premenstrual phase, and it returns to normal levels after menstruation (Jarrett & Graver, 1968; Roy, Ghosh, & Bhattacharjee, 1971). For diabetics, glucose tolerance also appears to change in response to the menstrual cycle (Cramer, 1942; Harrop & Mosentahal, 1918; Rosenbloom, 1921). Although these studies suggest glucose tolerance changes during the menstrual cycle, they do not explain differences in premenstrual distress among women.

Reid, Greenaway-Coates, & Hahn (1986) tested the theory postulating glucose intolerance was lower in women who suffer from alleged premenstrual "hypoglycemic" attacks. They compared five normal women to six women with who suffered from PMS. In addition to having higher scores on the Premenstrual Tension Self-Rating Scale (Steiner, Haskett, & Carroll, 1980) during the luteal phase compared to the follicular phase, the PMS sufferers (or their physicians) attributed their symptoms to hypoglycemia. In contrast to the studies mentioned above, Reid and associates found no differences in glucose tolerance during the follicular and luteal phases for the normal women. Likewise, they found no changes between the two phase for the women reporting to suffer from PMS. Thus, the Reid et al. study did not support the theory that hypoglycemia is related to premenstrual suffering.

Summary of physiological theories. Research on the physiological theories of PMS or LLPDD is full of inconsistencies. Despite this, there are several theories that

show promise. Raised estrogen levels have been consistently related to premenstrual distress, and a recent study confirms this relationship. Likewise, the theory that lowered levels of beta-endorphins are responsible for premenstrual distress has a fair amount of empirical support. Theories prolactin, aldosterone, progesterone deficiency, glucose intolerance, and vitamin deficiencies appear to be less viable.

Psychological Theories

Theories of PMS which emphasize cognitive and environmental factors will be discussed in this section. Much of the research in this area has focused on negative expectancies. Several of these studies imply that premenstrual distress is a figment of a woman's imagination, but they also suggest that negative thoughts exacerbate premenstrual distress. Stress, itself, has also been implicated as a variable which increases premenstrual suffering.

Negative expectancies and cognitions. Several researchers propose that premenstrual tension is due to negative expectancies (Clark & Ruble, 1978; Paige, 1973; Parlee, 1974; Sommer, 1973). Negative expectancies are hypothesized to result from prevailing cultural beliefs linking negative moods to the premenstrual phase of the menstrual cycle. A number of studies appear to support these assertions (AuBuchon & Calhoun, 1985; Clark & Ruble, 1978; Parlee, 1974; Parlee, 1982; Slade, 1984).

Both men and women seem to share the belief that menstruation is associated with negative moods. Parlee (1974) had a group of 25 females and 34 males, between the ages of 18 and 27, rate how they thought most women experience symptoms and mood

changes during the premenstrual, menstrual, and intermenstrual phases. Both male and female participants reported women experience greater symptom changes during the menstrual and intermenstrual phase. Moreover, the men and women reported almost identical patterns of symptoms and symptom changes. Similar results were found with adolescent boys and both pre- and post-menarchial girls (Clark & Ruble, 1978). The finding that symptom ratings between these three groups was strikingly similar indicates that self-reported symptoms associated with the menstrual cycle may reflect cultural stereotypes and these stereotypes are adopted at a fairly young age.

In the above studies, subjects were aware they were participating in a study designed to investigate the relationship between mood and the menstrual cycle. Using a different methodological approach, several studies have kept subjects blind to the intent of the study. For example, Parlee (1982) had seven normally menstruating women, who were unaware they were participating in a study on the menstrual cycle, monitor symptoms for 90 consecutive days. Time series analysis of the daily record revealed few significant fluctuations in mood. Analysis of group data indicated several symptoms, including confusion, depression, and fatigue, were lower during the premenstrual phase. After daily monitoring was completed, the women were asked retrospectively report the symptoms they experienced during the premenstrual phase of their cycle. The retrospective reports contradicted the results from the prospective daily reports. On the retrospective reports, women reported they felt anxious, depressed, irritable, and tense

during their premenstrual phase, even though their daily reports showed no increases in these moods premenstrually.

The results of Parlee's study imply that premenstrual tension is a fallacy or a societal myth. The sample size of this study is too small to conclude premenstrual tension is only a figment of a woman's imagination; however, results indicate some women retrospectively report they suffer from premenstrual tension, possibly because of a learned association between negative moods and the menstrual cycle, when in fact their moods are similar in all phases of their cycle.

In a similar study, 118 female nurses monitored 47 symptoms, from the Moos Premenstrual Distress Questionnaire, on a daily basis for eight weeks (Slade, 1984). Subjects were told they were participating a study of general health. Results showed pain and water retention were experienced premenstrually and menstrually, but there were no cyclic changes in concentration or emotions. A number of women showed elevations in symptoms during the menstrual and premenstrual phases, but the frequency of these occurrences was not beyond chance variation. Slade stated, "the results question whether higher levels of emotions premenstrual or menstrually are a genuine feature of a the normal female experience" (p.6).

Slade further suggested that women attribute negative chance occurrences, which normally are attributed to personal or environmental circumstances, to the premenstrual phase of their cycle. For example, a woman may respond differently to a small accident, such as starting a kitchen fire or cutting herself with a knife, when she is in the

intermenstrual phase as compared to when she is the premenstrual phase of her cycle. If the woman is intermenstrual, she may attribute the accident to lack of coordination, stupidity, or some external distraction (e.g., a small child). But, the same woman, when she is in the premenstrual phase of her cycle, may attribute this accident to an inability to concentrate due to premenstrual tension. This process maintains the belief that negative emotions are associated with the menstrual cycle. Slade acknowledged that the population in her study was young and healthy and may not have adequately represented the female population.

In a well controlled study, Olosov and Jackson (1987) directly manipulated negative expectancies by exposing 126 college women to information which posited either a biological or psychological view of PMS. These researchers found the biological information, which stated PMS was due to uncontrollable hormonal fluctuations, increased negative expectations while the psychological information, which stated PMS was due to self-fulfilling prophesies and negative expectations, decreased expectancies. In addition, individuals exposed to the biological information reported more negative moods on a daily mood measure during their premenstrual phase than those exposed to the psychological information.

Kudlas (1987) attempted to replicate these results with college students who reported at least moderate levels of premenstrual suffering. In addition, Kudlas hypothesized that changes in expectations were related to perceived feelings of being in control. Seventy-one subjects were randomly assigned to one of the following

conditions: (1) psychological (2) biological-passive (3) biological-active and (4) control. Like Olosov and Jackson's study, the psychological information stated premenstrual symptoms were related to increased negative expectancies, and the biological-passive information claimed PMS was related to uncontrollable hormonal fluctuations. The biological-active condition stated PMS was due to physiological factors, but information on how to treat or "control" premenstrual suffering (e.g. increasing exercise or making dietary changes) was also provided.

All subjects completed questionnaires measuring premenstrual expectancies and perceived control of premenstrual distress before the information was presented, immediately following the information presentation, and at a 40 day follow-up period. Subjects were also asked to monitor moods and symptoms associated with PMS daily. Both the biological-active and psychological groups were hypothesized to increase feelings of control and reduce negative expectancies, while the biological-passive group was expected to have higher negative expectancies and lower perceived feelings of control. Daily ratings of symptoms were expected to be higher for the biological-passive group compared to the biological-passive group.

Results of Kudlas's study supported the hypothesis relating expectancies to perceived feelings of control. Feelings of control were highly correlated with expectancies. Greater feelings of control were related to lower negative expectancy scores. Women in the biological-active and psychological group reported increased feelings of control and lower negative expectancy scores, while women in the

biological-passive condition reported higher negative expectancy scores and decreased feelings of control. Unfortunately, there were no significant changes on the daily measure. Kudlas suggested that negative expectancies or perceived feelings of control may play a larger role in the experience of premenstrual tension for mild PMS suffers, while having little effect on the symptoms of women who suffer severely.

In general, studies looking at negative expectancies have not included women who suffer severely from premenstrual distress. Thus, using this theory to explain LLPDD is questionable. However, this theory does have treatment implications, since it suggests that changing one's thoughts about the premenstrual phase and menstruation will reduce suffering.

Stress. Rather than looking at PMS as a figment of one's imagination" or as conditioned negative beliefs, it may be hypothesized that symptoms related to the menstrual cycle are exacerbated by stressful events, poor relationships, or previous traumatic events. Stress is unlikely to be the sole cause of LLPDD or PMS, but it may impact the presentation or magnitude of symptoms (Norris & Sullivan, 1983). Recent studies looking at the relationship between stress and symptom severity are mixed.

In a study looking directly at stressful events, marital satisfaction and PMS, Coughlin (1990) compared women who reported varying levels of premenstrual distress as rated retrospectively using the Moos Premenstrual Distress Questionnaire (Moos, 1968b). Results indicated those reporting the greatest amount of premenstrual distress also reported the highest scores on the Recent Life Events Scale (Holmes & Rahe, 1967),

but there were no significant differences between high suffers and low or non-suffers on a measure of marital satisfaction.

A study by Beck, Gevirtz, and Mortola (1990) was unable to demonstrate a relationship between psychosocial stress and premenstrual symptom severity. Twenty-five women with LLPDD monitored their premenstrual symptom and daily stress for three menstrual cycles. Serving as their own controls, only eight women experienced the greatest amount of emotional distress during the cycle that they also reported the greatest amount of stress. Nine subjects experienced the greatest amount of emotional distress during the cycle in which they reported the least amount of stress. The other eight subjects reported the greatest amount of emotional distress during the cycle they reported an intermediate amount of stress. Similar results were found for physical symptoms.

Premenstrual tension may also be related to traumatic events such as sexual or physical abuse. Miccio-Fonseca, Jones, and Futterman (1990) found that sexually traumatized women reported significantly greater premenstrual symptomatology than non-traumatized women. Miccio-Fonseca and associates suggested that "victims" may be less able to cope with negative symptoms associated with the menstrual cycle.

The concept that traumatized individuals have difficulty coping with symptoms frequently associated with the menstrual cycle is intuitively appealing, and it implies that interventions such as relaxation therapy, psychotherapy, or stress management may be effective in relieving premenstrual suffering. Unfortunately, the effect of "stress" on

symptom severity in women who meet diagnostic criteria for LLPDD remains unclear. Coughlin's study suggests women suffering from PMS experience more life stressors than women who do not appear to suffer from PMS, but the study by Beck et al. indicates level of stress experienced during various cycles does not directly impact symptoms severity. Miccio-Fonseca and associates, who found sexually traumatized woman are more likely to report premenstrual symptoms, explain their results not in absolute stress levels, but suggest "learning not to cope" results in higher premenstrual symptomatology.

Summary of Psychological Theories. A comprehensive theory by Morse and Dennerstein (1988, cited in Morse, Dennerstein, Farrell, & Varnavides, 1991) may be the best way to summarize the information presented above. Morse and associate propose that a woman's premenstrual symptoms are primarily related to cognitive processing. Although these theorists acknowledge negative changes associated with the "physical" aspect of the menstrual cycle occur, they assert that negative thoughts and anticipated behavioral deficits (or negative expectancies) lead to negative emotional responses, adverse changes in the self-concept, and an under utilization of coping skills. In other words, these women learn to be helpless. Failing to cope also decreases feelings of self-worth and self-efficacy (or feeling like one can cope). As a result, changes associated with the menstrual cycle are more likely to be interpreted negatively, and this pattern of negative thoughts becomes cyclical.

Treatment of PMS or LLPDD

Treatments for PMS have been derived primarily from physiological theories of this disorder. Numerous medications and vitamins have been suggested (Chakmakjian, 1983; Price, 1990; Robinson & Garfinkel, 1990; Rubinow & Roy-Byrne, 1984). Changing behavioral practices or health habits such as exercise and dietary change have also been recommended as ways to alter some underlying physical cause of PMS. Psychological treatment such as cognitive therapy and relaxation therapy have only recently been explored. Although research in this area is growing, the best approach to treating PMS or LLPDD remains unclear.

Medication and Vitamins.

Progesterone therapy. A highly publicized treatment of PMS is progesterone therapy, even though it's effectiveness is extremely questionable. Progesterone therapy is based on the pioneering research Katherine Dalton (1964), who strongly advocates the use of this medication in the treatment of PMS. Initial uncontrolled studies supported the effectiveness of progesterone (Greene & Dalton, 1953; Rees, 1953). However, a number of double-blind placebo-control studies have failed to show that progesterone is more effective than a placebo (Andersch & Hahn, 1985; Maddocks, Hahn, Moller, & Reid, 1986; Sampson, 1979; Van der Meer, Benedek-Jaszmann, & Van Loener, 1983).

Despite the lack of evidence to support progesterone as an effective treatment for PMS, it continues to be a common prescription for this disorder. Even reviewers who

clearly state there is no evidence to support the effectiveness of progesterone often suggest that this treatment should be tried (see Robinson & Garfinkel, 1990). This seems like a dangerous practice since the long term effects of progesterone use are unknown (Harrison, 1985; Robinson & Garfinkel, 1990).

Progestins. The effectiveness of synthetic progestins in the treatment of PMS has also been explored. Dydrogesterone, a chemically similar optical isomer of progesterone, was reported to be more effective than a placebo by Haspel (1981). However, Robinson and associate (1990) reviewed this data and concluded that dydrogesterone was not superior to placebo in alleviating symptoms of moderate or severe PMS. The effectiveness of dydrogesterone was tested in a double-blind, placebo-controlled, crossover study conducted by Sampson, Heathcote, Wordsworth, Prescott, and Hodgson (1988). Their sample size was relatively large (n=69) and subjects were required to prospectively monitor symptoms using the Moos Premenstrual Distress Questionnaire (Moos, 1968b). Sampson and associates reported dydrogesterone was no more effective than the placebo except for menstrual pain associated with bleeding. These researchers also reported that dydrogesterone tended to increase breast tenderness.

Diuretics. Hypothesizing that emotional and physical symptoms (e.g. weight gain, edema) of PMS are related to water retention, a number of researchers have tested the effectiveness of diuretics. With the possible exception of spironolactone, diuretics appear to be ineffective in the treatment of PMS (Robinson et al., 1990).

Spironolactone is a diuretic, but it may be effective because it is an aldosterone antagonist. Two studies have found spironolactone to be effective in relieving premenstrual suffering. O'Brien, Craven, Selby, and Symonds (1979), who studied 18 women with prospectively confirmed PMS, found spironolactone to be effective in reducing premenstrual symptoms in over 80% of the symptomatic group.

Aslaksen and Falk (1991) conducted a double-blind, placebo-controlled, crossover study to test the efficacy of spironolactone in the treatment of PMS. Askaksen and associate reported spironolactone was not more effective than the placebo in treating PMS in the first month of treatment, but that spironolactone was significantly more effective in reducing breast tension, headache, anxiety, and lethargy in the second cycle of treatment.

Dopamine Agonists. Dopamine agonists, such as bromocriptine, have been used to suppress prolactin levels. The use of this medication follows the theory relating raised prolactin levels to premenstrual suffering. Anderson, Larsen, Steenstrup, Svendstrup, and Nielsen (1977) conducted a double-blind placebo-controlled study. They found all premenstrual symptoms improved significantly, but only mastodynia or breast tenderness improved more than a placebo. Criteria for patient selection was not clearly specified. Similar results were found in another controlled study (Elsner, Buster, Schindler, Nessim, & Abraham, 1980). Results suggest bromocriptine may be effective in treating premenstrual breast tenderness, but is not very effective in treating other premenstrual symptoms.

Oral Contraceptives. Early studies suggested oral contraceptives were effective in minimizing premenstrual suffering (Herzberg, Johnson, & Brown, 1970; Kutner & Brown, 1972, Moos, 1968a). Price (1990) pointed out these studies may not have adequately distinguished premenstrual symptoms from dysmenorrhea, and symptoms of dysmenorrhea are greatly reduced with the use of oral contraceptives. Controlled studies have not found oral contraceptives to be superior to a placebo in the treatment of PMS (Cullberg, 1972; Goldzieher, Moses, Averkin, Scheel, & Taber, 1971; Morris & Udry, 1972; Silbergeld, Brast, & Nobel, 1971).

Vitamin Therapies. Vitamin B was initially proposed to be an effective, harmless treatment of PMS, but the efficacy and "safety" of this prescription is questionable. Two uncontrolled studies found vitamin B to be effective in alleviating premenstrual symptoms (Baumblatt & Winston, 1970; Kerr, 1977). A controlled study also found vitamin B to be more effective than a placebo (Abraham & Hargrove, 1980). On the other hand, two controlled studies found that vitamin B and a placebo were equally effective in reducing premenstrual distress (Hagen, Nesheim, & Tuntland, 1985; Stokes and Mendels, 1972). In addition to the possibility that vitamin B is an ineffective treatment of PMS, large doses of vitamin B may have deleterious effects. For example, Schaumburg et al. (1983) reported that large does of vitamin B may cause peripheral neuropathy.

Anti-depressant and anti-anxiety medication. Although PMS or LLPDD has been associated with affective and anxiety disorders (e.g. Warner, Bancroft, Dixson, & Hampson, 1991), comparatively few studies have looked at medications typically used

to treat affective and anxiety disorders as a treatment for PMS (Robinson et al., 1990). Robinson and Garfinkel (1990) noted that controlled studies of monoamine oxidase inhibitors or tricyclic antidepressants in the treatment of PMS are conspicuously absent from the literature.

Given the cyclic nature of PMS, a couple of controlled studies have examined the effectiveness of lithium carbonate. Singer, Cheng and Schou (1974) reported lithium and a placebo to be equally effective in reducing premenstrual distress. Steiner, Haskett, Osmun, and Carroll (1980) found lithium to be ineffective in reducing premenstrual symptoms, and most of the women in their study complained of side effects from the medication.

Several very recent studies have looked at the effectiveness of medications (e.g. fluoxetine, fluvoxamine, d-fenfluramine) which block the reuptake of serotonin. One double-blind controlled study reported no significant differences between fluvoximine and a placebo (Veeninga, Westenberg, & Weusten 1990), while two other controlled studies reported fluoxetine to be significantly more effective than a placebo in treating LLPDD (Rickels, Freeman, Sondheimer, & Albert, 1990; Stone, Pearlstein, & Brown, 1991). The two studies which reported significant differences between fluoxetine and a placebo used daily monitoring measures to assess symptom reduction and subjects met strict criteria for LLPDD. Veeninga and colleagues had subjects monitor symptoms on the 4th, 12th, 22nd, and 28th day of their menstrual cycle. Having subjects monitor on specific days instead of using daily measures may have created demand characteristic so

that those in the placebo group also reported reduced symptomatology. Thus, the differences in assessment procedures may account for the contrasting results of these studies.

Another medication that blocks the reuptake of serotonin is d-fenfluramine. Brzezinski et al. (1990) examined the effectiveness of this medication in the treatment of premenstrual depression. A double-blind, multiple-crossover study was performed where each subject received the active medication or placebo over six menstrual cycles. Results suggested d-fenfluramine was significantly more effective in reducing premenstrual depressive symptoms than a placebo as measured by the Hamilton Rating Scale (Hamilton, 1967). In addition, d-Fenfluramine appeared to reduce fat and carbohydrate consumption, which Brzezinski and associates found to be significantly elevated in the premenstrual phase during a baseline period. This study did not use prospective ratings of symptoms, but it did confirm that depressive symptoms were higher during the premenstrual phase compared to the intermenstrual phase.

Alprazolam, known for its anti-depressant and antianxiety qualities, was used as the active medication in a double-blind, placebo-controlled study (Harrison, Endicott, Rabkin, Nee, & Sandberg, 1987). The 34 women in this study, who met strict research criteria for LLPDD, rated alprazolam to be more effective than the placebo (73% versus 12%). Similar results were found in a double-blind, placebo-controlled, multiple-crossover study (Smith, Rinehart, Ruddock, & Schiff, 1987).

Summary of Medications. Many of the medication suggested to treat PMS or LLPDD do not appear to be more effective than a placebo. These include progesterone, progestin, lithium, and oral contraceptives. Moreover, these medications may have harmful side effects. The effectiveness of Vitamin B is questionable since results of controlled studies are mixed. Vitamin B may also have harmful side effects (e.g. peripheral neuropathy) if large doses are taken.

Results from controlled studies suggest that several medications may be effective in relieving premenstrual suffering. Those that block the reuptake of serotonin such as fluoxetine or d-fenfluramine show promise. Alprazolam and spironolactone may also be effective. Given the small number of studies in this area, the effectiveness of these medications is far from conclusive. Additional controlled studies are needed.

Although various medications may be helpful in alleviating premenstrual distress, many women prefer not to take medication. Medications may be expensive or may only be marginally effective. Thus, other treatments of PMS or LLPDD need to be researched. These treatments include psychological (social support, cognitive therapy, relaxation therapy) and behavioral treatments (changing exercise or diet).

Psychological Treatments

Moving away from a "medical" treatment of PMS or LLPDD, psychological treatments focus on social support, changing negative cognitions, or teaching patients to cope better with stress. There is a paucity of research in this area. The effectiveness

of social support is merely speculative. Controlled studies looking at the effectiveness of cognitive therapy and relaxation training in treating PMS or LLPDD are mixed.

Social Support. Levitt, Freeman, Sondheimer, and Rickels (1986) suggest group support may be helpful in alleviating premenstrual suffering. They recommend information be given on psychological, nutritional, and medical components of PMS. The goals of group support sessions include: (1) providing a forum for each woman to learn she is not alone with her symptoms and (2) discussing how patients have learned to cope with PMS. Three themes need to be addressed including guilt about PMS, fear of asserting needs, and alienation because of symptoms. Levitt and associates state group support sessions improve self-image and reduce stress which make PMS more manageable. Research is needed to empirically test this treatment method.

Cognitive Therapy. Only a couple of studies have looked at treating PMS or LLPDD with cognitive therapy. Byers (1986) compared the effectiveness of education versus education plus cognitive therapy in the treatment of PMS. Cognitive therapy worked on reducing negative or irrational beliefs associated with the menstrual cycle. Educational information about PMS was presented in a lecture to both groups. Each of the treatment groups met for 12 weeks. The education group met for one hour, while the cognitive therapy plus education group met for 2 hours. Fourteen women, who were self diagnosed as having PMS, were randomly assigned to each group. Byers (1986) found cognitive therapy and education were more effective than education alone in relieving depression and reducing number of dysfunctional attitudes. However, reports

of negative PMS symptoms, which were measured using prospective daily ratings on the Moos Menstrual Distress Questionnaire (Moos, 1968b), did not decrease with lower levels of depression.

Morse, Dennerstein, Farrell, and Varnavides (1991) compared three treatments of LLPDD: (1) hormone therapy (progestin), (2) relaxation therapy, and (3) coping skills training. Subjects in the hormone therapy group took 10 mg. of dydrogesterone twice a day on days 17 to 27 of an adjusted cycle. Relaxation therapy was provided by audiotape. Subjects in this group were instructed to practice daily on their own for the next three months. Coping skills training included rational emotive therapy (RET), problems solving, and assertiveness training. This therapy helped patients reduce negative emotions, cope with interpersonal relationships, and deal effectively with frustrating social situations. The coping skills groups were conducted in 10 sessions which lasted 90 minutes. Morse and associates randomly assigned 84 subjects to each of the three conditions. Fourteen women completed the hormone therapy treatment, 16 women completed the coping skills group, and only four subjects completed treatment to follow-up for the relaxation group. Thus, the relaxation group was excluded from final analysis.

Morse and associates had subjects monitor typical symptoms of PMS daily for the course of 5 menstrual cycles (one cycle prior to treatment or baseline, 3 cycles during treatment, one cycle following treatment or follow-up). The symptoms included: restlessness, headache, sore breasts, depression, aggression, hot flushes, well-being,

irritability, sex interest, and swelling. Scores from the seven days prior to menses were tabulated for each symptom at baseline and follow-up. Separate repeated measures ANOVAs were conducted for each symptom. Results revealed those in the hormone therapy group only had significant reductions in restlessness and depression. Those in the coping skills group reported significant improvements in several areas including sore breasts, depression, well-being, and irritability. Failure of those in the relaxation training group to complete treatment to follow-up may have been related to minimal personal contact with the therapist and/or the expectation that patients would practice relaxation on their own. Morse and colleagues concluded coping skills training, a form of cognitive therapy, was more effective than hormone therapy or relaxation therapy in the treatment of LLPDD.

The effectiveness of cognitive therapy in the Morse et al. study may be related to the combination of cognitive therapy techniques. Cognitive therapy in Byers' study looked primarily at changing irrational or exaggerated beliefs, while the study by Morse et al. combined RET with problem solving and assertiveness techniques. Differential results may have also been related to the subject population or assessment measures. Byers' subjects were self-diagnosed with PMS, while the subjects in the study by Morse and associates were diagnosed with LLPDD using prospective measures. Byers measured premenstrual symptoms by taking a total symptom score from the Moos Menstrual Distress Questionnaire (Moos, 1968b), while the Morse et al. study looked at individual symptom items. Looking at each item individually may have increased their

chances of finding significant results. Further research is greatly needed in this area to support the effectiveness of cognitive therapy in the treatment of LLPDD.

Relaxation Therapy. In addition to the study mentioned above, a couple of controlled studies have evaluated the effectiveness of using relaxation therapy or biofeedback in the treatment of PMS or LLPDD. Comparing biofeedback training (12 sessions) to an attention control group (one session), Konandreas (1989) found no significant changes in either group for premenstrual symptoms. Symptoms in this study were rated retrospectively. However, results indicated overall mood increased significantly for those in the biofeedback group as rated on the Profile of Mood States Rating Scale (POMS).

Goodale (1989) compared a relaxation group, which practiced relaxation techniques twice a day, to two control conditions: (1) a group that read twice a day and (2) a group just charting symptoms. Goodale reported a 50-60% reduction in symptoms for the relaxation group, a 2-40 % reduction for the reading group, and a 5-20 % reduction for the charting group. Goodale concluded relaxation was an effective treatment of PMS.

Although Goodale's study suggests relaxation may provide symptom relief for women suffering from LLPDD, this is not supported by the study conducted by Konandreas. Differential treatment effects may have been related to the amount of time spent relaxing. Goodale's study made sure subjects practiced relaxation twice a day, while subjects in Konandreas's were only seen once a week to practice relaxation and

were instructed to practice once a day. Further research is needed to support the effectiveness of relaxation in treating LLPDD. How long, and how many times a day an individual needs to practice relaxation procedures, appears to be an important question for this treatment.

Multi-Component Behavioral Treatment. Kuczmierczyk (1989) presented a case report on a 36-year-old woman who was diagnosed with LLPDD based on two months of prospective ratings. Before treatment the patient complained that she had intense feeling of anxiety and insomnia during her premenstrual phase. Treatment consisted of stress inoculation training, assertiveness training, and marital therapy. Patient reported a reduction in premenstrual symptoms. She was sleeping an average of 7 hours a night, instead of three, during her premenstrual phase. Treatment gains were maintained at three- and nine-month follow-up periods. Controlled studies are still needed in this area.

Changing Health Habits

Authors of PMS self-help books, such as Norris and Sullivan (1983) and Harrison (1985), recommend women with PMS increase their exercise and change their diet. Norris and Sullivan (1983) specifically recommend PMS suffers reduce their consumption of salt, sugar, and caffeine, while increasing their consumption of potassium, magnesium, and zinc. They also suggest that women with PMS increase their consumption of complex carbohydrates and decrease their consumption of fatty foods. There is some supporting evidence that exercise may be effective in relieving

premenstrual suffering. Research on dietary changes in the treatment of PMS or LLPDD is extremely scarce.

Exercise. A number of studies indicate that exercise can alleviate premenstrual tension. Reardon and Snyder (1987) had a group of eight previously sedentary women engage in a walk/jog exercise program three times a week for four months. While the control group (n=5) remained sedentary and showed no change in symptoms, women in the exercise group had a decrease in percentage of body fat (2.1%) and a significant reduction in PMS symptoms (64%). Unfortunately, Reardon and Snyder did not report how PMS symptoms were measured, and the initial level of premenstrual suffering was not presented. Moreover, these researchers failed to include a placebo control to see if treatment effects were the result of the exercise program or non-specific treatment factors.

In a similar study, Prior, Vigna, and Alojada (1986) had a group of 8 sedentary women engage in a running exercise program for three months. Compared to a control group (n=6) who remained sedentary, women in the treatment group reported significantly greater decreases in premenstrual symptoms, specifically those related to breast tenderness and fluid retention. The severity of premenstrual symptoms was measured by having subjects complete a questionnaire on days 2-5 of their cycle or during their menstrual phase. The questionnaire asks subjects to compare how they feel now compared to how they felt during the last two weeks. This method of reporting premenstrual symptomatology is questionable since it may create strong demand

characteristics (i.e. subjects assume that they should feel different now compared to their premenstrual phase). Like Reardon and Snyder, Prior et al. failed to include a placebo control group.

A recent study by Lemos (1990) compared the effectiveness of high intensity aerobic training to low intensity exercise in the treatment of moderate to severe PMS. Participants in the high intensity group exercised for thirty minutes, three times a week, at 70-85% of their determined maximum heart rate. Those in the control condition also exercised three times a week for 30 minutes, but they were not allowed to exceed 60% of their maximum heart rate. Treatment lasted for 10 weeks. Premenstrual symptoms in the high intensity group were significantly reduced from the first month of treatment to the 2nd month of treatment, and were significantly lower than the low intensity group. Results suggested treatment was more effective at relieving emotional symptoms than physical symptoms or cognitive/attentional symptoms. A reduction in stress and body fat was reported for both groups.

Caffeine. Norris and Sullivan (1983) recommended caffeine be reduced for PMS sufferers because it may increase the body's need for B vitamins. They further asserted that caffeine increases the severity of breast swelling, engorgement, and tenderness. In a large retrospective study of female college sophomores, Rossignol (1985) estimated the prevalence of premenstrual tension in high, moderate, and low consumers of caffeine-containing beverages. She found the prevalence of premenstrual tension was much higher in women who consumed more than 15 caffeine-containing beverages daily

than in those who drank less than this amount. Those women who stated that they experienced severe or moderate premenstrual symptoms reported that they consumed a larger quantity of caffeine than those who reported only mild symptoms.

In a more recent study, Rossignol and Bonnlander (1990) conducted another large (n=841) retrospective study and found a dose-dependent relationship between caffeine consumption and reported premenstrual distress. These researchers reported an odds ratio of 1.3 for consumers of one cup of a caffinated beverage per day, and an odds ratio of 7.0 for those consuming 8 to 10 cups a day.

Although the above results indicate there is a relationship between caffeine consumption and premenstrual tension, there is no strong evidence linking a reduction in caffeine with reduced premenstrual distress (Casey and Dwyer, 1987). In fact, higher caffeine consumption may be a response to suffering from PMS.

Sodium. Norris and Sullivan (1983) also recommended sodium or salt be reduced, especially for women who suffer from fluid retention (swelling of the face, hands, feet, breasts). Nissenbaum (1983) conducted a double-blind placebo-controlled study designed to investigate the effect of reduced dietary salt on PMS. After two baseline cycles, subjects started a reduced sodium diet. For the next two cycles, half the subjects took four grams of salt per day in capsule form, thus replacing salt removed from their low sodium diets. The remaining half were given capsules containing no salt and they also were instructed to take capsules daily. After two months a crossover took place. Subjects in the placebo condition were switched to the treatment condition, and

those in the treatment condition were given the placebo. Thus, all subjects participated in the three conditions: baseline, placebo, and treatment. Nissenbaum reported subjects did not show expected menstrual cycle patterns in any of the three conditions, which indicated her population of women did not suffer from PMS. Consequently, she found no significant premenstrual differences between baseline, placebo, and treatment. However, when measures were averaged across each cycle, both anxiety and weight were significantly lower during treatment than during baseline.

Sugar. Brody (1981) identified calories as the only as the only nutritional value in sugar and sugar consumption is related to tooth decay, obesity, diabetes, hypoglycemia, and heart disease to sugar consumption. Additionally, consumption of sugar results in the production of insulin from the pancreas. If the pancreas over-produces insulin, the body becomes drained of glucose, a condition called hypoglycemia or low blood sugar. Since cells need glucose to function, the glucose starvation can result in faintness, weakness, tremulousness, blurred vision, hunger, palpitations, fast pulse rate, sweating, tightness in the throat, restlessness, irritability, and anxiety.

Norris and Sullivan (1983) point out that many women with PMS have been misdiagnosed with hypoglycemia. As mentioned earlier, it has been hypothesized that glucose intolerance may be responsible for premenstrual distress. Despite this theory, Norris and Sullivan do not recommend a classic hypoglycemic diet because this diet is high in protein, calcium, and fat (products which they believe exacerbate premenstrual

symptoms). Instead, they recommend complex carbohydrates and foods high in certain minerals (potassium, magnesium, and zinc) and vitamins (B Vitamins). Although they do recommend that women reduce their intake of sugar, there is no evidence to show that the reduction of sugar intake does in fact decrease premenstrual symptoms.

Magnesium. Taking magnesium supplements or increasing the consumption of foods containing magnesium has also been suggested by Norris and Sullivan (1983) as a means of treating PMS. Magnesium deficiency may explain symptoms such as mood swings, nervous tension, bloating, and breast tenderness. Despite some evidence the PMS sufferers consume less magnesium than women who do not suffer from PMS (Gallant et al., 1988), treating PMS with magnesium supplements has not been empirically tested.

Potassium. PMS self-help books also recommend that women with PMS increase the amount of potassium and zinc in their diets. Potassium is critical to muscular contraction and nerve stimulation, and it regulates the body's water balance. Insufficient potassium may lead to body cramping. There needs to be a balance of potassium with sodium or muscle and nerve functioning suffer. An excess of either substance results in loss of both in the urine. High salt diets result in a potassium deficiency. Women taking diuretics to prevent water retention may also be depleting their potassium reserves (Norris & Sullivan, 1983). There is no firm evidence to indicate increasing potassium levels would be effective in treating PMS or LLPDD.

Zinc. Zinc plays a role in the synthesis of proteins and DNA and RNA (Norris & Sullivan, 1983). Zinc is required for the formation of connective tissues and cell growth. Zinc functions as a "traffic cop", policing the metabolism of various minerals such as copper, magnesium, and selenium. Zinc levels may be depleted by copper, estrogen, or diuretics. Although increasing zinc has been recommended in self-help books, there is no evidence to indicate that increasing the level of zinc would be effective in reducing premenstrual suffering.

Treating PMS with a Low-Fat Diet. Having women with PMS reduce their fat intake while increasing their intake of complex carbohydrates may alleviate premenstrual distress. Although hidden among the other dietary suggestions mentioned above, Norris and Sullivan (1983) tell PMS suffers to limit their intake of fat to less than 20 percent of their total caloric intake and to increase their consumption of complex carbohydrates to 50-65% of their total caloric intake. There appears to be some theoretical and empirical support for this suggestion.

A low-fat diet may be effective in relieving PMS symptoms by decreasing hormone levels. As mentioned previously, PMS may be related to raised levels of both estrogen and/or progesterone (e.g. Hammarback, Damber, and Backstrom, 1989). Several studies have noticed that estrogen levels are lower in populations consuming less fat in their diet (Armstrong et al., 1981; Goldin, et al., 1982; Goldin et al., 1986; Gorbach & Goldin, 1987; Shultz & Leklem, 1983). Dietary changes in fat consumption also appear to decrease estrogen levels (Ingram, Bennett, Willcox, & de Klerk, 1987).

Studies interested in the relationship between fat consumption and hormone levels among human females have typically compared vegetarian with non-vegetarian women or looked at racial differences. Fat consumption is assumed to be lower in vegetarian women. Armstrong et al. (1981) found lower urinary levels of estriol and total estrogens in vegetarian postmenopausal women compared to non-vegetarian postmenopausal women. Similarly, Golden et al. (1982) compared 10 vegetarian premenopausal women with 10 nonvegetarian premenopausal women and found urinary excretion of estriol was significantly lower in vegetarians. Shultz and Leklem (1983) found plasma levels of estrone and estradiol in Seventh Day Adventist vegetarian women were significantly lower than those of Seventh Day Adventist nonvegetarian women.

Fat consumption is also lower in Asian women compared to Caucasian American women. A study comparing Caucasian American women with immigrant women of Asian descent reported the Asian women consumed less fat in their diets and had lower levels of plasma estrone and estradiol concentrations than the Caucasian women (Goldin, et al., 1986).

Testing the hypothesis that a low-fat diet changes hormonal levels, Ingram and associates (1987) randomly placed thirty-three premenopausal women into a low-fat group or a standard diet group. After two months, these women were crossed over into the alternative diet for another two months. In the low-fat diet condition, subjects derived 20 percent of their calories from fat. In the standard diet condition, participants

derived 40 percent of their calories from fat. The low-fat diet significantly decreased levels of estradiol, testosterone, and cholesterol.

A low-fat diet has been found to be effective in treating cyclical mastopathy (severe breast tenderness and swelling), a common symptom of PMS (Rose et al., 1985; Boyd, et al., 1988). Rose et al. (1985) had ten women reduce their fat consumption to 20 percent of their total caloric intake over a three month period. Serum estradiol levels decreased by 28%, estrone by 36%, and prolactin by 29%. All ten women reported improvement in breast pain.

Boyd et al. (1988) randomly placed nineteen women suffering from cyclical mastopathy into the treatment or the control group. Members of the treatment group were told to reduce their fat intake to 15 percent of their total calories while increasing their carbohydrate intake to 65 percent of their total calories. Subjects in the control group were advised on the principles of a healthy diet based upon Canada's Food Guide, but were not instructed to change their dietary fat intake. Self-reports on the severity of breast tenderness and swelling were significantly lower for the intervention group compared to the control group. Physicians, who were blind to group assignment, reported swelling, tenderness, and nodularity had decreased in 6 of the 10 women in the intervention group and two of the nine women in the control group. None of the women in the women in the intervention group reported an increase in symptomatology while one woman in the control group reported increased suffering.

Increasing carbohydrate consumption may also reduce premenstrual suffering. Recent evidence suggests carbohydrate consumption can immediately relieve emotional distress associated with the premenstrual phase (Wurtman, Brzezinski, Wurtman, & Laferrere, 1989).

In sum, a low-fat diet may be effective in reducing premenstrual suffering by reducing estrogen levels. Increasing carbohydrate consumption may be effective in increasing the level of serotonin, and thus improving premenstrual symptoms. A controlled study indicated a low-fat diet is effective in relieving at least one symptom of PMS, breast tenderness. Theoretically, it seems possible a low-fat diet may also affect emotional and other physical symptoms of PMS.

Summary of Dietary Interventions. Many dietary suggestions have been recommended by physicians and self-help books, but these recommendations have not been empirically tested. There is little empirical evidence to suggest the common recommendations of reducing caffeine, salt, and sugar, while increasing magnesium, potassium and zinc will be effective in treating PMS or LLPDD.

Treating LLPDD or PMS with a low-fat diet appears to be more promising. Several studies suggest a low-fat diet will reduce estrogen levels. High levels of estrogen are hypothesized to be responsible for premenstrual suffering. In addition, research suggests a low-fat diet is effective in treating cyclical mastopathy or severe breast tenderness, which is a common symptom of PMS and LLPDD. Although the effectiveness of treating other physical symptoms and emotional symptoms of PMS or

LLPDD is highly speculative, it deserves to be empirically tested for a number of reasons. First, it may prove to be an effective treatment of LLPDD that provides other health benefits (e.g. reduction in blood cholesterol). Secondly, a low-fat diet intervention is also an inexpensive treatment which should have few side effects. Finally, this treatment is already being employed by a number of women who read self-help books or who have been told by various professionals to try this treatment as an option.

Chapter II

Methodology and Results

Premenstrual tension may be caused by high estrogen levels. A low-fat diet, which decreases estrogen levels, may reduce premenstrual suffering. Research indicates a low-fat diet is an effective treatment of cyclical mastopathy, a common symptom of PMS or LLPDD. Thus, it seemed plausible a low-fat diet would also alleviate other PMS or LLPDD symptoms. The purpose of the proposed study is to empirically test if a low-fat diet, which decreases fat consumption to 20% of total caloric intake and increases carbohydrate consumption to 65% of total caloric intake, is effective in treating LLPDD. It was hypothesized that a Low-Fat Diet intervention would be effective in reducing both emotional and physical symptoms of LLPDD.

Several researchers have suggested placebo effects are high for women suffering from PMS and LLPDD, thus it was important to include adequate control groups. This study included an Education-Support group, which provided support and gave information about PMS, and a Waiting-List control group, which did not receive treatment until PMS symptoms were rated prospectively for three menstrual cycles. The Education-Support Group was designed to be minimally effective, so that it could serve as a "quasi-placebo control group". It was hypothesized that the Education-Support Group would significantly reduce emotional symptoms of PMS compared to the Waiting-List Control Group, but not physical symptoms of PMS. In addition, it was hypothesized the Low-Fat

Diet Intervention would be significantly more effective in reducing physical and emotional symptoms of LLPDD compared to both the Education-Support Group and the Waiting-List Control Group.

Methods

Subjects

Subjects were solicited through a local newspaper advertisement and two mass mailings of flyers to all female employees at a large southern university. The flyers, which were sent through intercampus mail during the Spring semester of 1988 and the Fall semester of 1989, offered free assessment of PMS and a possibility of free treatment. Flyers were also posted at a nearby community college and a smaller state university in the area.

Forty-four women were solicited from the first round of advertising and twenty women were solicited through the second round of advertising. Women were included in the study provided they met the provisional diagnostic criteria for LLPDD (Appendix A). This was determined through a Structured Interview (Appendix B) which was conducted after informed consent (Appendix C) was obtained. Women on oral contraceptives or anti-depressant medication were allowed to participate provided they had been on this medication for at least two months prior to the intake interview, and they continued on the same medication during the course of the study. Based on

information from the Structured Interview, two of these women were excluded from the study. One woman had a severe eating disorder and was referred for treatment elsewhere and the other appeared to be suffering from dysmenorrhea and did not meet research criteria for LLPDD. The remaining women were randomly assigned to one of three treatment conditions: Low-Fat Diet (LF), Education Support (ES) and Waiting List control (WL).

Thirty-one women did not complete pre-treatment assessment, or daily monitoring of symptoms, and dropped out of the study before treatment began. Two women who completed treatment were excluded from group analyses because they changed their medication during the course of the study. One was in the LF condition and the other was in the ES condition. An additional woman was excluded from the LF group analyses because prospective monitoring strongly suggested she was suffering from cyclical mastopathy, and that she was not emotionally distressed at any time during her menstrual cycle. Two women in the LF condition were excluded because they did not provide any post-treatment assessment.

A total of 26 Caucasian women, who completed treatment and prospective monitoring of physical and emotional symptoms, were included for group analysis. The number of subjects in the LF, ES, and WL conditions was 10, 8, and 8, respectively. Subjects ranged from 23 to 46 years of age (Mean age = 35.8). Most of the women were married (57%). Thirty-eight percent had never been pregnant, and 54% had children. Education level was relatively high. All participants had high school degrees.

Twenty-six percent had graduate degrees and an additional 55% had bachelor degrees. Five subjects were on oral contraceptives and 3 subjects were taking anti-depressant medication. Table 1 provides information on the distribution of these demographic variables.

Demographic information on the women who dropped out of the study before treatment began was similar to those who participated for the entire length of this study. Mean age was 32.42 (range 19 to 49). Forty-eight percent of those who dropped out of the study were married. Of the women who dropped out, 58% had children and 32% had never been pregnant. The only apparent difference between the group of women who dropped out before treatment began and those that completed the study was education level. The majority of women who dropped out before treatment began only had high school degree. Sixteen percent of the women who dropped out had graduate degrees and an additional 29% had bachelor degrees.

Materials

Structured Interview. The Structured Interview (see Appendix B) was designed by this author to provide information in four general areas: (1) demographic characteristics, (2) premenstrual symptoms, (3) medical history, and (4) mental status.

Daily Monitoring Forms. Daily Monitoring Forms (See Appendix D) were designed by this author to measure daily moods and physical symptoms (Part I), and behavior (Part II).

Part I - Moods and Physical Symptoms: Part I consists of 18 items which were obtained from three subscales of the Moos Menstrual Distress Questionnaire (MDQ). The MDQ itself was designed as a standard measure of premenstrual symptoms. It consists of 47 items which are rated from 1 (no reaction at all) to 6 (acute or partially disabling). Moos (1968b) factor analyzed these items, and subsequently divided them into eight subscales: Pain, Water Retention, Negative Affect, Arousal, Control, Behavioral Change, and Concentration. The subscales of Negative Affect, Water Retention, and Pain were chosen for the Daily Monitoring Forms because items in these subscales reflect the most common symptoms experienced by women with PMS (Moos, 1985). Moreover, Moos (1968b) found that women reported different scores on these subscales in the intermenstrual, premenstrual, and menstrual phases. Based on a sample of 839 women, Moos (1985) reported the following internal consistency coefficients: Negative Affect ($\underline{r}$=.89), Pain ($\underline{r}$=.74), and Water Retention ($\underline{r}$=.67).

Part II - Behavior: This section of the Daily Monitoring Form was designed to measure behaviors, stress, and subjective ratings of relationships and/or activities. Behavior items were included to measure beverage consumption, chocolate consumption, number of cigarettes smoked, amount of sleep, amount of exercise, number of visits to fast food restaurants, and amount and type of medication taken. Beverages listed on the Daily Monitoring Forms included coffee, tea, soda, beer, wine, liquor, milk, juice, and water. These were included to obtain an estimate of caffeine and alcohol consumption. One item asked subjects to rate how stressful their day was on a scale of 1 (not stressful)

to 6 (very stressful). This section also provided a an opportunity to rate the quality of two relationships and/or activities on a scale of 1 (very poor) to 6(very good). The relationships and/or activities were determined during the structured interview. Subjects selected those activities or relationships that they thought were negatively influenced by their premenstrual symptoms. An additional item was included to determine the onset of menstruation.

Food Diary Form. The Food Diary Form (see Appendix E) asked subjects to list all food items consumed that day. Subjects were also asked the quantity or volume of each food item, how the food was prepared, and the brandname of each food item. Directions for the completion of the Food Diary Form were given on a separate sheet during the initial interview (see Appendix F).

Rating of Treatment Rationale Questionnaire. The Rating of Treatment Rationale Questionnaire (see Appendix G) asked subjects to rate how effective they thought treatment would be in alleviating their premenstrual symptoms. This questionnaire was given at the beginning of treatment, mid-treatment, and at the end of treatment.

Physician Release Form. The Physician Release Form (see Appendix H) gave permission for the experimenter to obtain information from the subject's physician.

Letter to Physician. The Letter to the Physician (see Appendix I) explained the nature of this study and asked the subject's physician to fill out a the Physician Questionnaire Form.

Physician Evaluation Form. The Physician's Questionnaire Form (see Appendix J) asked physicians (1) if there were any reasons why the subject should not participate in this study, (2) if the subject were on a prescribed diet, (3) if the subject were on any prescribed medication, and (4) the last date of examination.

Final Questionnaires: Two final questionnaires were created for the two treatment conditions: Low-Fat Diet Group (see Appendix K) and the Education Support Group (see Appendix L). Both questionnaires asked subjects to estimate the percentage of worsening or improvement of symptoms (-100% to +100%) following treatment. These questionnaires also asked what, if anything, they found helpful about treatment and if there were anything outside of treatment that may have increased or decreased premenstrual symptoms.

Final Questionnaire - Low-Fat Diet Group included questions about adherence to a low-fat diet, weight loss, change in diet habits, what helped change dietary habits, and what inhibited dietary changes.

Final Questionnaire - Education Support Group asked participants if they practiced any of the suggestions in group, such as relaxation techniques, problem-solving, cognitive restructuring, dietary habits, and exercise habits. The questionnaire then asked participants to rank the components of treatment: (1) group support, (2) learning not to feel guilty about suffering from PMS, (3) learning about relaxation techniques, (4) learning how to change "negative" cognitions, (5) learning how to problem-solve, (6) learning about the PMS diet (decreasing salt, sugar, and caffeine, and increasing

magnesium, potassium, and zinc), and (7) learning about exercise. Both questionnaires requested that participants give suggestions on how to improve treatment.

Conditions or Treatments

Low-Fat Diet Group (LF). The focus of the LF treatment was to change the subjects' dietary practices. Subjects in this group were instructed to reduce their dietary fat intake to 20% of their total calories and to increase their carbohydrate intake to 65% of their total calories. The LF group met once a week for eight weeks.

During the first session subjects were given a rational for using a low-fat diet intervention. Their own premenstrual symptoms were discussed briefly. Remaining sessions focussed entirely on changing dietary practices and premenstrual distress was not discussed.

Subjects were taught an exchange program (Cooper, 1988) and were encouraged to monitor food intake daily. Subjects were told to bring in three days of self-monitoring of food intake each week. Subjects were given computerized feedback on their self-monitoring of food intake. This feedback included percent fat, percent carbohydrates, and percent protein.

Subjects in the LF condition were also given information that would help them change their dietary intake. This information included how to eat at restaurants, how to structure their environment (e.g. putting left-over food in opaque containers), how to get family and friends to help, how to read food labels, and how to reward themselves for following the diet. Information on meal planning, low-fat snack foods, and low-fat

recipes were also provided. Subjects were given homework assignments and quizzes so that they would remember and follow the exchange program.

Education and Support Group (ES). The purpose of the ES group was to provide subjects with information and to give them social support for their premenstrual suffering. During the eight, weekly sessions, subjects in the ES group were given a rationale for treatment, provided with information on PMS, and were encouraged to discuss how premenstrual symptoms influenced their lives. Topics included: definition of PMS, physiology of the menstrual cycle, theories of PMS, overview of treatments, the impact of stress on PMS, strains on relationships and jobs, the PMS exercise program, and the PMS diet. Hand-outs were given out during the first seven sessions. Information given during the sessions and on the handouts was obtained primarily from two self-help books, Premenstrual Syndrome by Norris and Sullivan (1983) and Self-Help for Premenstrual Syndrome by Harrison (1985). The PMS exercise program and the PMS diet were discussed during the last two sessions to decrease the possibility that subjects would change their dietary or exercise practices during the course of treatment. Moreover, information on "the PMS diet" consisted of suggestions given by Norris & Sullivan (1983) to decrease sugar, salt, and caffeine intake while increasing their consumption of magnesium, potassium, and zinc. No suggestions were given on how to change their dietary practices, and subjects were not told to decrease their fat intake.

Waiting List Control (WL). The waiting list control group received no treatment during the first three menstrual cycles following the initial interview. The WL group

served as a no-treatment comparison group for the two intervention groups. The WL group controlled for the effects of receiving treatment and repeated testing. It did not control for all threats to internal validity, such as maturation or history, because the WL group did not monitor symptoms through the duration of treatment and post-treatment assessment.

Procedure

Once subjects signed the Consent Form and the Physician Release Form, they answered questions from the structured interview. Information obtained from the structured interview determined whether subjects were eligible to continue participation in the study. Subjects were to be excluded if: (1) they did not meet criteria for Late Luteal Phase Dysphoric Disorder (as indicated by the Structured Interview); (2) they felt they could not predict the onset of their menstruation within 5 days or if their menstrual cycles were longer than 35 days; (3) they were currently suicidal; (4) they exhibited a thought disorder; (5) they were a diabetic or on a prescribed diet; or (5) if they recently (within two month prior to the initial interview) started using birth control or anti-depressant medication.

Following the structured interview, subjects were instructed on how to complete Daily Monitoring Forms and Food Forms. Subjects were instructed to complete Daily Monitoring Forms every night before they went to sleep. They were required to complete Daily Monitoring Forms for at least one menstrual cycle prior to treatment. (Women in the control condition were required to complete daily monitoring for three

consecutive menstrual cycles before receiving treatment). Subjects were asked to hand in or mail their forms weekly.

Subjects were also instructed to complete food forms for three days during the luteal phase of their menstrual cycle and for three days during their follicular stage of their menstrual cycle. The three days of the luteal phase were designated as the Sunday, Monday, and Tuesday before the onset of their period, and the follicular phase was the Sunday, Monday, Tuesday following their period. Subjects were instructed to write down the consumption of any food or beverage immediately and to carry the Food Forms with them on the designated days for monitoring dietary intake. Subjects were instructed to hand in food forms with their Daily Monitoring Form for that week.

Treatment lasted for eight weeks and sessions for both groups were 75 minutes long. Following the first, fourth, and eighth treatment sessions, subjects were given the Rating of Treatment Rational Questionnaire to complete.

After treatment had been completed, subjects continued to monitor their symptoms and behaviors on the Daily Monitoring Forms and Food Forms (as described above) for two full cycles. Once this was completed, an exit interview was scheduled. Subjects were asked to complete their respective final questionnaire. They were then given feedback on any changes in premenstrual symptoms since the beginning of treatment. They were also informed about the other treatment condition. Women in the control condition were provided with group or individual treatment for eight weeks once they completed assessment information for three full menstrual cycles.

Results

Group analyses were performed to assess treatment effects and changes in dietary and other behavioral practices. Ratings of treatment rationale for the two treatment conditions was compared. The correlations between premenstrual symptoms and variables thought to influence premenstrual suffering (i.e. diet, exercise) are presented. Individual cases are discussed briefly at the end of this section to provide further insight and information. (Unfortunately, quality of relationships and/or work performance had to be excluded from final analyses because of insufficient data).

Symptom Ratings.

As indicated earlier, symptoms frequently associated with PMS were monitored on Part 1 of the Daily Monitoring Measure. In addition to a total score, there are three subscale scores: (1) mood, (2) pain, and (3) water retention. Three separate scores were tabulated for the total symptom score and the three subscale scores at the premenstrual, menstrual, and intermenstrual phases. The premenstrual phase consisted of the first four days monitored just prior to the onset of menses. The menstrual phase consisted of the first four days monitored after the onset of menses. Four days mid-cycle were used for the intermenstrual phase. Thus, each score for the following dependent measures represented a four day total: total symptom score, mood subscale score, pain subscale score, and water retention subscale score.

A 3 x 3 x 3 (group x phase x time) mixed factorial design was used to assess treatment effects. The independent variables were: (1) group, (2) menstrual phase, and (3) time. Group, which is a between subjects factor, represents the treatment conditions: Low-Fat (LF), Education-Support (ES), and Waiting-List Control (WL). Menstrual phase, a with-in subjects factor, designated the three phases of the menstrual cycle: premenstrual (four days before menses), menstrual (four days after menses), and intermenstrual (four days mid-cycle). Time, another within subjects factor, represents scores at pre-treatment (cycle prior to treatment), post-treatment (cycle following treatment), and follow-up (two cycles following treatment).

Total symptom score. A 3 x 3 x 3 (phase x time x group) repeated measures ANOVA was calculated for the total symptom score to assess: (1) changes in symptoms across time, (2) differences between the groups, (3) the interaction between group and time, and (4) differences in scores between the phases (see Table 2 and Figure 1). The main effect for time ($F(2,23)=2.1$, $p>.05$) and the interaction effect between group and time ($F(4,46)=1.33$, $p>.05$) were not significant. There was a significant main effect for group ($F(2,43)=3.29$, $p<.05$, Scheffé value=6.8, $\alpha=.05$). Post hoc tests using the Scheffé technique found significant group differences between the intermenstrual and premenstrual phases (see Figure 1 and Table 2).

At post-treatment during the premenstrual phase, the ES group was significantly different than the WL group. Mean scores were lower for the ES group ($\bar{x}=127.2$) than the WL group ($\bar{x}=194.8$). During the premenstrual phase at post-treatment, the ES

group was not significantly different than the LF group ($\bar{x}$=146.0), and the LF group was not significantly different than the WL group. There were no significant differences between the groups during the premenstrual phase at pre-treatment or at follow-up.

There were no other significant group differences during the menstrual phase; however, there were significant differences between groups during the intermenstrual phase at post-treatment and follow-up. At post-treatment, the ES group ($\bar{x}$=87.1) was significantly different than the LF group ($\bar{x}$=112.5), but the ES group was not significantly different from the WL group ($\bar{x}$=106.4). At follow-up the ES group ($\bar{x}$=87.9) was significantly different from the WL group ($\bar{x}$=118.3), but the ES group was not significantly different than the LF group ($\bar{x}$=95.7).

A significant main effect for phase was also found ($F(2,46)=29.72$, $p<.0001$, Scheffé value=6.8, $\alpha=.05$). Premenstrual and menstrual phases did not differ significantly from one another at any time. However, the premenstrual phase and the menstrual phase both differed significantly from the intermenstrual phase at pre-treatment, post-treatment, and follow-up. Mean scores obtained during the premenstrual and menstrual phases were higher than scores during the intermenstrual phase.

Results from the total symptom score suggest the women participating in this study were exhibiting physical and emotional distress related to the menstrual cycle because scores in both the premenstrual and menstrual phases were significantly higher than scores obtained in the intermenstrual phase at all times. There were no significant differences between the premenstrual phase and menstrual phases at any time. In

general, these results suggest emotional and physical distress for the women participating in this study were equally high for the premenstrual and the menstrual phases.

Results from the total symptom score scores further suggest that there was a slight advantage to being in ES group. Women in the education support group reported significantly less severe premenstrual symptoms overall compared to the women in the WL control group at post-treatment. However, these differences were not maintained at follow-up. Women in the ES group also reported less severe symptoms during the intermenstrual phase at post-treatment compared to the LF group, and significantly lower symptoms compared to the WL at follow-up. Thus, effects from participating in the ES treatment condition was not limited to the premenstrual phase.

Scores for women in the LF condition were not significantly different than those in the WL group, and they did not report a significant reduction in symptoms following treatment.

Mood symptom score. A 3 x 3 x 3 (phase x time x group) repeated measures ANOVA was calculated for the mood subscale score to assess: (1) changes in symptoms across time (2) differences between the groups and (3) the interaction between group and time and (4) differences in scores between the phases. There were no significant main effects for time ($F(2,46)=1.19$, $p>.05$) or for group ($F(2,23)=2.58$, $p>.05$). The interaction between time and group was not significant ($F(4,46)=.87$, $p>.05$). Thus, there were no significant group differences or changes across time (see Figure 2 and Table 3).

Similar to the total symptom score, there was highly significant main effect for phase ($F(2,46)=21.11$, $p<.0001$, Scheffé value=6.8, $\alpha=.05$) on the mood subscale score. Premenstrual and menstrual phases were not significantly different from one another at any time. The premenstrual phase was significantly different from the intermenstrual phase at all times during the study. Mean scores obtained during the premenstrual phase were higher than mean scores obtained during the intermenstrual phase. The menstrual phases differed significantly from the intermenstrual phase at pre-treatment and post-treatment, but not at follow-up. Mean scores obtained during the menstrual phase at pre-treatment and post-treatment were higher than means scores obtained during the intermenstrual phase.

Results from the mood subscale score suggest the women participating in this study were exhibiting emotional distress related to the menstrual cycle because scores obtained during the premenstrual phase were always significantly higher than those during the intermenstrual phase. In addition, scores obtained during the menstrual phase were significantly higher than the intermenstrual phase except at follow-up. There were no significant differences between the premenstrual phase and menstrual phases at any time, suggesting emotional distress for the women participating in this study was equally high for the premenstrual and the menstrual phases.

Unfortunately, negative emotional symptoms were not significantly reduced following treatment for either the LF or ES groups.

Pain symptom score. A 3 x 3 x 3 (phase x time x group) repeated measures ANOVA was obtained for the pain subscale to assess (1) changes in symptoms across time, (2) differences between the groups, (3) the interaction between group and time, and (4) differences in scores between the phases. There was no significant interaction effect between time and group ($F(4,46)=1.25$, $p>.05$). However, there were significant main effects for group ($F(2,23)=3.98$, $p<.05$, Scheffé value=6.8, $\alpha=.05$), and for time ($F(2,46)=3.99$, $p<.03$, Scheffé value=6.8, $\alpha=.05$). Post hoc tests using the Scheffé technique revealed that there were significant differences between groups during the premenstrual and intermenstrual phases (see Figure 3 and Table 4).

During the premenstrual phase at post-treatment, the ES Group was significantly different than the WL group. Mean scores were lower for the ES group ($\bar{x}=33.1$) than the WL group ($\bar{x}=59.0$). There was not a significant difference between the ES group and the LF group ($\bar{x}=43.3$) or the LF group and the WL group at post-treatment during the premenstrual phase. There were no group differences at pre-treatment or follow-up during the premenstrual phase.

There were no significant group differences during the menstrual phase. During the intermenstrual phase at post-treatment, scores on the pain subscale were significantly different for the ES group ($\bar{x}=28.0$) compared to the LF group ($\bar{x}=45.5$), but they were not significantly different than scores obtained by the WL group ($\bar{x}=36.4$). At follow-up, pain subscale scores were significantly different for both treatment conditions, LF ($\bar{x}=30.8$) and ES ($\bar{x}=29.1$), compared to the WL ($\bar{x}=41.4$) control group.

There were no significant differences found during the premenstrual or menstrual phases for any group when comparing scores from pre-treatment to post-treatment, pre-treatment to follow-up, and post-treatment to follow-up. During the intermenstrual phase, there was a significant difference from post-treatment to follow-up for the WL group.

A highly significant main effect for phase ($F(2,46)=19.96$, $p<.0001$, Scheffé value=6.8, $\alpha=.05$) was found. The premenstrual and menstrual phases were not significantly different from one another at any time. The premenstrual and menstrual phases were significantly different from the intermenstrual phase at pre-treatment, post-treatment, and follow-up.

Results from the pain subscale score suggest the women participating in this study were experiencing pain symptoms related to the menstrual cycle because scores obtained during the premenstrual and menstrual phases were always significantly higher than those during the intermenstrual phase. There were no significant differences between the premenstrual phase and menstrual phases at any time, suggesting physical pain for women participating in this study was equally high for the premenstrual and the menstrual phases.

There appeared to be a small advantage to being in the ES group. Women in the ES condition reported less severe pain symptoms at post-treatment compared to the WL group. The ES group also had significantly lower scores than the LF group during the intermenstrual phase at post-treatment, and significantly lower scores than the WL group

at follow-up. However, there were no group differences during the menstrual phase, which suggests that treatment may have impacted pain symptoms during the premenstrual and intermenstrual phases, but not the menstrual phase. Possibly, pain symptoms are qualitatively different during the menstrual phase.

The LF treatment appeared to have little effect on pain symptoms. However, scores during the intermenstrual phase were significantly lower than scores obtained by the WL group at follow-up. It is difficult to explain this difference considering the LF group was not significantly different than the WL group during the intermenstrual phase at post-treatment. As a matter fact, it was significantly higher than the ES group at post-treatment. Despite these mixed results during the intermenstrual phase, scores of the pain subscale during the premenstrual phase did not change significantly for the LF group following treatment, and they were not significantly different than the ES or WL groups.

Water symptom score. A 3 x 3 x 3 (group x phase x time) repeated measures ANOVA was calculated for the mood subscale score to assess (1) differences in scores between the phases (2) changes in symptoms across time (3) differences between the groups and (4) the interaction between group and time. Significant results were not found for the main effects for time ($F(2,46)=.33$, $p>.05$) or for the interaction effect between time and group ($F(4,46)=1.11$, $p<>.05$). Thus, there were no significant differences between groups or across time (see Figure 4 and Table 5).

There was a highly significant main effect for phase ($F(2,46)=29.19$, $p<.0001$, Scheffé value=6.8, $\alpha=.05$). Water retention scores obtained during the premenstrual

and menstrual phases were significantly different than water retention scores obtained during the intermenstrual phase. Mean scores were higher during the premenstrual and menstrual phases compared to the intermenstrual phase. There were no significant differences between the premenstrual and menstrual phases except at post-treatment. Mean scores at post-treatment were higher during the premenstrual phase than during the menstrual phase.

Results from the water retention subscale score suggest the women participating in this study were experiencing physical discomfort (i.e. breast tenderness, bloating) related to the menstrual cycle because scores obtained during the premenstrual and menstrual phases were always significantly higher than those during the intermenstrual phase. There were no significant differences between the premenstrual phase and menstrual phases except at post-treatment. In general, this suggests that water retention for women participating in this study was equally high for the premenstrual and the menstrual phases.

Unfortunately, negative symptoms associated with water retention were not significantly reduced following treatment for either the LF or ES groups.

Overview of Total and Subscale Scores. Women participating in this study exhibited physical and emotional distress related to the menstrual cycle. Symptoms appeared to be high in both the premenstrual and menstrual phases.

There appeared to be a slight advantage to being in the ES condition. For both the total symptom score and the pain subscale score, the ES group reported significantly

less severe symptoms than the WL group at post-treatment during the premenstrual phase, but these differences were not maintained at follow-up. Lower scores for the ES group compared to the WL group were also reported during the intermenstrual phase for both the total symptom score and the pain subscale scores.

In general, the group differences suggest that being in the ES group was better than receiving no treatment at all, but a number of factors suggest that even the ES treatment was only marginally "effective". First, there was no significant interaction effect for group by time, suggesting none of the groups changed significantly from pre-treatment to post-treatment, or from pre-treatment to follow-up. Second, the premenstrual phase was always significantly different than the intermenstrual phase. One goal of treating LLPDD or PMS is to reduce premenstrual symptoms to that of the intermenstrual phase. Lack of significant differences for the mood subscale score and water retention score is particularly disappointing since a disturbance in mood is suppose to be the primary symptom of LLPDD, and because the LF condition was hypothesized to have the greatest impact on symptoms in the water retention subscale. Finally, group differences observed in the premenstrual phase for the total symptom score and pain subscale score were not maintained at follow-up.

Verification of Behavioral Changes.

Additional analyses were performed to assess behavioral changes that may have influenced treatment effects. These analyses were performed to verify that only the women in the LF condition reduced their fat consumption, and to make sure that other

variables, such as stress, exercise, and caffeine intake, did not change in any of the groups. A 3 x 3 (group x time) mixed factorial design was used to assess changes in behaviors. The independent variables were group (LF, ES, and WL) and time (pre-treatment, post-treatment, and follow-up). The dependant variables were percentage of fat, grams of fat, percentage of carbohydrates, grams of carbohydrate, percentage of protein, grams of protein, average amount of caffeine consumed per day, average amount of exercise per day, and average rating of stress per day.

Food Diaries. All subjects were instructed to monitor their entire food consumption for six days pre-treatment, post-treatment and follow-up (three days in the luteal phase and three days in the follicular phase). Food Diaries were compared with Part 2 of the Daily Monitoring Measure. Any beverages or chocolate consumption listed on Part 2 of the Daily Monitoring Measure were added if they were not originally noted on the Food Diaries. The average of these six days was then calculated for the following dietary variables: (1) percentage of fat consumption, (2) grams of fat consumption, (3) percentage of carbohydrate consumption, (4) grams of carbohydrate consumption, (5) percentage of protein consumption, (6) grams of protein consumed, and (7) calories. (When fewer than six days were monitored, an average of the actual days monitored was used. Data was calculated from the food diaries even if they were completed on the wrong day).

A computerized program, Nutritionist III version 7, was used to compute these variables. A Masters-level student in nutrition, who was paid for her services, coded the

dietary information. All dietary information was double-checked for errors in coding.

One woman from the education support group had to be excluded from these analyses because she failed to monitor any of her dietary intake after treatment. Two women from the control group and one from the education support group only completed post-treatment assessment, and did not complete the follow-up assessment. Rather than exclude these women entirely, it was decided their averages obtained in the post-treatment assessment would be used again for their follow-up scores. Even though mean scores at follow-up are questionable for the WL and ES groups, mean scores at post-treatment are more accurately represented.

Percentage of fat. A 3 x 3 (group x time) repeated measures ANOVA was used to assess changes in percentage of fat consumed across time, differences between the groups, and the interaction between these variables. Significant effects for group ($F(2,22)=14.21$, $p<.0001$, Scheffé value=6.8, $\alpha=.05$) and time ($F(2,44)=3.32$, $p<.05$, Scheffe value=6.8, $\alpha=.05$) were obtained. There was also a significant group by time interaction effect ($F(4,44)=6.16$, $p<.001$, Scheffé value=6.8, $\alpha=.05$). Percentage of fat appeared to decrease for the LF group, while remaining about the same for the ES and WL groups (see Figure 5, Table 6, and Table 7).

Post-hoc tests using the Scheffe technique revealed that there were significant group differences at post-treatment, with the LF group being significantly different than the ES group and WL group. Mean scores were lower for the LF group ($\bar{x}=22.3$) compared to the ES group ($\bar{x}=35.3$) and WL group ($\bar{x}=38.9$). There were no significant

group differences at pre-treatment or follow-up. Mean scores at pre-treatment were 33.7, 26.3, and 33.0 for the LF, ES, and WL groups, respectively. At follow-up, mean scores for the LF, ES, and WL groups were 25.4, 34.6, and 31.1, respectively.

Further post-hoc analyses found that percentage of fat was significantly different from pre-treatment to post-treatment and from pre-treatment to follow-up for the LF condition. Mean scores at post-treatment ($\bar{x}=22.3$) and follow-up ($\bar{x}=25.4$) were lower than the mean score at pre-treatment ($\bar{x}=33.7$). There was also a significant difference from post-treatment to follow-up for the LF group. The mean score for percentage of fat was significantly higher at follow-up as compared to post-treatment. Mean scores at post-treatment and follow-up suggest that women in the LF condition were close to meeting the goal outlined in treatment of consuming less than 20% of their total calories from fat.

The percentage of fat did not change significantly from pre-treatment to post-treatment, pre-treatment to follow-up, or post-treatment to follow up for ES and WL groups. Mean scores for percentage of fat for the ES group were 36.3, 35.3, and 34.6 at pre-treatment, post-treatment, and follow-up, respectively. For the WL group, mean scores for percentage of fat were 33.0, 38.9, and 31.1 at pre-treatment, post-treatment and follow-up, respectively.

Grams of fat. A 3 x 3 (group by time) repeated measures ANOVA was used to assess changes in grams of fat consumed across time, differences between the groups, and the interaction between these variables. Significant effects for time ($F(2,44)=6.63$,

$p < .01$, Scheffé value=6.8, $\alpha = .05$), but not group ($F(2,22) = 3.21$, $p > .05$) were obtained. There was a significant group by time interaction ($F(4,44) = 3.42$, $p < .05$, Scheffé value=6.8, $\alpha = .05$). Grams of fat appeared to decrease for the LF group, while remaining about the same for the ES and WL groups (see Figure 6, Table 8, and Table 9).

Post-hoc tests employing the Scheffe technique revealed the LF group ($\bar{x} = 39.9$) consumed significantly less fat in grams than the ES group ($\bar{x} = 72.4$) or the WL group ($\bar{x} = 75.4$) at post-treatment. There were no significant group differences at pre-treatment or follow-up. Mean scores for grams of fat at pre-treatment were 70.8, 78.7, and 68.9 for the LF, ES, and WL groups, respectively. At follow-up, mean scores for grams of fat were 44.2, 69.6, and 59.1 for the LF, ES, and WL groups, respectively.

Further post-hoc analyses found that grams of fat consumed was significantly different from pre-treatment to post-treatment and from pre-treatment to follow-up for the LF condition. Mean scores for grams of fat were lower at post-treatment ($\bar{x} = 39.9$) and follow-up ($\bar{x} = 44.2$) than at pre-treatment ($\bar{x} = 70.8$) There was no significant difference from post-treatment to follow-up.

For the ES and WL conditions, there were no significant changes in grams of fat consumed from pre-treatment to post-treatment, pre-treatment to follow-up, or post-treatment to follow-up. Mean scores for grams of fat for the ES group were 78.7, 72.4, and 69.6 at pre-treatment, post-treatment, and follow-up, respectively. For the WL

group, mean scores for grams of fat were 68.9, 75.4, and 54.1 at pre-treatment, post-treatment, and follow-up, respectively.

Percentage of Carbohydrates. A 3 x 3 (group by time) repeated measures ANOVA was used to assess change in percentage of carbohydrates consumed across time, differences between the groups, and the interaction between these variables. Significant effects for group ($F(2,22)=3.48$, $p<.05$, Scheffé value=6.8, $\alpha=.05$) and time ($F(2,44)=4.96$, $p<.01$, Scheffé value=6.8, $\alpha=.05$) were obtained. There was also a significant group by time interaction ($F(4,44)=2.77$, $p<.05$, Scheffe value=6.8, $\alpha=.05$). The percentage of carbohydrates consumed appeared to increase for the LF condition while remaining about the same for the ES and WL groups (see Figure 7, Table 10, and Table 11).

Post-hoc tests revealed the percentage of carbohydrates consumed was significantly different for the LF group compared to the ES and WL groups at post-treatment. At post-treatment, the mean score for LF group ($\bar{x}=59.8$), was higher than the ES group ($\bar{x}=47.9$) and the WL group ($\bar{x}=50.1$). There were no significant group differences at pre-treatment or follow-up. Mean scores at pretreatment were 49.1, 49.6, and 45.5 for the LF, ES, and WL groups, respectively. At follow-up, mean scores were 56.7, 48.7, and 51.8 for the LF, ES, and WL groups, respectively.

Further analyses found that the percentage of carbohydrates consumed was significantly different from pre-treatment to post-treatment and from pre-treatment to follow-up for the LF group. Mean scores at post-treatment ($\bar{x}=59.8$) and follow-up

($\bar{x}$=56.7) were higher than mean scores at pre-treatment ($\bar{x}$=49.1). There were no significant differences from post-treatment to follow-up.

For the ES and WL conditions, there were no significant changes in percentage of carbohydrates consumed from pre-treatment to post-treatment, pre-treatment to follow-up, post-treatment to follow-up. Mean scores for the ES group were 49.6, 47.9, 48.7 at pre-treatment, post-treatment, and follow-up, respectively. For the WL group, mean scores were 45.5, 50.1, and 51.8 at pre-treatment, post-treatment, and follow-up, respectively.

Grams of Carbohydrates. A 3 x 3 (group by time) repeated measures ANOVA was used to assess change in grams of carbohydrates consumed across time, differences between the groups, and the interaction between these variables. Unlike percentage of carbohydrates consumed, there was no main effect for group ($F(2,22)=.02$, $p>.05$), or time ($F(2,44)=.27$; $p>.05$). The interaction of these variables was also not significant ($F(4,44)=.71$; $p>.05$). Thus, the consumption of grams of carbohydrates did not change significantly for any of the groups (see Figure 8 and Table 12).

Mean scores for grams of carbohydrates consumed for the LF group were 231.7, 246.7, and 226.2 at pre-treatment, post-treatment, and follow-up, respectively. For the ES group mean scores for grams of carbohydrates consumed were 238.9, 216.9, and 240.2 at pre-treatment, post-treatment, and follow-up, respectively. Mean scores for grams of carbohydrates consumed for the WL group were 228.3, 238.1, and 211.4 at pre-treatment, post-treatment, and follow-up, respectively.

Percentage of Protein. A 3 x 3 (group by time) repeated measures ANOVA was used to assess changes in percentage of protein consumed across time, differences between the groups, and the interaction between these variables. Main effects for group ($F(2,22)=.37$, $p>.05$) and time ($F(2,44)=1.30$, $p>.05$) were not significant. The interaction of these variables was also not significant ($F(2,44)=.21$ $p>.05$). Thus, percentage of protein did not change significantly for any of the groups (see Figure 9 and Table 13).

Mean scores for percentage of protein consumed for the LF group were 74.6, 68.9, and 67.1 at pre-treatment, post-treatment, and follow-up, respectively. For the ES group, mean scores for percentage of protein consumed were 16.1, 16.9, and 18.2 at pre-treatment, post-treatment, and follow-up, respectively. Mean scores for percentage of protein consumed for the WL group were 13.9, 16.3, and 15.4 at pre-treatment, post-treatment, and follow-up, respectively.

Grams of Protein. A 3 x 3 (group by time) repeated measures ANOVA was used to assess change in grams of protein consumed across time, differences between the groups, and the interaction between these variables. Like percentage of protein consumed, main effects for group ($F(2,22)=1.67$ $p>.05$) and time ($F(2,44)=.83$, $p>05$) were not significant. The interaction was also not significant ($F(2,44)=.32$ $p>.05$). Thus, grams of protein did not appear to change significantly for any of the groups (see Figure 10 and Table 14).

Mean scores for grams of protein consumed for the LF group were 74.6, 68.9, and 67.1 at pre-treatment, post-treatment, and follow-up, respectively. For the ES group mean scores for grams of protein consumed were 67.1, 73.0, and 66.3 at pre-treatment, post-treatment, and follow-up, respectively. Mean scores for grams of protein consumed for the WL group were 60.4, 58.0, and 53.3 at pre-treatment, post-treatment, and follow-up, respectively.

Calories. A 3 x 3 (group by time) repeated measures ANOVA was used to assess changes in caloric intake across time for the three groups, differences between the groups, and the interaction between these variables. There were no significant differences for time ($F(2,44)=2.56$, $p>.05$), group ($F(2,22)=.25$, $p>.05$), or the interaction of time by group ($F(4,44)=.95$, $p>.05$). Thus, caloric intake did not change significantly for any of the groups (Table 15).

Mean scores for calories consumed for the LF group were 1842, 1692, and 1549 at pre-treatment, post-treatment, and follow-up, respectively. For the ES group mean scores for calories consumed were 1896, 1786, and 1787 at pre-treatment, post-treatment, and follow-up, respectively. Mean scores for calories consumed for the WL group were 1764, 1876, and 1528 at pre-treatment, post-treatment, and follow-up, respectively.

Overview of Dietary Data From Food Diaries. Results suggested women in the LF condition significantly changed their dietary practices, while women in the ES and WL groups did not change their diets significantly.

Subjects in the LF condition significantly reduced their fat consumption. There was a significant reduction in both percent fat and grams of fat from pre-treatment to post-treatment and from pre-treatment to follow-up. However, the percentage of fat consumed at follow-up was significantly higher than percentage of fat consumed at post-treatment, suggesting that the women in the LF group were not following the low-fat diet as carefully at follow-up.

For the LF group, the percentage of carbohydrates increased significantly from pre-treatment to post-treatment, and this was maintained at follow-up. However, grams of carbohydrates did not change from pre-treatment to post-treatment or from pre-treatment to follow-up. Percentage of protein or grams of protein did not change throughout the study. There were no significant changes in caloric intake. These results suggested the subjects in the LF group lowered their fat intake, but did not significantly increase their carbohydrate or protein intake. This reduction in fat consumption was not great enough to significantly impact levels for total calories consumed. Calories consumed were relatively low throughout the study (less than 1850 calories per day). Reported reduction in percentage of fat consumed was close to the desired level of 20%, however this criterion was not met. Although the women in the LF group appeared to be consuming less fat, the adequacy of vitamin and minerals consumed is questionable because calorie consumption was low.

There were no significant changes in the above dietary variables from pre-treatment to post-treatment, or from pre-treatment to follow-up for the ES or LF groups.

Exercise. The average amount of exercise a participant engaged in per day was determined. This was done by adding the minutes of exercise monitored each day for an entire menstrual cycle and then dividing this number by the number of days in the menstrual cycle (or the number of days monitored during this menstrual cycle, thus controlling for any days missed). One subject from the WL group was excluded from this analysis because of missing data.

A 3 x 3 (time x group) repeated measures ANOVA was used to assess changes in exercise across time, between groups, and the interaction of these variables. No significant main effects were found for group ($F(2,22)=.89$, $p>.05$) or time ($F(4,44$, $p>.05$) but there was a significant interaction ($F(4,44)=.2.93$, $p<.05$). Post-hoc tests revealed that there were no significant changes from pre-treatment to post-treatment or from pre-treatment to follow-up for any of the three groups. The groups were not significantly different from one another at pre-treatment, post-treatment, or follow-up. However, the interaction effect remained significant for the LF and ES conditions. The mean amount of exercise per day increased for the ES group, while it decreased for the LF group. The interaction effect was not significant when comparing the WL group to the ES group or when comparing the WL group to the LF group (see Figure 11 and Table 16).

Caffeine. Consumption of caffeine was also determined by adding beverages containing caffeine and chocolate across an entire menstrual cycle. The resource book Food Values of Portions Commonly Used, 14th Ed. (Pennington & Church, 1985) was

used to estimate the milligrams of caffeine in each beverage and in chocolates. The average milligrams of caffeine consumed was then tabulated by dividing the total milligrams of caffeine consumed for entire menstrual cycle, by the number of days in the menstrual cycle (or the number of days monitored during that menstrual cycle). One women from the WL group was excluded from this analysis because of missing data.

A 3 x 3 (time x group) ANOVA for the average milligrams of caffeine consumed was used to assess differences in caffeine consumption between groups, changes in caffeine consumption across time, and the interaction between these variables. There were no significant main effects for group ($F(2,22)=.80$, $p>.05$) or time ($F(2,44)=.29$, $p>.05$). The interaction was not significant ($F(4,44)=.95$, $p>.05$). (See Figure 12 and Table 17).

Stress. Subjective ratings of stress were monitored daily on Part 2 of the Daily Monitoring Measure. These ratings were totaled for an entire menstrual cycle and then were divided by the number of days in that menstrual cycle (or the number of days monitored during that menstrual cycle) to yield an average rating of stress. One woman from the WL condition was excluded from this analysis because of missing data.

A 3 x 3 (time x group) repeated measures ANOVA was used to assess changes in the average rating of stress across time (pre-treatment, post-treatment, follow-up), between groups, and the interaction of these variables. There were no main effects for time ($F(2,44)=.83$, $p>.05$) or group ($F(2,22)=.58$, $p>.05$). The interaction between

group and time was not significant ($F(4,44)=1.44$, $p>.05$). (See Figure 13 and Table 18).

Rating of Treatment Rationale.

A 2 x 2 (time x group) mixed factorial design was used for rating of treatment rationale. The independent variables were group (LF and ES) and time (1st, 4th, and 8th sessions). The dependent measure was the total score from the Rating of Treatment Rationale Questionnaire.

A 2 x 2 (group x time) repeated measures ANOVA was conducted to assess differences between groups, change in ratings across time, and the interaction between these variables. There were no significant main effects for group ($F(1,16)=.65$, $p>.05$) or time ($F(2,32)=1.25$, $p>.05$). The interaction was not significant ($F(2,32)=1.69$, $p>.05$). Results suggest the two groups were equally convinced the treatment they received would be effective in relieving premenstrual suffering.

Relationship of Stress, Exercise, and Diet on Premenstrual Symptoms.

This study has presumed that various factors (i.e. diet, stress, and exercise) influence premenstrual suffering. To determine whether these variables were associated with symptoms, correlation coefficients were generated. Although no a priori hypotheses were specified, it was assumed that variables such as stress, caffeine consumption, and fat consumption would be positively correlated with symptoms, while exercise and carbohydrate consumption would be negatively correlated with symptoms (i.e. the more one exercises the less distress one feels). The following variables were correlated with

the total symptoms score, and the three subscale scores: (1) stress, (2) exercise (3) caffeine (4) percent fat, (5) grams of fat, (6) percent carbohydrates, (7) grams of carbohydrates, (8) percent protein, and (9) grams of protein. Critical r was determined using a two-tailed test of significance (see Table 18).

Results indicated that stress was positively and significantly correlated with both the mood subscale score ($r=.62$, $p<.001$) and the total symptom score ($r=.48$, $p<.05$).

No significant correlations were found for exercise or caffeine. Although nonsignificant, the correlation between caffeine consumption and the pain subscale was fairly high ($r=.37$, $p<.10$).

The association between grams of fat consumption and the mood subscale score was negative ($r=-.50$, $p<.01$), suggesting greater premenstrual emotional distress was related to lower fat consumption. This negative association was also found for total calories ($r=-.44$, $p<.05$) and grams of carbohydrates ($r=-.35$, $p<.10$).

Another interesting finding was the significant positive correlation between percentage of protein and the total premenstrual symptom score ($r=.42$, $p<.05$).

In sum, correlations obtained during this study suggest that stress and dietary variables are related to premenstrual distress. As predicted above, increased premenstrual suffering appeared to be related to higher reports of stress. Contrary to the predictions, fat consumption was negatively correlated with the emotional symptoms of PMS. Total calories consumed and carbohydrate consumption was also negatively

correlated. These correlations suggest that "malnutrition" is related to reports of negative emotions during the premenstrual phase.

Individual Cases

Anecdotal reports and information about individual cases are presented in this section because this information clarifies results obtained in the group analyses. Additionally, relevant information from the Final Questionnaire is presented.

Case from the Low-Fat Diet Group. As indicated above, the low-fat diet appeared to be ineffective in reducing premenstrual suffering, even though group averages suggest that fat consumption was reduced significantly. However, it is possible that group averages obscured results (i.e. some women may have failed to reduce percentage of fat consumption). To check this possibility, percentage of total symptom change (see Table 19) was calculated and compared to reduction in fat consumption and other dietary variables (see Table 20) for each case. The percentage of symptom change was calculated by subtracting the post-treatment (and follow-up) total symptom score from the pre-treatment total symptom score, and then dividing this number by the pre-treatment score. The hypotheses of this study suggested that only women who changed their fat consumption would report reduced premenstrual suffering, while those who did not change their diet would not report a decrease in premenstrual symptom severity.

Only two women (case #7 and case #10) failed to decrease their fat consumption to less than 25% of their total daily caloric intake at post-treatment and follow-up. One of these women (case #7) reported increased premenstrual distress, while the other

woman reported a dramatic decrease in symptom severity. The woman whose symptoms decreased (37% from pre-treatment to post-treatment and 37% from pre-treatment to follow-up) actually increased her fat consumption. She reported her fat consumption was 31%, 32% and 39% of her total caloric intake at pre-treatment, post-treatment, and follow-up, respectively.

Symptoms also increased in three of the women who appeared to reduce their fat consumption (case #2, case #4 and case #9). Two of these women (case #4 and case #9) reduced their fat consumption dramatically. They both reported they were consuming 39% of their calories from fat at pre-treatment, 20% of their calories from fat at post-treatment, and 22% of their calories from fat at follow-up. Grams of fat consumed was cut in half for both of these women. Their carbohydrate consumption was also close to the desired goal of 65% of their total caloric intake. However, for both women the percentage of protein consumed increased slightly and grams of protein consumed was also higher at post-treatment and follow-up, compared to pre-treatment. Reported caloric intake decreased for both women.

Another woman (case #2) whose symptoms increased dramatically despite apparent reductions in fat consumption, indicated that participating in the low-fat diet group was somewhat distressing to her. At pre-treatment she was consuming 26% of her total calories from fat, and she reduced her fat consumption to 20% and 22% of her total calories for post-treatment and follow-up, respectively. Food Diaries at post-treatment suggest that there were days when her caloric intake was extremely low. On the final

questionnaire, she claimed to follow the low-fat diet every day following treatment. During group meetings, this woman reported she had dieted frequently, and expressed a great deal of distress about eating large quantities of carbohydrates. She mentioned she had previously learned to avoid consumption of certain carbohydrates, and she expressed concern about increasing her weight. Despite her daily reports of increased premenstrual distress, this subject thought she was 80% improved at the end of treatment, possibly feeling that she must have improved given that she had followed the dietary suggestions.

The remaining five women (cases #1, #3, #5, #6, and #8) all reported improved symptoms. Improvement for one subject (case #1) was extremely marginal (3% post-treatment and 10% follow-up). The other four women reported percent decrease in symptoms between 23% and 25% at post-treatment, and 7% and 44% at follow-up. Some researchers (i.e. Stone, Pearlstein, & Brown, 1991) suggested that there should be a 50% reduction in symptom severity in order to assert treatment was effective in reducing premenstrual symptoms. None of these women met this criteria.

In sum, even when each case is looked at individually in the LF group, the low-fat diet intervention appeared to be ineffective. Individual cases suggest that increased protein consumption or failure to reduce protein may be influencing results. Another possibility is that body image concerns may be negating the effects or the low-fat diet intervention or they may actually increase symptom severity. It is still possible that a low-fat diet is effective in treating PMS or LLPDD, but the low-fat diet needs to be

conducted more carefully so that nutritional requirements are met, and the diet itself is personally satisfying.

Cases from the Education-Support Group. Group analyses presented above suggest there was an advantage to being in the ES group compared to receiving no treatment. Information presented in the Final Questionnaire for the ES group may provide insight into why this treatment appeared to be effective for some of the members in the ES group. Responses on the Final Questionnaire were compared to percentage of decrease or increase in total symptom severity (see Table 19).

Symptoms decreased for five members of the LF group (cases #11, #12, #13, #14, and #15) from pre-treatment to post-treatment and pre-treatment to follow. As was true for the LF group, none of the women in the ES group reduced their symptoms 50% from pre-treatment to post-treatment, but two reached this goal from pre-treatment to post-follow-up (case #12 and #15). Symptoms increased for two women (cases #16 and #18) from pre-treatment to post-treatment and from post-treatment to follow-up. For one subject (case #17), symptoms increased from pre-treatment to post-treatment, but decreased from pre-treatment to follow-up.

Responses on the Final Questionnaire for the Education-Support Group were different for the women who reported improved symptoms compared to those who reported increased premenstrual suffering. On the Final Questionnaire for the ES group, subjects were asked to rank the importance of the treatment components (group support, learning not to feel guilty for premenstrual suffering, cognitive restructuring, problem

solving, relaxation, exercise, and diet). Those women who reported improved symptoms all ranked group support and learning not to feel guilty for suffering from PMS as one of the top three components of treatment. On open ended questions, they all reported they practice problem-solving techniques, and most of them indicated they practiced relaxation procedures. When asked how treatment could be improved, a number of these women suggested treatment should be continued for a longer period of time. All of these women participated actively in group sessions and were open about their premenstrual symptoms and personal problems.

Of the three women whose symptoms increased, two of them (cases #17 and #18) ranked exercise as the most important component of treatment. One of these women (case #17) ranked group support as the least important component, and clearly stated that she did not learn anything new from treatment. The other woman (case #18) indicated she did not use problem- solving skills or relaxation techniques, but she did try to use cognitive restructuring to improve her self-image. These two women were in the same group, and the therapist of this group suggested they were incompatible and group discussion was stifled. This suggests group dynamics played a role in the effectiveness of the ES treatment. (See Appendix M for configuration of subjects in treatment groups). In sum, individual reports suggest group support and learning not to feel guilty about suffering from PMS were two very important components of treatment. Using problem-solving techniques may have improved symptoms. Group dynamics appeared to play a role in the efficacy of an education-support treatment. Even though five out

of the eight women reported improvements in symptoms, the percentage of improvement for these five women did not meet the 50% criteria suggested by Stone et al. (1991). Thus, the effectiveness of treatment appeared to be very minimal.

Cases from the Control Group. The eight women in the control group were monitored for three consecutive menstrual cycles. Percentage of change was calculated from the first cycle to the second cycle, and the first cycle to the third cycle (see Table 19). Percentage of symptom increase and decrease was relatively high for a number of the cases (greater than 20% change for 5 of the cases), suggesting that symptom severity can change greatly from one cycle to another. One woman in the control group (case #24) only had a 6% increase from the first cycle to the second cycle, but from the first cycle to the 3rd cycle she had a 47% decrease in symptoms. This result suggests that this subject had a relatively asymptomatic cycle. Another women in the control condition had a 50% increase in symptoms from the first cycle to the second cycle; there was a 31% increase from the first cycle to the third cycle.

The "large" changes in these two cases exemplify why it is difficult to assess treatment effects, and why 50% or greater symptom improvement is suggested as a criteria for determining the effectiveness of a treatment for LLPDD.

This also means that any treatment of LLPDD will need to be very powerful because of the variability in symptom severity from one menstrual cycle to the next menstrual cycle.

Summary of Results

Findings of this study suggest that participating in an education-support group may have a slight impact on premenstrual symptoms immediately following treatment, but this advantage was not maintained by the second cycle following treatment. Results further indicate that participating in a group designed to lower fat consumption was ineffective in treating PMS. Results suggested that women in the low-fat condition significantly reduced their dietary intake from 35% at pre-treatment to 22% at post-treatment and 25% at follow-up. Although this was a significant reduction, subjects did not reach the desired criteria of limiting fat intake to 20% of total caloric consumption.

Additional information gathered suggested that premenstrual symptoms were related to ratings of stress. There was also a negative correlation between caloric consumption and fat consumption, suggesting that lower levels of calories and lower fat intake is related to more severe premenstrual distress.

Chapter III

Current Findings and Future Directions

Discussion of Results

A treatment outcome study was conducted comparing a low-fat diet intervention with an education-support group and a waiting-list control group. A low-fat diet was hypothesized to be an effective intervention for reducing the severity of both physical and emotional premenstrual symptoms in women suffering from LLPDD. This was based on the theory relating low-fat diets to decreased estrogen levels and previous research suggesting low-fat diets are effective in treating severe breast tenderness, a common symptom of LLPDD or PMS.

The hypotheses that a low-fat diet would decrease suffering was not supported by the results of this study. However, there appeared to be an advantage to participating in a group which provided support and information on PMS compared to receiving no treatment.

Results from this study have implications for future research and treatment of LLPDD. These implications, as well as methodological issues and explanations of the results, will be discussed in the next sections of this chapter.

Dietary interventions for LLPDD.

The hypothesis that a low-fat diet would reduce emotional and physical symptoms of LLPDD was not supported by this study. This result may be attributable to a number

of factors. It is possible that subjects did not change their fat consumption or reductions in fat consumption were not large enough. Reductions in fat may have failed to lower estrogen levels, or raised estrogen levels may not be the "cause" of PMS. Other dietary factors, which were not changed, may be influencing premenstrual tension scores. Finally, participating in a low-fat group may conjure up negative body image concerns or other issues that may negate any positive effects of the low-fat diet.

Although women in the LF group may have failed to follow the low-fat diet, the data suggested that participants in the LF group did significantly reduce their fat consumption. This reduction appeared to be rather large. According to the LF group average, the percentage of total calories consumed by fat was 35% at pretreatment and only 22% and 25% at posttreatment and follow-up, respectively. Despite these results, subjects in the LF condition may have misrepresented their intake of fat by failing to report certain items or by only following the diet on days that they monitored their food intake. Sampling procedures for only six days during their menstrual cycle could have promoted inaccurate and/or nonrepresentative recording. Even though the reduction of fat intake was large, subjects did not meet the goal of less than 20% fat intake. Thus, fat intake may not have been reduced sufficiently to result in symptom reduction.

The reduction of fat may have been sufficient, but the low-fat diet may not have been maintained for a long enough period of time to result in symptom reduction. There were eight treatment sessions which were conducted in an eight to nine week period of time. Participants were encouraged to continue the low-fat diet after treatment sessions

ended, but no formal maintenance program was instituted. Subjects may have needed to maintain the low-fat diet for several more months before symptom relief would occur. However, the length of time participants followed a low-fat diet was similar to the study by Rose et al. (1985). This study found symptom reduction for cyclical mastopathy after three months on a low-fat diet. Subjects in this study were basically on the diet for three months if the post-treatment cycle is included, but there were no significant changes in premenstrual symptomatology from pre-treatment to follow-up for the group. Further research is necessary to determine if a low-fat diet would be effective if the length of treatment were increased or if subjects were to decrease fat intake to below 20% of their total caloric intake.

The above comments suggest that treatment was ineffective because the reduction of fat intake did not occur, the reduction of fat intake was not large enough, or the reduction of fat intake was not maintained for a long enough period of time. All of these comments imply that the diet intervention itself needed to be modified. These changes could include increasing the number of treatment sessions, establishing a maintenance program, or taking steps to ensure participants reduced fat intake to below 20% of their total caloric intake. Although these changes may make a low-fat diet intervention an efficacious treatment of LLPDD or PMS, it is also possible that the low-fat diet intervention was ineffective because the theoretical premises were incorrect.

Treatment may have been ineffective because estrogen levels were not reduced by the low-fat diet. Because of limited resources, this study could not measure estrogen

levels. However, Rose et al. (1985) found estrogen levels were reduced by 28% for women with cyclical mastopathy who were on a diet to reduce fat intake to 20% of their total calories. Further research is needed to see if a low-fat diet actually reduces estrogen levels in women who suffer from LLPDD.

It is also possible that estrogen levels were reduced for those following the low-fat diet, but the reduction of estrogen did not lead to reduced premenstrual distress. If this postulate is true, then fat consumption would not be related to premenstrual distress.

Fat consumption was negatively correlated with premenstrual emotional symptoms ($r=-.50$, $p<.01$), suggesting lowered fat intake was related to increased emotional distress during the premenstrual phase. In addition, calories consumed was negatively correlated with the mood subscale score ($r=-.44$, $p<.05$) as was grams of carbohydrates ($r=-.35$, $p=<.10$). These results suggest short-term malnutrition or low caloric intake was related to increased emotional distress during the premenstrual phase. Caloric intake did not significantly change for the women in the LF condition, but mean scores for calories were lower at post-treatment and follow-up, compared to pre-treatment. Thus, treatment may have been ineffective because total caloric intake was too low.

If low caloric intake is an etiological factor contributing to the severity of premenstrual distress, then dietary interventions for PMS and LLPDD need to focus on increasing calories consumed. However, this dietary suggestion needs to be tempered with other health concerns which typically face women. For example, the increase of fat consumption is contraindicated because it may increase the risk for obesity, diabetes,

breast cancer, hypertension, and coronary heart disease (see Labuza & Sloan, 1979). The rate of coronary heart disease is rising steadily among women, and women are already at greater risk for obesity and diabetes compared to men (see Rodin & Ickovics, 1990). Increased protein consumption is also contraindicated because correlations obtained in this study suggest high protein consumption is positively related to premenstrual symptoms. However, raising the consumption of carbohydrates, to maintain an appropriate caloric intake, may be an effective treatment for PMS.

Subjects were strongly encouraged to increase carbohydrate consumption in this study, but grams of carbohydrates consumed did not change significantly. In addition, calories consumed, grams of protein consumed, and percentage of total calories consumed by protein did not change significantly. Thus, treatment may have been ineffective because carbohydrates did not increase sufficiently, and/or protein consumption did not decrease sufficiently. Wurtman et al. (1989) found that various premenstrual symptoms were relieved immediately following the consumption of a carbohydrate-rich, protein-poor meal. This was hypothesized to be effective because carbohydrates increase serotonin synthesis and release (Fernstrom et al., 1979), while proteins prevent serotonin synthesis and release (Smith & Saunder, 1986). If treatment was ineffective because protein consumption was not lowered sufficiently, and/or because carbohydrate consumption was not increased sufficiently, it would suggest that changing serotonin levels should be the focus of treatment rather than reducing estrogen levels.

The explanations presented above focus on "physiological" reasons for failing to reject the null hypothesis, but there may be a "psychological" explanation. Lack of treatment effects may have been related to body image concerns. The low-fat diet may have changed physical factors that predispose a woman to experiencing premenstrual distress, but "dieting" itself may have conjured up negative emotions negating any treatment effects. Having women change their fat intake may have sensitized them to various body image issues. As indicated by Brownell (1991) many women pursue the "ideal body" by dieting or engaging in other behaviors, but physiological or genetic restraints may make it difficult, if not impossible, for them to obtain this ideal. Brownell suggested failure to meet the ideal body image leads to body dissatisfaction and emotional distress. Thus, the low-fat diet used in this study may not have been effective because of negative emotions associated with "dieting".

The low-fat diet used in this study was not effective in reducing the emotional and physical symptoms of LLPDD. Despite these results, a dietary intervention for LLPDD may be effective. Further research is needed to determine if a diet which in very high in carbohydrates and low in protein is effective in reducing premenstrual distress. Although the study by Wurtman et al. (1989) supported the efficacy of this type of diet, this study did not use daily prospective measures and it only assessed the immediate impact on emotional symptoms. The instant reduction of symptoms after consuming a high-carbohydrate, protein-poor meal suggests that this type of diet can be used as a coping mechanism during the premenstrual phase to reduce suffering. Further research

is needed to determine if consistently following a high-carbohydrate, low-protein diet throughout the menstrual cycle provides any additional benefits. Additionally, future research on dietary interventions of LLPDD should assess body image concerns or distortion to make sure this is not effecting the outcome of treatment.

Providing Education and Support: Is it enough to treat LLPDD?

This study suggests that women benefit from having access to a supportive environment where women are allowed to discuss their premenstrual symptoms and are given information on PMS. Group differences were found at post-treatment between the ES and WL groups for the total symptom score and the pain subscale score. The effect for the pain subscale score was not predicted. Rather it was hypothesized participating in the ES group would reduce emotional symptoms or symptoms on the Mood subscale. Comments made on the Final Questionnaire and statements made during treatment sessions give some insight into these results and provide information for developing a psychological treatment of PMS.

Even though there did appear to be a slight advantage to participating in ES condition, results suggest the ES condition was not a sufficient treatment for LLPDD. Education and support may be an important part of a psychological treatment, but coping skills (i.e. problem solving, relaxation training) may need to be taught and practiced outside of treatment sessions for significant reductions in symptoms to occur.

Validation of Premenstrual Suffering. Levitt et al. (1986) hypothesized providing education and support is effective in reducing premenstrual distress because this treatment

validates the PMS sufferers experiences and tells them that they are not "going crazy." Comments made during the ES sessions seemed to support this idea. Women in the ES group expressed that others, such as their physician, spouse, or boss, did not understand their problem. More importantly, they seemed to feel that their own feelings were not important and expressed feelings of guilt because they behaved irrationally during their premenstrual phase. In other words, they seemed to feel guilty because they suffered from PMS.

Numerous examples were given during sessions on how insensitive various individuals were to their premenstrual suffering. Most of the examples centered around the concept that PMS is not real, and PMS is just an excuse for poor performance or persistent emotional problems. One woman felt the worst statement she heard about PMS was from her female boss. Her boss made the comment that she did not have any emotional changes premenstrually, so she felt women complaining of PMS were just faking symptoms. Thus, PMS sufferers in this study felt others did not even believe they truly suffered from premenstrual distress, and most felt that they needed to hide their distress for fear others would view them as being emotionally flawed. Listening to other women discuss their premenstrual distress allowed the women in the ES group to believe their own symptoms were real.

Many of the sessions for the ES group were spent discussing relationship issues. Most of the women mentioned they tended to become extremely agitated and dissatisfied with their spouse's or boyfriend's behavior during their premenstrual phase. This

agitation often resulted in hostile behavior and/or guilty feelings. At first, women in the ES group felt their own behavior and feelings were unjustified and their irritation was related to hormonal changes associated with their menstrual cycle. After further discussion, however, many women in the ES group concluded they were dissatisfied with their significant other's behavior at other times during their menstrual cycle. ES members felt they could tolerate or cope with their dissatisfaction during their intermenstrual phase, but they could not tolerate this dissatisfaction during their premenstrual phase. They all admitted that solving relationship and other problems during the intermenstrual phase would reduce emotional distress felt during the premenstrual phase.

Rather than solving problems during the intermenstrual phase, members admitted that they tried to solve marital and other problems during the premenstrual phase. Increased emotional lability appeared to make it easier for them to complain or express their dissatisfaction. However, ES members indicated that their heightened state of arousal also led them to present their complaint(s) in an irrational fashion. Thus, they tended not to solve their problems, and they also received negative feedback from the person receiving the complaint. The negative feedback seemed to create or maintain the belief that the complaint itself, not the presentation of the complaint, was unjustified or irrational. Discussing this issue in a supportive group may have validated ES members feelings, reduced their feelings of guilt, and promoted problem solving behaviors.

Differences between the ES group and WL group for the total symptom score at post-treatment may be related to women in the ES group feeling supported. Feelings of guilt may have also been reduced. These ideas are supported by responses on the Final Questionnaire for the ES group. All the women whose symptoms improved in the ES group ranked "group support" and "learning not to feel guilty because they suffered from PMS" as one of the top three components of treatment. All of these women also indicated they were practicing problem solving skills. Having PMS sufferers consciously plan to solve problems at other times besides their premenstrual phase may be a good coping strategy.

Group Differences on the Pain Subscale. There were significant differences between the ES and WL group at post-treatment during the premenstrual phase on the pain subscale. These differences were not found during the menstrual phase. The pain subscale score includes the items: muscle stiffness, headache, cramps, backache, general aches and pains, and fatigue. Pain scores may have been influenced by two of the coping mechanisms discussed in group treatment sessions: (1) refraining from overcompensating and (2) practicing relaxation procedures.

Women in the group described how they felt less productive and fatigued at home or work during their premenstrual phase. Many expressed how they actually made themselves work harder during their premenstrual phase so that they would be as productive as they are during their intermenstrual phase. This extra effort seemed to result in increased fatigue, and these women continued to be feel less productive. One

coping strategy suggested in group was to lower their expectations of their work performance premenstrually, or at least not exacerbate symptoms by trying to overcompensate.

Relaxation procedures were also discussed and practiced during one session. Despite the fact that participants were not encouraged to practice these procedures outside of the group, a number of participants stated on the Final Questionnaire for the ES group that they were practicing relaxation procedures daily or at least when they felt stressed. This may have lowered pain scores since relaxation procedures have been found to be helpful in alleviating headaches (e.g. Blanchard, Andrasik, Ahles, Teders, & O'Keefe, 1980) and chronic pain (e.g. Keefe, 1982).

Interestingly, pain scores were different during the premenstrual phase, but not the menstrual phase. Pain experienced during these phases may be "qualitatively" different. The type of pain experienced in the premenstrual phase may be influenced by engaging in coping strategies or feeling supported, while pain experienced in the menstrual phase may not be influenced by these factors. This later point is supported by research on dysmenorrhea, or painful menstruation, which suggests that relaxation and other psychological techniques are relatively ineffective in reducing symptoms during the menstrual phase (Mohler, 1984).

No Significant Reductions in Symptoms. There were group differences at post-treatment during the premenstrual phase between the and WL groups for the total symptoms score and the pain score, but there were no significant changes across time for

the ES group. In other words, there were no significant changes from pre-treatment to post-treatment, or from pre-treatment to follow-up. This failure to significantly "reduce" symptoms suggests that merely providing support and information about coping strategies is not a sufficient treatment for LLPDD. However, education and support may be an important component of a more "powerful" psychological treatment in which women are encouraged to practice coping strategies (e.g. problems solving and relaxation techniques) during treatment sessions and outside of treatment sessions.

Stress, Exercise and Caffeine: Are These Variable Related to LLPDD?

There was a strong correlation between stress and the mood subscale score and the total symptom score. Although causality can not be inferred, this correlation is consistent with suggestions that stress impacts premenstrual suffering. Because both the premenstrual symptoms and stress were monitored on the Daily Monitoring Measure, these results may reflect a response bias. Although this may be true, it does not explain why the correlation between stress and the pain subscale is relatively small ($r=.13$, ns) compared to the mood subscale score ($r=.62$, $p<.001$), the water retention subscale score ($r=.37$, $p<.10$) or the total symptom score ($r=.48$, $p<.05$). Stress scores in this study also represented an entire cycle of monitoring stress prior to and during the premenstrual phase. Thus, stress scores reflect an accumulation of daily stressors across a menstrual cycle, not just how stressed one feels during the premenstrual phase. This relationship between stress and premenstrual symptoms suggests that managing stress

may be an effective treatment for LLPDD, especially for the emotional symptoms associated with LLPDD.

Why stress was significantly correlated with the total symptom score and the mood subscale score but not the pain subscale score is unclear. The nonsignificant correlation between the pain subscale score and stress appears to contradict previous comments in this paper that relaxation techniques (a form of stress management) may be helpful in reducing pain symptoms during the premenstrual phase. It is possible that stress is not associated with pain symptoms during the premenstrual phase and that relaxation and other coping strategies taught in session just helped subjects to cope with pain during the premenstrual phase. Another possibility for the nonsignificant correlation between stress and the pain subscale is that the measure of stress was unreliable. This is a possibility since the measure of stress was a single item, even though it ratings were made throughout the menstrual cycle. If this measure was not reliable, the correlations above would also be suspect. Further research is needed to clarify the relationship between stress and premenstrual distress, and the relationship between stress and the various types of premenstrual symptomatology.

The correlation between exercise and premenstrual symptoms was not significant. This is not surprising considering that few, if any, of the subjects engaged in extremely rigorous and consistent exercise programs. Research on exercise and PMS suggest PMS is lower in women who participate in intense exercise programs. For example, Berka (1990) found that intercollegiate athletes are less likely to report premenstrual distress

compared to recreational athletes and non-athletes. Moreover, treatment studies looking at the effectiveness of exercise in reducing premenstrual symptoms tend to have subjects engage in extremely rigorous exercise regiments (e.g. Lemos, 1990). Women who exercise intensely may learn to deal with pain or discomfort, or there may be a critical level of exercise intensity before this method is effective in relieving premenstrual distress.

Caffeine was also not significantly correlated with the total symptoms score or any of the subscale scores. However, the correlation between caffeine and the pain subscale score was relatively high ($r=.37$, $p<.10$). This result seemed to contradict previous findings. Rossignol (1985) and Rossignol and Bonnlander (1990) both reported a relationship between high caffeine consumption and premenstrual distress. Although the sample sizes were large in these two studies, retrospective reports were used. Further research using prospective monitoring is needed to assess the relationship between caffeine and premenstrual symptomatology.

Summary of Interpretations.

Reducing fat consumption, by itself, does not appear to be an effective treatment for LLPDD. However, changes in other dietary practices and nutrients may reduce premenstrual distress. Information obtained from this study and other research reports indicate that a high-carbohydrate, low-protein diet may be effective in relieving premenstrual distress. In addition, PMS or LLPDD may be related to malnutrition or

low caloric intake; thus, increasing caloric consumption may improve premenstrual symptom severity. Further research is needed to test these hypotheses.

Pursuing psychological treatments of PMS may prove to be beneficial. Results suggested there was a slight benefit to participating in the ES group as compared to receiving no treatment. This advantage may have been related to group support, learning not to feel guilty, and using coping skills such as problem solving and relaxation techniques. Even though the ES treatment was better than receiving no treatment, symptoms did not reduce significantly from pretreatment to post-treatment suggesting that the ES treatment in this study was only minimally effective.

Methodological Problems

This study had a number of methodological problems. These included methodological problems commonly seen in PMS research and errors associated with the assessment of dietary variables.

Methodological Problems and Issues Associated with Studying LLPDD.

Researching LLPDD is a complex task. Diagnosing and selecting the appropriate subjects is difficult, but important. A brief review of the literature suggests that attrition rates are very high when daily prospective monitoring is used. Thus, sample sizes reported in most studies tend to be small. Exclusion criteria may reduce sample size

even further. Length of time needed to adequately study LLPDD may promote response bias, poorly completed forms, and boredom with daily monitoring and treatment.

All the women in this study met the provisional criteria for LLPDD. Diagnosis was based on information obtained from the Structured Interview. Participants clearly reported premenstrual distress in which psychological symptoms were the predominant feature. Participants also expressed a period of symptom relief during the intermenstrual phase of their menstrual cycle and stated that premenstrual distress interfered with their social and/or occupational functioning. Selecting subjects based on the provisional criteria for LLPDD is an improvement over many treatment outcome studies with unclear entry criteria, but prospective monitoring is needed to confirm a diagnosis of LLPDD.

Ideally, subject selection should be based on two cycles of prospective monitoring. This criteria was not used in this study so that the amount of daily monitoring could be limited to one cycle prior to treatment. At the inception of this project, it was decided that increasing the length of daily monitoring would potentially lead to higher attrition rates. Recruitment of subjects and maintaining them in the study was an anticipated difficulty, so limiting daily monitoring to one cycle prior to treatment was expected to minimize this potential problem.

Despite the above reasoning, failure to use two cycles of prospective monitoring may have resulted in women with emotional distress throughout their menstrual cycle being included in the study. However, group analyses found very significant differences

between premenstrual and intermenstrual symptom scores for all groups, suggesting women in this study were suffering from a disorder related to their menstrual cycle.

When prospective monitoring is used as a selection criterion to determine if participants have LLPDD, daily ratings obtained during the premenstrual phase are compared to daily ratings obtained during the intermenstrual phase. A 30% reduction in symptoms from the premenstrual phase to the menstrual phase has been suggested as a cut off criterion to diagnose someone with severe premenstrual distress (see Anderson, Severino, Hurt, & Williams, 1988). If the total symptom scores for the pretreatment cycle in this study are used to compare the premenstrual to the intermenstrual phase, 65% of the participants met this strict criterion. An additional 19% of the subjects reported about a 20% reduction of symptoms from the premenstrual to the menstrual phase. These women may be described as having moderate to severe premenstrual distress. There were only four women who had less than a 10% reduction of symptoms from the premenstrual to the menstrual phase. Two of these women were in the ES group and the other two women were in the LF group. The two women in the LF group (case #8 and case #10, see Table 20) symptoms appeared to decrease after treatment, while the two women in the ES group (case #16 and *#18, see Table 20) symptoms appeared to increase after treatment. Thus, including these women in this study probably did not significantly influence the results of this study (i.e. the low-fat diet used in this study did not appear to reduce premenstrual distress, and the education-support group appeared to have a slight impact on premenstrual symptom severity).

Although a few women in this study may be more accurately diagnosed with an affective or anxiety disorder, this study appeared to exclude women with other common menstrual disorders (i.e. dysmenorrhea, cyclical mastopathy). A woman who reported severe premenstrual and menstrual cramping was excluded at the onset of the study. This woman said she was diagnosed by physician as having dysmenorrhea. One woman assigned to the LF condition was excluded from group analyses because she did not report emotional distress at any time during her menstrual cycle on the Daily Monitoring measure, and her daily reports suggested she was suffering from cyclical mastopathy. Interestingly, this was the only woman in this study who met the provisional diagnostic criteria for LLPDD and who reported a physical complaint (i.e. sore breasts) first when asked what premenstrual symptoms she experienced during the Structured Interview.

In addition to questions about premenstrual symptoms, subjects were also asked on the Structured Interview if they suffered from any gynecological or physical problems. All of the participants in this study denied having any serious gynecological problems including endometriosis and pelvic inflammatory disease. However, yeast infections or a history of yeast of infections were common. Of course, a medical evaluation would be necessary to confirm the absence or presence of these disorders (including dysmenorrhea and cyclical mastopathy). Gynecological exams and physical have been performed in several studies to confirm the general health of participants (e.g. Stone et al., 1991), but they were not performed in this study. Requiring physical or

gynecological exams for inclusion into a LLPDD study is an added burden to the participant, and it may further increase attrition rates. Moreover, a simple physical exam can not definitively rule out some gynecological problems such as endometriosis (surgery is needed to make a conclusive diagnosis: see Yankauskas, 1990). Although there is a possibility that women with a physical ailments were included in this study, its important to remember that emotional symptoms were the predominant feature. Some type of physical pain tends to be the primary symptom of the disorders mentioned above.

Another diagnostic criteria for LLPDD is the remission of symptoms shortly following the onset of menstruation. Exactly when symptoms should end is unclear. Some researches purport that symptoms should dramatically disappear at the onset of menses (e.g. Clayton, 1984). However, others have suggested that symptoms may linger into the menstrual phase (Reid, 1991). In general, this study (which looked at the four days prior menses and the four days following menses) found no significant differences between the premenstrual and menstrual phases. These results suggested symptoms began during the premenstrual phase and continued into the menstrual phase. one could assert that women in this study did not meet diagnostic criteria for LLPDD, but this implies that subjects really suffered from another emotional or menstrual disturbance. Given the discussion above, this seems unlikely for the majority of subjects in this study. Another interpretation is that symptoms of LLPDD can continue into the menstrual phase. This diagnostic issue will probably be debated for some time.

Contrary to the above criterion, researchers generally agree that premenstrual distress must interfere with social or occupational functioning for LLPDD to be diagnosed. This criterion distinguishes LLPDD from "normal" variations of mood and physical symptoms associated with the menstrual cycle. Although subjects in this study reported that premenstrual symptoms interfered with their personal relationships, social activities, and/or work performance on the Structured Interview, no standardized measure of this disturbance was obtained. This oversight is common, but there are a few exceptions. For example, Stone et al. (1991) used the Global Assessment Scale (Endicott & Spitzer, 1978) to assess social and occupational functioning.

Measuring social and occupational functioning prospectively or daily has also been recommended for accurate diagnosis and as a dependant measure. This study did try to measure quality of relationships and/or work performance on the Daily Monitoring Measure, but insufficient data made it impossible to analyze the results of this measure. Subjects were asked to select two relationships (e.g. husband, children) or activities (e.g. work) that they felt were negatively influenced by their premenstrual distress. This type of procedure was used in a previous study which assessed the effects of teaching women time-management skills (e.g. King, Winett, & Lovett, 1986). Unfortunately, this method of assessment was not an appropriate technique in this study. Although subjects rated the quality of relationships and/or activities daily, they often did not engage in the activity or relationship during their premenstrual phase; thus, lack of sufficient data made it impossible to use this as a dependent measure or as a diagnostic tool. A prospective

measure of occupational and social functioning will probably need to be more global. This type of measure may be helpful for assessing the impact of treatment and as a diagnostic instrument.

Methodological concerns related to diagnostic issues are compounded by the small sample size of this study. Although a fair number of subjects were recruited for this study, attrition rates were high, possibly because prospective monitoring of symptoms was used to assess the effectiveness of the LF and ES treatments. Approximately 50% of the women who were initially interviewed dropped out before treatment began. This rate of attrition is common when prospective monitoring is employed (e.g. Morse, et al., 1991; Stone et al. 1991). Stone et al. (1991) assert that using prospective measures actually screens out women who do not suffer from LLPDD and that this procedure reduces the number of placebo responders. Women who are willing to monitor their symptoms and behaviors daily may also be more willing to follow treatment recommendations. These comments suggest that there is an advantage to high attrition rates, even though high attrition rates result in small sample size.

Exclusion criteria can also reduce sample size. Trying to avoid this problem, exclusion criteria for this study were not as stringent as many studies in this area. For example, a number of studies exclude women taking oral contraceptive or anti-depressant medication, while women on these medications were allowed to participate in this study. It may be argued that inclusion of these women obscured results. On the other hand,

inclusion of these women promotes generalizability of results because many women who suffer from LLPDD take these medications.

Extremely strict exclusion criteria and daily monitoring may result in very large numbers of women being excluded from participating in a research projects studying LLPDD. For example, Stone et al. (1991) solicited 487 women from a newspaper ad to participate in a treatment-outcome study testing the effectiveness of fluoxetine. Four hundred-fortythree of these women appeared to meet the provisional criteria for LLPDD as indicated over a telephone interview. These women were asked to monitor their symptoms daily for two full menstrual cycles, only 152 completed this task. Eighty-one of these women were excluded because: (1) they did not have a 30% reduction in symptoms from the premenstrual to the intermenstrual phase for two consecutive menstrual cycles as indicated by a daily monitoring measure; (2) they met criteria for a current psychiatric disorder as determined by the Schedule of Affective Disorders and Schizophrenia (Endicott & Spitzer, 1978); (3) they did not report social or occupation impairment on the Global Assessment Scale (GAS); or (4) they were taking prescribed medication. Of the remaining 71 women, 25 declined to participate in the study because they did not want to take medication or because they did not want to be involved in a placebo-control study. Only twenty women completed the entire research protocol. Although this is probably a "model" study for LLPDD because subject selection was so selective, one has to wonder if the results of this study can be generalized to most women who report suffering from premenstrual distress.

The Stone et al. study is also considered a model study because prospective daily monitoring was used as a dependent measure. Participants in this study were required to monitor symptoms daily for at least five menstrual cycles. This consistent monitoring could promote boredom and response bias. This study and the Stone et al. study did not address this issue, but Morse et al. (1991) used three separate versions of their daily monitoring measure to overcome monotony and to reduce the tendency toward response bias. The necessity of this technique is questionable, and this procedure may be burdensome to the researcher (e.g. increase time to code data).

In general, studies on LLPDD are demanding for both researchers and subjects. Methodological flaws are common, but more recent studies are trying to overcome previous mistakes. Sample size and diagnostic issues seem to be the primary concerns of many studies of PMS and LLPDD; unfortunately, they are also major concerns of this study.

Methodological Concerns Related to Dietary Assessment.

There were also methodological errors associated with the reporting of dietary intake. Directions on how to complete the food diaries may have been inadequate. Moreover, this study only had subjects monitor food intake for six days out of the cycle which may have resulted in inaccurate estimates of dietary variables (e.g. grams of fat, calories).

Directions for completing the Food Diary were given verbally after subjects answered questions from the Structured Interview. For most subjects the Structured

Interview lasted about an hour; thus, concentration may have been poor when directions were given. Even though typed directions were also provided, many subjects failed to follow these instructions.

Subjects were asked to monitor food intake for the Sunday, Monday, and Tuesday prior to menses (3 days luteal phase) and for the Sunday, Monday, and Tuesday after the onset of menses (3 days follicular phase). Monitoring food consumption was limited to six days because additional monitoring was expected to increase the attrition rate. This concern appeared to be legitimate because many subjects indicated that they hated completing the food diaries. Unfortunately, this procedure may have resulted in inaccurate estimates of dietary variables. For example, one subject stated they she did not eat much during the days she monitored her food intake because she did not want to write down what she ate. Although sampling of dietary data may have been inadequate, it was not much different than the sampling procedures used in the two treatment studies of cyclical mastopathy described in Chapter I (Boyd et al., 1988; Rose et al., 1985). Four and three day food records were used in these two studies.

Unlike the two studies for cyclical mastopathy, this study also tried to control for different eating patterns associated with the menstrual cycle (see Gong, Garrel, & Calloway, 1989). This study choose specific days (i.e. Sunday, Monday, Tuesday) to control for possible eating pattern differences during the different days of the week. Although six days centering immediately around the onset of menses was considered, this option was not chosen because onset of menstruation tends to vary by a couple of days;

thus overlap of the two phases would occur if the predicted day on menses was inaccurate. It was thought that predicting the Sunday, Monday, and Tuesday before menses would be relatively easy because all of the subjects reported during the Structured Interview that they were able to predict the onset of menstruation within two or three days. Overlap between the two phases was not an anticipated problem using this procedure, but it was problem. For example, several women started menstruating while they were monitoring food intake for the luteal phase. Thus, this study did not truly control for variations in eating patterns related to the menstrual cycle.

Failure of several women in the ES and WL conditions to even monitor their food intake at post-treatment and follow-up another problem. These women basically decided that monitoring their food consumption was too bothersome or that it was not important because they did complete the Daily Monitoring Measure. Because these women were in the ES and WL groups, they probably did not realize the importance of monitoring their food intake.

Even those who completed their food records may have become bored or resistant to completing the Food Forms towards the end of treatment. This may have led to inaccurate or incomplete recordings of food intake at follow-up. Less accurate recording at follow-up may explain why there were no significant group differences at follow-up for grams of fat or for percentage of fat, even though there was a significant reduction in fat from pre-treatment to follow-up for the LF group. Inaccurate or incomplete

recording of food records is a common problem; however, there may have been ways to improve the reliability of recorded dietary intake.

Reliability of food monitoring may have been enhanced if a trained nutritionist gave instructions on how to complete food records on a separate day from the intake interview. Having subjects use specific measuring tools and scales to weigh food would also improve reliability. Increasing the number of days food intake was monitored would also have improved estimates, especially if consecutive days were used instead of three days one week and three days on another week (see Block, 1982). Subjects may have been willing to complete Food Forms more accurately if they were paid to complete the forms. Despite the fact that these techniques may have improve reliability estimates, they may also have sensitized women in the ES and WL groups to the importance of diet in this study. This emphasis may have resulted in ES and WL group members changing their diets.

Concluding Remarks

Late Luteal Phase Dysphoric Disorder (LLPDD) was recently included in the DSM-III-R (American Psychiatric Association, 1987) as a provisional diagnostic category. This diagnoses was intended to represent a subset of women suffering from premenstrual distress in which emotional symptoms are the predominant feature, and the diagnosis was established to help improve treatment and advance research by providing

specific symptom criteria (Hamilton & Gallant, 1990). Accurate diagnosis and differentiation from other disorders related to the menstrual cycle is important because response to treatment and etiology of these disorders may differ. Although most treatment-outcome studies of PMS and LLPDD have explored the efficacy of various medications, behavioral and psychological interventions may be effective, and these types of treatments deserve further investigation.

Testing the effectiveness of a behavioral treatment, this study compared the efficacy of a low-fat diet in treating LLPDD to an education-support group and a waiting-list control group. The low-fat diet used in this study was not effective in relieving emotional and physical distress experienced by women suffering from LLPDD, despite previous research which indicated that a low-fat diet was effective in relieving cyclical mastopathy.

Failure of this study to find a low-fat diet effective in treating LLPDD may be related to a number of factors including the design of the treatment intervention, negative emotions associated with "dieting", or methodological problems. The treatment design of the treatment program may have been inadequate. For example, the length of treatment may have needed to be increased, or dietary change may have needed to be monitored more closely. Other factors such as negative emotions associated with dieting may have negated any treatment effects associated with the low-diet. Methodological problems including small sample size, high attrition rates, and less stringent exclusion criteria may have influenced results. On the other hand, a low-fat diet may truly be an

ineffective in treatment for LLPDD. The theoretical assumptions relating raised estrogen levels to LLPDD and low-fat diets to lowered estrogen levels may be incorrect.

Raised estrogen levels were hypothesized to cause premenstrual distress, and a low-fat diet was predicted to lower estrogen levels. A study on cyclical mastopathy (Rose et al., 1985) suggested that a low-fat diet could reduce estrogen levels and reduce the severity of breast tenderness. This result indicates that cyclical mastopathy is related to raised estrogen levels. If results of this study are correct, and the low fat diet did reduce estrogen levels (which is unknown because this was not measured), then estrogen levels may not be the "cause" of PMS. Although results of this study imply that a low-fat diet is not an efficacious treatment of LLPDD, testing the effectiveness of this treatment again under more ideal circumstance (i.e. measuring estrogen levels, careful control of fat intake) may provide additional insight into the etiology of LLPDD.

Although a low-fat diet in this study did not appear to be an effective treatment of LLPDD, other dietary changes, which are based on different theoretical premises, may reduce premenstrual suffering. Negative correlations between calories consumed and premenstrual symptoms suggest that malnutrition may play a role in the severity of premenstrual distress. Thus, increasing caloric consumption, or the consumption of carbohydrates, may improve premenstrual distress. Carbohydrates have been found to promote the synthesis and release of serotonin (Fernstrom et al., 1979). This type of treatment suggests that changing serotonin levels should be the focus of treatment of LLPDD. In support of this idea, well controlled treatment outcome studies have found

medications which block the reuptake of serotonin (i.e. fluoxetine, d-fenfluramine) to be effective in treating LLPDD (e.g. Stone et al., 1991).

A well-controlled dietary study also suggests that serotonin may play a role in the reduction of premenstrual distress. Wurtman et al. (1989) reported that a high-carbohydrate, low-protein meal was effective in immediately reducing premenstrual distress. This diet was thought to be effective because carbohydrates promote the synthesis of serotonin while protein consumption inhibits the production of serotonin (Fernstrom et al., 1979). The present study did not contradict these findings because there were no significant changes in grams of carbohydrates or grams of protein consumed. Moreover, there was a significant positive correlation between percentage of protein consumed and premenstrual symptoms, and a negative correlation between grams of carbohydrates consumed and the emotional symptoms of LLPDD. Further research is needed to see if a highcarbohydrate, low-protein diet is an effective treatment of LLPDD. Additional research is needed to confirm Wurtman et al.'s report that a high-carbohydrate, low-protein meal immediately reduces premenstrual suffering, thus suggesting that this type of meal can be used as a coping strategy to temporarily reduce premenstrual distress.

Dietary changes outlined above may reduce premenstrual symptomatology, but "psychological" techniques also appear to be promising. There were significant differences between the Education-Support group and the Waiting-List control group at post-treatment, suggesting that there was a slight advantage to participating in a group

that provided support and education compared to receiving no treatment. However, this treatment approach can only be considered minimally effective (or not effective at all), because there were no significant reductions in symptoms from pre-treatment to post-treatment.

The education and support treatment was actually designed to be only marginally effective, because it was serving a "quasi-control group" for the low-fat diet intervention. Subjects in the Education-Support group were not encouraged to practice various coping strategies such a problem solving, relaxation, and cognitive restructuring outside of the session. However, information obtained following treatment suggested that those who used these techniques on their own also reported a reduction in premenstrual distress.

Information gathered in group sessions suggested that several specific coping strategies may be helpful in reducing

premenstrual symptoms including: (1) teaching women to solve problems at other times besides their premenstrual phase, (2) teaching them to not to feel guilty because they suffer from PMS, and (3) teaching women that they don't need to overcompensate for perceived deficits in work productivity.

Research reports on the effectiveness of "psychological" and "behavioral" treatments of LLPDD are relatively scarce. Psychological treatments may need to include a number of techniques to be effective. For example, Morse et al. (1990) reported that a treatment using rational emotive therapy, problem solving, and assertiveness training was effective in reducing premenstrual distress. As indicated

above, dietary changes which increase carbohydrate consumption my be helpful in alleviating symptoms of premenstrual distress. Accumulating body of evidence suggests that intense exercise programs are effective in reducing premenstrual symptomatology (e.g. Lemos, 1990). Further treatment-outcome studies for behavioral and psychological treatments are greatly needed.

The above recommendation and comments suggest that there is a "standardized treatment" which will be effective in reducing symptoms of LLPDD for most women suffering from this disorder. Although this may be true, it is possible that treatment of LLPDD may need to be more individualized. For example, one woman with LLPDD may benefit from marital therapy and relaxation training, while another LLPDD sufferer may respond better to an intensive exercise program.

An individualized treatment program of LLPDD may be effective if it is based on a comprehensive assessment of: (1) situations (or stimuli) that seem to exacerbate or reduce LLPDD symptoms, (2) responses to feeling premenstrual distress, and (3) consequences of these responses. A case study presented by Kuczmierczyk (1989) exemplifies this point. Kuczmierczyk describes a woman whose premenstrual symptoms appeared to be exacerbated by marital problems and work-related stressors. The patient response to premenstrual suffering was to take sick leave for 3-4 days. The consequence of this behavior was immediate relief from work, and she also elicited positive supportive behavior from her husband. Thus, positive "rewards" for being sick during her premenstrual phase may have maintained her symptoms. Based on this assessment,

treatment focussed on teaching the patient how to alleviate marital difficulties and helped the woman cope with work related problems by teaching her assertiveness skills and relaxation techniques. This individualized treatment approach appeared to be very effective in relieving premenstrual symptoms for this case.

The suggestion that treatment may need to be more individualized implies that the manifestation and maintenance of LLPDD may depend on upon a complex set of etiological factors. For the most part, this paper has presented a number of etiological theories (e.g. elevated estrogen, reduced beta-endorphin levels, psychosocial factors) as if these factors were mutually exclusive. A more accurate conceptualization of the "cause" of LLPDD probably includes a combination of various physiological and psychological factors. This biopsychosocial perspective and is intuitively appealing but extremely difficult to study. Further research is needed to clarify the role of various etiological factors and the interaction of these factors.

Unfortunately, conducting etiological and treatment-outcome studies for LLPDD is extremely difficult. Daily prospective monitoring is recommended, but this increases attrition rates. Exclusion of women on prescribed medication has also been suggested, but this also limits sample size and reduces generalizability of results. Gynecological and physical examinations to rule out physical disorders have been conducted in a number of studies, but this is an additional expense that may also decrease sample size. Studies that have employed these methods have been supported by pharmaceutical companies and have been located in densely populated areas. Thus, following all the recommended

methodological suggestions for LLPDD is going to severely limit who conducts LLPDD studies and where these studies are conducted.

Fortunately, a number of well controlled studies have been conducted despite the problems mentioned above. Better controlled studies seemed to be published after LLPDD was included into the DSM-III-R as a provisional category. Diagnostic issues may be very important for making treatment recommendations. For example, this study suggested that a low-fat diet was not a good prescription for LLPDD, while other studies strongly suggested that a low-fat diet was an excellent treatment option for those suffering from cyclical mastopathy. Despite the advantages of a clear diagnostic category, a number of individuals and women's organizations are concerned that diagnosing severe premenstrual distress as a "mental disorder" will lead to work discrimination and other prejudicial practices against women (see Spitzer et al., 1989). Although there may be some merit to this concern, women who suffer from severe premenstrual distress deserve to be accurately diagnosed and given treatment recommendations that will be helpful in alleviating their suffering. These recommendations should be empirically based, rather than on unsubstantiated claims that a particular treatment is effective. A standardized definition is needed to accomplish this goal.

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Table 1. Demographic Information

	Low-Fat n=10	Education-Support n=8	Waiting-List n=8
Marital Status			
Single	n=1	n=0	n=1
Married	n=5	n=7	n=5
Separated/ Divorced	n=3	n=1	n=2
Highest Degree Earned			
Graduate	n=4	n=3	n=2
Bachelor	n=5	n=3	n=4
Associates	n=1	n=1	n=0
High School	n=0	n=1	n=2
Oral Contraceptive	n=1	n=2	n=2
Anti-Depressant	n=2	n=0	n=1
Non-parous	n=4	n=3	n=3
Children			
Two Children	n=3	n=4	n=3
One Child	n=2	n=0	n=2
No Children	n=5	n=4	n=3
Age	Mean=35.6 (Range 23-46)	Mean=37.5 (Range 25-43)	Mean=34.25 (Range 24-40)

Table 2. Mean Total Symptom Score and ANOVA Results

Group Phase	Time: Pre-Treatment Mean	Pre-Treatment S.D.	Post-Treatment Mean	Post-Treatment S.D.	Follow-up Mean	Follow-up S.D.
Low-Fat						
Premen.	159.4	47.3	146.0	36.9	140.0	45.4
Menstrual	166.2	60.3	150.0	50.3	137.8	39.9
Intermen.	115.3	38.0	112.5	23.2	95.7	18.2
Education-Support						
Premen	148.9	47.0	127.2	24.3	116.2	26.1
Menstrual	129.6	34.0	128.1	39.8	114.6	33.3
Intermen.	96.6	25.8	87.1	8.7	87.9	15.0
Waiting-List						
Premen.	181.5	50.1	194.8	74.5	175.8	78.5
Menstrual	159.1	41.7	152.5	51.8	170.1	64.5
Intermen	103.1	20.0	106.4	16.4	118.3	30.0

Note: Group x Time interaction $F(4,46) = 1.33$, ns
Main effect for group $F(2,23) = 3.29$, $p<.05$
Main effect for time $F(2,46) = 2.10$, ns
Main effect for phase $F(2,46) = 29.72$, $p<.0001$
ns-nonsignificant at $p<.05$

Table 3. Mean Mood Symptom Score and ANOVA Results

Group Phase	Time					
	Pre-Treatment		Post-Treatment		Follow-up	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Low-Fat						
Premen.	66.2	21.6	62.8	20.4	60.3	21.1
Menstrual	71.3	28.2	58.3	21.1	57.5	20.1
Intermen.	54.5	28.9	47.3	13.2	45.0	12.2
Education-Support						
Premen	70.5	24.2	59.8	13.5	52.6	23.5
Menstrual	54.5	19.0	59.5	22.7	49.7	21.3
Intermen.	44.6	14 9	39.4	5.4	39.6	9.9
Waiting-List						
Premen.	84.5	28.0	89.1	44.9	82.1	43.0
Menstrual	70.4	26.1	71.8	24.6	73.9	37.0
Intermen	48.1	12.9	50.0	11.4	56.9	27.3

Note: Group x Time interaction $F(4,46) = .87$, ns
Main effect for group $F(2,23) = 2.58$, ns
Main effect for time $F(2,46) = 1.19$, ns
Main effect for phase $F(2,46) = 21.11$, $p<.0001$
ns - not significant at $p<.05$

Table 4. Mean Pain Symptom Score and ANOVA Results

Group Phase	Time: Pre-Treatment Mean	Pre-Treatment S.D.	Post-Treatment Mean	Post-Treatment S.D.	Follow-up Mean	Follow-up S.D.
Low-Fat						
Premen.	58.3	22.7	43.3	13.9	43.2	14.0
Menstrual	59.7	20.5	58.2	25.9	51.7	15.5
Intermen.	40.0	11.1	45.5	19.8	30.8	5.2
Education-Support						
Premen	46.3	18.2	33.1	6.8	35.6	8.3
Menstrual	46.6	15.8	40.0	10.5	37.8	7.3
Intermen.	32.0	12.7	28.0	4.4	29.1	5.4
Waiting-List						
Premen.	54.5	15.3	59.0	23.4	48.1	20.5
Menstrual	56.1	14.1	46.5	19.5	53.5	19.0
Intermen	34.5	11.0	36.4	6.8	41.4	9.1

Note: Group x Time interaction $F(4,46) = 1.25$, ns
Main effect for group $F(2,23) = 3.98$, $p<.05$
Main effect for time $F(2,46) = 3.99$, $p<.05$
Main effect for phase $F(2,46) = 19.96$, $p<.0001$
ns - nonsignificant at $p<.05$

Table 5. Mean Water Retention Symptom Score and ANOVA Results

Group Phase	Time					
	Pre-Treatment		Post-Treatment		Follow-up	
	Mean	S.D.	Mean	S.D.	Mean	S.D.
Low-Fat						
Premen.	34.9	16.3	39.9	15.3	36.5	16.5
Menstrual	35.2	20.5	33.5	13.7	28.6	13.4
Intermen.	20.8	5.7	19.7	4.8	19.9	5.1
Education-Support						
Premen	32.1	11.1	34.4	13.3	28.0	8.4
Menstrual	28.5	10.1	28.6	14.9	27.1	12.0
Intermen.	20.0	5.1	19.8	4.8	19.1	5.2
Waiting-List						
Premen.	42.5	15.3	46.6	14.9	45.5	19.9
Menstrual	32.6	15.0	34.3	13.1	42.8	20.5
Intermen	20.5	4.4	20.0	4.2	20.0	3.8

Note: Group x Time interaction $F(4,46) = 1.11$, ns
Main effect for group $F(2,23) = 1.62$, ns
Main effect for time $F(2,46) = .33$, ns
Main effect for phase $F(2,46) = 29.19$, $p<.0001$
ns - not significant at $p<.05$

Table 6. Mean Scores for Percentage of Fat and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(2,44)
Low-Fat	33.70	5.40	22.30	4.64	25.40	5.89	6.16**
Education Support	36.29	2.43	35.29	6.87	34.57	2.99	
Waiting-List	33.00	8.59	38.88	6.31	31.13	9.43	

Note: ** $p<.001$

Table 7. Scheffé Pairwise Comparisons Across Time for Percent Fat

Group	Pre-Treatment to Post-Treatment	Pre-Treatment to Follow-up	Post-Treatment to Follow-up
Low-Fat	*	*	*
Education-Support	ns	ns	ns
Waiting-List	ns	ns	ns

Note: ns = nonsignificant
* $\alpha<.05$

Table 8. Mean Scores for Grams of Fat and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	70.80	21.02	39.90	9.24	44.20	14.61	3.42*
Education Support	78.71	36.34	72.42	29.64	69.57	27.66	
Waiting-List	68.88	23.34	75.38	21.29	54.13	18.10	

Note: * $p<.05$

Table 9. Scheffé Pairwise Comparisons Across Time for Grams of Fat

Group	Pre-Treatment to Post-Treatment	Pre-Treatment to Follow-up	Post-Treatment to Follow-up
Low-Fat	*	*	*
Education-Support	ns	ns	ns
Waiting-List	ns	ns	ns

Note: ns = nonsignificant
* $\alpha<.05$

Table 10. Mean Scores for Percentage of Carbohydrates and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	49.10	4.65	59.80	5.81	56.70	6.04	2.77*
Education Support	49.57	2.51	47.86	9.16	48.71	3.64	
Waiting-List	45.50	13.28	50.13	4.82	51.75	10.84	

* $p<.05$

Table 11. Scheffe Pairwise Comparisons Across Time for Percent Carbohydrates

Group	Pre-Treatment to Post-Treatment	Pre-Treatment to Follow-up	Post-Treatment to Follow-up
Low-Fat	*	*	ns
Education-Support	ns	ns	ns
Waiting-List	ns	ns	ns

Note: ns = nonsignificant
* $\alpha<.05$

Table 12. Mean Scores for Grams of Carbohydrates and ANOVA results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group xTime
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	231.7	62.7	246.7	60.7	226.2	68.6	.71
Education Support	238.9	101.3	216.9	84.7	240.3	82.9	
Waiting-List	228.35	84.4	246.0	78.0	211.4	61.3	

Note: No significant differences were found at $p<.05$.

Table 13. Mean Scores for Percentage of Protien and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group xTime
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	16.10	3.84	16.90	3.24	18.20	5.14	.21
Education Support	13.86	1.77	16.29	4.50	15.43	4.35	
Waiting-List	13.88	2.75	17.13	14.73	15.75	17.52	

Note: No significant differences were found at $p<.05$.

Table 14. Mean Scores for Grams of Protein and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group xTime
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	74.60	21.02	68.90	16.27	67.10	18.85	.32
Education Support	67.14	27.23	73.00	23.40	66.29	20.25	
Waiting-List	60.38	19.08	58.00	20.23	53.25	20.40	

Note: No significant differences were found at $p<.05$.

Table 15. Mean Scores for Calories and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	1842	442.6	1602	289.2	1549	413.9	.95
Education Support	1896	802.6	1787	567.3	1787	664.8	
Waiting-List	1764	559.9	1876	629.2	1528	369.9	

Note: No significant differences were found at $p<.05$.

Table 16. Mean Average of Exercise and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	19.21	16.36	18.43	15.76	17.54	16.04	2.93*
Education Support	12.21	11.92	17.58	19.59	28.33	23.28	
Waiting-List	13.01	9.51	8.74	8.06	10.17	11.86	

Note: * $p<.05$

Table 17. Mean Scores for Milligrams of Caffeine Consumed and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	239.0	333.1	192.2	204.5	141.2	162.3	.95
Education Support	316.6	175.9	296.7	199.1	310.1	202.1	
Waiting-List	295.7	251.5	301.9	306.3	337.9	338.3	

Note: No significant differences at $p<.05$.

Table 18. Mean Scores for Average Rating of Stress and ANOVA Results

Group	Pre-Treatment		Post-Treatment		Follow-up		Group x Time
	Mean	S.D.	Mean	S.D.	Mean	S.D.	F(4,44)
Low-Fat	2.363	.924	2.317	1.451	2.476	1.209	.236
Education Support	2.915	1.106	2.971	1.104	2.697	.971	
Waiting-List	2.670	.801	2.151	.550	2.820	.746	

Note: Higher scores indicate greater ratings of stress. No significant differences were found at $p<.05$.

Table 19. Correlations of Variables Thought to Influence Premenstrual Distress

	Daily Symptom Ratings During the Premenstrual Phase			
Variable	Mood	Pain	Water	Total
Stress	.6203***	.1263	.3663	.4760*
Exercise	-.1746	-.1147	.0201	-.1289
Caffeine	.1805	.3662	.1148	.2723
Calories	-.4393*	.0081	-.0311	.2333
Percent Fat	-.2112	.0253	-.1907	-.1577
Grams of Fat	-.5011**	-.0076	-.0627	-.2811
Percent Carbohydrates	-.0740	-.1414	-.2677	-.1768
Grams of Carbohydrates	-.3476	-.0661	-.0488	-.2177
Percent Protein	.3282	.3783	.3274	.4215*
Grams of Protein	-.1473	.3626	.2184	.1417

Note: Two-tailed test of significance *p<.05 **p<.01 ***p<.001 Negative correlations between calories (and grams of fat) and mood suggest that the lower the caloric intake (and intake of fats) the more negative the premenstrual mood. Positive correlations between stress and mood indicated that the higher the rating of stress the more negative the premenstrual mood.

Table 20. Percentage of Symptom Change for Total Symptom Score

Case #	Percentage Change Pre-Tx to Post-Tx	Percentage Change Pre-Tx to Follow-up
Low-Fat		
#1	3% decrease	10% decrease
#2	24% increase	66% increase
#3	24% decrease	44% decrease
#4	22% increase	3% decrease
#5	32% decrease	24% decrease
#6	25% decrease	17% decrease
#7	27% increase	7% decrease
#8	23% decrease	18% decrease
#9	27% increase	2% increase
#10	37% decrease	37% decrease
Education-Support		
#11	17% decrease	7% decrease
#12	45% decrease	56% decrease
#13	47% decrease	30% decrease
#14	24% decrease	10% decrease
#15	30% decrease	50% decrease
#16	19% increase	5% increase
#17	29% increase	19% decrease
#18	40% increase	22% increase
Control		
#21	23% decrease	6% decrease
#22	26% increase	23% increase
#23	20% increase	6% increase
#24	6% increase	47% decrease
#25	24% decrease	34% decrease
#26	50% increase	31% increase
#27	3% increase	4% increase
#28	5% decrease	21% decrease

Note: Decrease indicates a reduction in premenstrual suffering, while increase indicates raised levels of premenstrual distress.

Table 21: Summary of Dietary Variables for Individual Cases in the Low-Fat Diet Condition.

Case#	Fat		Carbohydrates		Protein		Calories
	%	Grams	%	Grams	%	Grams	
Case #1							
Pre-Tx	41%	59gm	43%	140gm	15%	49gm	1269Kc
Post-Tx	24%	50gm	62%	299gm	12%	59gm	1860Kc
Fol.-Up	23%	49gm	61%	285gm	16%	75gm	1836Kc
Case #2							
Pre-Tx	26%	34gm	53%	153gm	21%	62gm	1117Kc
Post-Tx	20%	28gm	59%	188gm	15%	48gm	1238Kc
Fol.-Up	22%	20gm	60%	124gm	14%	29gm	800Kc
Case #3							
Pre-Tx	32%	70gm	32%	230gm	18%	90gm	1931Kc
Post-Tx	19%	32gm	60%	233gm	21%	84gm	1531Kc
Fol.-Up	27%	50gm	55%	225gm	17%	71gm	1605Kc
Case #4							
Pre-Tx	39%	95gm	47%	256gm	13%	73gm	2154Kc
Post-Tx	20%	42gm	63%	297gm	16%	77gm	1802Kc
Fol.-Up	22%	51gm	57%	293gm	17%	87gm	2022Kc
Case #5							
Pre-Tx	27%	51gm	48%	210gm	24%	104gm	1699Kc
Post-Tx	21%	42gm	57%	268gm	22%	101gm	1832Kc
Fol.-Up	20%	40gm	59%	267gm	21%	98gm	1778Kc
Case #6							
Pre-Tx	37%	106gm	47%	305gm	15%	100gm	2559Kc
Post-Tx	18%	41gm	69%	361gm	14%	73gm	2057Kc
Fol.-Up	25%	42gm	60%	230gm	15%	56gm	1498Kc
Case #7							
Pre-Tx	36%	65gm	47%	191gm	16%	65gm	1566Kc
Post-Tx	29%	40gm	52%	160gm	20%	61gm	1217Kc
Fol.-Up	32%	30gm	46%	96gm	23%	47gm	824Kc
Case #8							
Pre-Tx	29%	74gm	58%	329gm	12%	68gm	2228Kc
Post-Tx	20%	28gm	66%	214gm	15%	48gm	1287Kc
Fol.-Up	22%	44gm	63%	284gm	16%	71gm	1790Kc
Case #9							
Pre-Tx	39%	83gm	46%	217gm	14%	65gm	1866Kc
Post-Tx	20%	37gm	60%	247gm	16%	67gm	1626Kc
Fol.-Up	22%	41gm	60%	249gm	18%	77gm	1627Kc
Case #10							
Pre-Tx	31%	72gm	55%	286gm	13%	69gm	2032Kc
Post-Tx	32%	57gm	50%	200gm	18%	71gm	1574Kc
Fol.-Up	39%	76gm	46%	200gm	15%	66gm	1712Kc

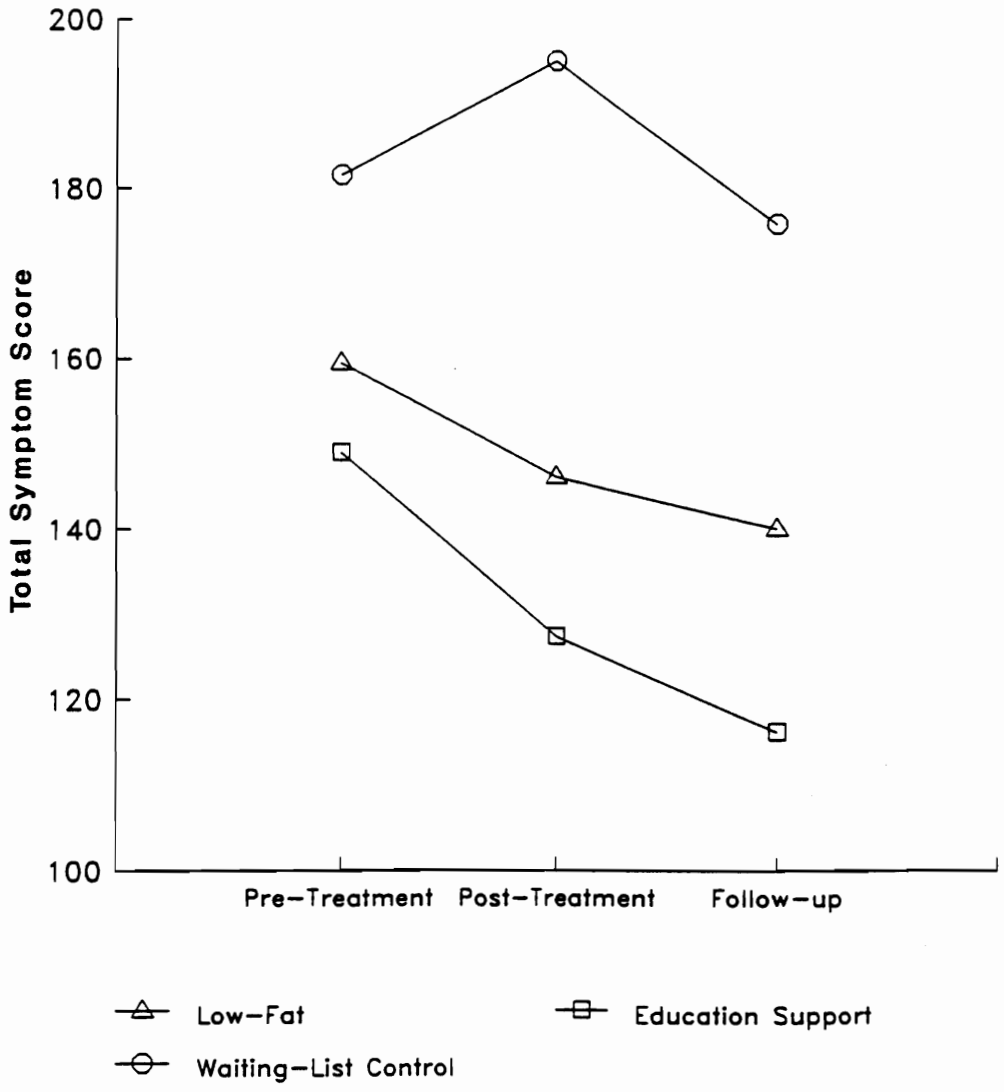

Figure 1. Mean Premenstrual Symptom Score

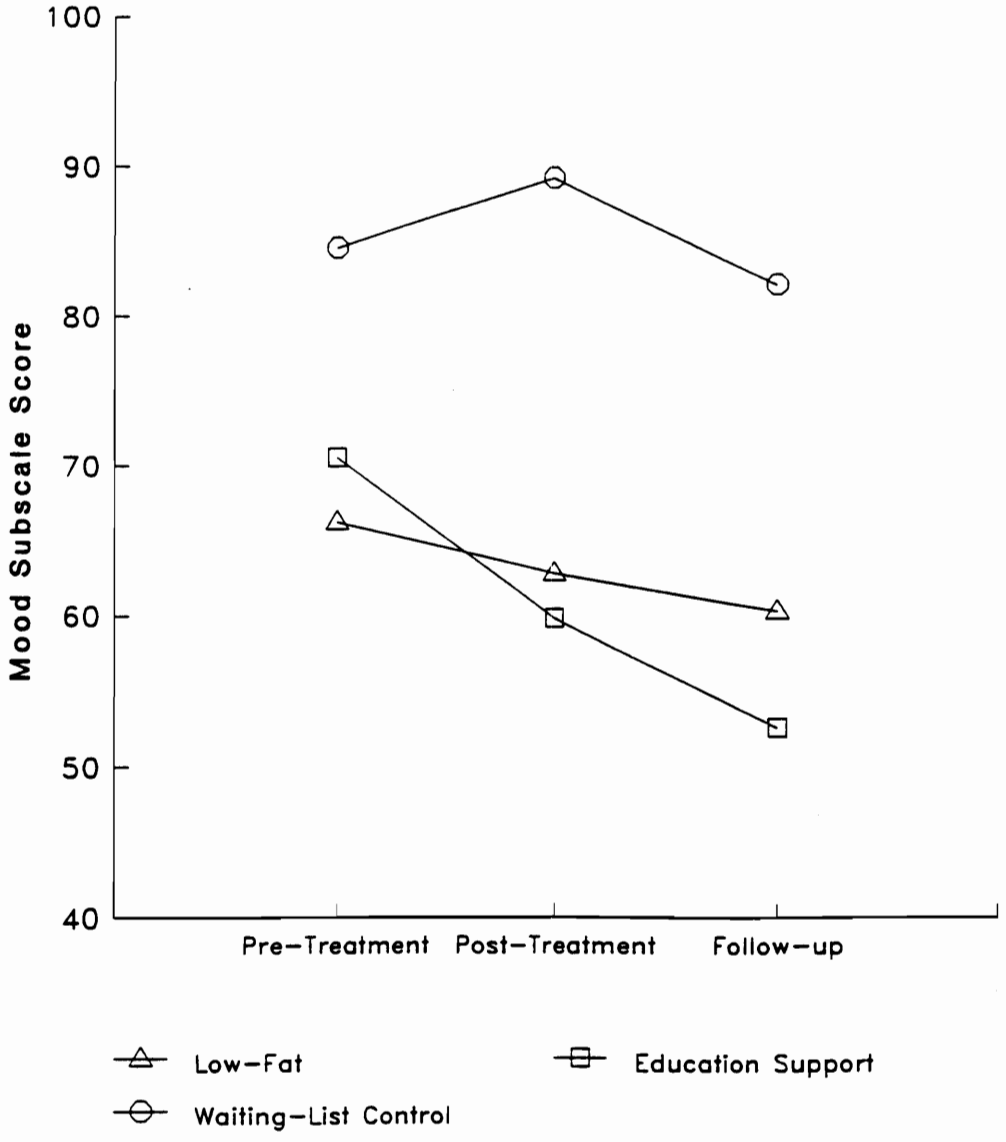

Figure 2. Mean Premenstrual Mood Score

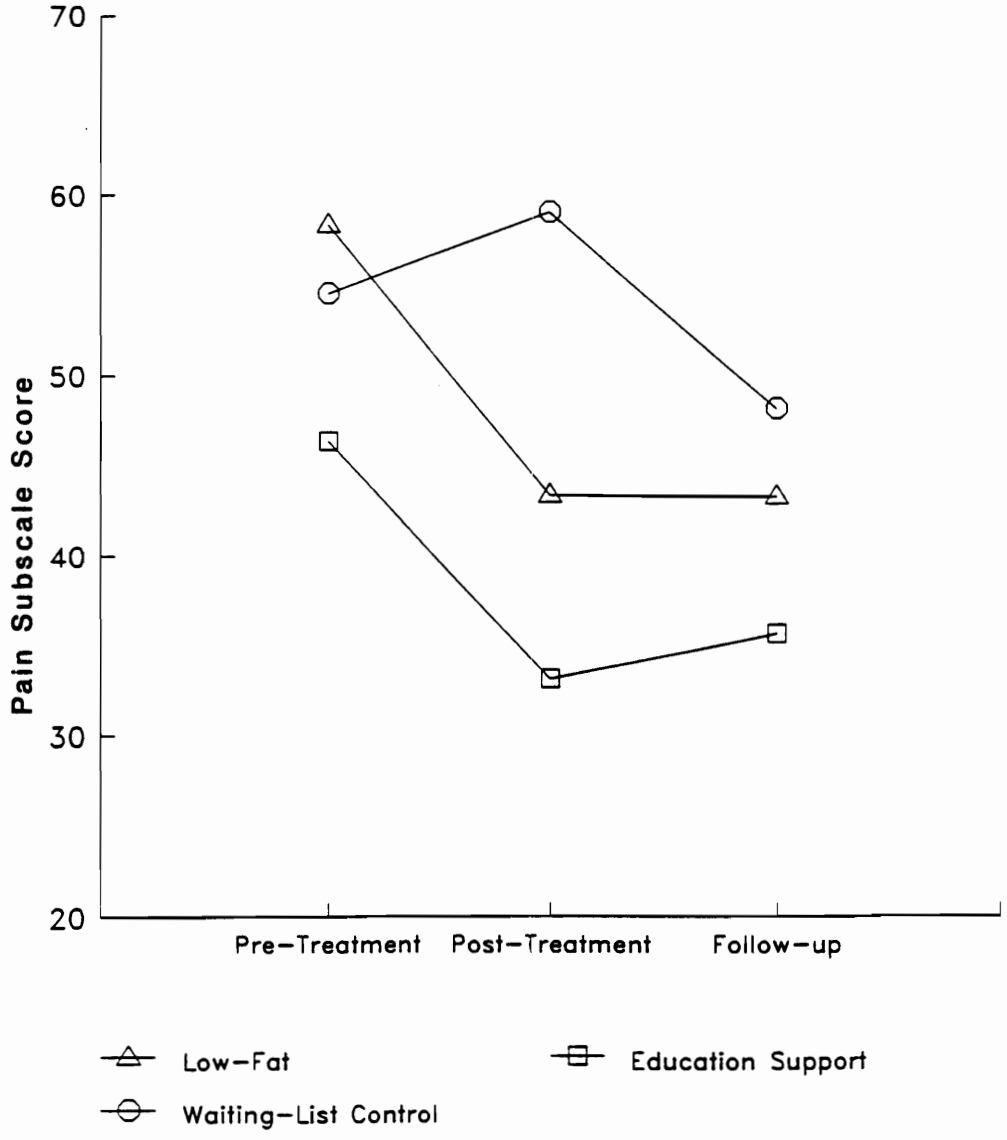

Figure 3. Mean Premenstrual Pain Score

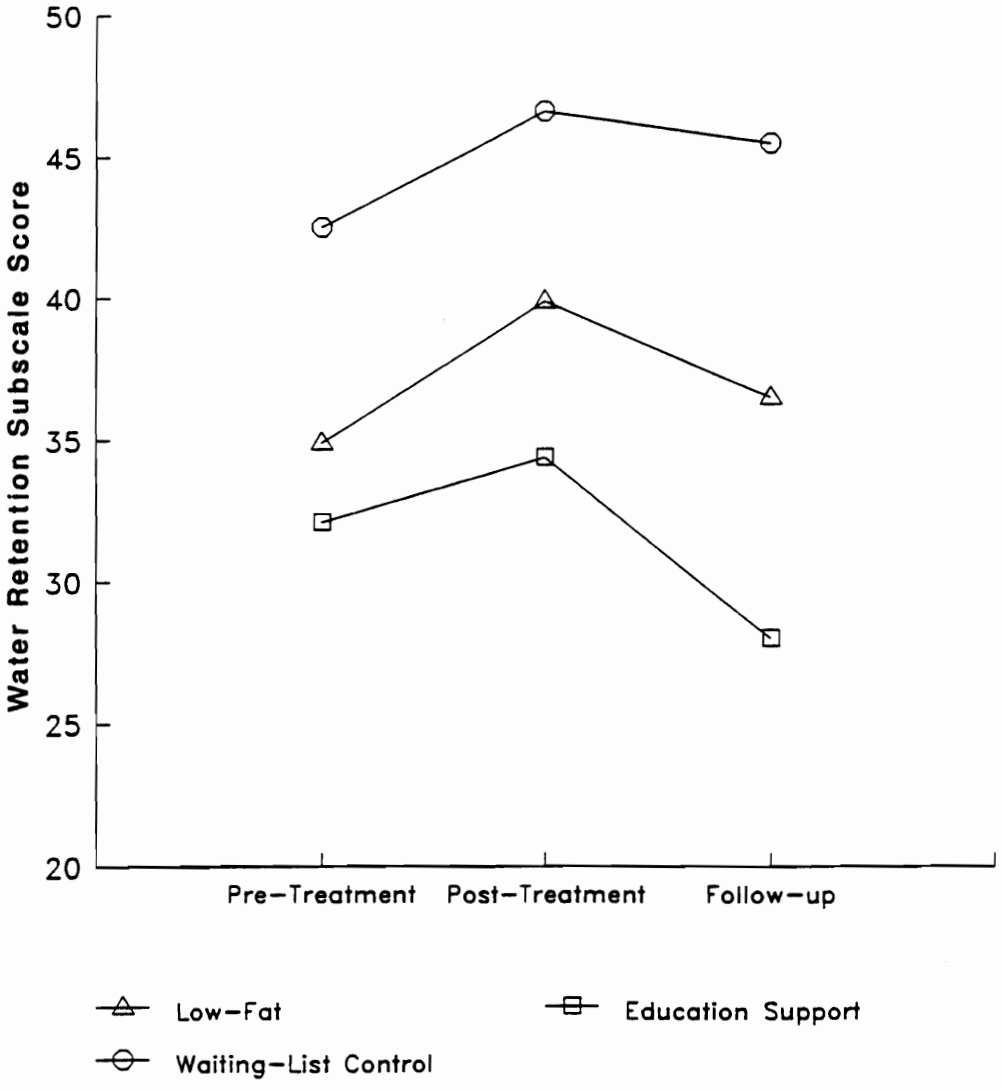

Figure 4. Mean Premenstrual Water Retention Score

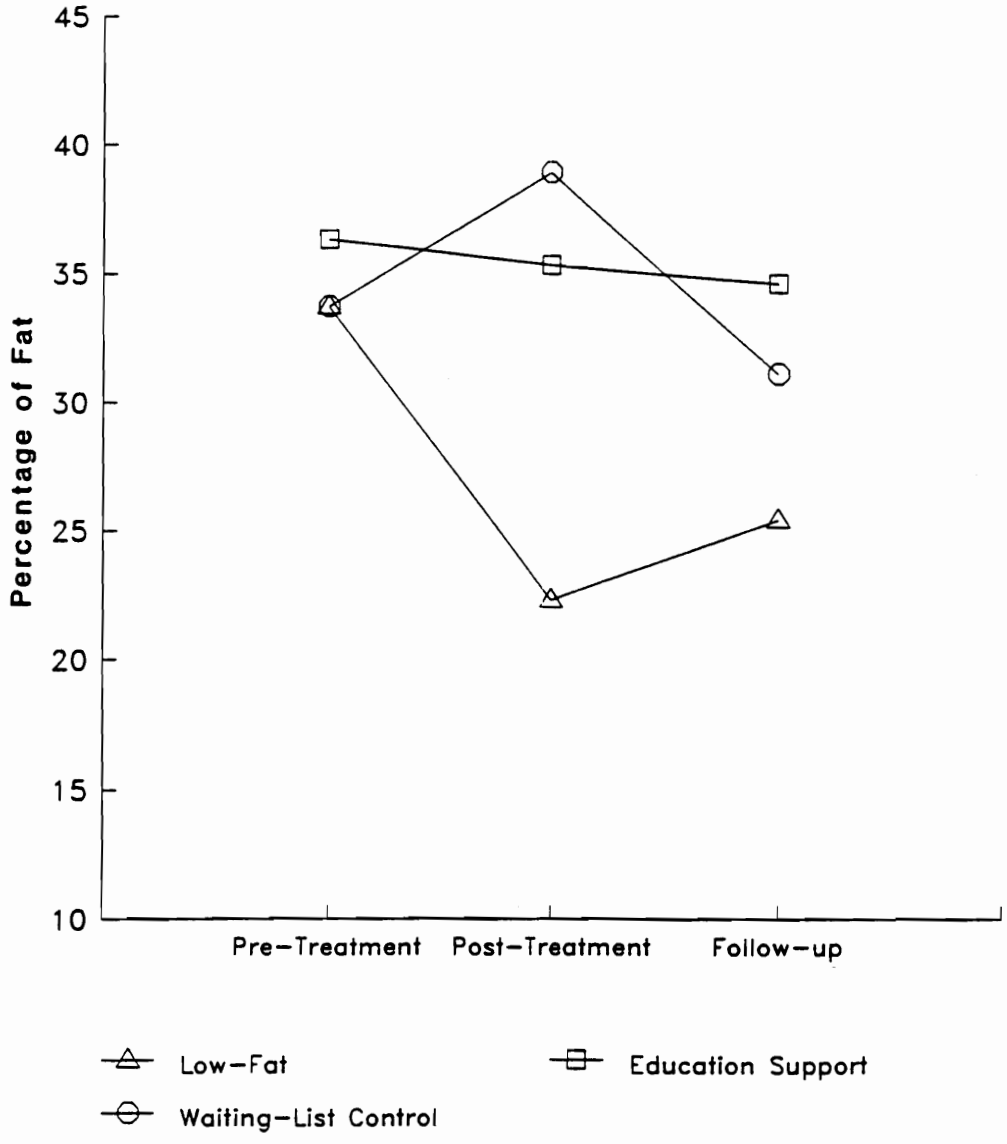

Figure 5. Mean Fat Percentage

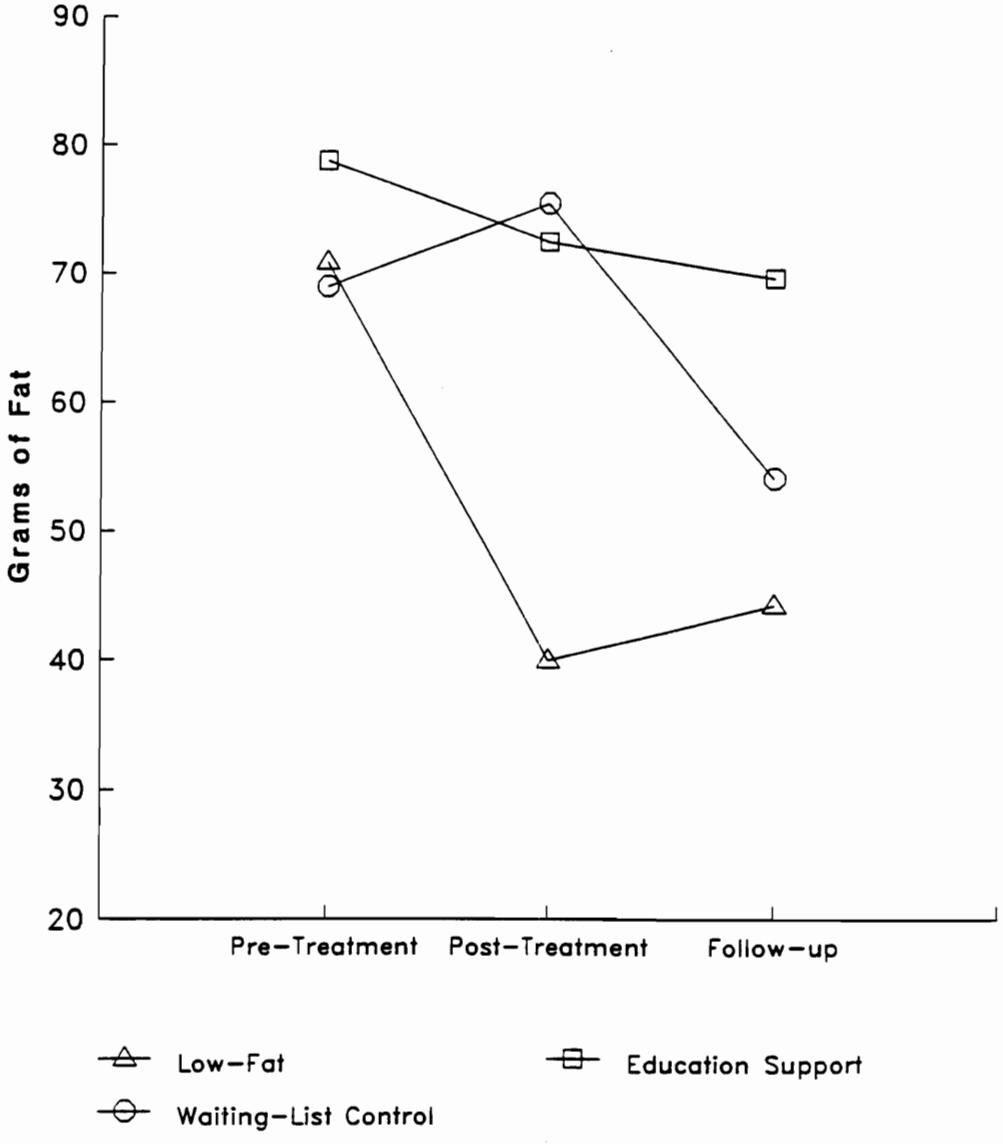

Figure 6. Mean Grams of Fat

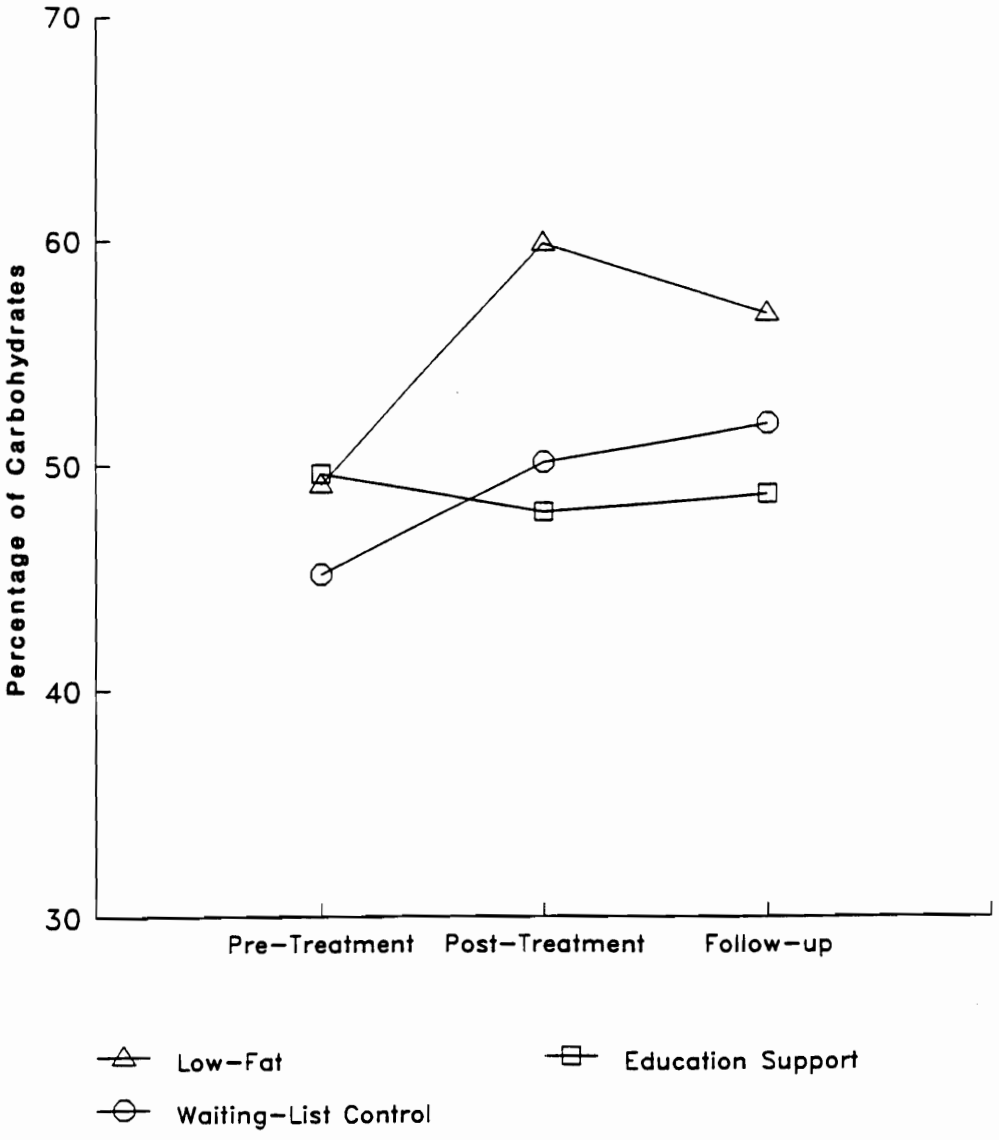

Figure 7. Mean Carbohydrate Percentage

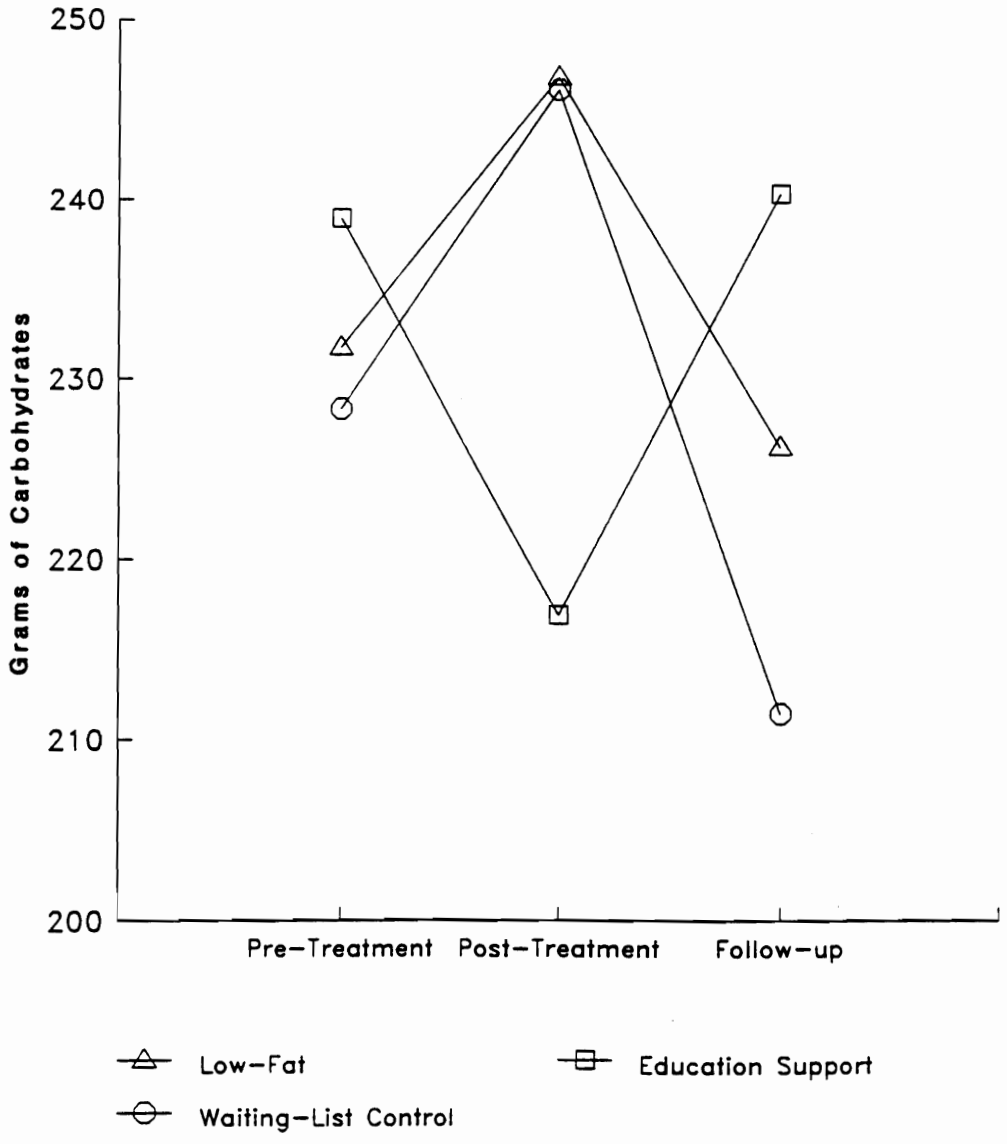

Figure 8. Mean Grams of Carbohydrates

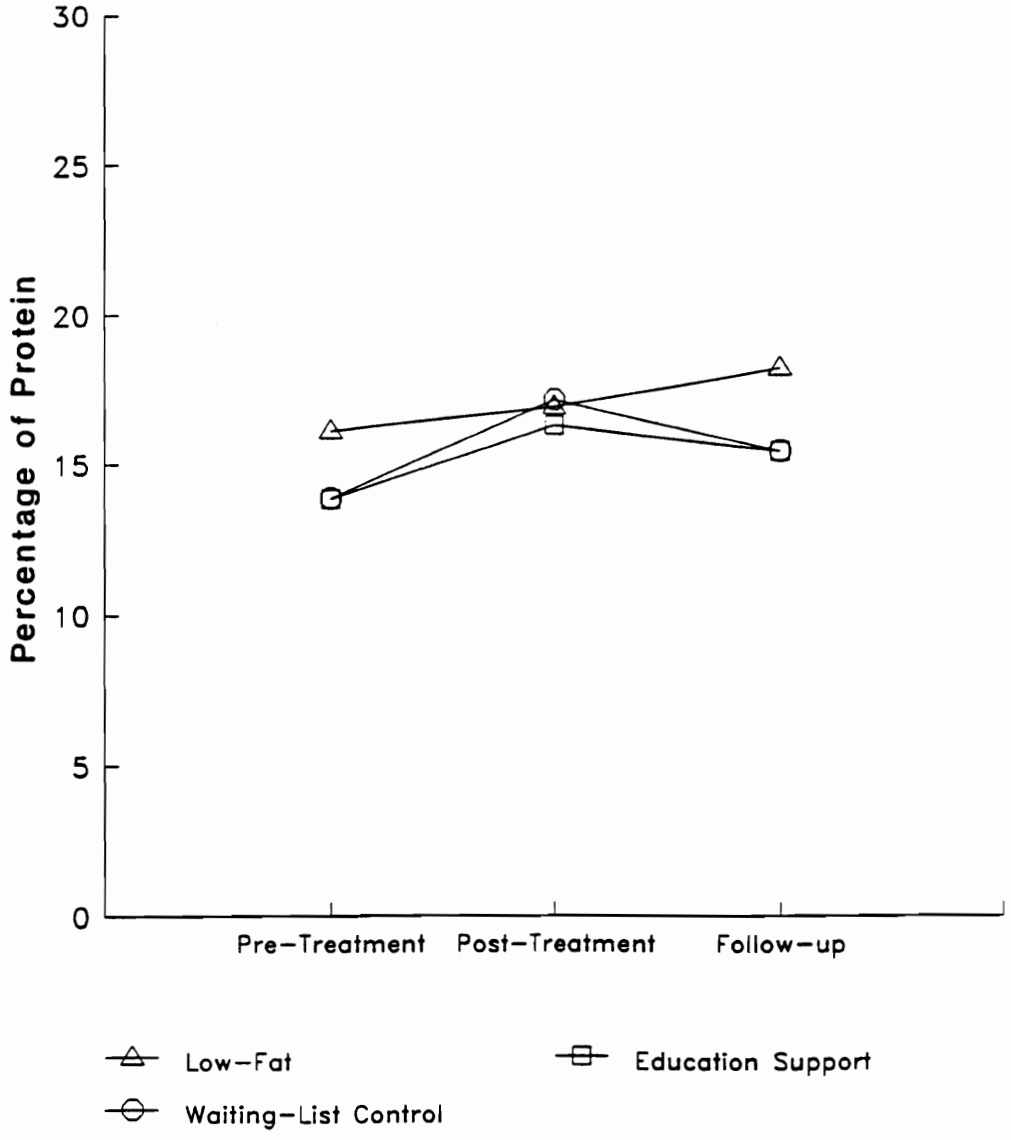

Figure 9. Mean Protein Percentage

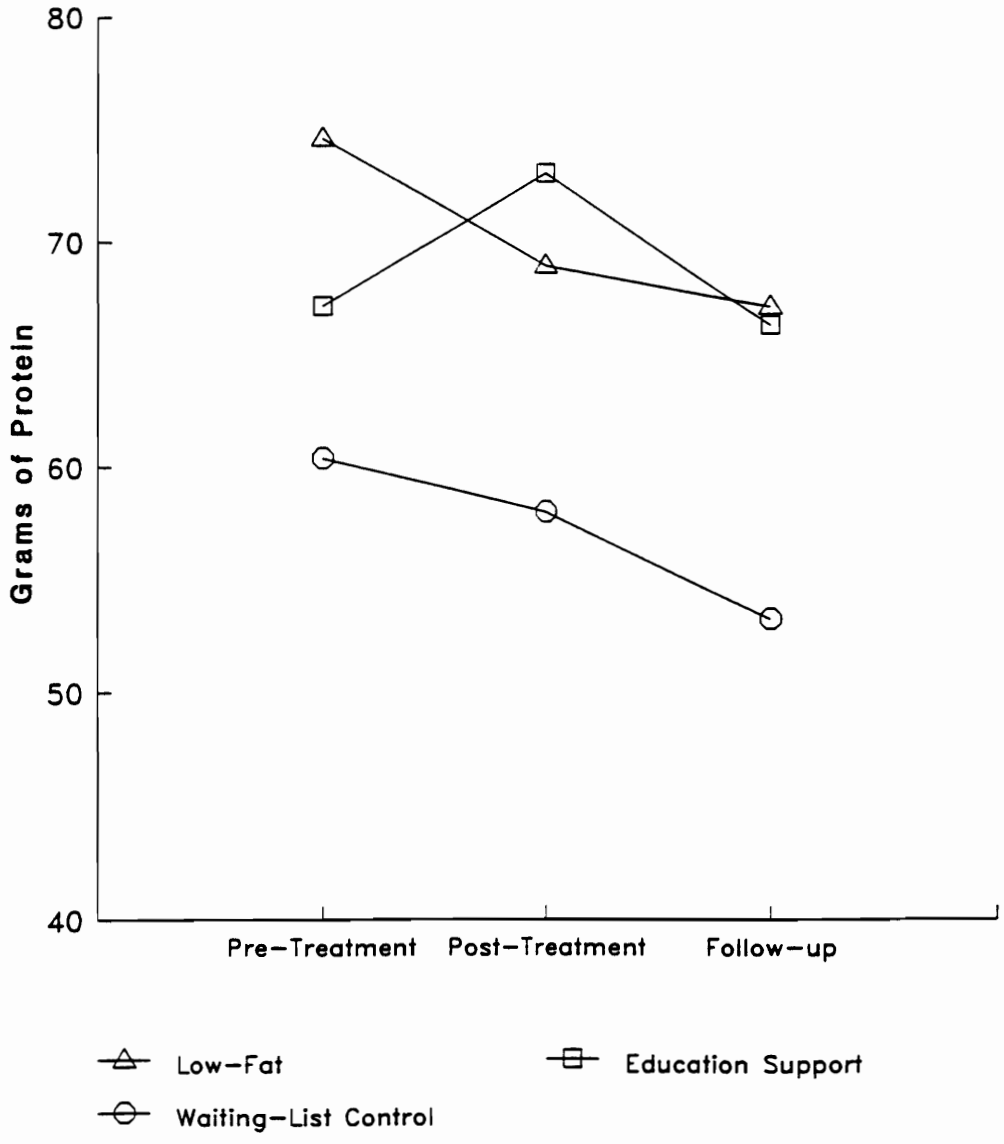

Figure 10. Mean Grams of Protein

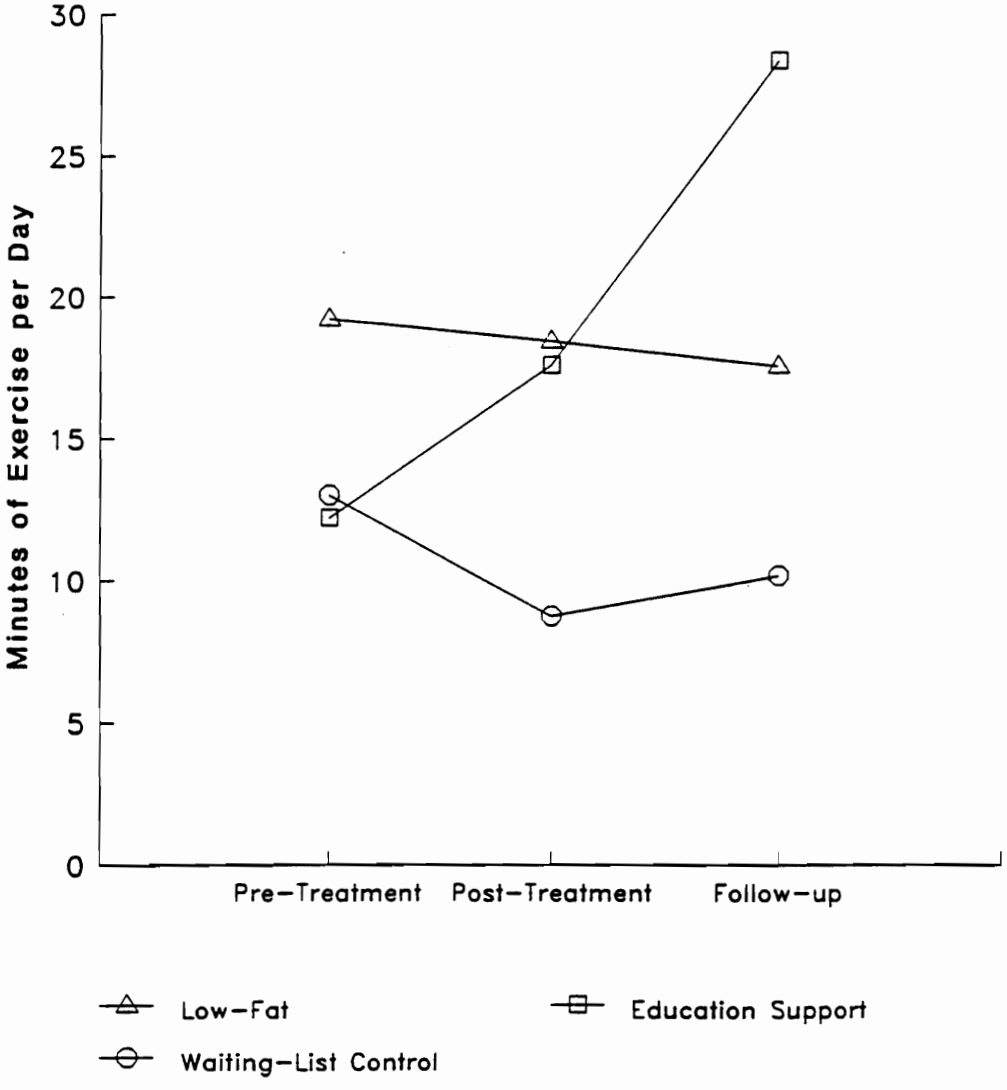

Figure 11. Average Amount of Exercise in Minutes per Day

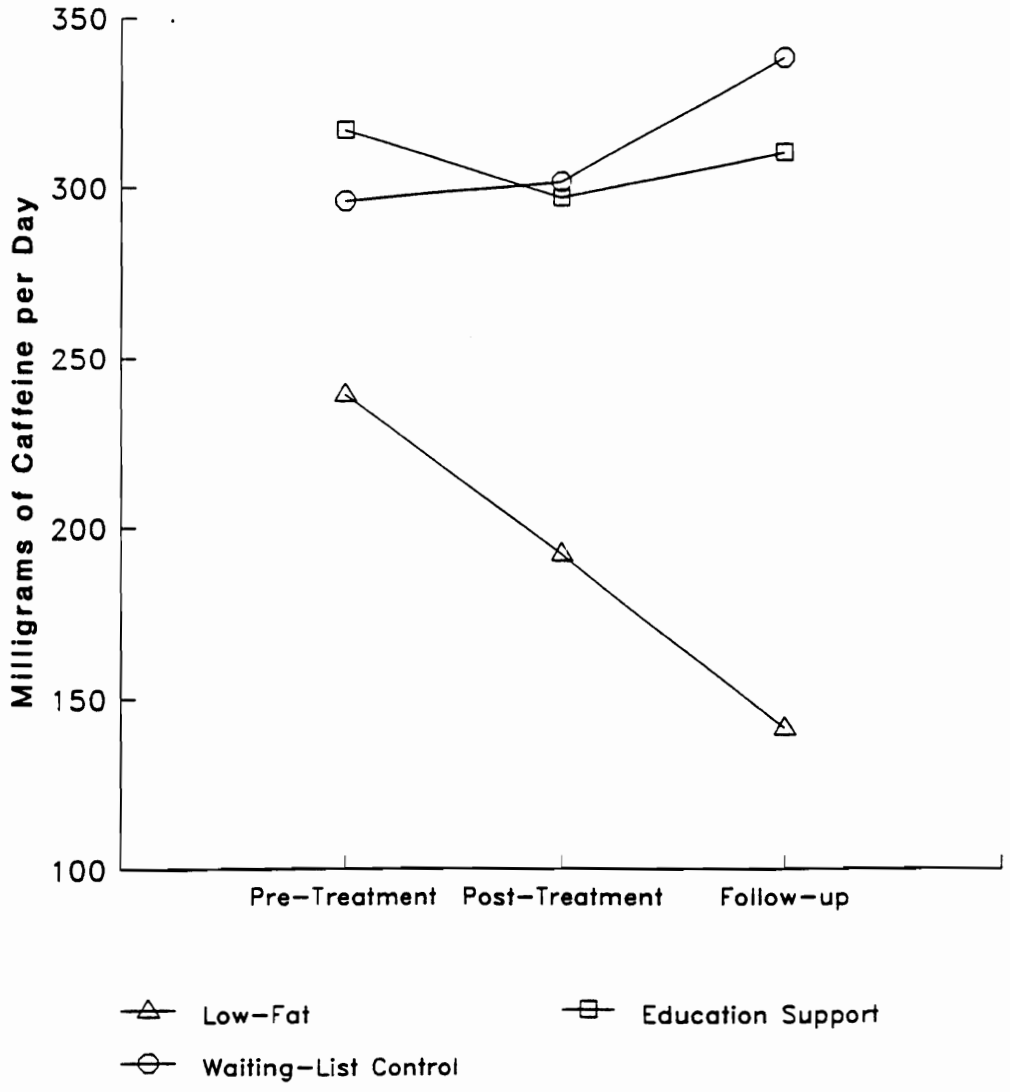

Figure 12. Average Amount of Caffeine in Milligrams per Day

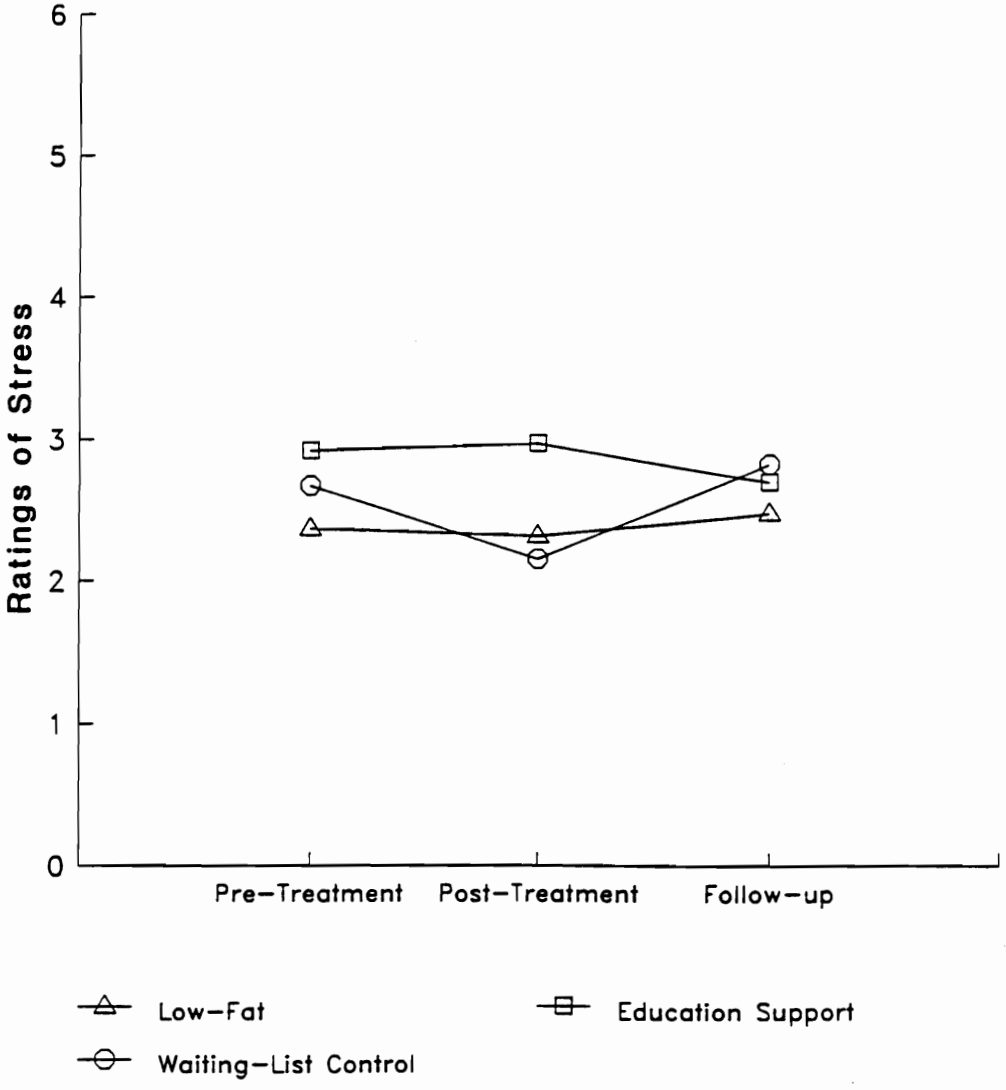

Figure 13. Average Rating of Stress per Day

Appendix A

Diagnostic Criteria for Late Luteal Phase Dysphoric Disorder

DIAGNOSTIC CRITERIA FOR LATE LUTEAL PHASE DYSPHORIC DISORDER

A. In most menstrual cycles during the past year, symptoms in B occurred during the last week of the luteal phase and remitted within a few days after onset of the follicular phase. In menstruating females these phases correspond to the week before, and a few days after, the onset of menses. (In nonmenstruating females who have had a hysterectomy, the timing of luteal and follicular phases may require measurement of circulating reproductive hormones).

B. At least five of the following symptoms have been present for most of the time during each symptomatic late luteal phase, at least one of the symptoms being either (1), (2), (3), or (4):

(1) marked affective lability e.g., feelings of suddenly sad, tearful, irritable, or angry
(2) persistent and marked anger or irritability
(3) marked anxiety, tension, feelings of being "keyed up" or "on edge"
(4) markedly depressed mood, feelings of hopelessness, or self-deprecating thoughts
(5) decreased interest in usual activities, e.g. work, friends hobbies
(6) easy fatigability or marked lack of energy
(7) subjective sense of difficulty in concentrating
(8) marked change in appetite, overeating, or specific food cravings
(9) hypersomnia or insomnia
(10) other physical symptoms such as breast tenderness or swelling, headaches, joint or muscle pain, a sensation of "bloating or weight gain.

C. The disturbance seriously interferes with work or with usual social activities or relationships with others.

D. The disturbance is not merely an exacerbation of the symptoms of another disorder, such as Major Depression, Panic Disorder, Dysthymia, or a Personality Disorder (although it may be superimposed on any of these disorders).

E. Criteria A, B, C, and D, are confirmed by prospective daily self-ratings during at least two symptomatic cycles. (The diagnosis may be made provisionally prior to this confirmation.)

Appendix B

Structured Interview

STRUCTURED INTERVIEW

Demographic Information

1. Age:_____

2. Race: a) Caucasian b) Black c) Hispanic d) Oriental
e) Other_____

3. Marital status: a) Single b) Married c) Divorced

4. Occupation:_____

5. Education:_____

Premenstrual Symptoms

1. What premenstrual symptoms do you experience?_____

_____

_____

(Ask subject if she experiences the following symptoms unless she has already stated that she suffers from that symptom)

Yes	No	
___	___	Suddenly feeling sad, tearful, irritable or angry
___	___	Anger or irritability (persistent or marked)
___	___	Anxiety, tension, feeling "keyed up"
___	___	Depressed, hopeless feelings, self-depreciating thoughts
___	___	Hypersomnia or insomnia
___	___	Breast tenderness or swelling, headaches, joint or muscle pain, sensation of bloating, weight gain
___	___	Change in appetite, overeating, specific food cravings
___	___	Difficulty concentrating
___	___	Lack of energy
___	___	Decreased interest in usual activities

2. During a single menstrual cycle, when do your premenstrual symptoms start (How many before menstruation?)_____
When do they end (e.g. at menstruation, a day after menses)?

_____

3. At what age did you start experiencing premenstrual symptoms?

_____

4. Did symptoms start after a precipitating event or events (e.g. birth of a child, oral contraceptives, stressful event)?
Yes No, If so, what was (were) the event (events)?

_____

_____

5. Does premenstrual tension interfere with your life?
Yes No
If yes, What two "activities" or relationships does it interfere with the most?

_____

6. Using the following scale provide, rate the degree to which your premenstrual symptoms interfere with your life?

_____

0 1 2 3 4 5
not at all very much

a. ____ your career
b. ____ your education
c. ____ your personal life
d. ____ family relations
e. ____ general home-life satisfaction
f. ____ marital satisfaction, if married

7. How long have you experienced severe premenstrual tension or when did premenstrual tension start to interfere with your life?

_____

_____

8. Have there been times when your premenstrual symptoms have been more intense? Yes No If so, when?_____

_____

_____

less intense? Yes No If so, when?_____

_____

9. Have there been cycles when you have been symptoms free? Yes No
If so when?_____

10. If you are feeling discomfort during your premenstrual phase what do you do (e.g. take naps, take medication)?

_____

11. What makes your premenstrual symptoms better?

_____

What makes them worse?_____

_____

12. Do you do things to prevent premenstrual suffering? Yes No
If so, what do you do?_____

13. In general, how often would you do the following four days prior to menses? (thus, answers can range from one to four)

____ take a nap
____ go to bed early
____ leave work or school
____ not go to work or school
____ slow down o become less active
____ forgo activities (sex, outings, etc.)

14. Image that you have 12 full cycles in a year: how often would you do the following, in one year, four days prior to menses? (thus, your answers can range from 0 to 48)

____ go to bed
____ go to bed early
____ leave work or school
____ not go to work or school
____ slow down o become less active
____ forgo activities (sex, outings, etc.)

15. Can others tell if your in your premenstrual phase?
Yes No
If yes who?

Husband Yes No N/A How?_____

Children Yes No N/A How?_____

Co-workers Yes No N/A How?_____

Friends Yes No N/A How?_____

16. Do they express help or concern or give special help?
Yes No
If yes who?

Husband Yes No N/A How?_____

Children Yes No N/A How?_____

Co-workers Yes No N/A How?_____

Friends Yes No N/A How?_____

17. How long is your menstrual cycle (e.g. 28 days)?_____

When will your next period begin?_____

How accurate is this prediction(within 5 days)?_____

Medical Information:

1. Have you ever sought medical treatment for premenstrual tension? Yes No
If yes, what were you informed or how was it treated?

_____

_____

2. Have you ever been diagnosed as having any of the following:

Diagnosis:	Yes	No	Course of Illness and Treatment:
Asthma	___	___	_____
Allergies	___	___	_____
(Name)_____			
Diabetes	___	___	_____
Hypertension	___	___	_____
Heart Disease	___	___	_____
Dysmenorrhea	___	___	_____
Endometriosis	___	___	_____
Pelvic Inflam-itory Disease	___	___	_____
Hypoglycemia	___	___	_____
Yeast Infection	___	___	_____
Bladder Infection	___	___	_____
Breast Cancer	___	___	_____
Other "female" Problem (Name)	_____		_____

3. When was the last time you had a breast examination (to check for breast cancer)?_____

Have you had a mammogram? Yes No
If so, how many times?_____

Have you had a biopsy? Yes No
If so, how many times?_____

4. Have you ever had any serious illnesses? Yes No (explain)

_____

_____

5. Have you ever been operated on? Yes No (explain):

_____

_____

6. How often have you consulted your physician concerning your general health:
In the past 3 months?_____
In the past year?_____
In your lifetime?_____

How often have you seen your gynecologist or general practitioner concerning "female problems" or routine pelvic exams:
In the past 3 months?_____
In the past year?_____
In your lifetime?_____

7. Have you ever used oral contraceptives? Yes No If yes, when did you take them (month/yr to month/yr)?_____

_____

8. Have you ever taken any other medication which contained hormones? Yes No If yes, what was the name of the medication?

_____
When did you take the medication?

_____

9. Are you currently taking any prescribed or non-prescribed medication? Yes No
If yes:
Name of medication Dosage Frequency

_____

_____

10. Have you ever been pregnant? Yes No
Do you have any children? Yes No How many?_____
Have you ever miscarried? Yes No
Have you ever had an abortion? Yes No

11. Do you exercise regularly? Yes No (Explain):

_____

12. Are you on a prescribed diet? Yes No (Explain):

_____

Mental Status

1. Have ever received any psychological or psychiatric treatment for emotional problems? Yes No
If yes, include when, approximate number of sessions, helpfulness, and circumstances under which treatment was sought:_____

_____

2. Have you ever been depressed? Yes No
If yes, are you depressed now? Yes No
Have you ever thought about harming yourself or killing yourself? Yes No

3. Have you ever had a problem with alcohol? Yes No
or with other drugs? Yes No

The following questions are somewhat different than the previous questions:

4. What is today's date?_____
   The day of the week?_____
   Do you remember my name?_____

5. I am going to say some numbers. Listen carefully, then repeat them back to me. (Give digits approximately one per second).
   forwards: (i) 5-8-2 (ii) 6-4-3-9 (iii) 4-2-7-4-1
   backwards: (i) 6-9-2 (ii) 7-2-8-6 (iii) 7-5-8-3-6

6. Who is the president of the United States?_____

7. Who is the governor of Virginia?_____

8. Have you ever had any strange experiences? Yes No (explain):

_____

9. Have you ever heard things other people could not hear or heard things when other people were not around? Yes No (explain):

_____

10. Do you believe you have special powers? Yes No (explain):

_____

11. Have you ever felt or thought people were out to get you Yes No (explain):

_____

_____

Appendix C

Consent Form

Assessment and Treatment of Premenstrual Tension Syndrome

I, _____, freely and voluntarily consent to participation in a research program entitled "Assessment and Treatment of Premenstrual Tension Syndrome" to be conducted by Jane M. Kudlas, M.S. and Richard Winett, PhD. The procedures to be followed have been explained to me and I understand them. They are as follows:

1. I understand that will be interviewed by a graduate student in clinical psychology and be required to complete a number of questionnaires to:
   (a) determine if I fit diagnostic criteria for Late Luteal Phase Dysphoric Disorder and am appropriate for treatment.
   (b) gain information on my medical history and health behaviors. Assessment will take approximately one and one-half hours.

2. If I am appropriate for treatment and desire treatment, I understand that I will be assigned to one of two treatment groups for a period of eight weeks. Treatment will focus on information, group support, and a change in lifestyle. Treatment will be conducted in small groups. I will be assigned to a treatment group one to three menstrual cycle following the initial interview (Approximately 4 to 16 weeks).

3. Prior to treatment, during treatment, and following treatment I will be required to:

   (a) keep a daily diary of my moods, premenstrual symptoms, and various other behaviors (e.g. beverage consumption, sleep, exercise). Daily monitoring will take about five minutes of my time each day.
   (b) record everything I eat and drink three days during the luteal phase of my menstrual cycle and three days during the follicular phase of my menstrual cycle.

4. I understand that I will give consent for my physician to be contacted to request that he give medical information on my medical history and current health. Treatment will not begin unless my physician sends back desired information.

5. I understand that I can withdraw my consent at anytime without penalty.

6. I understand that all information obtained from me will be held as confidential as legally possible, and my records will be stored in a locked cabinet. Furthermore, in any scientific report of this project, there will be no way to identify me.

7. I understand that I may be contacted by phone each week by a research assistant who will give me feedback on my form completion. This research assistant will also keep me informed of further assessment and when treatment will begin.

8. I understand that treatment sessions will be audiotaped.

9. I understand that if I consistently fail to complete daily monitoring measures or do not attend treatment sessions my participation in this study will be ended.

There are no major risks associated with this study other than the possible discomfort involved in answering some of the questions. However, it is anticipated that the overall effects of treatment, if completed, will be beneficial.

_____ _____

Signature Date

_____ _____

Address Phone

_____

If you have any questions about this study, contact any of the following people:

Jane M. Kudlas, M.S., Principle Investigator
(Office 961-6630 and Home 951-8610)
Richard Winett, Ph.D., Professor (961-6275)
Helen Crawford, Ph.D., Chairperson, Human Subjects Committee, Department of Psychology (961-6520)
Charles D. Waring, Chairperson Institutional Review Board (961-5283)

Appendix D

Daily Monitoring Forms

DAILY MONITORING FORMS

Please rate numbers 1-18 on the following scale:

1	2	3	4	5	6
not at all	not too much		somewhat		very much

PART I	Date: / /	Date: / /	Date: / /	Date: / /	Date: / /	Date: / /	Date: / /
1. Crying	1.	1.	1.	1.	1.	1.	1.
2. Loneliness	2.	2.	2.	2.	2.	2.	2.
3. Anxiety	3.	3.	3.	3.	3.	3.	3.
4. Restlessness	4.	4.	4.	4.	4.	4.	4.
5. Irritability	5.	5.	5.	5.	5.	5.	5.
6. Mood Swings	6.	6.	6.	6.	6.	6.	6.
7. Depression	7.	7.	7.	7.	7.	7.	7.
8. Tension	8.	8.	8.	8.	8.	8.	8.
9. Muscle Stiffness	9.	9.	9.	9.	9.	9.	9.
10. Headache	10.	10.	10.	10.	10.	10.	10.
11. Cramps	11.	11.	11.	11.	11.	11.	11.
12. Backache	12.	12.	12.	12.	12.	12.	12.
13. Fatigue	13.	13.	13.	13.	13.	13.	13.
14. General aches/pain	14.	14.	14.	14.	14.	14.	14.
15. Painful Breasts	15.	15.	15.	15.	15.	15.	15.
16. Swelling	16.	16.	16.	16.	16.	16.	16.
17. Weight gain	17.	17.	17.	17.	17.	17.	17.
18. Skin Disorder	18.	18.	18.	18.	18.	18.	18.

PART II (Cont.)	Date: /	Date: /	Date: /	Date: /	Date: /	Date: /	Date: /
How many ounces of Chocolate did you eat?	36.	36.	36.	36.	36.	36.	36.
How may cigarettes did you smoke?	37.	37.	37.	37.	37.	37.	37.
Exercise:							
38. Type	38.	38.	38.	38.	38.	38.	38.
39. Time spent (min)	39.	39.	39.	39.	39.	39.	39.
40. Intensity (Hi, Med, Low)	40.	40.	40.	40.	40.	40.	40.
How many hours of sleep did you get last night?	41.	41.	41.	41.	41.	41.	41.
Prescribed or nonprescribed medication (Include vitamins, motrin, decongestives, birth-control pill, etc.) Write on back if necessary							
42. Name Dosage: Number of pills:	42.	42.	42.	42.	42.	42.	42.
43. Name: Dosage: Number of pills	43.	43.	43.	43.	43.	43.	43.
Time spent in the following activity or with the given person (people)							
44.	44.	44.	44.	44.	44.	44.	44.
45.	45.	45.	45.	45.	45.	45.	45.
Rate the quality of time spent in activity or with person (people) 1=very poor 2=poor 3=OK/poor 4=OK/good 5=good 6=very good							
46.	46.	46.	46.	46.	46.	46.	46.
47.	47.	47.	47.	47.	47.	47.	47.
Rate how stressful your day was based on the given scale: 1=Not 2=fairly 3=sort of 4=moderately 5=stressful 6=very stressful							
48. Stress	48.	48.	48.	48.	48.	48.	48.
Did you eat at a fast food restaurant? yes or no	49.	49.	49.	49.	49.	49.	49.
1-Spotting 2-Slight Flow 3-Moderate Flow 4-Heavy Flow with Clots							
50. Menstrual Flow	50.	50.	50.	50.	50.	50.	50.

PART II (Cont.)	Date: /	Date: /	Date: /	Date: /	Date: /	Date: /	Date: /
How many ounces of Chocolate did you eat?	36.	36.	36.	36.	36.	36.	36.
How may cigarettes did you smoke?	37.	37.	37.	37.	37.	37.	37.
Exercise:							
38. Type	38.	38.	38.	38.	38.	38.	38.
39. Time spent (min)	39.	39.	39.	39.	39.	39.	39.
40. Intensity (Hi, Med, Low)	40.	40.	40.	40.	40.	40.	40.
How many hours of sleep did you get last night?	41.	41.	41.	41.	41.	41.	41.
Prescribed or nonprescribed medication (Include vitamins, motrin, decongestives, birth-control pill, etc.) Write on back if necessary							
42. Name Dosage: Number of pills:	42.	42.	42.	42.	42.	42.	42.
43. Name: Dosage: Number of pills	43.	43.	43.	43.	43.	43.	43.
Time spent in the following activity or with the given person (people)							
44.	44.	44.	44.	44.	44.	44.	44.
45.	45.	45.	45.	45.	45.	45.	45.
Rate the quality of time spent in activity or with person (people) 1=very poor 2=poor 3=OK/poor 4=OK/good 5=good 6=very good							
46.	46.	46.	46.	46.	46.	46.	46.
47.	47.	47.	47.	47.	47.	47.	47.
Rate how stressful your day was based on the given scale: 1=Not 2=fairly 3=sort of 4=moderately 5=stressful 6=very stressful							
48. Stress	48.	48.	48.	48.	48.	48.	48.
Did you eat at a fast food restaurant?							
yes or no	49.	49.	49.	49.	49.	49.	49.
1-Spotting 2-Slight Flow 3-Moderate Flow 4-Heavy Flow with Clots							
50. Menstrual Flow	50.	50.	50.	50.	50.	50.	50.

Appendix E

Food Diary

FOOD DIARY

Day of Week_____

Date_____

Type of Food	Quantity		Brand Name	How Prepared
	Number	Volume		

Appendix F

Directions for Completing Food Diary Forms

DIRECTIONS FOR COMPLETING FOOD DIARY FORMS

1. WRITE DOWN EVERYTHING YOU EAT OR DRINK FROM THE TIME YOU GET UP UNTIL YOU GO TO BED. This includes all meals any time of the day or night, snacks, coffee breaks, soda pop breaks, cocktails, beer nibbling, refrigerator raiding . . .

2. The best way to remember what and how much you are eating or drinking is to write it down in your food record IMMEDIATELY after a meal or a snack.

3. BE EXACT AS TO AMOUNTS, I.E. report in terms of level tablespoons, cups, dimensions of slices of meat, size of fruit, etc.

4. DESCRIBE ACCURATELY THE KIND OF FOOD RECORDED:

   a. State brand name whenever possible
   b. Type of product. For example:
      1). milk: whole, 2%, evaporated, whole, etc.
      2) squash: summer, acorn, etc.
      3) lettuce: romaine, iceberg etc.
      4) bread: whole wheat, white, rye etc.

5. BE SURE TO RECORD EVERYTHING

   a. Vitamin preparations.
   b. "Hidden foods". - sugar on cereal or fruit, butter, oleo, or mayonnaise on bread, vegetables, popcorn, crumb, or cheese toppings etc.
   c. Candy, soda, potato chips, (state kind and amount).

Appendix G

Rating of Treatment Rational Questionnaire

Rating of Treatment Rationale

A technique designed to treat your premenstrual tension was just explained to you. We are interested in obtaining your impressions about the treatment procedure at this time. Please answer each question below as honestly and openly as possible. Circle the number which best reflects your feelings right now.

1. How logical does this type of treatment seem to you for helping women with premenstrual tension.

1	2	3	4	5	6	7	8	9
Not at all Logical								Very Logical

2. How confident are you that this treatment will be successful in treating your premenstrual suffering?

1	2	3	4	5	6	7	8	9
Not at all Confident								Very Confident

3. How confident would you be to recommend this treatment to a friend with premenstrual tension?

1	2	3	4	5	6	7	8	9
Not at all Confident								Very Confident

4. How important do you think it is that we make this treatment available to others who experience premenstrual tension?

1	2	3	4	5	6	7	8	9
Not at all Important								Very Important

5. How successful do you believe this treatment would be in decreasing other problems like general anxiety, depression etc.?

1	2	3	4	5	6	7	8	9
Not at all Successful								Very Successful

Appendix H

Physician Release From

RELEASE OF RECORDS

I, _____, hereby request that _____ furnish the medical records relating to my general health and diagnosis and/or treatment of medical conditions, illnesses or diseases, gynecological problems and other medical problems to:

Jane M. Kudlas, M. S.
Psychological Services Center
Department of Psychology
Virginia Tech

_____
Physicians Name

_____

_____
Physicians Address

_____
Participant's Signature date

_____
Witness's Signature date

Appendix I

Letter to Physician

Re:

The individual whose name is listed above has presented to by research project on the treatment of Premenstrual Tension Syndrome or Late Luteal Phase Dysphoric Disorder. In order to meet certain human subjects protection requirements of our Human Subjects Committee of Research, I am writing to obtain information about this individual. The individual has consented to our contacting you; a copy of a signed release of information form is enclosed.

Your assistance in completing the enclosed "Physician Evaluation Form" and returning it in the enclosed envelope would be appreciated. Treatment will not begin until this information is received; thus your prompt attention will be greatly appreciated.

The information needed concerns the following: (1) Your opinion as to whether any of the treatment procedures would be contraindicated for the individual (2) whether the individual is on a prescribed diet (3) current medication and (4) the date of your last evaluation.

There will be two eight week treatments provided. Your patient will be randomly assigned to one of these groups. The first treatment is designed specifically to modify patient's diet. Subjects in this group will be asked to decrease their fat consumption to 20% of their total caloric intake and to increase their carbohydrate consumption to 65% of their total caloric intake The second treatment group will be given information about PMS. Information will include the definition of PMS, physiology of the menstrual cycle, theories of PMS, overview of treatments, the impact stress on PMS, and strains on relationships and jobs. Subjects in this group will also be given the opportunity to discuss premenstrual symptomatology and problems associated with having PMS.

I hope this information is sufficient. If not, please feel free to contact me at the Psychological Services Center.

Sincerely,

Jane M. Kudlas, M.S.
Graduate Student in
Clinical Psychology

Richard Winett, Ph.D.
Professor of Clinical Psychology

Appendix J

Physician Evaluation Form

Re:_____ Physician _____
Signature

Phone_____

1. Is there any medical information which would indicate that this person should not be permitted to participate in the program outlined in the accompanying letter? No___ Yes___

If yes, please specify._____

_____

_____

2. Is this person on a prescribed diet? No___ Yes___

3. What medications are currently being prescribed to this individual?
(Please List)

_____

_____

4. Date of last physical examination:_____

5. Is there anything else of importance you can tell us about thispatient?_____

_____

_____

_____

Appendix K

Final Questionnaire for the Low-Fat Diet Group

FINAL QUESTIONNAIRE
Low-Fat Group

Do you think your PMS symptoms have (circle one):
Improved, Increased, or Stayed the Same.

On the following scale rate the degree of improvement (with 100 meaning 100% improvement) or degree to which you symptoms have increased (with -100 meaning that your symptoms are 100% worse than before treatment).

-100 -80 -60 -40 -20 0 20 40 60 80 100

What, if anything, did you find helpful about this treatment?

_____

_____

Was there anything outside of treatment that may have decreased your premenstrual suffering?_____(Explain)_____

_____

_____

Was there anything during or outside of treatment that may have increased your premenstrual suffering?_____(Explain)

_____

_____

Have you lost any weight since the beginning of treatment? ___

If so, how much weight have you lost? _____

_____

Did you follow the "PMS" diet after treatment was over? _____

If yes, how may days during the week do you think you met the low-fat diet goal (less than 20% fat and 65% carbohydrate)?

_____

Did you follow the low-fat diet during treatment? ___ If yes, how many days during the week did you meet the low-fat diet goal?_____

On the average, how many days out of the week did you really "cheat" on the "PMS" diet (more than 30% fat)?_____
(more than 40% fat)?_____

Do you think that your diet habits have changed since the beginning of treatment? _____ (Explain)

_____

_____

What was most helpful in getting you to change your dietary habits?

_____

_____

Was there anything external (e. g. husband, children) or internal (feelings that you couldn't change your diet) that inhibited you making the suggested dietary changes (Explain)?

_____

_____

What do you think can be done to improve this treatment?_____

_____

_____

Appendix L

Final Questionnaire for the Education Support Group

FINAL QUESTIONNAIRE
Education Support Group

Do you think your PMS symptoms have (circle one):
Improved, Increased, or Stayed the Same.

On the following scale rate the degree of improvement (with 100 meaning 100% improvement) or degree to which you symptoms have increased (with -100 meaning that your symptoms are 100% worse than before treatment).

-100 -80 -60 -40 -20 0 20 40 60 80 100

What, if anything, did you find helpful about this treatment?

_____

_____

Was there anything outside of treatment that may have decreased

your premenstrual suffering?_____(Explain)_____

_____

_____

Was there anything during or outside of treatment that may have

increased your premenstrual suffering?_____(Explain)_____

_____

_____

How often did your practice relaxation training?_____

_____

Did you ever use Problem Solving Skills?____Was it helpful?

_____

_____

Do you think that you've changed any "exaggerated" or "irrational" thoughts?____(Explain)_____

_____

_____

_____

Did you change your eating habits (explain)_____

_____

_____

Did you change your exercise habits (explain)_____

_____

_____

Rank the components of treatment with 1 being the most important and 7 being the least important:

___ Group support (knowing that there are others out there that suffer from PMS and being able to talk about it).

___ Learning that I don't need to feel guilty for feeling bad or being less productive during my premenstrual.

___ Learning about relaxation techniques.

___ Learning how to problem solve.

___ Learning about how to change my diet (decrease salt, sugar, caffeine, and increasing magnesium, potassium, and zinc).

___ Learning about how exercise can help my PMS symptoms.

What do you think could be done to improve treatment?_____

_____

_____

Appendix M

Configuration of Treatment Conditions

CONFIGURATION OF TREATMENT CONDITIONS

Low-Fat Diet Condition

Group A:
Case #1, #2, #3, #4, #5, & #6

Group B:
Case #7
(plus one individual who was excluded because she changed medication).

Group C:
Case #8 & #10
plus woman who was excluded because she appeared to have cyclical mastopathy
plus a woman from the control condition who had already completed three cycles of monitoring.

Group D:
Case #9
plus two women who did not complete post-treatment assessment
plus a woman who was assigned to the control condition, but did not complete all three cycles of assessment.

Education Support Group

Group E:
Case #10 & #11
plus one woman who was excluded because she changed medication.

Group F:
Case #12, #13, & #14

Group G:
Case #16
plus a woman who was assigned to the control condition but did not complete all three cycles of monitoring

Group H:Case #17 & #18

JANE MICHELE KUDLAS
Maiden Name: Jane Michele Walsh
476-82-9323

MAILING ADDRESS

Route 4 Box 650A
Hollywood, MD 20636
(301) 373-3749

OFFICE ADDRESSES

Charles County Mental Health
P.O. Box 1037
LaPlata, MD 20646

DATE AND PLACE OF BIRTH: May 16, 1963, Minneapolis, Minnesota

EDUCATIONAL BACKGROUND

Degree	Date	University	Area of Emphasis
B.A.	7/85	University of St. Thomas St. Paul, Minnesota	Psychology
M.S.	11/87	Virginia Polytechnic Institute and State University Blacksburg, Virginia	Clinical Psychology
Ph.D.	12/92	Virginia Polytechnic Institute and State University Blacksburg, Virginia	Clinical Psychology

HONOR AND AWARDS

1981-1985 Recipient of an academic scholorship from the Alliss Education Foundation

1981-1985 Member of Delta Epsilon Sigma National Honor Society

1983-1984 President of Psychology Club

1985 Graduated Summa Cum Laude - University of St. Thomas

PROFESSIONAL AFFILIATIONS

American Psychological Association
(Student Affiliate from 1984 to 1990)

Association for the Advancement of Behavioral Therapy

TEACHING EXPERIENCE

Visiting Professor: Taught three courses for the Human Development Department at St. Mary's College of Maryland. Courses included: Life Span Development, Exceptionality, and Social and Emotional Development. Duties included lecturing, creating exams and homework assignments, and grading students. In addition, supervision was provided to a student conducting an independent research project for college credit. [January 1991 - August 1991]

Seminar Lecturer: Taught cognitive-behavioral theory and treatment methods to 3rd year Psychiatry Residents at the Medical University of South Carolina. [November, 1989]

Guest Lecturer: Presented information on Premenstrual Tension Syndrome to interested individuals at Lewis Gale Hospital in Salem, Virginia. [April, 1986]

Laboratory Instructor/Teaching Assistant: Taught Introductory Psychology Lab for the Psychology Department at Virginia Polytechnic Institute and State University. Duties included lecturing on controversial topics in psychology (e.g. intelligence testing with minorities, use of lie detectors in criminal cases), creating test questions, grading students, and tutoring individual students on information from the Introductory Psychology class. [September, 1984 - June, 1985]

CLINICAL EXPERIENCE

Psychologist Associate: Provided supervised psychological services for the School-Based Day Treatment Program. The School Based Day Treatment Program is a governor's initiated project that assesses and treats elementary school children with emotional and/or behavioral problems. Services included individual therapy conducted at the elementary school, family therapy, and teacher consultations.

Charles County Mental Health [November 1990 - present]
LaPlata, MD

Psychologist Associate: Provided supervised psychological services for the Family Counseling Center. Services included individual, family, and marital therapy for adults, children, and adolescents.

Family Counseling Center [October 1990 - present]
LaPlata, MD

Psychology Intern: Provided supervised psychological services to fulfill requirement for doctoral degree in clinical psychology. (1) Crime Victim's Research and Treatment Center: Assessment and treatment of crime victims (child sexual and physical abuse, incest, rape, physical assault, domestic violence, and family members of homicide victims), (2) Wellness Center: Student Counseling Center that provides psychological services, individual and group psychotherapy, to students at the Medical University of South Carolina, (3) Weight Management Center: Use of behavioral change interventions in conjunction with a liquid protein diet in the treatment of over weight individuals.

Medical University of South Carolina
Charleston, South Carolina [September 1989 - August 1990]

Clinical Trainee/Therapist: Provided supervised psychological services including assessment, evaluation, and therapy (individual, marital, and family) to children, adolescents and adults. Observed forensic evaluations.

Mental Health Center of the New River Valley
Radford, Virginia [September 1987 - May 1988]

Academic Counselor: Provided information, guidance, and advice to undergraduate students on course requirements, academic majors, and careers.

Arts and Sciences Advising Center
Virginia Polytechnic Institute and State University
Blacksburg, Virginia [September 1986 - August 1989]

Clinical Trainee/Therapist and Supervisor: Provided supervised psychological services including assessment, evaluation, and therapy (individual, marital, family, and group) to children, adolescents, and adults. (As a fourth year graduate student I supervised the clinical work of 1st and 2nd year graduate students).

Psychological Services Center [September 1985 - August 1989]
Virginia Polytechnic Institute and State University
Blacksburg, VA

RESEARCH

Research Interests: (1) Women's health and mental health issues: Treatment and assessment of Late Luteal Phase Dysphoric Disorder or Premenstrual Tension Syndrome, pregnancy, rape prevention, and assessment and treatment of incest survivors, (2) Prevention and treatment of child sexual abuse, (3) Consumer satisfaction of mental health services.

Research Experience: Involvement in the following research projects: (1) Consumer Satisfaction of the School Based Therapy Project [November 1990], (2) Assisting Physically Injured Crime Victims' in a Health Care Setting [September 1989 - February, 1990], (3) Assessment and Treatment of Tension Headaches [September 1987 - January 1989], (4) Suicide Treatment Project [September, 1988 - January, 1989], (5) Treatment of Irritable Bowel Syndrome [June, 1986 - January, 1987], (6) Correlates of self-esteem [September, 1984 - January 1985]. Participation in these research projects included literature review, development of research idea, data collection, being a therapist for treatment groups, and/or data analysis.

PROFESSIONAL PRESENTATIONS

Buri, J., Walsh, J., & Kirchner, P. (1986). Parental Nuturance and Self-Esteem. Paper presented at the 58th Annual Convention of the Midwestern Psychological Association.

Neff, D., Edwards, M., & Walsh, J. (1987, November). Relaxation Training vs. Cognitive Skills Training in the Treatment of Irritable Bowel Syndrome. Paper presented at the Association for Advancement of Behavior Therapy, 21st Annual Convention, Boston, Massachusetts.

Neff, D., Broyles, S., Edwards, M. Sikkema, K., Crowe, H., Gould, R., Jasie, D., Kudlas, J., Bonner, M., & Keister, L. (1989, April). The Influence of Success Experiences on Chronic Tension Headache Treatment outcome. Paper presented at the 10th Annual Meeting of the Society of Behavioral Medicine, San Francisco, California.

Walsh, J., & Neff, D. (1987, November). The Influence of Information on Premenstrual Expectancy and Control of Premenstrual Symptoms. Paper presented at the Association for the Advancement of Behavior Therapy, 21st Annual Convention, Boston, Massachusetts.

PUBLICATIONS

Buri, J., Kirchner, P., & Walsh, J. (1986). Familial Correlates of Self-Esteem, Journal of Social and Psychology, 127, 583-588.

Neff, D., Broyles, S., Edwards, M., Sikkema, K., Crowe, H., Gould, R., Jasie, D., Kudlas, J., Bonner, M., & Keister, L. (Manuscript in preparation). The influence of success experiences on chronic tension headache treatment outcome.

Master's Thesis: A controlled Study of the Effects of Information on Premenstrual Expectancy and Daily Mood Ratings.

Dissertation: Low-Fat Diet vs. Education-Support in the Treatment of Late Luteal Phase Dysphoric Disorder.

REFERENCES

Richard A. Winett, Ph.S., Professor of Psychology
Director of the Center of Research in Health Behavior
Psychology Department
Virginia Polytechnic Institute and State University
Blacksburg, VA 24061
(703) 231-8747
[Dissertation Chair]

Debra F. Neff, Ph.D., Assistant Professor
1H241 University of Michigan
East Medical Center Drive
Ann Arbor, Michigan, 48109-0050
(313) 936-7052 or (313) 662-9482
[Master's Thesis Chair]

Connie L. Best, Associate Professor
Crime Victim's Research and Treatment Center
Department of Psychiatry and Behavioral Sciences
Medical University of South Carolina
Charleston, SC 29425
(703) 792-2945
[Major Supervisor on Internship]