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THE TREATMENT VALIDITY OF IDENTIFYING AND TREATING DEPRESSION
AND BEHAVIOR CHANGE SYMPTOM CLUSTERS IN WOMEN COMPLAINING
OF THE PREMENSTRUAL SYNDROME

The University of North Carolina at Greensboro

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THE TREATMENT VALIDITY OF IDENTIFYING AND TREATING
DEPRESSION AND BEHAVIOR CHANGE SYMPTOM
CLUSTERS IN WOMEN COMPLAINING OF
THE PREMENSTRUAL SYNDROME

by

Susan Ruth Leonard

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Approved by

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APPROVAL PAGE

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The main goal of the present study was to test a treatment validity hypothesis in relation to premenstrual syndrome (PMS): that selection of specific target behaviors and matching treatments to them would enhance treatment outcome. Thus, this study assessed the treatment validity of identifying depression and behavior change symptoms within the general classification of PMS. This study also addressed the question of whether, on a general measure of PMS, in comparison to a no-treatment control group, it is more effective to use an intervention designed to treat depression or behavior change symptoms of PMS.

Thirty-six women reporting premenstrual symptoms of depression and behavior change were randomly assigned to one of three treatment groups: cognitive therapy for depression, operant intervention for behavior change, and no-treatment control. Treatment occurred between the first and second menses. Control subjects engaged in record keeping only. All subjects completed ratings of depression and behavior change daily, and recorded crying frequency and time resting daily throughout three menstrual cycles. Within one week of each menses onset, subjects also retrospectively completed the Menstrual Symptom

Questionnaire (MSQ), an overall measure of PMS, and the Menstrual Distress Questionnaire (MDQ) which included negative affect and behavioral change symptom clusters.

It was predicted that the MDQ negative affect cluster, daily depression rating, and crying frequency would differentially improve in the depression treatment group; the MDQ behavioral change cluster, daily behavior change rating, and time resting would differentially improve in the behavior change treatment group; and the MSQ would differentially improve for one of the treatment groups. Multivariate and univariate analyses of variance did not support these predictions.

Four of the seven dependent measures showed significant changes over time: the MSQ, both MDQ clusters, and the daily behavior change rating. There were no significant differences between groups, including the control group.

Lack of support for the experimental hypotheses is discussed in light of the confound of potentially ineffective treatments with the treatment validity of identifying symptom clusters. The change over time regardless of intervention is discussed as consistent with placebo or common factor effects within PMS. Directions for future treatment validity and PMS research are suggested.

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CHAPTER I

INTRODUCTION

Three relatively recent developments contributed to the formulation of this experiment. First, fairly recently, behavioral psychology has entered the domain of medicine with growing respect and mutual benefit. Their integration has resulted in the development of a field called behavioral medicine. Second, women have gained prominence within the professions of both psychology and medicine. They have redirected research on women's problems. Female scientist-practitioners in behavioral medicine approach common gynecological complaints from a behavioral perspective, eschewing "anatomy is destiny" and pursuing relief of discomfort (Calhoun & Sturgis, 1984). One such complaint is premenstrual syndrome (PMS). Third, and finally, the application of behavioral approaches to traditionally medical problems brings with it issues inherent in behavioral research. One such issue presently of concern is the utility of behavioral assessment.

The central concern of this dissertation is the utility of behavioral assessment. This concern is more precisely characterized as the value of matching treatment procedures to target behaviors as evaluated specifically through the use of treatment validity methodology. To date, treatment

validity has been examined primarily using the disorder of depression. This dissertation is an attempt to apply the methodology of treatment validity to a different disorder, PMS.

The present chapter reviews and elaborates upon the factors leading to the choice of this disorder. First, the philosophical underpinnings of the field of behavioral medicine are addressed. Second, the specific disorder of premenstrual syndrome (PMS) is reviewed. Previous research pertaining to the nature of PMS precludes the identification of the specific symptoms of PMS addressed in the present study (depression and behavior change). A review of the etiological considerations in PMS and various treatment procedures precedes the proposed management procedures used in the present study (cognitive therapy of depression and operant intervention for behavior control). Third, and central to this dissertation, issues in the evaluation of behavioral assessment are addressed and the particular topic of interest here, treatment validity, is reviewed. Finally, these topics (PMS and treatment validity) are integrated in the statement of purpose.

Behavioral Medicine

Behavioral medicine has been conceptualized as a clearly interdisciplinary field which emphasizes the integration of behavioral and biomedical knowledge (Schwartz

& Weiss, 1978). Behavioral psychology has been credited with providing the means to change behavior that affects health and disease (Agras, 1975). In behavioral medicine, psychological intervention is applied to physiological processes.

It has been demonstrated that psychological processes can exacerbate physical disorders (Blanchard & Ahles, 1979; Melamed & Siegel, 1980). The Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM III) delineates specific conditions under which psychological factors are judged as affecting physical conditions. The diagnostic criteria for the category are the following:

- A. Psychologically meaningful environmental stimuli are temporarily related to the initiation or exacerbation of a physical condition.
- B. The physical condition has either demonstrable organic pathology (e.g., rheumatoid arthritis) or a known pathophysiological process (e.g., migraine headache, vomiting).
- C. The condition is not due to a Somatoform disorder (no organic pathology or process). (DSM III, pp. 303-304).

This focus on an interaction between behavioral and biomedical events suggests that psychological interventions may be applied to normal physiological processes, as well as to pathophysiological processes. The psychological interventions may not directly affect the cause or physical basis of the process. Nonetheless, behavioral interventions

have been effective with a variety of physical disorders; for example, vascular (Kallman & Gilmore, 1981), muscular (Bird, Cataldo, & Parker, 1981), and central nervous system (Mostofsky, 1981). This success suggests that there are psychological effects on physical systems, for probably both diseased and nondiseased processes. In the present study, psychological approaches were used with components of premenstrual syndrome, which may be considered a nondiseased physiological process.

The DSM III criteria suggest an interaction between psychological and physical conditions rather than the dichotomous view promoted by defining a specific set of disorders as psychogenic. Psychosomatic medicine is differentiated from behavioral medicine as it classically has been separate from the rest of medicine. Psychosomatic medicine is presumed to deal exclusively with disorders of psychological origin (psychogenic disorders), clearly differentiated from disorders of organic origin. Schwartz and Weiss (1978) suggest that the integrative focus of behavioral medicine transcends the "...mind/body dualism that plagued the early development of psychosomatic medicine" (p. 250). The focus of psychological interventions on physical systems, the basis of behavioral medicine, clearly has bearing on the mind/body issue.

Mind/body Issue

The integration of the behavioral and biomedical fields relates to the philosophical stances on the mind/body issue. These stances may be classified as monistic or dualistic (Bunge, 1980). The mind/body issue concerns the nature of the psychological (mind) and its relationship to the physical (body). Dualistic positions maintain that both aspects of the mind/body distinction are necessary to provide an adequate explanation of experience. Dualistic positions include psychophysical parallelism, interactionism, and emergentism or epiphenomenalism. Monistic positions maintain that one aspect of the mind/body dyad is sufficient to explain experience. Monistic positions include mental monism and physical reductionism.

The dualistic approaches differ in terms of their interpretation of the relationship between mind and body. Psychophysical parallelism acknowledges the existence of both the psychological and the physical, contending they are separate and synchronous processes which do not interact. Psychophysical parallelism requires the acceptance of the mind separate from the physical world. The unobservable and unmeasurable aspects of this separatist view and its denial of the impact of the physical on the psychological and the psychological on the physical are difficult to incorporate in an empirically based scientific position (Bunge, 1980).

The philosophical position of interactionism attempts to resolve the mind/body issue by contending that mind and body are separate, but interacting. Behavioral medicine demonstrations of psychological interventions effective with physical functioning appear to suggest such a relationship between mind and body. This position, however, seems more closely related to the field of psychosomatic medicine which epitomizes the separation of organic and psychological origins (Schwartz & Weiss, 1978). Interactionism accepts a distinction between the psychological and physical, but suggests an impact of one upon the other. Whether the impact is psychological to physical or physical to psychological or reciprocal, the argument suffers mostly from separatism (Pomerleau, 1979). From a scientific approach, the interaction seems reasonable, because available data are interpretable as suggesting psychological impact on physical processes. The separation of the two, however, seems arbitrary. What is physical versus psychological may be indistinguishable.

An alternate attempt to allow the psychological to be separate, but related to the physical is the position contending that mind is an emergent of the brain, epiphenomenalism. This position suggests that the psychological are emergent brain processes, that is, the psychological are based on physical processes, but are

different from and more than the physical events from which they emerge. According to this view, psychological processes are emergent from physical brain processes in the same sense that water (a liquid) is an emergent of the gases hydrogen and oxygen; the parts are known, but the outcome is not predictable from the parts. Epiphenomenalism is presumed to maintain a scientific approach even as chemistry must deal with emergents (Sperry, 1969). This suggests the whole is greater than the sum of the parts, a stance consistent with current thinking in some disciplines (Bindra, 1970; Sperry, 1970). It seems incompatible with behavioral medicine, however, as it maintains a separatist stance and tends to remove the psychological portion of behavioral medicine from the realms of science. In general, Bunge (1980) suggests the dualistic positions are antithetical to a scientific approach.

The monistic positions seem to overcome the dualism criticism that separation of psychological and physical processes is arbitrary; the two processes may be indistinguishable. Essentially, the monistic positions differ in terms of the process which is necessary to account for experience. Mental monism assumes all that exists is the mind which creates only the impression of the physical world. This is a difficult position to uphold if one desires to maintain a scientific approach; science depends

on sensory data. This stance has been offhandedly discounted as unscientific (Bunge, 1980). This position is, thus, untenable from the perspective of behavioral medicine.

An alternate monistic position is physical reductionism. This position suggests that ultimately all that is called psychological will be reduced to and explained by physical processes. From this perspective, psychological processes are considered a product of physical function, to be explained by biological/physiological processes. This is considered a popular stance among the sciences (Sperry, 1970).

Physical monism is the commonly accepted philosophical stance in behavioral medicine. It is a monistic position which views the mental and physical as indistinguishable and, thus, is consistent with the use of psychological interventions with physical and health-related disorders. While explanations may ultimately be derived from biomedical research, the presence of organic etiology does not establish the ineffectiveness of psychologically based interventions. Since we know that psychological interventions can be effective with physical processes (Blanchard & Ahles, 1979), it seems reasonable to pursue the demonstration and explanation of these effects within behavioral medicine.

This study addressed the specific health-related problem of premenstrual syndrome (PMS). Psychologically based interventions were used with PMS symptoms. This approach is consistent with the preferred philosophical stance within behavioral medicine, physical monism.

Premenstrual Syndrome

Dysmenorrhea is a noun of Greek origin defined simply as "painful menstruation." A number of authors have found this simplistic view to be insufficient to encompass the full range of menstrually related distress reported by women. This study is primarily concerned with one subset of menstrually related distress: premenstrual syndrome (PMS). PMS is a topic which has received much attention in the popular press recently (Paige, 1973). There is a dearth, however, of controlled empirical work on the topic. Most of the work that has been done is presented in relation to other subsets of dysmenorrhea.

Premenstrual syndrome is ill-defined, and its existence as a distinct disorder is questioned. There are three issues central to making a distinction between menstrual and premenstrual problems: (a) the basis upon which the distinction is made, (b) the exclusiveness of the respective syndromes in different women, and (c) whether there is a functional distinction between syndromes. There are basically three criteria upon which the distinction has been

made: the cause of the symptoms, the nature of the symptoms, and the temporal occurrence of the symptoms. Various researchers contend that women can have only one or both of the syndromes. The functional nature of the distinction between the syndromes in prescribing treatment has also been disputed. The definitions and distinguishing issues related to perimenstrual symptoms are discussed.

Behrman and Gosling (1966) delineated four categories of dysmenorrhea: primary, secondary, membranous, and premenstrual tension syndrome. Primary and secondary dysmenorrhea are distinguished essentially by their relationship to identifiable pelvic disease; no organic pathology is associated with primary dysmenorrhea, and secondary dysmenorrhea is caused by another disease state. Membranous dysmenorrhea is rare and gains its name from the appearance of the material passed from the uterus during the painful menses. Their premenstrual tension syndrome is viewed as a complex of cyclic and recurrent physical and psychological symptoms.

More recently, Chesney and Tasto (1975a) also distinguished between secondary and primary dysmenorrhea. Additionally, they upheld Dalton's (1969) distinction of spasmodic and congestive forms of primary dysmenorrhea. Spasmodic dysmenorrhea refers to abdominal cramping pains beginning on the first day of menstruation. Congestive

dysmenorrhea is considered to be a variation of the premenstrual syndrome and refers to dull, aching pains and affective changes beginning prior to the onset of menstruation.

Dalton (1977) proposed a faulty progesterone feedback pathway as the physiological process underlying "congestive" dysmenorrhea. Thus, she contended that insufficient progesterone is the cause of premenstrual (congestive) problems and that excess progesterone is the cause of spasmodic dysmenorrhea. Chesney and Tasto (1975a) upheld the congestive-spasmodic distinction through the development of a questionnaire (the Menstrual Symptom Questionnaire or MSQ) which clearly distinguished among 48 women whose menstrual complaints parallel Dalton's dichotomous descriptions. The MSQ consists of 24 statements which describe symptoms associated with the menstrual period. Twelve of the items are associated with congestive dysmenorrhea, and 12 with spasmodic dysmenorrhea. Each item is rated regarding the subject's typical menstrual period on a 5-point scale ranging from never to always. In addition, the 25th item offers the subject a choice between two descriptions of menstrual discomfort: one is characteristic of congestive and the other of spasmodic complaints. The subject chooses the description which most closely matches her experience. Chesney and Tasto (1975a) found no subjects

with both kinds of symptoms. They also found a differential effect of treatment on the two groups (Chesney & Tasto, 1975b).

The distinction between congestive and spasmodic dysmenorrhea has been questioned, however. Cox (1977) used Chesney and Tasto's (1975a) MSQ with 14 women and did not find dichotomous groups. He found a fairly even distribution of respondents throughout the range of scores on the MSQ. He identified scores of 48-68 as congestive, scores of 69-80 as mixed, and scores of 81 and above as spasmodic. Chesney and Tasto (1975a & b) reported no subjects in the middle range, and Dalton's (1977) theory does not account for mixed symptomatology.

Webster, Martin, Uchalik, and Gannon (1979) also disputed Dalton's theory of hormonal imbalance. They factor analyzed MSQ's from 275 women and found seven (not two) factors which accounted for 62% of the variance: premenstrual negative affect, menstrual pain (cramping), premenstrual pain, menstrual back pain, water retention, and two factors without clear labels. This breakdown does not support the congestive-spasmodic distinction proposed by Dalton. Stephenson, Denney, and Aberger (1983) also factor analyzed the MSQ using the responses of 423 women and cross-validated the analysis on the responses of 294 other women. They used a severity scale rather than the frequency scale

used with the MSQ. Their analyses also revealed seven factors which accounted for 64% of the variance. These factors did not parallel the congestive-spasmodic distinction considered inherent in the MSQ. Their clearly labelled factors were essentially the same as those of Webster and colleagues (1979). Their factors included menstrual pain, premenstrual negative affect, water retention, premenstrual pain, and menstrual back pain. The congestive-spasmodic dichotomy appears to have little support.

Instead of distinguishing between premenstrual and menstrual disorders on the basis of symptoms, an alternative way is temporally. Moos (1969) in developing the Menstrual Distress Questionnaire (MDQ), used a time of menstrual flow-premenstrual week distinction. Each of the 839 women in Moos (1969) study rated their experience of 47 symptoms for the menstrual, premenstrual, and intermenstrual time periods, and for their worst period. Their responses were factor analyzed separately for each time period, and the same eight clusters emerged for all the analyses (Moos, 1969). The clusters were pain, concentration, behavioral change, autonomic reactions, water retention, negative affect, arousal, and control. The control cluster is made up of symptoms derived from menopausal complaints, not menstrual complaints. These clusters are quite similar to

those derived from the MSQ by factor analyses (Stephenson et al., 1983; Webster et al., 1979). This temporally based distinction, menstrual versus premenstrual, also seems to be related to symptoms. Women complain of discomfort accompanying their menstrual flow and of discomfort preceding their menstrual flow; they tend to characterize the nature of this discomfort differently (Dalton, 1969; Moos, 1969). Moos (1969) suggests his pain scale reflects symptoms usually associated with dysmenorrhea (menstrual complaints) and the negative affect scale reflects symptoms "almost definitional" of premenstrual complaints (p. 392). There appears to be an inconsistency in Moos' (1969) report of similar clusters in menstrual and premenstrual phases and the association of different clusters with each time period. It seems that while the same clusters were demonstrated in each phase, they had different levels of severity. On the average the pain cluster received greater endorsement during the menstrual phase of the cycle and the negative affect cluster received greater endorsement during the premenstrual phase. Although menstrual and premenstrual discomfort may be characterized differently, women do not seem to suffer exclusively from one or the other; a variety of symptoms at a variety of times can occur in the same woman (Moos, 1969).

The issue of primary concern in this study is premenstrual syndrome (PMS). The temporal distinction and

the symptoms most commonly associated with discomfort preceding the menstrual flow seem to provide an operational means of distinguishing the premenstrual syndrome from strictly menstrual complaints.

Importance of PMS

It is difficult to estimate the prevalence of PMS (Calhoun & Sturgis, 1984). Reports of menstrual problem frequency often combine menstrual and premenstrual complaints. In addition, premenstrual complaints are often categorized as symptomatic of other disorders or traits. The estimates which have been made, however, range from 50% to 95% of the adult female population (Dalton, 1979; Hoes, 1980; Reid & Yen, 1981; Widholm, 1979; Wood, Larsen, & Williams, 1979). Varying degrees of severity are used in computing frequency of the syndrome which may account for the discrepancy in estimates. Estimates of severe or debilitating premenstrual problems are usually restricted to 5-10% of the adult female population (Dalton, 1979; Moos, 1969). Unlike dysmenorrhea, PMS tends to increase with age (Gough, 1975; Hoes, 1980; Moos, 1968) and does not appear to respond to a popular treatment for dysmenorrhea: childbirth (Dalton, 1969; Kistner, 1971).

Estimates of the impact of perceived premenstrual changes seem to be primarily based upon self-report. Women believe that they are less effective premenstrually (Brooks

et al., 1977; Parlee, 1973; Sommer, 1973). Studies using actual measures of overt behavior, however, do not support the conclusion that performance is impaired in cognitive and academic tasks (Berstein, 1977; Dor-Shav, 1976), speech fluency and behavior changes (Silverman & Zimmer, 1976; Zimmerman & Parlee, 1973), and in other cognitive and perceptual-motor behavior (Sommer, 1973). The detrimental impact of PMS, as it is perceived by women, however, should not be discounted. The belief that the premenstruum is responsible for lessened effectiveness and competence may lead women to be more anxious than usual which may impair their performance, or it may lead women to avoid particular activities in which they believe their performance will be impaired (Brooks et al., 1977). Thus, while the impact on performance may not have been demonstrated, the negative impact of the belief that performance is impaired is still possible. Woman hours, although not clearly documented, are surely lost due to premenstrual discomfort.

Nature of Premenstrual Symptoms

An important question related to the examination of PMS is: Are the nature and timing of premenstrual symptoms unique to the syndrome? The timing of symptoms has been used in the definition of the syndrome. This, however, is insufficient to demonstrate that the nature of these symptoms is exclusively premenstrual or cyclic.

Demonstrations that these particular symptoms occur consistently during the premenstruum are necessary to establish the periodic nature of the symptoms.

First, research suggesting various explanations for the apparent periodic nature of these symptoms is discussed. Factors other than actual menstrual changes have been used to account for the apparent periodic nature of premenstrual symptoms including cultural stereotypes, expectation, social reinforcement, and methodological problems. Second, the literature dealing with the consistency of premenstrual symptoms is reviewed. Essentially the same symptom complex is consistently reported. Reports examining the nature of premenstrual symptoms also are discussed. Premenstrual symptoms have been compared to other responses to stress, characteristic response patterns, and major psychological disorders.

Periodic Nature of Symptoms. Some researchers suggest the symptoms reported as premenstrual do not occur exclusively during the premenstruum. Similar symptomatology in response to periods of stress other than the premenstruum has been reported (Halbreich & Kas, 1977; Moos, 1969). Many studies using the MDQ report correlations between the menstrual, premenstrual, and intermenstrual time periods on the MDQ (Moos, 1969). These reports suggest the symptoms may not be unique to PMS.

Premenstrual symptoms have also been viewed as characteristic responses from the perspective of personality trait theory. Women with certain personalities are seen as more likely to show characteristic symptoms premenstrually, as well as at other times. Studies have been done which seek psychological patterns or personality characteristics to account for premenstrual complaints. The relationship between the severity of menstrual symptoms and personality traits has been examined with conflicting results. Gough (1975) reported menstrual distress was related to the California Personality Inventory measure of femininity; others have found no such relationship with similar measures of personality (Berry & McGuire, 1972; Slade & Jenner, 1980). A relationship between high neuroticism on the Eysenck Personality Inventory and perimenstrual suffering has been found (Slade & Jenner, 1980), and greater menstrual symptom complaints have been reported among women considered to be neurotic (Coppen & Kessel, 1963). Others, however, have found no relationship between personality inventory measures of neuroticism and menstrual symptomatology (Awaritefe, Awaritefe, & Ebie, 1980). Higher state anxiety levels, however, among women instructed to imagine that they were in the perimenstrual time period have been reported (Awaritefe et al., 1980).

Attempts have also been made to relate premenstrual symptoms to other types of disorders. In general, premenstrual negative affect and major psychological disorders are considered to be different (Birtchnell & Floyd, 1975; Blechman & Galland, 1983; Diamond, Rubinstein, Dunner, & Fieve, 1976). Many researchers, however, feel that premenstrual affective changes are related to more serious disorders. These affective changes have been characterized as related to affective disorders or depressive syndromes (Kashiwagi, McClure, & Wetzel, 1976; Wetzel, Reich, McClure, & Wald, 1975) and as mild forms of an affective disorder (Endicott, Halbreich, Schacht, & Nee, 1981). More commonly, the menstrual cycle is seen as exacerbating an already existing disorder (Haskett et al., 1980; Zola, Meyerson, Rezinoff, Thornton, & Concool, 1979). The view of premenstrual symptoms as characteristic responses or as related to major psychological disorders suggests the symptomatology is not unique to the premenstruum. The views also imply continuity between premenstrual affective symptoms and other affective changes.

Researchers have also reported no cyclic pattern of symptoms (Golub & Harrington, 1981). Males and females completed symptom surveys: no differences between females in various phases or between males and any of the females were found. Koeske and Koeske (1975) have implied that

other factors might be responsible for the report of cyclic symptoms. There seems to be a tendency to attribute any behavioral changes occurring near the time of menstruation to PMS or menstrually related changes.

Factors other than actual menstrual changes have been used to account for the apparent periodic nature of premenstrual symptoms. Cultural stereotypes, expectation, social reinforcement, and methodological problems have been suggested (Brooks, Ruble, & Clark, 1977; Gannon, 1981; Woods et al., 1982). Researchers have suggested effects of cultural stereotypes on menstrual symptom ratings (Paige, 1973). Koeske and Koeske (1975) suggested that negative mood, including depression, is likely to be attributed to menstruation. Parlee (1974) had both men and women rate symptoms of menstruation. Subjects were instructed to complete the MDQ as they believed most women would complete it for the menstrual, premenstrual, and intermenstrual time periods. The similarity of their responses suggests pervasive cultural beliefs about menstrual symptom experience. In addition, using deception regarding cycle phase resulted in distinctly different symptom estimates. EEG measures were presented as evidence of a subject's actual menstrual cycle phase. In fact, all subjects were actually in the same phase of the menstrual cycle, six to seven days before menstruation. All reported that they

believed the EEG measure manipulation. Women who were led to believe that they were premenstrual reported increased incidence of physical symptoms such as water retention as compared to women who were led to believe they were not premenstrual (Ruble, 1977). Similarly, when the purpose of the study (examining menstrual changes) was disguised, no difference in mood state was reported by women whose retrospective reports of the premenstruum included increased negative affect (Vila & Beech, 1980). These studies support the prevalent influence of expectation in the experience of PMS. Women may actually be reporting culturally expected stereotypes rather than actual premenstrual symptoms.

The behavior of others may also influence women's perception of and response to the premenstruum. The influence of others may be indirect through expectation of performance changes or direct through social reinforcement or consequence of "sick" behavior. If a woman is excused from activities and from expectations of mood control and "rational" behavior by those around her during the perimenstrual time, she may be less likely to participate in activities, or to display controlled mood or "rational" behavior. If she is reinforced for "sick" behavior, she is likely to display it. Examination of the impact of environmental factors on menstrually related behavior has been recommended (Devany & Leonard, 1979), but rarely

directly investigated. Environmental variables should not be minimized, however, as they are consistently demonstrated to be controlling factors in a variety of physiological disorders and processes (e.g., Melamed & Siegel, 1980). Treatment of menstrual distress has utilized environmental factors in at least one reported case: Mullen (1968) had a client's spouse ignore her pain-related behavior to help decrease it. Further research is needed into the impact of environmental variables on PMS.

Methodological problems have also been noted in menstrual problem research (Gannon, 1981; Parlee, 1973). Large discrepancies between retrospective ratings and daily ratings have been demonstrated (Parlee, 1974; Rouse, 1977; Woods et al., 1982). These discrepancies suggest the effect of recall bias on premenstrual symptom reports. This bias may reflect a tendency among women to forget premenstrually related symptoms (Gannon, 1981) or to report them consistently with cultural stereotypes (Ruble, 1977). Sampson and Prescott (1981) recommend the use of daily ratings, in a review of PMS assessment and response to treatment, in order to demonstrate cyclicity of symptom occurrence. The assessments of personality traits and menstruation are consistently retrospective and have been criticised for possible confounding due to the similarity of instruments (Gannon, 1981). Measures of neuroticism and

measures of menstrual discomfort share specific items. Correlations between these measures may reflect these similar items, not actual relationships between neuroticism and PMS. Causal interpretations of correlational data are also common (Parlee, 1973), suggesting experimenter bias and misleading conclusions.

Consistency of Symptom Clusters. PMS has been viewed as an ill-defined set of symptoms (Russell, 1972). Moos (1968) suggested that conflicting data exist regarding menstrual symptomatology. Despite the many differences among identified menstrual symptoms which may be attributed to individual (Moos, 1968) and methodological differences (Parlee, 1973), a review of the literature dealing specifically with premenstrual complaints suggests there is a certain amount of consistency in the symptom clusters associated with the complaint of PMS. There is no consensus regarding the essential symptoms of PMS, but there is a substantial amount of repetition of specific symptoms (Haskett, Steiner, Osmun, & Carroll, 1980).

Behrman and Gosling (1966) identified premenstrual weight gain as diagnostic for PMS. Based upon later work, it appears to be neither necessary nor sufficient for the diagnosis, but seems to be fairly consistent (Chesney & Tasto, 1975a; Moos, 1968; 1969; Stephenson et al., 1983). Weight gain is frequently subsumed within a factor

independently identified as water retention (Moos, 1969; Stephenson et al., 1983; Webster et al., 1979). This factor or symptom category also includes breast tenderness and swelling, symptoms which are also fairly consistently reported (Chesney & Tasto, 1975a; Moos, 1968; 1969; Russell, 1972). Although these physical changes are not unique to the premenstruum, they appear characteristic of that time period.

In addition to these physical symptoms which have been consistently associated with PMS, similar psychological symptoms have also been independently identified. Symptoms of negative affect including mood swings, tension, depression, and irritability are common to the Chesney and Tasto questionnaire (1975a) and the Moos questionnaire (1969). These same mood changes are reported by others attempting to delineate PMS (Golub, 1976; Haskett et al., 1980; Kutner & Brown, 1972; Schuckit, Daly, Herrman, & Hineman, 1975).

Depression is among the most common affective symptoms rated premenstrually on the MDQ (Woods, Most, & Dery, 1982). It has been estimated that one out of ten women experiences premenstrual depression (Kessel & Coppen, 1963). Depression is also among the most frequently cited premenstrual symptoms in gynecology textbooks (e.g., Behrman & Gosling, 1966; Kistner, 1971; Novak, Jones, & Jones, 1975). Although

depression is not exclusive to the premenstruum, it appears characteristic of that phase of the menstrual cycle.

In addition to the physical and affective symptoms reported during the premenstruum, behavior change complaints are also common. Symptoms of fatigue or lethargy and bed rest are common to the Moos questionnaire (1969) and to the Chesney and Tasto questionnaire (1975a). Such changes in behavior or activity are not unique to the premenstruum. They have been reported during both the premenstrual and menstrual time periods. The Moos questionnaire (1969) is rated for both premenstrual and menstrual experience of symptoms and the Chesney and Tasto questionnaire (1975a) relates to all perimenstrual symptoms. In addition, specifically menstrually related changes in activity level or resting behavior have been reported (Amodei, 1983). Although behavior change is not exclusively symptomatic of the premenstruum, reports of it seem characteristic of that time period.

PMS appears to consist of a complex assortment of symptoms. Prior to embarking on an examination of the treatment validity of matching treatment procedures to specific symptoms clusters (the present study), identification of orthogonal symptom clusters was deemed necessary. In order to identify orthogonal symptom clusters subsumed by the label PMS, and amenable to psychological intervention, a pilot study was conducted.

Identification of Clusters (Pilot Study)

In order to identify specific symptom clusters for use in an examination of the treatment validity of matching specific treatments to specific symptoms, the following pilot study was conducted. A number of methodological issues in perimenstrual research were addressed in the design of this pilot study.

Woods, Most, and Dery (1982) compared daily blind ratings of health with a Menstrual Distress Questionnaire (MDQ, Moos, 1969) that had been completed retrospectively. They found major differences between prospective and retrospective ratings of negative affect, pain, and water retention. Rouse (1978) found the same clusters to be rated differently prospectively and retrospectively in another comparison of daily and retrospective ratings of menstrually related symptomatology. She used the retrospective and "at present" forms of the MDQ, and her subjects were aware of the focus on menstrual distress prospectively as well as retrospectively. Neither of these studies factor analyzed in order to identify symptom clusters; they used the symptoms clusters originally identified by Moos (1969). These clusters of symptoms derived from the MDQ appear to be fairly consistent across analyses (Stephenson, Denney, & Aberger, 1983; Webster, Martin, Uchalik, & Gannon, 1979). Despite the apparent consistency of these clusters, it seems

necessary to derive symptom clusters empirically from a blind daily report of symptoms. "Blind" is used here to indicate that the subjects are not told that the study deals specifically with menstrual symptoms. The clusters have historically been derived from retrospective assessments of menstrually related symptoms. Such assessments have been demonstrated to be inconsistent with daily blind ratings which help to control the fairly clear impact of social expectation on the experience of perimenstrual complaints (Ruble, 1977; Vila & Beech, 1980). In order to derive empirically symptom clusters from blind daily ratings, the following three pilot study analyses were conducted.

Subjects completed a Self-Assessment Questionnaire (Appendix A-1) daily for 40 days without the explicit knowledge that the purpose of the study was to collect information regarding their menstrual cycles. Of the 107 Introductory Psychology students who signed up for the study, only four did not receive credit as they did not complete the study. Of the 103 subjects who received credit, the data from only 97 could be analyzed as six subjects recorded no menses within the 40 days of self assessment. A factor analysis using a varimax rotation resulted in 13 factors with eigenvalues greater than one. Although a perfect replication of Moos' original factors was not obtained in the first pilot study factor analysis,

similar factors were found. The similar or overlapping factors include Moos' factors: control, negative affect, behavioral change, arousal, and water retention (Appendix A-2). The Moos factors are identified in the right hand column of Appendix A-2.

The factors identified by this pilot study analysis of all menstruating subjects were not used to identify clusters to be targeted in the treatment validity study, the focus of this disseration project, in part, because of other aspects of the sample. Of the 97 subjects reporting menses during the 40 days of self-assessment, 24 were using oral contraceptives, a possible confounding variable, and 15 rated themselves as aware of the purpose of the study all along. Awareness was rated on a 7-point Likert-type scale; 1 = not at all aware of the experimenter's interest in menstrually related problems and 7 = very much aware. Subjects who rated themselves above 4 on the scale were not considered "blind." Four was an arbitrarily selected cut off (see Appendix A-3).

Based upon this sample information, only 58 subjects were considered "pure," that is, not on oral contraceptives and unaware of the experimental focus on menstrually related complaints. A second factor analysis on just the 58 "pure" subjects using a varimax rotation also resulted in 13 factors with eigenvalues greater than one. Again, although

a perfect replication of Moos' factors was not obtained, similar factors were revealed. These similar or overlapping factors include items on Moos' control, negative affect, arousal, water retention, and behavioral change factors (Appendix A-4). The Moos factors are identified in the right-hand column of Appendix A-4.

The clusters derived from the analyses of all menstruating subjects (Appendix A-2) and the subset of those subjects who were not taking oral contraceptives and not aware of the experimental focus on menstrually related issues (Appendix A-4), while not identical, did reveal substantial similarity. The analyses also reveal clusters similar to those reported by Moos. The similar clusters include: control items, arousal items, water retention items, negative affect items, and behavioral change items.

The factors derived from the second pilot study analysis of the "pure" subjects also were not used to determine the symptom clusters to be targeted in the treatment validity study, the focus of this dissertation project, in part, because of the goal of the pilot study. The goal of the pilot study was to confirm through replication of Moos' factor analysis two prominent factors amenable to psychological intervention in a population complaining of PMS. The daily self assessment data were reanalyzed using only the data from the 33 subjects (out of

the 58 "pure" subjects) who reported that they suffered from premenstrual syndrome or premenstrual problems on the debriefing questionnaire (Appendix A-3). A varimax rotation was used and again resulted in 13 factors with eigenvalues greater than one.

The factors derived from the final factor analysis of the responses of the subjects reporting PMS were similar to those revealed by the first two pilot study analyses. It is clear that while the variance accounted for differed, the items tend to cluster in a consistent manner particularly in relation to the factors derived by Moos. The three pilot study analyses revealed clusters similar to the Moos clusters. The similar clusters include: control items, arousal items, water retention items, negative affect items, and behavioral change items.

The factors identified by the final pilot study analysis of subjects complaining of PMS were not directly used to identify clusters to be targeted in the treatment validity study, the focus of this dissertation project, because of the small sample size. The number of subjects did not exceed the number of items factored by a ratio of 4 to 1 as considered necessary by Cattell (1952). Although Moos' factors were not prospectively derived from a "blind" population sample, his factor analysis did meet the criteria of a 4:1 ratio. Moos used 839 subjects and the

questionnaire includes 47 items. The pilot study factors do provide an indication and verification of which factors derived by Moos could serve as the basis of symptom cluster identification for a treatment validity study.

Although a perfect replication of Moos' original eight factors was not obtained in the pilot study factor analyses, similar factors and factors which share a sizable proportion of the same items were found. The similar or overlapping factors specifically with the final analysis of the subjects reporting PMS include Moos' factors: control, arousal, water retention, negative affect, and behavioral change (Appendix A-5). The Moos factors are identified in the right hand column of Appendix A-5. The first three similar factors seem inappropriate to serve as target symptom clusters in a treatment validity study of psychological interventions. The control factor seems inappropriate as those items were originally included to account for general complaining, the arousal factor seems inappropriate as the items indicate positive affective events which do not require treatment, and the water retention factor seems inappropriate for a psychological intervention. In addition, arousal and water retention are not among the first two to three factors identified in the first factor analysis of all menstruating subjects (Appendix A-2) or in the final factor analysis of the PMS subjects in the pilot study (Appendix A-

5); the first two to three factors account for most of the variability in factor analysis (e.g., Conte & Plutchick, 1981). The other two similar factors are the second and third factor in the final pilot study analysis of subjects reporting PMS. They are similar to Moos' factors negative affect and behavioral change. A portion of the items which make up Moos' factor, negative affect, loaded on the second pilot study factor, depression. The shared items include 3. crying, 38. mood swings, 40. depression, and 45. tension. These overlapping items appear amenable to a standard psychological intervention for depression. The last similar factor, Moos' behavioral change factor, overlaps with the third factor in the PMS subjects' pilot study. The shared items include 4. lowered work or school performance, 8. take naps; stay in bed, and 15. stay at home. This behavioral change factor appears amenable to a standard psychological intervention designed to alter activity level.

Based upon the results of the pilot study and their interface with the factors originally derived by Moos, it appears reasonable to target Moos' factors, negative affect and behavioral change, in an evaluation of matching treatments to targeted symptom clusters. These factors are amenable to psychological intervention, and they have support from factor analyses of prospective, "blind" ratings of premenstrual symptoms.

Etiology and Treatment

Physiological Intervention. A number of theories of the physiological etiology of the affective and behavioral symptoms of PMS have been proposed. No conclusive evidence implicating a particular physiological malfunction in individuals' subjective experience of premenstrual distress is available, however (Steiner & Carroll, 1977). These theories are briefly reviewed. There is no necessary link between etiology and treatment from a monistic philosophical stance. Physically based treatment procedures, however, have been associated with physiological explanations of etiology. These physically based interventions are inconsistently effective or have undesirable side effects or both. These treatment procedures and demonstrations of their effectiveness are also discussed.

At present a surgical procedure eliminating the presumed hormonal source of the physiological process is technically possible. Fluctuations in estrogen and progesterone levels would be terminated by bilateral oophorectomy, that is, removal of the ovaries. Even this invasive procedure has not been determined to be consistently effective (Novak et al., 1975). Even if it were, the side effects such as the complications of major surgery, infertility, and the systemic impact of estrogen loss may be deemed undesirable.

Oral contraceptives have been suggested as a means of controlling PMS as anovulatory cycles are frequently considered to have fewer and less severe accompanying symptoms (Clare, 1979; Novak et al., 1975). The side effects, again, may be undesirable, e.g., infertility, increased risk of cervical cancer, pulmonary embolism, cerebral thrombosis, thrombophlebitis, and neuro-ocular lesions. Morris and Udry (1972), however, found no difference in perimenstrual symptom complaints between users and nonusers of oral contraceptives in a controlled double blind study. Others have also found a continuation of perimenstrual symptoms with the use of oral contraceptives (Cullberg, 1972; Herzberg, Draper, Johnson, & Nicol, 1971). Smith (1975) concluded there are four possible, nonexclusive results with the use of oral contraceptives: (a) cessation of perimenstrual symptoms, (b) decreased severity of perimenstrual symptoms, (c) side effects of the oral contraceptive, and (d) no change. Cullberg (1972) reported that a small number of women experience exacerbations of their symptoms from oral contraceptives. Oral contraceptives are not a highly reliable method of treating perimenstrual discomfort. In addition, premenstrual depression and dysphoria have been reported in anovulatory cycles (Adamopoulos, Loraine, Lunn, Coppen, & Daly, 1972), further disputing the role of ovulation in premenstrual distress.

Dalton (1969; 1977) has been a great proponent of hormone imbalance theories. She advocates a progesterone deficiency theory and has promoted the use of supplemental progesterone in the treatment of PMS. There are, however, undesirable side effects, for example, the systemic effect of progesterone, inconvenience of injections and suppositories, unpleasantness, and expense (Clare, 1979). In addition, few controlled trials have been performed examining the effect of progesterone on menstrual symptoms. Those that have been done do not support the uncontrolled reports of effectiveness. Smith (1975) reported little difference in plasma progesterone levels and no improvement with progesterone injections in women with premenstrual depression. Taylor (1979) demonstrated no differences in absolute or relative progesterone levels between symptomatic and asymptomatic women. Sampson (1979; 1980) using double blind procedures demonstrated no difference between progesterone and placebo on a variety of premenstrual symptoms.

Various dietary changes have been suggested for control of PMS. High protein and low carbohydrate diets have been suggested by Dalton (1977) to control mood swings believed to be caused by altered sugar metabolism during the premenstruum. No controlled studies have been done (Calhoun & Sturgis, 1984). Various vitamin deficiencies have been

implicated, most noticeably pyridoxine (vitamin B6). Functional pyridoxine deficiency has been linked to depression associated with oral contraceptive use and to premenstrual depressed affect (Winston, 1973). The effectiveness of pyridoxine in alleviating premenstrual depression has not been confirmed in controlled studies (Stokes & Mendels, 1972). Dietary restriction and supplements may be undesirable for some women.

Electrolytes and water retention have been implicated in PMS by Janowsky, Berens, and Davis (1973). They report that activation of the renin-angiotensin-aldosterone system causes premenstrual depression. They further suggest that premenstrual negative affect is caused by the impact of angiotensin on the nervous system. The activity of the renin-angiotensin-aldosterone system does not occur in anovulatory cycles (Steiner & Carroll, 1977); thus, the causal nature of angiotensin is unlikely since premenstrual symptoms may occur during anovulatory cycles (Adamopoulos et al., 1972). If the negative symptoms of PMS are viewed as a response to water retention and its associated discomfort, then diuretics become a reasonable symptomatic treatment. Salt restriction has also been suggested, but not experimentally assessed. These interventions also may have undesirable side effects, e.g., dehydration, sodium-potassium imbalance, and possible teratogenic effects. The

use of diuretics is clinically reported to be idiosyncratically effective (Smith, 1975). In addition, no conclusive demonstration of a relationship between severity of premenstrual symptoms and weight gain or water retention is available (Bruce & Russell, 1962; Golub, Menduke, & Conley, 1965; Reeves, Garvin, & McElin, 1971; Russell, 1972).

Levels of prolactin (a pituitary hormone) have also been implicated in PMS (Carroll & Steiner, 1978). Serum prolactin have been found to be higher in women complaining of PMS (Halbreich, Ben-David, Assael, & Bornstein, 1976). Symptomatic women had higher prolactin levels in all phases of the cycle and proportionately greater increases at the time of the premenstruum. The reported effectiveness of bromocriptine (a prolactin secretion suppressor) in decreasing premenstrual symptoms provides much of the support for the prolactin hypothesis (Benedek-Jaszmann & Hearn-Sturtevant, 1976; Carroll & Steiner, 1978). Others, however, have demonstrated only partial effectiveness, that is, inconsistent effects across symptoms, and no more relief of symptoms than by use of a placebo (Andersen, Larsen, Sttenstrup, Svendstrup, & Nielson, 1977; Elsner, Buster, Schindler, Nessim, & Abraham, 1980). O'Brien and Symonds (1982) reported no consistent changes in serum prolactin level across the menstrual cycle, no difference in level in

symptomatic and asymptomatic women, and no correlation between mood changes and prolactin level. In addition, it has been found that prolactin secretion may be influenced by a variety of other factors including diet (Hill & Wynder, 1976), stress, sleep (Frantz, 1978), and vitamin B6 (Foukas, 1973; McIntosh, 1976). The impact of these other substances and situations on prolactin levels is considered indirect evidence of the prolactin hypothesis (Carroll & Steiner, 1978). Any effects of these other factors on PMS is believed to be mediated by prolactin. The inconsistency of the data, however, suggests prolactin is not a primary mediator of PMS (Blechman & Galland, 1983).

Uterine prostaglandins, in general, have been implicated in the etiology of dysmenorrhea, not PMS (Denney & Gerrard, 1981; Fraser, 1980; Gonzalez, 1980). Blechman and Galland (1983) propose a model which includes the role of excessive premenstrual pain sensitivity and uterine prostaglandins in PMS. They propose a testable dual behavioral-biological mechanism for the acquisition and maintenance of PMS. Central to their proposal is the coexistence of excessive uterine prostaglandin production and acute pain sensitivity in PMS sufferers. They proposed that pain sensitivity is centrally mediated with uterine prostaglandins or is acquired through interoceptive conditioning. They attribute acquisition of premenstrual

symptoms to amplification of routine physiological and psychological experiences, and maintenance of premenstrual symptoms to generalized anticipatory anxiety about the menstrual pain experience. This proposal provides an interesting attempt at incorporating current physiological hypotheses regarding PMS with psychological hypotheses regarding PMS. Much of this proposal lacks empirical support; and, unfortunately, while there is some evidence that heightened pain sensitivity is characteristic of dysmenorrheic women (Haman, 1944), there is also evidence which refutes this position. Aberger, Denney, and Hutchings (1983) report no greater pain sensitivity among dysmenorrheic women grouped using the Menstrual Symptom Questionnaire into spasmodic, congestive, mixed, and nondysmenorrheic groups. They did, however, support a previous finding that women seem to have higher pain thresholds during the premenstruum (Tedford, Warren, Flynn, 1977). There appears to be a problem in Blechman and Galland's proposal with hypothesized pain sensitivity as well as some confusion regarding the role of uterine prostaglandins in PMS versus dysmenorrhea.

Decreasing the production of uterine prostaglandins involves the use of pharmacological treatment which may have a number of side effects. Many of these side effects are the same as premenstrual symptoms, including mood

disturbances, edema, and gastrointestinal distress (Larkin, Van Order, Poulson, & Scott, 1979). All the more reason women with premenstrual symptoms might choose not to try these prostaglandin inhibiting drugs.

Psychological Intervention. Many of the interventions derived from theories of organic etiology are effective with some women who complain of PMS. None of the presently available interventions has been demonstrated to be consistently effective. Some women with PMS cannot or choose not to try these treatments because of side effects. Psychological interventions provide an alternative, and they do not necessarily imply the absence of organic etiology. At this time, a clear pathophysiological basis for PMS is unknown. It is, however, known that psychological factors can influence physical conditions. While PMS does not strictly meet the DSM III criteria for psychological factors affecting physical conditions, a physiological, if not a pathophysiological, process appears to underlie the complaints of PMS. The presence or absence of organic etiology, or knowledge thereof, provides proof of neither the effectiveness nor ineffectiveness of psychologically based interventions. Effectiveness is an empirical question.

The dearth of behavioral studies of PMS has been attributed to the vagueness of the complaint (Calhoun &

Sturgis, 1984). The generality of the syndrome seems to make it a challenging issue from a behavioral perspective since PMS consists of a cluster of symptoms which occur frequently in the population, but are not consistent across individuals. One goal of behavioral studies would be to identify the variables controlling the PMS responses. Alteration of cultural expectation and stereotype is an option which has not yet been attempted (Calhoun & Sturgis, 1984). Modification of the environmental consequences for the sick role has also been recommended (Devany & Leonard, 1979).

Another goal of behavioral studies might be to decrease symptomatic responses. Intervention through control of symptomatic responses is a reasonable intervention even if there is a physiological basis for the premenstrual mood and behavior changes. Interventions at the physiological level may be too invasive and have undesirable side effects. Distress, from cultural expectation or physiological process, and response to it, can be lessened through gaining a sense of control over the situation or pain. A sense of control has been reported to decrease subjective discomfort (Melzack & Wall, 1982). Pain management procedures are effectively being presented as self-control coping strategies (Denney, 1980; Turk & Genest, 1979). Quillen and Denney (1982) report effective management of perimenstrual

pain and discomfort using a self-control procedure similar to anxiety management training. Interventions which offer a means of controlling premenstrual symptoms (a response to what is culturally described as an uncontrollable physiological event) seem to have a high probability of success.

Using techniques tailored for specific symptoms has been suggested (Clare, 1979). Chesney and Tasto (1975b) found a differential response to desensitization treatment between congestive and spasmodic dysmenorrheic women. They used hierarchies specifically directed at spasmodic symptoms. Spasmodic subjects decreased their experience of dysmenorrheic symptoms; congestive women did not. Dusen (1976) replicated this differential effect with a desensitization procedure, but not with a cognitive restructuring procedure. Cox and Meyer (1978) did not replicate this differential effect with desensitization, nor did Rosenthal (1978). Rosenthal (1978), however, used hierarchies which were demonstrably different for congestive and spasmodic subjects. Although the desensitization procedure was not differentially effective, the hierarchies used appeared to address different dysmenorrheic symptoms. These different hierarchies functioned as different treatments which were equally effective with different dysmenorrheic symptoms. The importance of tailoring

treatments is suggested by both the differential impact of hierarchies addressing spasmodic symptoms (Chesney & Tasto, 1975b) and hierarchies addressing different symptoms for congestive and spasmodic women (Rosenthal, 1978).

The present study utilized psychological, noninvasive interventions. The approach taken through the use of these treatments is one of symptomatic management of PMS. The expectation was that subjects would gain skills with which they could control the adverse symptoms they reported that they experienced during the premenstruum. The interventions used were modified versions of the Beck (1979) cognitive therapy of depression and the Fordyce (1976) operant treatment program for behavior control. These interventions were designed specifically for use with affective and behavioral changes commonly associated with the premenstruum. The treatments have been previously demonstrated to be effective with nonmenstrually related depression and behavior changes, the target symptoms of this study. The cognitive therapy (depression treatment) involved identifying and disputing negative thoughts. Negative thoughts were considered to be characteristic of the experience of premenstrual affective symptoms, particularly depression. The operant intervention (behavior change treatment) involved planning contingent rewards for completion of scheduled activities. Avoidance of scheduled

or required activities were considered to be characteristic of premenstrual behavior changes. Differential impact of the two treatments was expected. The cognitive therapy of depression was expected to decrease affective symptoms, and the operant intervention for behavior changes was expected to decrease behavior change premenstrual symptoms.

Proposed Management

Symptomatic treatment for PMS was used in this study for three reasons. First, etiologically based treatments have been shown to have undesirable side effects and inconsistent effectiveness. The physiological treatment of PMS is based upon theories of etiology which have only sparse support in the literature. These treatments purport to treat the cause of a syndrome which has no clearly established physical basis. In addition, these treatments have undesirable nontherapeutic side effects. The second reason a symptomatic treatment was used is that symptomatic treatments have been shown in the past to be effective with other disorders. Symptomatic treatment has demonstrable positive effects, even in medicine. Aspirin, although not expected to alter the course of a bacterial infection, is frequently used to control the symptom of fever. In behavioral medicine, a popular example of symptomatic treatment is the use of temperature biofeedback for migraine headaches (Melamed & Siegel, 1980). Migraine sufferers are

trained to increase the temperature of an area of their periphery, usually the hands, through the use of thermal biofeedback techniques. The ability to increase hand temperature has resulted in decreases in migraines. Third, and finally, there is little evidence to suggest adverse effects of symptomatic treatment. The most commonly postulated negative effect of symptomatic treatment is symptom substitution. The fear of symptom substitution requires the belief that there are underlying causes, physiological or psychological, of premenstrual symptoms which must be behaviorally expressed. The critical question, when faced with the possibility of symptom substitution, is whether it is likely to occur. The evidence derived through reviews of empirical findings is against symptom substitution (Bandura, 1969; Lazarus, 1971; Sloane, Staples, Cristol, Yorkston, & Whipple, 1975). Symptomatic treatment is reasonable when dealing with a syndrome of unknown origin.

The interventions used here sought to provide the individual with a means of controlling her behavior such that the targeted problem of depression or behavior change was resolved or became more manageable. In selecting treatments to match the target behaviors of depression and behavior change, the assumption of continuity between premenstrual symptoms and other forms of negative affect or

behavior was made. If depression is viewed from a behavioral perspective as a complex of responses which can be subsumed by the label of depression, the heterogeneous cluster of depressed behaviors which constitutes the psychological diagnostic category of depression also constitutes the premenstrual symptom of depression. Similarly, if avoiding responsibilities or required activities is viewed from a behavioral perspective as a complex of responses subsumed by the label behavior change, the heterogeneous cluster of activities which constitutes the behavioral category of avoidance behaviors also constitutes the premenstrual symptoms of behavior change. Treatments which have been demonstrated to be effective with other forms of these two heterogeneous clusters of behavior have been selected and are discussed. Beck's cognitive therapy of depression (Beck, Rush, Shaw, & Emery, 1979) was modified for use with premenstrual depression, and Fordyce's operant treatment for behavior control (Fordyce, 1976) was modified for use with premenstrual behavior changes.

Empirical support for the effectiveness of Beck's cognitive therapy of depression is available. The cognitive therapy of depression procedure involves three steps: (a) learning to identify negative thoughts or self-statements, (b) learning to dispute the negative thoughts, and (c) practice in disputing the negative thoughts. Shipley and

Fazio (1973) demonstrated the treatment procedure was more effective than a supportive treatment control. Morris (1975) demonstrated that a cognitive-behavioral treatment was more effective than an insight-oriented treatment or waiting list control. Shaw (1977) demonstrated cognitive modification was more effective than drug therapy, specifically imipramine (Rush, Beck, Kovacs, & Hollon, 1977).

Empirical support for Fordyce's operant treatment program for chronic pain behavior is also available. The operant treatment program involves application of contingent positive consequences for increases in activity level or engaging in required activities and withdrawal of positive consequences for "sick" behavior or staying in bed and avoiding responsibilities. The goal of the treatment program is to increase activity level and simultaneously to decrease pain behaviors such as staying in bed. Essentially, patients are taught to make more adaptive or active responses which will elicit positive responses of others (Fordyce, 1976). One of the first operant-based treatment programs reported decreased reported pain, medication use, and time in bed, and increased activity levels in patients followed from five to 175 weeks after treatment (Fordyce, Fowler, Lehman, Delateur, Sand, & Trieschman, 1973). An eight-week learning theory-based

program reported that 74% of their patients were medication free and leading normal lives six months to seven years after treatment (Anderson, Cole, Gullickson, Hudgens, & Roberts, 1977). At a ten-month follow-up of another similar program, 75% of the patients reported decreased pain or increased activity level, and 58% reported being medication free (Cairns, Thomas, Mooney, & Pace, 1976).

These demonstrations of the effectiveness of cognitive therapy of depression and operant treatment for behavior control, although not with menstrually related symptoms, suggested that these treatments would be effective in alleviating the premenstrual symptoms of depression and behavior change. The effectiveness of these treatments with premenstrual symptoms of negative affect and behavioral change assumes continuity between depression and behavior change under other circumstances and the clusters of behavior viewed as depression and behavior change during the premenstruum.

Evaluation of Behavioral Assessment

Behaviorists generally assume that correct identification of target behaviors leads to more effective intervention (Nelson & Hayes, 1981). There have been few empirical investigations, however, that demonstrate the importance of correct target behavior identification or of matching treatment to assessment results (Nelson & Hayes,

1979). The treatment validity of the selection of target behaviors (depression or behavior change) in PMS sufferers was assessed.

Treatment Validity

At present, there is some debate regarding the basis upon which to evaluate behavioral assessment. If the difference between traditional and behavioral assessment is seen primarily as conceptual, rather than methodological, then presumably the same methodology by which traditional assessment is evaluated should be sufficient and appropriate for behavioral assessment. Alternately, the conceptual differences may be seen as affecting methodology, requiring different evaluative criteria.

Cone (1977) has suggested that the differences between behavioral and traditional assessment are primarily conceptual. The methodological criteria used to evaluate traditional assessment procedures, therefore, apply to behavioral assessment. The psychometric criteria used to evaluate traditional assessment procedures are the validity and reliability (consistency) of measurement. These criteria are believed to be important in behavioral assessment as well (Hartmann & Wood, 1982). Cone (1977) proposed recasting traditional validity and reliability in terms of generalizability theory. He suggested that evaluation of behavioral assessment devices across universes

of generalizability (e.g., items, time) provides sufficient criteria upon which to judge the devices. Others agree and believe these procedures will lead to more adequate assessment devices (Hartmann & Wood, 1982). These psychometric and generalizability theory approaches to the evaluation of assessment devices assume that people respond consistently across dimensions or universes. When dealing with a traditional approach to psychological functioning, this assumption is reasonable. A good assessment device would be expected to reflect the theoretically proposed consistency of responses emanating from a trait, conflict, or disorder.

In traditional psychometric evaluations of assessment procedures, the consistency of responses measured by a traditional assessment device is examined: (a) across time by test-retest reliability and predictive validity; (b) across situations by parallel forms reliability and concurrent validity; and (c) across response systems by convergent validity. The assumptions of behavioral assessment are, however, different. They are that behavior is modifiable, situation-specific, and varies across response systems (Nelson, 1983). In behavioral assessment, inconsistent responding may indicate real changes in behavior, rather than a poor assessment device (Nelson, Hay, & Hay, 1977). Therefore, the psychometric criteria of

validity and reliability, alone, seem to be inappropriate or insufficient to evaluate behavioral assessment (Nelson & Hayes, 1979). Since the concepts of behavioral assessment differ from traditional assessment, perhaps the evaluative methodology should also differ. A functional approach to evaluation is more in keeping with a behavioral analysis.

Nelson and Hayes (1979) recommended evaluating behavioral assessment functionally using the criteria of conceptual validity and treatment validity. These criteria are felt to better reflect the assumptions of behavioral assessment. Conceptual validity reflects one functional value of behavioral assessment: the long-term contributions to the understanding of behavior. This involves both descriptions of phenomena and consistent conceptual explanations of why the phenomena interact as they do. Conceptual validity comes with time. Treatment validity reflects another functional value of behavioral assessment: the contribution of assessment procedures to treatment effectiveness. Treatment validity is concerned with the impact of assessment decisions on treatment effectiveness; for example, targeting one specific behavior may be more parsimonious in attaining the treatment goals than targeting another behavior. To demonstrate, Hay, Hay, and Nelson (1977) showed that reward of academic performance increased both academic accuracy and on-task behavior, while reward of

on-task behavior increased on-task behavior, but had no impact on academic performance. Treatment validity of all stages of the assessment procedure may be evaluated, including selection of assessment device, choice of target behavior, classification or diagnosis of clients, the use of functional analysis, and choice of treatment procedure. Demonstrations that particular assessment decisions lead to more effective treatment are needed at all stages of behavioral assessment to establish treatment validity. The evaluation of treatment validity of various stages of behavioral assessment procedures with various populations can lead to a greater understanding of behavior and, thus, to greater conceptual validity (Nelson & Hayes, 1979; 1981).

Recently, treatment validity studies have been recategorized into three general types of studies (Hayes, Nelson, & Jarrett, in press). These types of studies are: post hoc, a priori single dimension, and a priori multiple dimension. The post hoc type of treatment validity study addresses the relationship between patient characteristics and outcome of treatment. This type of study usually addresses this relationship subsequent to the actual experimental procedure, perhaps in an attempt to explain the pattern of results. Hayes and his colleagues consider this to be appropriate primarily as a means of generating treatment validity hypotheses.

The a priori, single-dimension type of study is divided into three subcategories indicating the dimension or the portion of the assessment process which is experimentally varied. These subcategories are manipulated assessment, manipulated match, and observed differences. The a priori, single-dimension, manipulated-assessment type of treatment validity study addresses the effect of assessment devices, strategies, and methods on outcome of treatment. Essential to this subcategory is that subjects be randomly divided into groups and one aspect of assessment be systematically varied. The a priori, single-dimension, manipulated-match type of treatment validity study addresses the effects of different use of available assessment data on outcome of treatment. What is varied in this subcategory is the "correspondence" between the assessment data and the chosen treatment procedure. The a priori, single-dimension, observed-differences type of treatment validity study addresses the relationship between patient types and outcome of treatment. Subjects are divided into groups based upon assessment differences (nonrandomly), and the impact of treatment, if differential, demonstrates the treatment validity of the assessment differences. This is considered a more common approach than the preceding a priori, single-dimension subcategories (Hayes et al., in press).

The a priori, multiple-dimension type of study is

divided into five subcategories indicating the two or more dimensions or the portions of the assessment procedure which are experimentally varied. These subcategories are manipulated assessment-manipulated match, manipulated assessment-observed differences, manipulated match-observed differences, manipulated assessment-manipulated match-observed differences, and observed differences with two or more treatments. The manipulated assessment-manipulated match combination addresses the effect of assessment procedures on the impact of different uses of assessment information on outcome of treatment. Essentially it is a design allowing each of the two single-dimension questions to be asked along with the question of their impact on each other. The manipulated assessment-observed differences combination addresses the effect of assessment procedures on the outcome for distinct subject types. Again, studies which fall in this subcategory address the single-dimension questions and their interaction. The manipulated match-observed differences combination addresses the effect of the use of assessment information on outcome of treatment for distinct subject types. The triple combination addresses the effect of assessment procedures on the impact of different uses of assessment information on outcome of treatment for distinct subject types. The final subcategory of the a priori, multiple-dimension type of treatment

validity study is observed differences using two or more treatment procedures. This subcategory addresses the effect of different treatment procedures on the outcome for distinct subject types.

According to Hayes and his colleagues, all of the above types of treatment validity studies can be performed through within-subject designs with the exception of the single-dimension, observed-differences subcategory as treatment does not vary and no differences can be manipulated.

One additional type of study is discussed in this delineation of types of treatment validity studies; it is termed the manipulated target (Hayes et al., in press). Hayes and his colleagues suggest that this type of study does not evaluate an assessment procedure, but rather explores the nature of a disorder which contains a number of possible target behaviors. The essential feature casting the manipulated target type of study from the treatment validity subtypes seems to be that any type of assessment may be used in the exploration of the nature of a disorder (Hayes et al., in press). Since the role of treatment validity studies is the evaluation of the various aspects of behavioral assessment, the view that manipulated target studies offer no information regarding the value of the assessment procedure clearly rules this type of study from

the classification of treatment validity studies. An alternate view considers the choice of target behavior a part of the assessment process essential to the effectiveness of treatment. While this particular type of study may not permit direct assessment of the quality of behavioral assessment, "it may evaluate the quality of our theories of disorders which may lead us to target one behavior over another. This could be called a kind of 'treatment validity.'" (Hayes et al., in press, p. 32). Perhaps, in the same way that the post hoc type of study is permitted in the classification of treatment validity studies, the manipulated target type of study could be included for its value in generating treatment validity hypotheses.

Two pragmatic considerations deserve attention in discussing the use of the manipulated target type of treatment validity methodology for the present study. First, while it is experimentally ideal to identify "pure types," the ability to do so may differ for different disorders. In some disorders, they may not be readily available as an experimental population, they may not exist at all, and the results obtained through the study of such pure types may not actually offer clinically useful information, particularly if they are sufficiently uncommon in the naturally occurring population. The treatment

validity of the spasmodic versus congestive dysmenorrhea distinction demonstrated by Chesney and Tasto (1975a) is an example. The ease with which one can find women who readily assign themselves to one category or the other and the actual distinction between the categories, per se, has been repeatedly questioned (Cox, 1977; Stephenson et al., 1983; Webster et al., 1979). In addition, in the initial screening for the present study, all of the first 20 volunteers demonstrated experience of a combination of the two symptom clusters (depression and behavior change), despite their statistically orthogonal nature. That is, they all experienced both clusters at least mildly rather than displaying only one of the clusters. The second pragmatic issue is the unexplored nature of the disorder studied here, PMS. This issue fits nicely with the view of Hayes and his colleagues (in press) that the manipulated target type of study is useful for the exploration of the nature of the disorder. When the symptom pattern of a disorder is unclear, and the patient population is undefined, as in PMS, it may be more valuable to use the manipulated target procedure. With this type of study, it is possible to evaluate the effectiveness of treatment, to get a preliminary picture of the course and symptoms of disorder, and to generate other treatment validity hypotheses for future research. Based upon these concerns,

the manipulated target appears to be the most appropriate design for the present study despite its step-sibling status to the other types of treatment validity studies.

A number of studies have been done which may be classified as treatment validity studies. These studies assess the impact of the use of behavioral assessment on treatment effectiveness. These treatment validity studies are reviewed and presented by problem area to better characterize the contribution of treatment validity studies to conceptual validity.

Treatment Validity Literature Review

There are select studies within particular topic areas which demonstrate the impact of behavioral assessment on enhancing treatment outcome. Various components of the assessment procedure have been examined by these studies. For example, the importance of subject characteristics or classification in enhancing treatment outcome has been demonstrated through the differential treatment effects of relaxation training in idiopathic and pseudoinсомniacs as classified by EEG measures (Borkovec, Grayson, O'Brien, & Weerts, 1979). As subjects were categorized as idiopathic and pseudoinсомniacs after completion of the study, this would fit in the post hoc type of treatment validity study delineated by Hayes and his colleagues (in press). The treatment validity of target behavior selection has also

been assessed. Aggressive behavior was shown to decrease more in response to contingency contracting for solitary as opposed to social play (Wahler & Fox, 1980). This study appears to be a manipulated target type of study as treatments directed at specific target behaviors were applied in the same individuals. The assessment of depression also has been examined using the treatment validity question: does the selection of target behavior enhance treatment outcome? Depressed women were classified as those with irrational cognitive responses and those with overt skills deficits in a manipulated match type of study (McKnight, Nelson, Hayes, & Jarrett, 1984). Cognitive therapy and social-skills training were used with both groups and were found to have differential treatment effectiveness. Not surprisingly from a logical perspective, cognitive therapy was more effective with irrational cognitive responders, not only in altering irrational cognitions but also in ameliorating depression. Social-skills training was more effective with those with overt skills deficits, in improving both social skills and depression. These are select examples within different areas. Treatment validity studies, in general, need to be done for different disorders, different phases of the assessment process, and different assessment techniques. Treatment validity studies within the areas of (a) social

behavior and (b) perimenstrual distress are discussed with an eye to the component of the assessment procedure being evaluated and the type of study performed.

Social behavior is a complex cluster of complaints and behaviors. Problems in social behavior have commonly been attributed to social anxiety and/or skills deficits. Trower, Yardley, Bryant, and Shaw (1978) compared the impact of social skills training and systematic desensitization on individuals classified as deficient in social skills or as socially anxious. This study appears to fit into the treatment validity category of observed differences with two treatments. Distinct subject types are crossed with two distinct treatment approaches, testing the treatment validity of the subject types and the theories which distinguished them and their implied treatments. Not surprisingly, individuals deficient in social skills improved more with social skills training. Socially anxious individuals, however, improved with either intervention in a comparable manner. It seems more important to successful treatment selection to identify social-skills deficits than social anxiety. Within social-skills deficits, the treatment of target behaviors has been demonstrated. Kupke, Calhoun, and Hobbs (1979), in what is likely to be classified as a manipulated target type of study, showed higher ratings of female attraction resulted from training

males in personal attention skills (e.g., use of the pronoun "you") over minimal encouragement skills (e.g., brief statements like "Oh, really?").

Others have found differential effects of treatment within social anxiety. Ost, Jerremalm, and Johansson (1981) assessed the impact of matching treatment to individual response patterns in social phobics. They classified their subjects as behavioral or physiological responders and evaluated the effect of social skills versus relaxation training on the response patterns. In general, social skills training was better for behavioral responders (six of ten measures were significantly different), while relaxation training was better for physiological responders (three of ten measures were significantly different). Specifically, on the overall change score measure, there was no significant difference between treatments for behavioral responders, but the physiological responders improved significantly more ($p < .025$) with the relaxation treatment than with the social skills treatment. This study demonstrated the treatment validity of the behavioral-physiological differentiation among social phobics using an observed differences with two types of treatment type study. Socially anxious individuals have also been subdivided into those with increased autonomic perceptions plus increased physiological reactivity and those with increased autonomic

perceptions and no increase in physiological reactivity (Shaher & Merbaum, 1981). This is similar to the behavioral-physiological distinction used by Ost and colleagues (1981). Shaher and Merbaum (1981), however, focus more on a physiological-cognitive distinction. Individuals in these classifications or subdivisions of social anxiety responded differentially to desensitization and cognitive restructuring procedures; those with increased physiological reactivity improved more with desensitization and those with no increase in physiological reactivity improved more with cognitive restructuring. This study demonstrated the treatment validity of the physiological-cognitive differentiation among socially anxious subjects using an observed differences with two types of treatment type study.

Another study which may be retrospectively classified as a treatment validity study further clarifies the value of specifying social behavior problems through assessment procedures. Meichenbaum, Gilmore, and Fedoravicius (1971) demonstrated differential responding to treatment by individuals with generalized social anxiety and those with specific speech anxiety. Cognitively oriented insight treatment was more effective with generalized social anxiety, and desensitization was more effective with specific speech anxiety. This suggests the treatment

validity of distinguishing general from specific anxiety using what appears to have been a post hoc type of treatment validity study. Similar to Shaber and Merbaum's (1981) treatment of social anxiety, others have examined the differential responding of speech anxious individuals divided into cognitive and somatic responders (Altmaier et al., 1982). These researchers examined the impact of components of stress inoculation on these two types of anxiety responders using an observed differences with two or more treatments type study. They found the relaxation component and the complete stress inoculation treatment to be significantly better ($p < .001$) than the coping statement component or no treatment for somatic responders, but only on the cognitive indices of anxiety. The behavioral indices of anxiety showed no differential effects; all three components were significantly better than no treatment ($p < .006$). There was no significant differential treatment effect for the cognitive responders. This study supports the importance of assessing the impact of assessment decisions on different response modes when performing treatment validity studies (Nelson & Hayes, 1979).

In general these treatment validity studies of social problems stress the importance of differentiating skills deficits versus anxiety excesses and then identifying the specific pattern of anxiety (e.g., general or specific, and

cognitive, behavioral, or physiological) in order to maximize treatment impact. In terms of general understanding of behavior referred to as social anxiety and social problems, we have gained a relatively clear picture through these studies that social problems constitute a class of complex response patterns which benefit differentially from specified treatment. The use of alternative types of treatment validity studies could help to provide an even clearer picture of the value of behavioral assessment procedures in the study of social problems and their treatments.

Another area which has been examined from the perspective of the treatment validity of behavioral assessment is dysmenorrhea. These studies have focused primarily on the treatment validity of the Menstrual Symptom Questionnaire in classifying subjects as experiencing congestive versus spasmodic dysmenorrhea. Using the classification of congestive versus spasmodic dysmenorrhea, Chesney and Tasto (1975b) demonstrated a differential response to desensitization using an observed differences with two or more treatments type study. Women classified as spasmodic dysmenorrhea sufferers improved significantly more ($p < .001$) from desensitization than from placebo-discussion or no treatments; there were no differential treatment effects for congestive dysmenorrhea

sufferers. Duson (1976) found similar differential responding to desensitization, but not to a cognitive restructuring procedure. The treatment validity of the congestive-spasmodic distinction, however, has not been upheld in other studies. Cox and Meyer (1978) found systematic desensitization effective with fourteen women with primary dysmenorrhea, but found no significant difference between congestive and spasmodic groups using what appears to be a post hoc type of treatment validity study; they consider it an arbitrary dichotomy. Rosenthal (1978) also reported no significant treatment effect and no interaction between congestive and spasmodic dysmenorrhea classifications and treatment. Quillen and Denney (1982), using essentially a single-dimension, observed-differences type of study with a no-treatment control group, found pain management training to be effective in treating dysmenorrhea, but found no differential response to treatment based upon the subject classification of congestive and spasmodic dysmenorrhea. Amodei (1983) also found little support for a differential effect of treatment on congestive and spasmodic responders using an observed differences with two or more treatments type treatment validity study. These results suggest the congestive-spasmodic distinction proposed by Dalton (1969), in general, is not predictive of treatment effectiveness. The treatment validity of the

congestive-spasmodic classification based on the Menstrual Symptom Questionnaire has not been clearly demonstrated. The effectiveness of desensitization using hierarchies and images specifically matched to the subjects' symptoms (Rosenthal, 1978; Quillen & Denney, 1982), however, suggests the importance of matching treatment to specific target behaviors in order to enhance treatment outcome. Research examining all components of the assessment procedure and a variety of the types of treatment validity studies are needed in the area of perimenstrual distress.

Statement of Purpose

The purpose of this study was to evaluate the treatment validity of identifying specific target behaviors within the premenstrual syndrome. This treatment validity issue is concerned with the question: does selection of specific target behaviors and matching treatment to them enhance treatment outcome?

Selection of specific target behaviors is considered a manipulated target type study which is not considered to have direct bearing on the quality of assessment (Hayes et al., in press). Therefore, there is a potential controversy in calling the present study a treatment validity study without qualification. The qualification of applying the label treatment validity study may be reasonably reiterated here. Hayes and his colleagues (in press) suggest that the

selection of target behaviors when evaluated in a manipulated target type study does not offer information directly related to the evaluation of behavioral assessment and, therefore, does not clearly fit within the classification of treatment validity study. They do offer, however, that such a study may evaluate the quality of our theories of disorders which may lead us to select specific target behaviors and, thus, may be viewed as a kind of treatment validity. In addition, in the same way that the post hoc type of study is permitted in the classification of treatment validity studies, the manipulated target type of study could be included for its value in generating other treatment validity hypotheses and in addressing issues related to treatment validity. Therefore, the present study is referred to here as a treatment validity study of targeting specific symptoms.

Treatment Validity of Targeting Specific PMS Symptoms

The central question posed here is: does the selection of a specific target behavior from among the many symptoms associated with PMS and matching it to a treatment enhance treatment outcome? This is a question of the treatment validity of identifying specific target behaviors within the general classification of PMS. Within an individual woman with symptoms of depression and behavior change, is it important to the outcome of treatment to use a treatment

directed at her depression symptoms? or is it important to the outcome of treatment to use a treatment directed at her behavior change symptoms? More generally, is it important to treatment effectiveness to target specific symptoms within PMS? It may not be important to treatment effectiveness; this is an empirical question (Nelson & Hayes, 1979).

The prediction regarding this issue is that symptoms will change only if they have been targeted by the specific treatment procedure matched to them. Thus, in this study, the cognitive therapy of depression was expected to decrease complaints of the symptoms associated with depression, and the operant intervention for behavior control was expected to decrease complaints of the symptoms associated with behavior change. The interventions used were expected to be effective in treating only the symptoms for which they were intended as they have been demonstrated to be effective, previously, with similar, although nonmenstrually related problems (Beck et al., 1979; Fordyce et al., 1973). These procedures are noninvasive and provide the subject with a means of controlling the discomfort she experiences premenstrually.

These predictions have treatment validity implications. If the cognitive therapy of depression is effective with depression symptoms and the operant intervention for

behavior control is effective with behavior change symptoms, then, in the future, a woman with predominant depression symptoms would be better helped by the depression treatment than by the behavior change treatment. Similarly, a woman with predominant behavior change symptoms would be better helped by the behavior change treatment than by the depression treatment. These implications for the future management of PMS symptoms actually offer testable hypotheses regarding the treatment validity of targeting specific symptom clusters.

An additional question is addressed by this study: will changing one specific symptom (depression or behavior change) have more impact than changing the other on changing PMS as a whole, as measured by the Menstrual Symptom Questionnaire? This question also has treatment validity implications. If the overall measure, the MSQ, responds differentially to the depression treatment, then the depression symptom cluster may be more central to the disorder of PMS. It may be more effective to target depression symptoms than behavior change symptoms. Alternately, if the overall measure responds differentially to the behavior change treatment, then the behavior change symptom cluster may be more central to the disorder of PMS. It may be more effective to target behavior change symptoms. These implications also provide testable hypotheses regarding the nature of PMS. If over time and trials,

treatments that target depression are consistently superior to those that target behavior change, then selecting depression as a target behavior within PMS may be said to have "treatment validity." Or if treatments that target behavior change are consistently superior to those that target depression, then selecting behavior change as a target behavior within PMS may be said to have "treatment validity." While this does not provide a direct evaluation of assessment, it would provide information on the nature of the disorder (Hayes et al., in press).

In summary, this study asks the questions: (a) does the selection of a specific target behavior (depression or behavior change) from among the many symptoms associated with PMS and matching it to treatment (cognitive therapy of depression or operant intervention for behavior control) enhance treatment outcome? and (b) will changing a specific symptom (depression or behavior change) have more impact on changing PMS as a whole, as measured by the MSQ? Although this study may be considered a manipulated target type which does not directly evaluate behavioral assessment (Hayes et al., in press), it is considered a kind of treatment validity study here as the answers it provides may generate further treatment validity hypotheses and information regarding the nature of the disorder (PMS) against which our theories may be tested.

CHAPTER II

METHOD

Subjects

Thirty-six postpubescent female volunteers, complaining of premenstrual discomfort, and solicited from the local community, served as subjects. The subjects (age range 21-41 years, mean 31.9) were not oral contraceptive users. They denied use of physician-prescribed medication for their premenstrual discomfort, and were not experiencing severe psychological or psychiatric disturbances. All subjects reported at least a one-year history of premenstrual discomfort. Hoes (1980) suggested that a four-cycle history of symptoms is necessary for a diagnosis. Subjects were solicited by newspaper, radio, or television public service announcements. Any volunteer determined to be ineligible during the screening assessment phase of the study was offered a list of referral sources including local physicians and psychologists.

Screening Assessment

One hundred and two women responded to the advertisements for subjects. Of these, 27 were deemed inappropriate for participation in the study during the initial telephone contact; of these, 10 were taking oral contraceptives, 9 were taking prescription medication for

premenstrual syndrome, and 8 had other medical conditions (e.g., previous hysterectomy). The remaining 75 women from the community scheduled screening assessment appointments with the principal investigator. They all reported that they were at least 18 years old and denied use of oral contraceptives and physician-prescribed medication for premenstrual symptoms. Nineteen of the women who initially scheduled appointments either cancelled or failed to come in for their appointments. The remaining 56 volunteers met with the principal investigator for their screening assessment appointments. Two of these were determined ineligible by virtue of psychological/psychiatric disturbances. Another eleven were determined to be ineligible by their scores on the Menstrual Distress Questionnaire which was used to assess severity of symptoms. Five subjects withdrew following the screening assessment and prior to the first (baseline) menses; three had been assigned to the control group, one to the depression treatment group, and one to the behavior change treatment group. Two additional subjects withdrew after completing their participation in the treatment sessions: one in the depression treatment group and one in the behavior change treatment group. Thus, thirty-six subjects completed the study, twelve in each group.

Volunteers were interviewed by the principal investigator to ascertain the presence of premenstrual discomfort and the absence of serious psychological disturbance (see Appendix B). Each subject complained of at least a one-year history of premenstrual discomfort, including depression and behavior change. The duration of discomfort ranged from 4 to 14 days prior to the onset of a menstrual period. Sutherland and Stewart (1965) consider the diagnosis of premenstrual syndrome to be based upon the report of physical or psychological symptoms beginning at least four days before menstruation.

Following the interview, volunteers still eligible then completed the Menstrual Distress Questionnaire (MDQ) (Moos, 1969). The MDQ (see Appendix C) consists of 47 items presented as symptoms which women experience. For the screening assessment, each item was rated regarding a typical week preceding the subject's menstrual flow onset. The items are rated on a 6-point Likert-type scale ranging from no experience of the symptom to acute or partially disabling experience of the symptom. The original version of the MDQ rates the most recent menstrual cycle and includes two additional ratings of each symptom: the period of the actual menstrual flow and the intermenstrual time period. The intermenstrual time refers to the rest of the subject's cycle excluding her days of flow and the preceding

week. These ratings of the menstrual and intermenstrual time periods were considered unnecessary for the present study. The focus here was on the the premenstruum and associated symptomatology. The 47 items on the MDQ are divided into eight factors by Moos' original analyses (1969). Each of the eight factors reflects an intercorrelated group of symptoms associated with phases of the menstrual cycle. The factors are pain, concentration, behavioral change, autonomic reactions, water retention, negative affect, arousal, and a control scale. The control scale includes six items which are not considered to be symptoms of menstrual distress, but are included as a measure of complaining.

Eligibility for participation required a score of 15 or greater on the Moos Behavioral Change cluster (items: 4, 8, 15, 20, and 41), an average rating of at least 3 on each of five items; and a score of 24 or greater on the Moos Negative Affect cluster (items: 3, 11, 21, 27, 36, 38, 40, and 45), an average rating of at least 3 on each of eight items. The score of 3 was an arbitrary cut-off chosen because it indicates the symptom is present and noticeable although mild. The Moos Behavioral Change and Negative Affect clusters were chosen because they appear to be consistent clusters based upon Moos (1969) findings and are supported by the pilot data reported in the first chapter

(the Introduction); they also appeared to be amenable to psychological intervention. If the individual met these criteria, she was eligible to participate in the study.

The mean score on the screening assessment MDQ Behavioral Change cluster for the 36 subjects who completed the study was 19.1 and the range was 15 to 26. Their mean score on the Negative Affect cluster was 35.6 and the range was 25 to 48 (Table 1; Table 1 and all subsequent tables are located in Appendix D).

After eligibility had been determined through the interview and the MDQ, during the screening session, a data deposit of \$20.00 was collected. Refundable deposits have been shown to result in fewer absences and more consistent data (Ersner-Hershfield, Connors, & Maisto, 1981). Each subject was asked to make out four checks for \$5.00 each to the principal investigator and her supervisor. One of the four checks was returned to the subject at each of the four previously agreed upon times; these times were explicitly stated in the initial consent form (Appendix E1). Subjects signed an additional consent form which specifically referred to the treatment group to which each had been assigned (Appendix E2).

Design

A 3 (treatment) X 3 (measurement occasions) experimental design was used. The between-groups factor was

treatment. The treatments were cognitive therapy of depression, operant intervention for behavioral change, and a no treatment data collection only control. Subjects were randomly assigned to treatment within the constraint that one third of the subjects were in each treatment group. Twelve of the subjects participated in the cognitive therapy of depression, 12 participated in the operant intervention for behavioral control, and 12 served as a no-treatment control group. The two experimental treatment procedures (cognitive therapy of depression and operant intervention for behavioral control) were administered in four sessions which occurred between the baseline menses and the posttreatment menses. The control group members were offered a choice between the two treatment procedures after three cycles of data collection. The within-subjects factor (measurement occasions) refers to the time periods for which the collected dependent measures were analyzed. All dependent measures were analyzed for the baseline premenstruum (the four days preceding each subject's first menstrual period), the posttreatment premenstruum (the four days preceding each subject's second menstrual period, the first following intervention), and the follow-up premenstruum (the four days preceding each subject's third menstrual period).

Dependent Measures

Each subject completed a daily self-assessment every evening at bedtime throughout her participation in the study (three menstrual cycles). The daily rating included self-reports of subjective and objective measures of the two targeted behaviors (depression and behavioral change). Both subjective measures (depression and behavior change) used Likert-type scales with 0 = no experience of the symptom and 6 = extreme experience of the symptom. Although subjects completed daily self-assessments throughout their participation in the study (three menstrual cycles), only the four days prior to the onset of menses were used in the data analyses. The objective measures used a frequency count of discrete episodes of crying for depression and duration of time resting, not engaged in required activities for behavior change. The sum of each of the ratings (subjective Likert-type and objective frequency count or amount of time) for the four days immediately preceding the onset of menstrual flow served as the dependent variable scores for the analyses. Thus, each subject had four daily self-rating scores for each premenstruum (Appendix F). Each subject received instructions regarding the completion of the daily self-assessment as needed from the principal investigator.

In order to provide an additional means of assessing the impact of treatment on the two targeted symptom clusters, each subject also completed an MDQ for each premenstruum included in the study. Each subject completed the MDQ three times following the screening assessment; the items were rated regarding the week prior to the most recent menstrual flow onset. The MDQ was completed within one week after the onset of each subject's first (baseline) menstrual period; then within one week after the onset of each subject's second menstrual period; and, finally, within one week after the onset of each subject's third (follow-up) menstrual period. The assessments were administered after the onset of each subject's menstrual period as the onset of menstrual flow is considered to be symptom free for premenstrual syndrome (Dalton, 1977). In addition, the onset of menstrual flow is more easily equated across subjects than is the premenstruum, and it is close in time to the experience of premenstrual symptoms which aids in reliability of ratings. The MDQ provided a score for the Behavioral Change symptom cluster and a score for the Negative Affect symptom cluster.

In addition to the daily self-assessment and the MDQ measures which specifically assessed the targeted symptoms, each subject also completed the MSQ (Chesney & Tasto, 1975) three times to provide a global measure of change which is

not directly tied to the criteria for inclusion in the study. The MSQ consists of 24 statements which describe symptoms associated with the menstrual period. Twelve of the statements are associated with congestive dysmenorrhea (premenstrual symptoms), and 12 are associated with spasmodic dysmenorrhea (menstrual symptoms). The MSQ, usually scored for frequency of symptom occurrence, was scored for the purpose of this study for severity of symptoms during the present/most recent premenstruum. Severity was rated on a Likert-type scale ranging from 1 = not at all to 5 = severe experience of the symptom (Appendix G). Sums of the subject's severity ratings on the congestive (premenstrual) items were compared in order to have a general measure of change in symptom severity across the three menstrual cycles included in the study. The MSQ was completed with the MDQ within one week after the onset of each of each subject's three menstrual periods.

Therefore, each subject had a total of seven scores for each premenstruum; four daily self-assessment scores, two MDQ targeted symptom cluster scores, and one MSQ congestive symptom global score (Table 2).

Treatment

Each subject participated in one of two treatment programs (or was in the control group): One program was designed to address the symptom of depressed mood and the

other was designed to address the symptom of behavior change or decreased activity level. A modified version of Beck's (1979) depression treatment (see Appendix H) was used as the depression intervention. A modified version of Fordyce's (1976) operant program for behavior control (see Appendix I) was used as the behavior change/activity level intervention.

Beck's cognitive therapy of depression (Beck, Rush, Shaw, & Emery, 1979) was modified for application within four treatment sessions (see Appendix H). The treatment procedure involved restructuring of negative thought patterns. This method of restructuring negative thought patterns takes two forms: (a) demonstrating the falsity of the beliefs through empirical hypothesis testing and (b) disputing the negative beliefs through logical arguments. Both forms of restructuring negative thoughts were explained, demonstrated and, practiced within the treatment sessions. Subjects received instructions to practice the technique daily and to apply it during the premenstruum.

Fordyce's operant intervention for pain behavior control (Fordyce, 1976) was modified for use with decreased activity level and behavior change during the premenstruum and for application within four sessions (see Appendix I). The treatment procedure involved learning to increase desired/required activity level through the application of operant principles and the use of scheduling. Increased

required activity level is accomplished through identification of potential rewards and scheduling these rewards contingent upon completion of the required activity (see Appendix I-4). Both parts of this operant program for behavior control were explained, demonstrated, and practiced within the treatment sessions. Subjects received instructions to practice the technique daily and to apply it during the premenstruum.

The subjects who had been randomly assigned to the control group were told that they needed to collect daily assessment data for three cycles. After completing the data collection, they would then have the opportunity to participate in four experimental treatment sessions conducted by the principal investigator. Following data collection, control subjects were debriefed. The debriefing involved an explanation of the experimental hypothesis and the use of and need for control groups (see Appendix J). Subjects were then presented with the rationales for both treatments and were given the opportunity to choose the treatment procedure they would prefer. Eight control subjects chose to terminate their involvement prior to participating in one of the treatments; two chose the cognitive therapy of depression; and two chose the operant intervention for behavior control.

Each of the interventions was administered within 14 to 20 days of the onset of the first menstrual flow in four sessions. The final session occurred at least one week prior to the onset of the subjects' second menstrual flow. The interventions were individually administered by the principal investigator and one other female graduate student in psychology who served as therapists. The principal investigator served as a therapist to five of the subjects in the behavior change treatment group and seven of the subjects in the depression treatment group; the other therapist saw seven subjects in the behavior change treatment group and five subjects in the depression treatment group. The assignment of subjects to therapists was based upon compatibility of schedules and balance across therapists of the two treatment groups. The principal investigator provided the interventions to all of the control subjects who participated in treatment. The therapists were trained in the administration of the treatment packages.

Therapist Training and Monitoring

Each therapist participated in four hours of training with the two treatment packages. The training involved review of the components of each treatment package and rehearsal of administration of each of the eight treatment sessions (see Appendices H & I). The training also involved

clarification and rehearsal of any aspect of the sessions that the therapists felt they needed to practice further.

Each therapist was periodically audiotaped. These tapes were reviewed by the author and her supervisor to ensure consistency in the administration of the interventions. As a check on the independent variable, tapes of 11 percent of the sessions were reviewed by both the principal investigator and her supervisor. Each identified each taped session as the "behavior change" or "depression" treatment. One hundred percent agreement was demonstrated suggesting the interventions were identifiably different.

Procedure

Each subject participated in a screening interview during which she completed the MDQ, was determined to be eligible for treatment, and was randomly assigned to a treatment group. At that time, she made her data deposit, received instructions regarding data collection, and began data collection. She completed daily rating sheets for at least four days prior to the onset of her first (baseline) menstrual period. She continued to self-record daily throughout the duration of her participation in the study. Within one week after the onset of her baseline menstrual period, each subject contacted the principal investigator and completed the MDQ and the MSQ. All subjects except the

twelve control subjects were then assigned to a therapist and participated in four treatment sessions within the next 14 to 20 days. Within one week after the onset of her second menstrual period, each subject again contacted the principal investigator and completed the MDQ and the MSQ. Within one week after the onset of her third (follow-up) period, each subject completed her last MDQ and MSQ for this study. At that time, each subject also received a list of referrals for further treatment, if she so desired.

CHAPTER III

RESULTS

Experimental Design

A 3 (treatments) by 3 (measurement occasions) experimental design was used. The between-subjects factor was treatments (cognitive therapy of depression, operant techniques for behavioral control, and no treatment control). Subjects were nested in treatment. The within-subjects factor was measurement occasions or time.

Data were collected for three premenstrual time periods: pretreatment or baseline, posttreatment, and follow-up. Discrete scores for the three premenstrual time periods assessed in the study were derived by summing each subject's daily ratings across the four days immediately preceding the onset of menses. The sums of the ratings for the four days prior to the onset of the baseline menstrual period, the sums of the ratings for the four days prior to the onset of the posttreatment menstrual period, and the sums of the ratings for the four days prior to the onset of the third (follow-up) menstrual period provided the scores for the measures of depression and behavior change: subjective depression rating, crying episode frequency,

subjective behavior change rating, and amount of time resting. An additional measure of each targeted symptom cluster was provided by the negative affect and behavioral change symptom cluster scores derived from the Menstrual Distress Questionnaire (MDQ) completed by each subject for each premenstruum during the study. A seventh dependent measure consisted of a score derived from the Menstrual Symptom Questionnaire (MSQ) which provided an overall measure of change not directly related to the targeted symptom clusters. Decreasing scores on all measures indicate improvement, i.e., lower scores reflect endorsement of less symptom severity or fewer symptoms.

The results of the analyses are presented in the context of the experimental questions to which they pertain. The central question was "did the treatments differentially affect the types of symptoms?" This question is further divided into the specific differential effects of treatment on the depression symptom measures and on the behavior change symptom measures. The second question was "did the treatments differentially affect overall PMS complaints?"

Did Treatments Differentially Affect

Types of Symptoms?

Insofar as the experimental hypothesis was concerned with the differential effect of the treatments on the specific targeted symptom clusters, it was necessary to

analyze the impact of the treatments on the specific symptom clusters separately. Therefore, two separate analyses were conducted; one on the dependent variables used to measure depression and one on the dependent variables used to measure behavior change. Thus, one multivariate analysis of variance (MANOVA) with Groups as the between-subjects factor, Time as the within-subjects factor, and three dependent measures (the MDQ negative affect symptom cluster measure, the subjective daily depression rating, and crying episode frequency) was used to assess the impact of the treatments on the depression symptom cluster; and one MANOVA with Groups as the between-subjects factor, Time as the within-subjects factor, and three dependent measures (the MDQ behavioral change symptom cluster measure, the subjective daily behavior change rating, and time resting) was used to assess the impact of treatments on the behavior change symptom cluster. These results are discussed in relation to the specific experimental questions to which they pertain.

Did the Treatments Differentially Affect the Depression Measures?

The depression measures included the MDQ negative affect cluster measure, the daily depression rating measure, and the crying frequency measure. The depression symptom cluster MANOVA revealed no significant effects for Group,

Wilks Lambda = .91, which is equivalent to $F(6,62) = .53$, $p > .05$; Time, Wilks Lambda = .71, which is equivalent to $F(6,28) = 1.93$, $p > .05$; or the interaction of Group by Time, Wilks Lambda = .55, which is equivalent to $F(12,56) = 1.61$, $p > .05$ (Table 3).

Consistent with the MANOVA results, none of the ANOVAs on these measures showed the predicted significant group by time interaction. Only the ANOVA on the MDQ negative affect symptom cluster showed any significant effect. There was a significant main effect for Time, $F(2,66) = 4.79$, $p < .05$. There was no significant effect for Group, $F(2,33) = .41$, $p > .05$, and no significant effect for the interaction of Group by Time, $F(4,66) = 1.32$, $p > .05$ (Table 4; Figure 1; Figure 1 and all subsequent figures are in Appendix K). The Newman-Keuls post hoc comparison among the means comprising the significant effect for Time (Table 5) revealed significant differences between the baseline time period and the post-treatment time period and between the baseline time period and the follow-up time period. The difference between the means of the posttreatment and follow-up time periods were not significant.

There were no significant effects demonstrated in the analyses of the daily depression rating measure (Table 6; Figure 2). There were no significant effects for Group, $F(2,33) = .39$, $p > .05$, Time, $F(2,66) = .69$, $p > .05$, or the interaction, $F(4,66) = .89$, $p > .05$.

There were no significant effects demonstrated in the analyses of the crying frequency measure (Table 7; Figure 3). There were no significant effects for Group, $F(2,33)=1.06$, $p>.05$, Time, $F(2,66)=.38$, $p>.05$, or the interaction, $F(4,66)=2.02$, $p>.05$.

These results offer no support for the prediction that the treatments would differentially affect the depression measures.

Did the Treatments Differentially Affect the Behavior Change Measures?

The behavior change measures included the MDQ behavioral change symptom cluster measure, the daily behavior change rating measure, and the time resting measure. Similarly to the MANOVA performed on the depression symptom measures, the behavior change symptom cluster MANOVA also revealed no significant effects for Group, Wilks Lambda = .95, which is equivalent to $F(6,62)=.28$, $p>.05$ or the interaction of Group by Time, Wilks Lambda = .66, which is equivalent to $F(12,56)=1.10$, $p>.05$. The behavior change symptom cluster MANOVA, however, did reveal a significant effect for Time, Wilks Lambda = .59, which is equivalent to $F(6,28)=3.21$, $p<.05$ (Table 8).

Similar to the findings on the depression measure analyses, none of the ANOVAs performed on the behavior

change measures showed the predicted significant group by time interaction. There was, however, a significant effect on the MDQ behavioral change symptom cluster measure, consistent with the results of the MANOVA on the behavior change symptom measures. This was a main effect for Time, $F(2,66)=4.25$, $p<.05$. There was no significant effect for Group, $F(2,33)=.71$, $p>.05$, and no significant effect for the interaction of Group by Time, $F(4,66)=.68$, $p>.05$ (Table 9; Figure 4). The Newman-Keuls post hoc comparison among the means comprising the significant effect for Time (Table 10) revealed significant differences between the baseline time period and the posttreatment time period and between the baseline time period and the follow-up time period. The difference between the means of the posttreatment and follow-up time periods were not significant.

There was also a significant effect on the daily behavior change rating measure, a main effect for Time, $F(2,66)=6.43$, $p<.01$. There was no significant effect for Group, $F(2,33)=.39$, $p>.05$, and no significant effect for the interaction of Group by Time, $F(4,66)=2.27$, $p>.05$ (Table 11; Figure 5). The Newman-Keuls post hoc comparison among the means comprising the significant effect for Time (Table 12) revealed significant differences between the baseline time period and the posttreatment time period and between the baseline time period and the follow-up time period. The

difference between the means of the posttreatment and follow-up time periods were not significant.

There were no significant effects demonstrated in the analyses of the time resting measure (Table 13; Figure 6). There were no significant effects for Group, $F(2,33) = .11$, $p > .05$, Time, $F(2,66) = 1.00$, $p > .05$, or the interaction, $F(4,66) = .02$, $p > .05$.

These results offer no support for the prediction that the behavior change treatment would differentially affect the behavior change measures.

In general, the multivariate analyses and the univariate analyses demonstrate no support for the treatment validity hypothesis that the treatments would differentially affect the type of symptoms.

Did the Treatments Differentially Affect Overall PMS Complaints?

The congestive scores on the MSQ were included as an overall measure of PMS symptom complaints. The congestive items on the MSQ are those items which pertain to symptoms occurring prior to the onset of the menstrual flow and, thus, are analogous to premenstrual symptoms. The ANOVA performed on the MSQ measure showed no differential effect by treatment group.

There was one significant finding on the MSQ measure. It was a significant main effect for Time, $F(2,66) = 7.52$,

$p < .01$. There was no significant effect for Group, $F(2,33) = .06$, $p > .05$, and no significant effect for the interaction of Group by Time, $F(4,66) = 1.77$, $p > .05$ (Table 14; Figure 7). The Newman-Keuls post hoc comparison among the means comprising the significant effect for Time (Table 15) revealed significant differences between the baseline time period and the posttreatment time period and between the baseline time period and the follow-up time period. The difference between the means of the posttreatment and follow-up time periods were not significant.

In general, these results offer no support for the hypothesis that the treatments would differentially affect an overall measure of PMS complaints.

In summary, there was no indication in the multivariate or univariate analyses of a differential effect by treatments on either type of symptom or the overall measure of PMS complaints. The only significant effects were for Time on the MDQ measure of depression and behavior change, on the daily behavior change rating, and on the MSQ overall measure of PMS symptom complaints.

Correlation Among Dependent Measures

The degree of correlation among the seven dependent measures is of interest for a number of reasons. The measures were concomitantly collected and are presumed to be measuring various aspects of the same problem, PMS. Pearson

correlation coefficients were computed for the pretreatment or baseline scores on all seven measures. The degree of correlation among the measures is discussed in relation to the aspects of PMS (depression symptom cluster, behavior change symptom cluster, overall PMS complaints) they were presumed to be assessing. The correlation matrix for all measures is presented in Table 16.

Correlation Among Depression Measures

The three measures used to assess the depression symptoms were the MDQ negative affect symptom cluster, the daily depression rating, and crying frequency. The MDQ negative affect symptom cluster measure showed a significant correlation with the daily depression rating, $r = .42$, and with the crying frequency measure, $r = .47$, for both $p < .01$. The daily depression rating and crying frequency measures also showed a significant correlation, $r = .46$, $p < .01$.

Correlation Among Behavior Change Measures

The three measures used to assess behavior change were the MDQ behavioral change symptom cluster measure, the daily behavior change rating, and time resting. The MDQ behavioral change symptom cluster measure showed no significant correlation with the daily behavior change rating, $r = .23$, or time resting, $r = -.06$, for both $p > .05$. The daily behavior change rating and time resting measures, however, showed a significant correlation, $r = .32$, $p < .05$.

Correlation Between Depression and Behavior
Change Measures

The MDQ negative affect and behavioral change symptom cluster measures showed a significant correlation, $r = .55$, $p < .01$. The daily depression and behavior change ratings also showed a significant correlation, $r = .62$, $p < .01$. The objective measures, crying frequency and time resting, however, showed no significant correlation, $r = .00$, $p > .05$. The preceding three comparisons are the logical associations which might be expected among the measures; the two measures taken from the MDQ questionnaire were compared, the two subjective measures taken from the daily rating form were compared, and the two measures considered to be the objective measures were compared. There was only one other significant correlation among the measures compared across targeted symptom clusters: the MDQ negative affect measure showed a significant correlation with the daily behavior change rating, $r = .34$, $p < .05$. There was no significant correlation between the MDQ negative affect measure and the time resting measure, $r = -.10$, $p > .05$. The MDQ behavioral change measure showed no significant correlation with the daily depression rating, $r = .24$, or with the crying frequency measure, $r = .23$, for both $p > .05$.

Correlation Between the Overall Measure of PMS
and the Symptom Cluster Measures

The MSQ was the overall measure of PMS. It showed significant correlations with both the MDQ negative affect measure, $r = .37$, $p < .05$, and the MDQ behavioral change measure, $r = .43$, $p < .01$. The MSQ also showed a significant correlation with the daily depression rating, $r = .33$, $p < .05$. There were no other significant correlations; daily behavior change rating, $r = .19$, crying frequency measure, $r = .14$, and time resting measure, $r = .24$, for all $p > .05$.

Individual Subject Data and
Nonparametric Sign Tests

To examine the influence of individual subjects' responses on the apparent change over time and to assess the relative number of subjects improved in each group, nonparametric sign tests (Siegel, 1956) were performed on the frequency of subjects improving or worsening from baseline. Improving or worsening were defined as a change of at least plus or minus one unit of measurement relative to the baseline score. The analyses pertaining to the overall change (change over time) are presented first, and then the analyses reflecting changes within individual treatment groups are presented.

Change over Time Sign Tests

Nonparametric sign tests were performed on the number of subjects who changed from baseline to posttreatment and

from baseline to follow-up for each dependent measure (Table 17). Consistent with the results of the ANOVAs, the sign tests on the MSQ overall measure of PMS were significant. From baseline to posttreatment, 35 subjects' scores changed: eleven subjects worsened and 24 subjects improved ($p < .05$). From baseline to follow-up, 33 subjects' scores changed: 8 subjects worsened, and 25 improved ($p < .01$) (Figures 8-10).

On the MDQ negative affect symptom cluster measure, the sign test effects were weak at best. Weak, throughout this discussion, refers to significance levels greater than $p > .05$. From baseline to posttreatment, 34 subjects' scores changed: 11 subjects worsened, and 23 improved ($p < .10$). From baseline to follow-up, 33 subjects' scores changed: 13 subjects worsened, and 20 improved ($p > .25$) (Figures 11-13).

On the MDQ behavioral change symptom cluster, the sign test effects were also relatively weak. From baseline to posttreatment, 35 subjects' scores changed: 13 subjects worsened, and 22 improved ($p < .25$). From baseline to follow-up, 32 subjects' scores changed: 9 worsened, and 23 improved ($p < .05$) (Figures 14-16).

The daily behavior change rating showed effects on the sign tests consistent with the ANOVAs. From baseline to posttreatment, 36 subjects' scores changed: 11 worsened, and 25 improved ($p < .05$). From baseline to follow-up, 36 subjects' scores changed: 11 worsened, and 25 improved

($p < .05$) (Figures 17-19). The sign tests on the measures which did not show the significant change over time on the ANOVAs (the daily depression measure, crying frequency, and time resting) were consistent with those analyses; none of the sign tests showed a significant effect (Table 17).

Individual Treatment Group Sign Tests

Nonparametric sign tests were performed on the number of subjects in each treatment group (depression treatment, behavior change treatment, and control) who changed from baseline to posttreatment and from baseline to follow-up for each dependent measure (Table 17). Only two dependent measures showed significant effects on the sign test in individual treatment groups; these are the MSQ overall measure of PMS (Figures 8-10) and the daily behavior change rating (Figures 17-19). Both showed significant effects at posttreatment for the behavior change treatment group and significant effects at follow-up for the depression treatment group; for all four effects, 12 subjects' scores changed: two worsened, and 10 improved ($p < .05$).

Weaker effects were evident in four dependent measures: the MSQ overall measure, the MDQ negative affect symptom cluster, the MDQ behavioral change symptom cluster, and the daily depression rating. In addition to the significant effects on the MSQ overall measure, weaker effects were revealed at posttreatment for the depression treatment group

and at follow-up for the behavior change treatment group; for both, three subjects worsened, and nine improved ($p < .25$). The MDQ negative affect cluster and the daily depression rating cluster show the same pattern for the weak effect as demonstrated for the significant sign test effects. The effect was for the behavior change treatment group at posttreatment and for the depression treatment group at follow-up; in each case, three subjects worsened ($p < .25$). The same pattern of effects was evident on the MDQ behavioral change symptom cluster with the addition of an effect for the behavior change treatment group at follow-up, in which only nine subjects' scores changed, with one worsening ($p < .25$).

Thus, the depression treatment group showed significant or weak effects on five dependent measures at follow-up and the behavior change treatment group showed significant or weak effects on five dependent measures at posttreatment. In contrast, the control group showed essentially equal numbers of subjects' scores worsening and improving on all measures at posttreatment and follow-up ($p > .25$) (Table 17).

CHAPTER IV

DISCUSSION

The main goal of the present study was to test the hypothesis that matching specific treatments to the symptom clusters of depression and behavior change within premenstrual syndrome (PMS) would enhance the effectiveness of treatment. This is the treatment validity hypothesis that targeting specific symptom clusters within a disorder will improve treatment effectiveness. Thus, this study assessed the treatment validity of identifying depression and behavior change symptoms within the general classification of PMS. The prediction regarding this issue was that symptoms matched to the specific treatment procedure used would change. A modified version of Beck's cognitive therapy for depression was expected to decrease scores on the three measures of depression: (a) ratings of symptoms included on the negative affect symptom cluster of the Menstrual Distress Questionnaire (MDQ), (b) premenstrual daily ratings of depression, and (c) reported premenstrual frequency of crying. A modified version of Fordyce's operant intervention for control of pain behavior was expected to decrease scores on the three measures of behavior change: (a) ratings of symptoms included on the

MDQ behavioral change symptom cluster, (b) premenstrual daily ratings of behavior change, and (c) reported premenstrual time resting. The results of the multivariate and univariate analyses did not support this prediction.

This study also addressed the question: does targeting a specific symptom (depression or behavior change) have more impact on changing PMS as a whole, as measured by the Menstrual Symptom Questionnaire (MSQ)? In other words, in comparison to a no-treatment control group, on a general measure of premenstrual symptoms, is it more effective to target depression symptoms of PMS or behavior change symptoms? The prediction regarding this question was that one treatment (Beck's cognitive therapy of depression or Fordyce's operant intervention for behavior control) and its associated target symptom (depression or behavior change) would be more effective than both targeting the other symptom and the control procedure, in reducing the scores on the MSQ overall measure of PMS. The univariate analysis of the MSQ overall measure revealed no such differential treatment effect.

The results of the analyses pertaining to both questions are discussed below. The implications of the various analyses for the treatment validity hypothesis are addressed, followed by a discussion of the overall treatment effects on PMS. Subsequently, the limitations of the

present study are summarized, directions for future research are suggested, and conclusions are offered.

Treatment Validity Hypothesis

The treatment validity hypothesis in this study was that identifying the specific target behaviors of depression and behavior change and matching the the cognitive therapy of depression and operant intervention for behavior control to them, respectively, would enhance treatment outcome. This hypothesis was tested using two MANOVA's, one on the three depression measures and one on the three behavior change measures. Differential effects for treatment group by time period were expected in support of the hypothesis. The outcome of these analyses are reviewed below, followed by a discussion of the potential impact of the choice of treatment and the choice of symptom cluster on the findings.

Outcome

The treatment validity hypothesis predicted that the MANOVA's would show significant effects for the treatment group by time period interactions. Specifically, the MANOVA on the three depression measures was expected to reveal significantly greater improvement by the depression treatment group. The depression treatment group was expected to improve over time relative to the no-treatment control group and the behavior change treatment group. There was no significant interaction effect. Thus, there

was no evidence of the treatment validity of targeting the depression symptom cluster within PMS and matching a cognitive therapy of depression to it. Similarly, the MANOVA on the three behavior change measures was expected to reveal significantly greater improvement by the behavior change treatment group. The behavior change treatment group was expected to improve over time relative to the no-treatment control group and the depression treatment group. There was no significant interaction effect. Thus, there was no evidence of the treatment validity of targeting the behavior change symptom cluster within PMS and matching an operant intervention for behavior control to it.

The nonparametric analyses of the number of subjects in each individual treatment group who changed from baseline to posttreatment and from baseline to follow-up may suggest a different conclusion. There were differential significant effects by the treatment groups on two of the dependent measures (the MSQ and the daily behavior change rating). At posttreatment, the behavior change treatment group shows significantly more subjects improved than worsened; the depression treatment group and the control group do not show this significant effect. At follow-up, however, the depression treatment group shows significantly more subjects improved than worsened; the behavior change treatment group and the control group do not show this significant effect.

These differential effects do not show a clear relationship between specific treatment procedures and specific symptom clusters, but do suggest differential effects of the two interventions. These differential effects seem to be related to how quickly or how long the intervention will be effective. It appears that while the behavior change intervention results in significantly more subjects improving at posttreatment, this improvement does not last through follow-up; and it appears that while the depression treatment does not result in significantly more subjects improving at posttreatment, it does at follow-up. These are weaker findings than the parametric statistics offer, but they do suggest differential effects of the two interventions on two of the dependent measures. These effects, however, do not seem to differentiate in terms of the specific symptom clusters. It appears, therefore, that there is little empirical support for the experimental hypothesis that the treatments would differentially effect the symptom clusters to which they were matched.

There are three potentially confounding factors which may have bearing on the nonsignificant findings. The experimental hypothesis states that the identification of a specific symptom cluster and matching a treatment to it will enhance treatment outcome. The factors which may result in nonsignificant findings are the treatment procedures chosen,

the target behaviors chosen, and the match between the treatments and the targets. The assessment of the appropriateness of the match requires that the appropriateness of the treatments and targets be previously established. Discussions of the choice of treatment and the choice of the cluster, in the present study, follow.

Choice of Treatment

The effectiveness of the chosen treatment procedures with premenstrual symptoms has not been directly demonstrated. The modified form of Fordyce's (1976) operant intervention for pain behavior control was assumed to be effective with PMS symptoms based upon research with other physical disorders that limited activity. Similarly, the modified form of Beck and his colleagues' (1979) cognitive therapy of depression was assumed to be effective with PMS symptoms based upon research with depression in general. It may be that these assumptions were incorrect, and that the treatment procedures were ineffective with premenstrual symptoms. The problem, then, may lie with the specific treatments selected, rather than in the match between specific symptom clusters and specific treatments. Targeting the specific symptom clusters with treatments that have been demonstrated to be effective for premenstrual symptoms of behavior change or depression may well enhance treatment outcome.

Treatment validity is considered to be "based upon a nexus of assessment devices, theoretical distinctions, and treatment approaches" (Hayes et al., in press, page 19). Each of these three aspects is necessary in order to demonstrate treatment validity. The absence or incorrectness of any one aspect prevents treatment validity from being established. It is, therefore, inherent in the treatment validity methodologies that the treatment procedures used be effective with the disorder in question. Correct assessment and effective treatment are both required in order to address the question of the match between the two. Treatment validity demonstrations are always dependent upon the effectiveness of the treatments.

It cannot be said based upon the results of the parametric statistical analyses that the treatments were any more effective than the control procedure. There were significant improvements across time on four of the seven dependent measures; there was no significant difference among the three treatment groups: depression treatment, behavior change treatment, and control procedure.

Nonparametric analyses of the number of individuals whose scores changed from baseline to posttreatment and from baseline to follow-up, however, suggest that there may indeed have been a greater effect by the experimental treatment procedures than the control procedure. A number

of the dependent measures showed significantly more treated subjects (at various levels of significance) improved than worsened in the depression and behavior change treatment groups. The control group subjects, however, showed no significant differences between the number who improved and the number who worsened. These data do suggest greater effectiveness (more subjects improved) in the experimental treatment groups than in the control group. Unfortunately, these differences appear to have been too weak to result in demonstrable significance on parametric statistical analyses. The issue of power is discussed later. In light of the relative weakness of these findings, ineffectiveness of the treatment procedures still warrants discussion.

If the treatment procedures were ineffective (that is, no more effective than a control procedure) with premenstrual symptoms, reasons for their ineffectiveness merit discussion. The extension of the Fordyce (1976) operant intervention, usually used to control pain behavior, to the inactivity associated with premenstrual complaints appears logically consistent, as does the extension of the Beck et al. (1979) cognitive therapy for depression to the negative affective changes associated with premenstrual complaints. It is clear, however, that logic does not guarantee effectiveness. The effectiveness of a treatment procedure is a matter for experimental demonstration. The

present study may provide one instance of the experimental ineffectiveness of the treatment procedures for PMS symptoms, despite the nonparametric findings.

In addition, there are points of logic which may be argued. The effectiveness of Fordyce-like procedures has been primarily demonstrated with chronic pain patients (Anderson et al., 1977; Cairns et al., 1976; Fordyce et al., 1973). While the decreased activity level reported by women who complain of PMS appears to be the same as the inactivity reported in chronic pain patients, it may well be different in a variety of ways essential to treatment effectiveness. Similarly, the effectiveness of Beck-like procedures has primarily been demonstrated with pervasive, chronic, and endogenous depression (Beck et al., 1979). While the depression reported by women with PMS appears to be the same as the depression reported by individuals suffering with more pervasive depression, it may well be different in a variety of ways essential to treatment effectiveness.

An additional issue pertaining to treatment effectiveness warrants attention. In order for a treatment procedure to be effective, it must be implemented. When the procedure is patient applied, the compliance and cooperation of the patient are required. If the women in the present study did not implement the procedures, they had little chance of being effective. In the present study, it is

likely that the subjects were implementing the procedures during their posttreatment premenstrual days as the post-treatment premenstruum usually occurred within one week of the completion of the treatment program. During the treatment program (four visits within two weeks), subjects in the behavior change treatment group completed practice activity schedules, and subjects in the depression treatment group used homework forms for daily practice of the procedure. These practice sheets were reviewed by the therapist during the treatment sessions. It is, of course, possible that the subjects lied regarding practice, but it would have required more effort in filling out the homework sheets.

There is less certainty that subjects actually implemented the treatment procedures for the follow-up premenstrual days; subjects were merely instructed to continue daily practice of their procedure throughout the remainder of their participation in the study. There is, in fact, anecdotal evidence suggesting that subjects in the behavior change treatment group were not applying the treatment technique during the follow-up premenstruum. A number of women in the behavior change treatment group voluntarily reported noncompliance to the author. At the final data collection visit, they admitted that during their most recent premenstruum they had not implemented the

procedure they had been taught. This would essentially result in a return to baseline for an effective treatment technique. In comparison, none of the women in depression treatment group made such reports. There was, however, no parametric statistical evidence of such a return to baseline distinguishing the behavior change treatment group from the other groups. The nonparametric sign tests performed on the individual treatment group data, however, show a pattern of results which may support this return to baseline evidence of noncompliance. The two dependent measures showing significant effects on the sign tests (the MSQ and the daily behavior change rating) both showed a significant effect at posttreatment for the behavior change treatment group, but no significant effect at follow-up. This may be interpreted as a return to baseline.

The variety of factors which may play into noncompliance (divulged or not) were not elucidated by the present study, but certainly merit further investigation. Methodologically, it would be better to ensure through some form of check on homework compliance that the treatment procedures were indeed implemented during the posttreatment and follow-up premenstruum.

The behavior change treatment group showed improvement at posttreatment and not follow-up on the nonparametric analyses. It appears that the lack of effectiveness of the

behavior change treatment may be accounted for by noncompliance. The pattern of results on the nonparametric sign tests for the depression treatment group show the reverse of the behavior change treatment group results. The depression treatment group data show significant improvement at follow-up, and not at post-treatment. This may be accounted for by the amount of practice required for effective application of the procedure. It may take time (more than one menstrual cycle) for subjects to learn how to effectively apply the cognitive therapy for depression. The length of practice and assessment may influence the apparent effectiveness of the depression treatment procedure. An investigation of the amount of practice necessary to effectively utilize the procedure would elucidate this suggested interpretation of these findings.

One alternative explanation of the ineffectiveness of previously effective treatment procedures concerns the timing of administration of the treatment procedures. Both treatments in the present study were administered within two weeks following the onset of each subject's baseline menses. This is technically considered an asymptomatic time period (Dalton, 1979). Therefore, treatment was administered during a symptom-free time period, and depended upon generalization of the intervention techniques to a symptomatic time period. It could be argued that it is

ineffective to teach a skill designed to cope with symptoms during an asymptomatic time period.

Other effective treatment procedures, however, also teach skills during asymptomatic time periods for application when symptoms arise. For example, systematic desensitization is taught and practiced before the phobic scene or object is actually encountered. In systematic desensitization, the encounter with phobic scene is rehearsed imaginally; similarly, application of the coping skills taught in the present study were practiced while imagining that it was the premenstruum (Appendices H and I). Relaxation training provides another example of training of a coping technique prior to actual experience of a symptom. Relaxation exercises are taught prior to facing a painful or stressful experience in order to prepare for it. Similarly, Lamaze breathing exercises for childbirth are taught and practiced prior to delivery in order to prepare the woman with the skills to handle that situation. The intervention techniques taught in the present study were also taught prior to the premenstrual symptoms in order to prepare for them.

In addition, with all these interventions it would seem that training might be less effective if it were to occur initially when the symptoms were in full force. The theoretical underpinnings of systematic desensitization as

well as the clinical experience with phobic responses suggest that training in the face of the phobic stimulus will not be particularly effective. Certainly, the introduction to Lamaze breathing exercises during labor is less effective than having learned the skills prior to that time. With PMS, the decreased activity level and depression experienced during the premenstruum are antithetical to the skills necessary for acquisition of a new coping response (Cassara, 1984).

It may be that practice in applying the techniques in response to the symptoms in question may enhance their effectiveness, but the initial training appears to require training prior to the symptomatic time period. The relative merits of training during symptom flare-ups versus prior to them could certainly be empirically investigated. Despite the evidence generalized from other treatments and other disorders, it may be necessary with PMS to learn and/or practice the application of treatment procedures during the experience of symptoms.

Choice of Cluster

In addition to the choice of the treatments that may have been no more effective than the no-treatment control, the choice of the specific cluster may have contributed to nonsignificant findings. It is possible that the specific clusters identified, depression and behavior change, are

nonessential in the treatment of PMS. The symptom clusters targeted in the present study were chosen for experimentally pragmatic reasons: (a) consistency with previously identified clusters in PMS, and (b) potential responsiveness to noninvasive psychological interventions.

Guidelines for selecting from among alternative socially acceptable target behaviors have been proposed and summarized by Nelson and her colleagues (Nelson & Barlow, 1981; Nelson & Hayes, 1979). These guidelines are, by no means, rigid rules; they do, however, offer a point of departure for subsequent empirical demonstrations of the treatment validity of target behavior selection. Some of these guidelines may have been violated by the selection of the specific symptom clusters for targeting within the present study. While the present study provides no empirical evidence for such violations, they may have bearing on nonsignificant or nonsupportive findings for the treatment validity hypothesis.

The first guideline recommends targeting the most irritating symptom first (Tharp & Wetzel, 1969); it is likely that the behavior change symptoms were not the most irritating for the women in the present study. Targeting the behavioral change symptom cluster may have violated this guideline. All of the participants reported both negative affect and behavior change symptoms of at least

mild severity. In addition, only eight of the 36 subjects reported that behavior change symptoms were more bothersome than negative affect symptoms, based upon percentage of the total possible symptom severity score on each symptom cluster (Table 18). Each subject's screening assessment scores on the MDQ negative affect and behavioral change symptom clusters were calculated as percentages of the total possible score on each cluster (negative affect: 48, and behavioral change: 30). These percentages make up Table 18. Most subjects have higher negative affect percentage scores than behavioral change percentage scores.

Another guideline which may have been violated suggests targeting responses at the beginning of a response chain (Angle, Hay, Hay, & Ellinwood, 1977); it is possible that either the negative affect or behavior change symptoms endorsed by women complaining of PMS may be part of longer response chains. If there are other behaviors which precede symptoms in a response chain, targeting the first behaviors is assumed to be more effective than targeting the subsequent symptoms.

Two other guidelines were summarized by Nelson and her colleagues (Nelson & Barlow, 1981; Nelson & Hayes, 1979) which could also be empirically investigated prior to target behavior selection. One is targeting behaviors which will result in beneficial response generalization (Hay et al.,

1977). This is done in order to maximize the effect of the intervention procedure, thereby reducing the overall amount of treatment time and enhancing the therapeutic gains. The other guideline suggests targeting a behavior which is easy to change first (O'Leary, 1972) in order to lay a groundwork of successful change upon which to build further changes.

The use of such guidelines is assumed to enhance treatment effectiveness. This is clearly an empirical question for each specific disorder and each choice of target behavior. Attention to these guidelines prior to empirical investigations of the relationship between the assessment of target behaviors and treatment outcome, however, may be helpful in confirming or disconfirming a particular treatment validity hypothesis.

The particular choice of treatment procedures and symptom clusters makes it difficult to reject the tested treatment validity hypothesis. Further investigation is needed to clarify the role of these two factors in the treatment validity of targeting behavior change symptoms in PMS. The results of the present study suggest overall that treatment outcome may not be enhanced by targeting negative affect or behavior change symptoms and matching them to the specific treatment procedures used.

Overall Treatment Effects on PMS

Two general classes of findings pertain to the overall treatment effects on PMS: (a) the differences between the number of subjects who improved as compared to the number who worsened within each treatment group, and (b) the differences between the entire groups.

The nonparametric sign tests on the individual treatment groups provide analyses of the relative improvement of subjects within treatment groups. These analyses demonstrate that significantly more subjects improved than worsened in both treatment groups on at least two dependent measures. There was no significant difference between the number of subjects in the control group who improved and the number who worsened. This suggests that while subjects in the control may have improved, the change was random compared to subjects in the treatment groups of whom significantly more improved than worsened. This is an interesting finding and it does suggest that the interventions warrant further investigation with PMS symptoms.

The more conservative approach would be to examine and focus on the differences between the groups, rather than the differences within the groups. The parametric analyses addressed the relationship between the groups and revealed no significant differences between the groups. On the average, all three appear to have improved over time.

From this conservative perspective, the discussion of overall treatment effects on PMS may address three general concerns: (a) why there was change across time, (b) why there was no statistical difference between the experimental treatments and the control group, and (c) why there was change on only four of the seven dependent measures. These concerns are discussed following a summary of the significant main effects for time.

Significant Main Effects for Time

There were significant main effects for time on four dependent variables: the MDQ negative affect symptom cluster, the MDQ behavioral change symptom cluster, the daily behavior change rating, and the MSQ overall measure. The significant effect for time simply reflects a decrease in symptom severity over time regardless of treatment. The experimental treatment procedures were not statistically more effective than the record keeping of the control group.

Change over Time

The improvements over time may reflect the most consistent finding demonstrated in other studies of the treatment of PMS: improvement attributable to a placebo effect. A high placebo response in the treatment of PMS has been reported in controlled double blind trials (Clare, 1979). Simple acknowledgement of the existence of menstrually related behavior and mood changes has been

reported to be essential in the effective management of PMS (Cassara, 1984). Simply seeking help through participation in a research project, attending systematically to the symptoms, and having one's concerns professionally heard may be the "placebo" causing the decline of symptom severity and complaints over time.

Classically, placebos have been considered inert pharmacological products; this restrictive definition has been extended, however, to include nonspecific treatment effects in various forms of psychotherapy (Critelli & Neumann, 1984; Shapiro & Morris, 1978). Explanation of "placebo" responses may be given at various levels: cognitive, behavioral, and physiological.

One common cognitive explanation of placebo responding is expectancy. A number of different specific explanations for the expectancy mechanism in placebogenesis have been proffered (Shapiro & Morris, 1978). Among them are cognitive dissonance and feelings of control.

The theory of cognitive dissonance (Festinger, 1957), suggests that a generalized drive state is established when two beliefs are dissonant (logical opposites). This drive state will lead the individual to alter the weaker belief in order to achieve a state of cognitive consonance. In the case of placebogenesis, dissonance will result if the belief that the therapy will be effective is confronted with the

experience of no improvement. If the belief in therapeutic effectiveness is strong, the belief regarding lack of improvement will be changed in order to achieve consonance. A dissonance-induced placebo effect may have occurred in the present study. The belief that participation would help PMS symptoms when confronted with lack of improvement may have resulted in a state of cognitive dissonance. If beliefs that could restore consonance (e.g., the treatment is ineffective, the researcher is wrong) were not acceptable in the subject's value system, then the belief regarding improvement could have changed in order to achieve consonance.

Feelings of control may also serve as an expectancy mechanism of placebogenesis. The expectation that therapy will be effective may give people a feeling of control over their lives (Gatchel, 1980). Such a sense of control may reduce perceived discomfort (Melzack & Wall, 1982). A common complaint among women reporting PMS symptoms is that they feel out of control (Dalton, 1969). Entering into the present research project may have offered the expectancy of a means of control to the subjects, and thus the experience of symptom improvement.

Behavioral explanations of placebogenesis have also been offered (Shapiro & Morris, 1978). Classical and operant conditioning both offer viable explanations of improvement in response to nonspecific effects.

In a classical conditioning model, a neutral stimulus may come to serve as a conditioned stimulus for the response of improvement due to previous temporal association with unconditioned stimuli that produced improvement. Classical conditioning accounts have been used for physiological reactions to drugs occurring faster than they can be pharmacologically induced (Shapiro & Morris, 1978; Stanley & Schrosberg, 1953). In the present study, subjects may have experienced improvement during their previous experiences with psychological professionals, in research projects, or at universities.

Intentionally or not, operant conditioning principles may control the responses of experimental subjects and patients (Frank, 1968; Shapiro & Morris, 1978). Reinforcement has been implicated in independently and incrementally increasing placebo effects (Buckalew, 1972). Subjects in the present study may have been differentially reinforced for improvement by subtle experimenter cues. The situation of the study (i.e., a therapeutic setting) also may have served as a discriminative stimulus for positive regard or other reward for symptom improvement.

A physiological mediator in placebo responses has also been suggested. A system of endogenous opiates, including the analgesia-inducing beta-endorphin sequence of the beta-lipotrophin amino acid chain, may account for

placebo analgesia (Houston, Bee, & Rimm, 1985). Levine, Gordon, and Fields (1978) demonstrated, in patients displaying a placebo effect, an increase in reported pain due to injection of an opiate receptor-blocking drug. Thus, mediation of the placebo effect by an endogenous opiate system was inferred. Such a mechanism may be generalized to a psychotherapy placebo effect. Decreased ratings of pain or discomfort may be the result of increased production of endogenous opiates in response to the experimental or therapeutic situation. Increased beta-endorphin levels in response to social settings have been reported, albeit in animal research (Houston et al., 1985). By virtue of participation in the present study and accompanying cognitive or behavioral events, subjects may have produced greater quantities of endogenous opiates and, thus, reported the experience of improvement.

One additional influence on placebo effects deserves mention: it is the impact of evaluation (Shapiro & Morris, 1978). The assessment procedure, per se, may cause reactive changes in the participants. Simple pretesting has been found to sensitize respondents and result in alteration of responses (Haase & Ivey, 1970; Mungus & Walters, 1979). Experimental designs have been created to account for such effects; the main purpose of the Solomon four-group design is to evaluate the effect of pretesting on the impact of a

specific treatment (Solomon, 1949). This has been called an "ideal design for social scientists" (Campbell, 1957, p. 303) suggesting the importance of attending to the impact routine measures may have on what they assess. The simple use of pretests and repeated measures may have altered subjects' responding.

In the present study, it is possible that the daily self recording resulted in a decrease in reported symptom severity. Self-recording has been shown to be reactive, with desirable behaviors increasing in frequency, and undesirable behaviors decreasing in frequency (Nelson, 1977). It is believed that reactivity is due to either positive or negative self-evaluation (Kanfer, 1974) or to positive or negative naturally occurring environmental consequences (Rachlin, 1974).

Lack of Differential Treatment Effects.

There were no significant group by time effects demonstrated by the statistical analyses in the present study. This lack of treatment procedure effectiveness compared to a no-treatment control raises, again, the issues of placebo factors and treatment effectiveness.

The "control group" in the present study has been referred to as a "no-treatment" group. In actuality, this control group may be considered a "placebo" control group. An effect sufficient to result in significant changes across

time occurred in the control group to the extent that it could not be distinguished from the "treatment" groups. Some common events may have been causing the common effects.

In order to consider the control group a placebo control group, a definition of placebo within psychotherapy research must be established. A number of different positions exist regarding the appropriate definition of placebo within psychotherapy research. These definitions range from treatments considered theoretically inert from the perspective being tested (Rosenthal & Frank, 1956), to treatments without theoretical support in general (O'Leary & Borkovec, 1978), to the view that all treatments may be theoretically explained via Bandura's (1977) self-efficacy theory. Critelli and Neumann (1984) provide a review of these and alternative positions which define placebo based upon the concept of nonspecific treatment effects. They conclude that the concept of common factors offers the most useful definition of placebo for psychotherapy research. Common factors means those factors that are common to most forms of therapy (Kazdin, 1979; Wilkens, 1979).

In the present study, this common-factors definition of placebo offers a workable explanation of the apparent lack of differential treatment effects. It may be that improvement over time in all groups resulted from those factors which are common to all three groups (e.g., data collection, expectancy, experimenter contact).

Treatment effectiveness is another issue raised by the apparent lack of differential treatment effects. The treatment procedures were not statistically more effective than the control procedure. The reasons for this apparent lack of treatment effectiveness are discussed previously and include possible lack of compliance and inappropriateness of the procedures for PMS-related symptoms. Most simply, however, the present study may provide one instance of the experimental ineffectiveness of the treatment procedures with PMS symptoms.

Differential Responsiveness of Measures.

Only four of the seven dependent variables showed any significant effect on the parametric analyses. These four measures were the MSQ overall measure, both MDQ symptom clusters (negative affect and behavioral change), and the daily behavior change rating. They showed significant improvement over time regardless of intervention. The three measures which showed no significant effects were the daily depression rating and the two "objective" daily measures (reported crying frequency and reported time resting). In the face of consistent effects among the three treatment groups (the two treatments and the control procedure), the inconsistency in responsiveness of the dependent measures merits discussion.

Only two of the seven dependent variables showed a significant effect for time on the nonparametric analyses. These two measures were the MSQ overall measure and the daily behavior change rating. The two MDQ symptom clusters (negative affect and behavioral change) showed weaker effects (Table 17). There were no significant effects on the daily depression rating and the two "objective" daily measures (reported crying frequency and reported time resting). These results are consistent with those of the parametric analyses. Thus, the nonparametric analyses regarding change over time are consistent with the more conservative parametric analyses. Therefore, the parametric analyses will be specifically addressed in the discussion of the differential responsiveness of the dependent measures.

Two general issues are apparent in evaluating the differential responsiveness of measures: the validity of the MSQ as an overall measure of PMS, and the relationship between prospective and retrospective measures. In addition to the general topic of prospective versus retrospective measures are the specific problems of inconsistency of the measures within and between symptom clusters.

Validity of the MSQ as an Overall Measure of PMS. The MSQ was devised as a measure of dysmenorrhea. It was designed to distinguish between spasmodic and congestive

dysmenorrhea (Chesney & Tasto, 1975a). While this distinction has been questioned (Cox, 1977; Stephenson et al., 1983; Webster et al., 1979), the use of the congestive measure on the MSQ as an overall measure of PMS in the present study seemed appropriate. In the original distinction, spasmodic dysmenorrhea referred to menstrual cramping and discomfort accompanying menses; congestive dysmenorrhea referred to bloating and discomfort preceding the onset of menses (Dalton, 1969; 1979). Congestive dysmenorrhea, therefore, had been likened to PMS.

If the MSQ is a valid overall measure of PMS, conceptually there should be a relationship among the results of the MSQ analyses and the results of the analyses of the other measures of PMS. If the MSQ is an overall measure of PMS and the specific symptom clusters targeted account for a large proportion of the variance within PMS, then it would be expected that the results of treatment on the three measures would be similar. The results on the MSQ and on the MDQ specific symptom clusters all showed a significant effect for time and no differential effect by treatment group. In addition, both MDQ symptom clusters were significantly correlated with the MSQ overall measure, although each accounted for less than 20 percent of the variance. This similarity and correlation among these measures suggest that the MSQ does provide an overall

measure of PMS and reflects the targeted symptom clusters, at least, retrospectively.

The retrospective measures, the MSQ and the MDQ measures, appear to be consistent among themselves. The differential responsiveness of measures, therefore, may be due to general differences between prospective (daily ratings) and retrospective (MSQ and MDQ measures) measures.

Prospective Versus Retrospective Ratings. The relationship of prospective and retrospective ratings for PMS, in general, is discussed. Possible explanations for the inconsistency of the measures within and between symptom clusters also are discussed. Because the inconsistencies among the measures are different for the two symptom clusters they are discussed separately. Within this context, issues of definition, interpretation, and differences in the time periods assessed are addressed.

The only prospective dependent measure which showed the significant effect for time was the daily behavior change rating. The other three prospective measures (daily depression rating, crying frequency, and time resting) showed no significant effects. The other three measures which showed the significant effect for time (the MSQ overall measure, and both MDQ symptom cluster measures) were all retrospective measures. The method of measurement may account for the differential responsiveness of the measures.

The inconsistent findings between most of the prospective daily ratings and the retrospective measures may reflect the general inconsistency between prospective and retrospective measures of premenstrual symptoms. Woods and her colleagues (1982) suggest that report of perimenstrual symptoms is influenced by the method of data collection. They compared a prospective daily health diary with a retrospective MDQ for 73 women. Their analyses revealed that the women were much more likely to report negative affect symptoms and water retention symptoms on the retrospective device. Among the symptoms these researchers found reported on the MDQ, but never on the daily health diary, were crying and avoiding social activities. The items crying and avoiding social activities are included in the symptom clusters targeted in the present study. Crying was also the objective daily measure of the depression cluster and avoiding social activities may be related to the objective daily measure of behavior change, time resting. It is possible that even when specifically solicited in daily ratings these behaviors are not of sufficient frequency or importance to warrant notice or reflect change.

One problem in comparing the findings of Woods and her colleagues (1982) with those of the present study is the nature of the prospective ratings. In the Woods study, blind prospective ratings were used, whereas in the present

study the subjects were aware that premenstrual symptoms were of interest. The discrepancy between prospective and retrospective ratings, however, still seems relevant. Others have also reported that retrospective self-report measurement devices maximize the reporting of negative moods and minimize the reporting of positive moods during the premenstrual time period (Englander-Golden, Whitmore, & Dienstbier, 1978). Even when using the same questionnaire prospectively and retrospectively, differences between the ratings on various symptom clusters have been reported (Rouse, 1978). Differences attributable to prospective versus retrospective methodology may well have had an impact on the results of the present study.

Differential Responsiveness of Depression Measures.

If taken at face value, it certainly seems reasonable to assume that daily ratings of depression and crying frequency would be related to retrospective ratings of depression. In fact, the three measures are significantly correlated. In terms of content, both the objective measure, crying frequency, and the subjective daily depression rating were items included in the retrospective MDQ negative affect symptom cluster (item numbers 3. crying and 40. depression). The similarity of the items on the prospective and retrospective measures may account for the intercorrelation among the three depression measures. While all three

depression measures were significantly correlated, only the retrospective MDQ negative affect symptom cluster measure showed the significant change across time. This discrepancy may be due to the prospective-retrospective measurement distinction (Woods et al., 1982). As previously mentioned, the daily ratings of these behaviors may not be of sufficient frequency or strength to reflect change.

Differential Responsiveness of Behavior Change Measures. It seems reasonable to assume that daily ratings of behavior change and time resting would be related to retrospective ratings of behavior change. On one hand, consistent with the prospective-retrospective distinction, only the two daily measures of behavior change were significantly correlated. On the other hand, both the MDQ behavioral change symptom cluster measure (retrospective) and the daily behavior change measure (prospective) showed the significant change over time.

The time resting measure showed no significant effects. It was the only measure of the behavior change cluster which did not change significantly over time. The time resting measure was significantly correlated with the other prospective measure of the behavior change cluster: the daily behavior change measure (accounting for ten percent of the variance). This similarity among the prospective measures and concomitant dissimilarity to the retrospective

measures suggests some support for a prospective-retrospective distinction. It seems most parsimonious, however, to ascribe the lack of significant change over time on the time resting measure to insufficient strength or importance to reflect change, an explanation consonant with that applied to the daily depression ratings.

The lack of significant correlation between the daily behavior change rating and the retrospective MDQ behavioral change symptom cluster measure is complicated by the concomitant significant change over time on both measures. Three possible differences between these measures are described. The first is concerned with time, the second with interpretation, and the third with definition.

Time may account for differences between the daily and retrospective measures. On the retrospective questionnaire, the subjects were instructed to rate the symptoms for the week prior to their most recent menstrual period. The prospective daily ratings were analyzed only for the four days immediately preceding the onset of the subjects' menstrual periods. This discrepancy could have resulted in inconsistent findings. Severe behavior changes may have occurred five or six days prior to menstrual onset resulting in a retrospective rating of severe lowered school or work performance. As only the four days preceding the onset of menses were included in the daily rating score, the

prospective rating may have been only mild. Thus, time differences may be an artifactual cause of differences between the daily and retrospective behavior change measures.

Interpretation of the measures also may account for differences between the daily and retrospective measures. Interpretations may vary between the prospective and retrospective measures. They may also vary between subjects; one woman's severe behavior change may be only mild to another, and one's moderate decreased efficiency may be debilitating to another. The repeated measures design hopefully helped to account for such individual differences. Other inconsistencies in the interpretation of the rating scales may not be accounted for by design. On one measurement occasion, a woman may have retrospectively reported "avoids social activities" as severe in response to only mild prospectively reported behavior change. On another measurement occasion, the same woman may have retrospectively reported "avoids social activities" as mild in response to severe prospectively reported behavior changes. The former time period may have been the occasion of avoiding only one, but an important social activity (a business lunch), while the latter time period may have included avoiding multiple social gatherings, none of which were recalled as a great loss. Similarly, one day of

prospectively rated severe behavior change could be retrospectively rated as moderate decreased efficiency on one occasion while five days of prospectively rated mild behavior change could be retrospectively rated as moderate decreased efficiency on another occasion. These types of inconsistency in the interpretation of the rating scales were not accounted for in the present study and could play a part in the lack of correlation between the daily and retrospective behavior change measures.

Similar to interpretation, differences in definition may account for the differences between the daily and retrospective behavior change measures. Unlike the depression symptom cluster, the items which make up the daily behavior change ratings actually may not be part of the identified MDQ behavioral change symptom cluster. The identified cluster actually included specific types of behavior change, as opposed to a global rating of how different one's behavior appeared compared to "normal." The retrospective MDQ cluster included lowered school or work performance, take naps and stay in bed, stay at home, avoid social activities, and decreased activity; it did not include a rating of behavior change, per se. It may well be that the global term "behavior change" reflects a different set of behaviors than are assessed by the retrospective MDQ behavioral change symptom cluster measure.

While there is no significant correlation between the daily behavior change rating and the MDQ behavioral change symptom cluster measure, there is a significant correlation between the daily behavior change rating and the MDQ negative affect symptom cluster measure. It appears that the daily behavior change rating accounts for 12 percent of the variance on the MDQ negative affect symptom cluster measure. This across cluster correlation is consistent with previously demonstrated significant correlations between depressed mood and a low frequency of pleasant events (Lewinsohn & Graf, 1973; Lewinsohn & Libet, 1972). This suggests that the daily rating of behavior change has a different definition than the MDQ behavioral change symptom cluster. Subjects may have defined the daily behavior change rating as decreases in pleasant events (relating to items on the MDQ negative affect cluster) rather than as a global rating of the specific behavior changes delineated by the items on the MDQ behavioral change symptom cluster. Different definitions of the measures may account for the lack of correlation between the prospective and retrospective behavior change measures.

The lack of correlation between the behavior change prospective and retrospective measures may be accounted for by differences in time, interpretation, and definition of the measures. The issue is, then, why only the daily

behavior change rating showed the significant change over time. The other measures, which were significantly correlated with the MDQ negative affect cluster, did not show the significant change over time. As previously discussed, the other prospective measures may not have been of sufficient importance or sensitivity to reflect the change, whereas the daily behavior change rating was interpreted consistently with the retrospective measures. The measures which did not show the significant change over time (daily depression rating, crying frequency, and time resting) may have been defined or interpreted as similar to comparable single items on the retrospective devices. Single items may be relatively weak or insensitive to change compared to the clusters. Similarly, the behavior change daily rating may have been interpreted or defined in a more global manner reflecting mood or affective changes in general. Measures which incorporate a number of specific items do appear sensitive to change (e.g., the MSQ overall measure, both MDQ symptom cluster measures). Differential strength of measures may account for their differential responsiveness.

One additional point concerning the "objective" daily measures (crying frequency and time resting) relates to this issue of strength. The baseline frequency of both of these measures was extremely low. They appear to be irrelevant

behaviors and are subject to floor effects. This decreases their utility as measures of change and may account for the differential responsiveness of, at least, these measures.

The overall treatment effects on PMS demonstrated in the present study do not support the experimental hypotheses. No differential effect on symptom clusters for treatment group by time period was revealed and no enhancement of outcome resulted from targeting a specific symptom cluster. The results demonstrate a significant change over time on four out of seven dependent measures regardless of specific intervention. These effects are interpreted as placebo effects or the effects of common therapeutic factors seen specifically on retrospective general-concept measures as opposed to prospective single-item measures. Because of the potential impact of other variables on the present study, the inability to rule out treatment ineffectiveness, and the differential (although relatively weak) effects demonstrated by the nonparametric analyses, it can only be stated that the present findings are inconclusive. The treatment procedures, in light of the nonparametric findings, certainly warrant further investigation, as do treatment validity hypotheses which have been neither confirmed nor belied. No definitive statement regarding the effectiveness of targeting depression or behavior change symptom clusters in enhancing treatment outcome has been made.

Limitations of the Present Study

Possible explanations and interpretations of the results of the present study were discussed previously. It remains important to address the limitations of the present design in answering the experimental questions proposed. Limitations within the rubric of treatment validity research as well as design issues in the study of premenstrual symptoms and in general are discussed.

The purpose of this study was to evaluate the treatment validity of identifying specific target behaviors within PMS. This treatment validity issue is concerned with the question: does selection of specific target behaviors and matching treatment to them enhance treatment outcome? One limitation of the present study may be that it does not offer direct evidence regarding the evaluation of behavioral assessment as is expected of treatment validity studies.

As previously discussed, selection of specific target behaviors is considered a manipulated-target type of study which is not considered to have direct bearing on the quality of assessment (Hayes et al., in press). The controversy in calling the present manipulated-target type of study a treatment validity study is that such studies do not offer information directly related to the evaluation of behavioral assessment. Such studies may be included as treatment validity studies, however, as they evaluate the

quality of our theories of disorders which may lead us to select specific target behaviors and have value in generating other treatment validity hypotheses (Hayes et al., in press). The role of the present study in evaluating the quality of a theory of PMS as a whole and in generating other treatment validity hypotheses is discussed.

A major limitation of the present study is that two unknowns were addressed simultaneously. In addressing two unknowns simultaneously the results are equivocal and, thus, the interpretation difficult. In addition to the treatment validity question (were the correct symptom clusters targeted?), the present study was also asking outcome questions. The outcome questions were also being asked as no previous work has been done addressing the effectiveness of the treatment procedures with premenstrual symptoms. For example, the lack of a differential treatment effect in the present study is difficult to interpret. Whether identifying the specific symptom clusters offers little to enhance treatment outcome or the treatment procedures were ineffective is unclear. Treatment validity questions may be answered clearly only if outcome questions pertaining to the effectiveness of specific treatments with specific disorders are answered first. When outcome questions have been answered, treatment validity or the impact of various aspects of assessment may be clearly demonstrated.

An implication of the equivocal findings in the present study is that it may be too early in our empirical knowledge of premenstrual disorders to use them to assess the impact of assessment on treatment outcome. So little is known of the disorder in general and its treatment in particular that the present attempt to evaluate the quality of behavioral assessment using the disorder of PMS is clearly premature. Treatment validity studies need to be done, but will provide useful information about the value of various aspects of behavioral assessment only with fairly well-known disorders with already established treatment procedures.

The present study rests upon the theory that PMS is a multisymptom disorder which may respond to symptom specific intervention procedures (Clare, 1979; Moos, 1969). Two psychologically relevant symptom clusters were chosen for targeting and logically matched to two intervention procedures. The consistency of findings among the retrospective measures and their intercorrelation suggest that the MDQ negative affect and behavioral change symptom clusters are related to the MSQ overall measure of congestive dysmenorrhea or PMS. This finding provides a connection between the specific type and timing of premenstrual symptoms. Thus, in a small way, this study provides support for the theory that PMS is a multisymptom disorder. It provides only limited support, however, for the

responsiveness of the symptom clusters to the selected treatment.

In terms of generating other treatment validity hypotheses for testing, the questions raised by the present study may be addressed from a treatment validity perspective. For example, a manipulated assessment type of treatment validity study could address the value of assessing specific symptoms of PMS at all, regardless of particular cluster. The placebo effect explanation that record keeping in general and a sympathetic professional ear are sufficient to result in decreased severity of symptoms suggests that assessing specific symptoms may not enhance outcome; all women complaining of PMS will respond to record keeping and serious attention. Women complaining of PMS could be randomly assigned to two groups. The single aspect of assessment to be varied in this example would be the formulation of specific treatment plans based upon specific symptoms endorsed. One group would see therapists who have access to an assessment device upon which they specify symptoms and would receive treatment based upon the assessment of those specific complaints, and the other group would see therapists who do not have access to the assessment device upon which the subjects specified their symptoms and would receive treatment which does not account for the specific complaints assessed. Differential outcomes

for the two groups would delineate the treatment validity of assessing specific symptoms within PMS. This is an empirical question within the rubric of treatment validity research. Additional treatment validity hypotheses generated by the present study are presented later (in Directions for Future Research).

Additional limitations of the present study include assumptions which in retrospect appear to be faulty. One faulty assumption relates to the equivocal findings of the present study and their relationship to the lack of effectiveness of the treatment procedures. The treatment procedures were chosen based upon the assumptions that premenstrual depression was the same as other depression and that premenstrual behavior change was the same as other illness-induced behavior change. The treatment procedures have been demonstrated to be effective with nonmenstrually related depression and behavior change. Their lack of effectiveness in the present study calls into question the assumption of equivalence between premenstrual and nonmenstrually related depression and behavior change.

A second faulty assumption relates to factors derived in the pilot study which were statistically established as orthogonal. The choice of negative affect and behavioral change clusters was based in part upon their orthogonal nature. This orthogonality, however, was statistically

established by a factor analysis varimax rotation. It was not a reflection of "real" orthogonality or actually independent symptom clusters. Moos' (1968; 1969) original reports of these factors suggests that they are, in fact, highly correlated particularly within subjects. Thus, it appears that any subject experiencing both negative affect and behavioral change symptoms does not experience them as independent symptoms, but experiences them as related symptoms unlikely to respond differentially. This is in direct contradiction to the experimental hypothesis which assumes the symptom clusters were independent and predicts differential responding.

Lack of information about the disorder results in additional limitations of the present study. The subjects used in the present study were self-diagnosed volunteers. Any woman who believed that she had PMS and who met the experimental criteria was eligible to participate. Post hoc examination of the daily depression and behavior change ratings do demonstrate that subjects showed an elevation of symptoms during the premenstruum compared to the postmenstruum (Figure 20). The postmenstruum is considered to be symptom free in women with PMS (Dalton, 1979).

Many of the researchers working with menstrual disorders feel that much more stringent criteria are needed. These criteria include assessment of multiple menstrual

cycles and professional rather than self-diagnosis. The specific subject eligibility criteria recommendations range from two to six menstrual cycles of specific records demonstrating symptom occurrence premenstrually (Dalton, 1979; Sampson & Prescott, 1981). The value and importance of prospective and retrospective assessment of specific symptom clusters in PMS cannot be ruled out on the basis of the present study, in part, because it is possible that the women participating in this study were not actually suffering from PMS. The use of more stringent criteria in future research is recommended.

In addition, in the present study only one baseline assessment and two posttreatment assessments were used. Calhoun and Sturgis (1984) recommend multiple pre- and post-treatment measures due to the fluctuation in menstrually related symptoms across cycles. Others also support the use of multiple pre- and posttreatment measures in order to provide a greater sample of behavior upon which to base experimental conclusions (Dalton, 1979; Sampson & Prescott, 1981).

The issue of power as related to sample size is another limitation of the present study. Power is the probability of rejecting the null hypothesis appropriately and is determined by the interaction of sample size, effect size, and significance level (Cohen, 1977). In the present

study, using the treatment group sample size (12), a medium effect size (.25), and the arbitrary standard significance level (.05), the power of the ANOVA's used was only .23-.28 depending upon the degrees of freedom (Cohen, 1977). That is, the rate of Type II error, or failing to reject a false null hypothesis, is between 72 and 77 percent. This rate of Type II error suggests that failure to reject a false null hypothesis is likely. Sample size may also be determined by the other three variables: effect size, significance level, and power. Using the same effect size (.25, medium), the standard significance level (.05), and the proposed standard power level (.80), the necessary sample size to demonstrate significant effects would be 52 subjects per treatment group (Cohen, 1977). It is apparent that the power level and sample size in the present study were insufficient to reveal even medium size effects; to reveal small effects a sample size of 322 per treatment group would be necessary and even large effects (unlikely in psychological treatment evaluation research) would require a sample size of 21 subjects per treatment group (Cohen, 1977). Thus, the sample size in the present study is clearly a limitation.

Overall, the limitations of the present study serve to temper the tendency to reject the experimental hypothesis. At the same time, it is clear that the treatment validity of targeting specific symptom clusters within PMS has not been

demonstrated in the present study. The results, however, may be viewed as intriguing and may serve to spur further treatment validity and PMS research. Attention to these limitations in future research is advised.

Directions for Future Research

In light of the limitations of the present study, directions for future research must be carefully considered. It is clear that studies examining both treatment validity and PMS are needed. Prior to embarking on another research project which addresses treatment validity questions in the assessment of PMS, more research on PMS is necessary. In general, future directions for research include a) examination of treatment validity questions with better known disorders which have treatments empirically demonstrated to be effective, and b) descriptive and simple outcome research on PMS.

Various issues have been raised in the interpretation of the results of the present study. A number of these issues lend themselves to treatment validity studies, assuming the limitations of the present study and the state of empirical knowledge of PMS can be overcome. These and other directions for future research are discussed.

The present study was concerned with the importance of matching treatment to identified symptom clusters. An alternative design which could be used to examine the

correspondence between identifying specific symptom clusters and outcome is the manipulated match type of treatment validity study. Hayes and his colleagues (in press) suggest that this may be more powerful in revealing such relationships between assessment and treatment than other designs. In such a design, individuals would be randomly assigned to groups based upon the method of matching assessment to treatment. Those whose symptoms are matched to treatment would be compared with those whose symptoms are mismatched. Differences in outcome would be interpreted to reflect the treatment validity of different use of available assessment data. Such a design answers the question: is outcome enhanced by matching treatment to symptoms or is assessment of those symptoms irrelevant?

Such a design could offer a more powerful evaluation of the value of targeting the depression and behavior change symptom clusters within PMS. Subjects would be randomly assigned to one of the three treatment groups: depression treatment, behavior change treatment, and control. Half of the subjects in each group would have been determined to have depression symptoms, but no behavior change symptoms; and the other half would have been determined to have behavior change symptoms, but no depression symptoms. This design would address the same question as the present study through the more powerful treatment validity design:

manipulated match. One difficulty with such a design is the availability of such "pure" types of subjects. In addition, in light of the limited information on PMS in general and its response to various treatments in particular, even this more powerful design would be premature.

An alternative treatment validity approach might sidestep the limited information on the characteristics of PMS. This approach would compare an idiographic and a yoked approach to treatment of PMS. This would require that subjects be randomly assigned to one of two groups: idiographic and yoked. The idiographic group would be individually assessed and symptomatically treated based upon the specific symptoms complained of by each subject. The yoked group would receive specific symptomatic treatment which was mismatched with her specific symptomatic complaints. This would meet the criteria of a manipulated match type of treatment validity study; the value of behavioral assessment in matching versus mismatching symptoms to treatment would be evaluated. The difficulty inherent in the absence of empirically effective treatments, however, remains.

In light of the potential differences on prospective and retrospective assessments, an evaluation of the treatment validity of these two forms of assessment would be valuable. A manipulated assessment type of treatment

validity study could be used. All subjects could be assessed using both prospective and retrospective devices, and randomly assigned to a prospective or retrospective group. Those in the prospective group would receive a treatment based upon only the prospective assessment and those in the retrospective group would receive a treatment based upon only the retrospective assessment. Differential outcomes between the groups would confirm the treatment validity of the prospective versus retrospective aspect of assessment. Such a design would answer the question: is outcome enhanced by the prospective or retrospective method of assessment?

An additional issue related to the prospective assessment of PMS lends itself to a treatment validity question. Insofar as it has been recommended that three months of prospective assessment are required to establish the diagnosis of PMS (Calhoun & Sturgis, 1984; Dalton, 1979), it would be interesting to examine the treatment validity of such assessment. A simple observed-differences type of treatment validity study could be used. Subjects whose PMS had been confirmed by three months of prospective ratings could be compared to subjects whose PMS had not been confirmed by the three months of prospective rating. Pragmatically speaking, one would need to use subjects all of whom retrospectively report PMS. Any differential

response to the same treatment procedure would demonstrate the treatment validity of three months of prospective ratings to confirm PMS. This is a treatment validity study in that the impact of prospective assessment on treatment would be evaluated. Again, this study may be premature in the face of no empirically effective treatment procedures.

Although it does not directly lend itself to a treatment validity question, another direction for future research was suggested by the results of the present study. It was suggested that the lack of treatment effect could have been due to noncompliance. In light of the potential effect of noncompliance, assessing the impact of the treatment procedures when compliance is ensured would be helpful in clarifying both present and future results. One way to address the question of the impact of compliance is to ensure compliance through a variety of measures in future research. Alternatively, the the impact of compliance could be directly evaluated. Most simply, although not a treatment validity study, a group in which compliance is ensured could be compared to a group in which compliance is not ensured. Such a treatment outcome study could clarify the impact of compliance on the outcome of the behavior change treatment.

Although the results of the present study were not supportive of the experimental hypothesis, the issues

raised by the findings offer multiple directions for future research. Primary among those directions are the many different types of treatment validity study which could elucidate the present findings and further the evaluation of behavioral assessment.

Conclusions

The main goal of the present study was to test the hypothesis that the selection of a specific target behavior within PMS and matching it to a treatment would enhance treatment outcome. Overall, the results did not support this treatment validity hypothesis.

This study also addressed the question: in terms of a general measure of premenstrual symptoms, is it more effective to target depression symptoms of PMS or behavioral change symptoms? The results of the parametric statistical analyses of this study suggest that neither experimental treatment was significantly more effective than a control procedure on the overall measure of PMS.

The major difficulty in comfortably rejecting the experimental hypothesis is the confound of the importance of identifying the specific symptom clusters and the effectiveness of the treatment procedures used. If the treatment procedures were ineffective in ameliorating or managing the symptoms of PMS, then there is no way to determine whether targeting the specific symptom clusters

would enhance treatment outcome. It would be necessary to use proven treatment procedures and ensure their application in order to effectively evaluate the contribution of identifying specific symptom clusters to treatment effectiveness. The treatment procedures used in the present study had been effective with similar symptoms of other disorders, but had not previously been established as effective with PMS symptoms. The lack of significant findings may reflect ineffective treatment procedures rather than a lack of treatment validity for identifying specific symptom clusters. Alternatively, it may be that identifying the depression and behavioral change symptom clusters within PMS is irrelevant in treatment effectiveness. Further investigation regarding the impact of the treatment procedures and the treatment validity of targeting specific symptoms is needed to clarify these issues.

Due to the limitations of the present study, the findings are essentially equivocal. The experimental hypothesis was not supported, but cannot be discarded or ruled out. The major contribution of the present study appears to be two-fold. First, the present study provides an example of the necessity of using treatments whose effectiveness has been previously empirically demonstrated in a treatment validity study. Second, the present study demonstrates the need for descriptive and outcome research

with PMS prior to its use as a vehicle for the elaboration of treatment validity issues.

Due to the present state of empirical knowledge of PMS it is clear that research focusing on treatment validity questions are premature with this disorder. When more has been demonstrated regarding effective interventions for PMS, treatment validity questions could be better addressed through investigations of the disorder. For the present, treatment validity questions are best addressed through research using disorders which are better known and have tried and true treatment procedures available. Treatment validity studies address higher order research questions: thus, they require basic knowledge of treatment effectiveness and a firm theoretical base in order to provide useful or meaningful results as Hayes and his colleagues have suggested (in press).

In summary, the most salient results suggest only that PMS symptom severity does not respond differentially to an operant intervention for behavior control, a cognitive therapy of depression, or record keeping only. While there was no substantial support for the experimental hypothesis, it cannot be rejected outright. The confound of treatment effectiveness and symptom cluster choice requires clarification. The only firm statement that can be made is that there were no significant differences among groups and

there was an general decline in symptom severity from baseline to posttreatment and follow-up. Overall, this study serves as a demonstration of the necessity of using disorders with known effective treatments in examining the treatment validity of specific aspects of behavioral assessment.

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Appendix A
Pilot Study Materials

Appendix A-1

ID# _____ Day _____ Date _____

Self Assessment Questionnaire

The following is a list of symptoms which women sometimes experience. Please describe your experience of these symptoms today. Rate each item on the following scale: 1=no experience of the symptom; 2=barely noticeable; 3=present, mild; 4=present, moderate; 5=present, strong; 6=acute or partially disabling.

Weight Gain	_____	Lowered judgement	_____
Insomnia	_____	Fatigue	_____
Crying	_____	Nausea, vomiting	_____
Lowered school or work performance	_____	Restlessness	_____
Muscle stiffness	_____	Hot flashes	_____
Forgetfulness	_____	Difficulty concentrating	_____
Confusion	_____	Painful breasts	_____
Take naps; stay in bed	_____	Feelings of well being	_____
Headache	_____	Ring in the ears	_____
Skin disorders	_____	Distractible	_____
Loneliness	_____	Swelling	_____
Feeling of suffocation	_____	Accidents	_____
Affectionate	_____	Irritability	_____
Orderliness	_____	General aches and pains	_____
Stay at home	_____	Mood swings	_____
Cramps	_____	Heart pounding	_____
		Depression	_____
		Decreased efficiency	_____

Appendix A-1, page 2

Dizziness, faintness	_____	Lowered motor coordination	_____
Excitement	_____	Numbness, tingling	_____
Chest pains	_____	Change in eating habits	_____
Avoid social activities	_____	Tension	_____
Anxiety	_____	Blind spots, fuzzy vision	_____
Backache	_____	Bursts of energy, activity	_____
Cold sweats	_____	Cold	_____
Menstrual bleeding	_____	Flu	_____
Allergies	_____		

Appendix A-2

Prospective Rating Factors
(N=97; all menstruating subjects)

	MOOS FACTORS	
Factor 1:		
	5. muscle stiffness	pain
	8. take naps; stay in bed	behavioral change
	17. dizziness	autonomic reactions
	19. chest pains	control
	23. cold sweats	autonomic reactions
	32. ringing in the ears	control
	39. heart pounding	control
	43. numbness, tingling	control
	44. change in eating habits	n/a
	46. blind spots, fuzzy vision	control
Factor 2:	Depression	
	3. crying	negative affect
	7. confusion	concentration
	38. mood swings	negative affect
	40. depression	negative affect
	45. tension	negative affect
Factor 3:		
	4. lowered school or work performance	behavioral change
	15. stay at home	behavioral change
	25. fatigue	pain
	29. difficulty concentrating	concentration
	33. distractible	concentration
Factor 4:	Positive Affect	
	14. orderliness	arousal
	18. excitement	arousal
	31. feeling of well being	arousal
	47. bursts of energy	arousal
Factor 5:		
	24. lowered judgement	concentration
	26. nausea, vomiting	autonomic reactions
	37. general aches and pains	pain
	41. decreased efficiency	behavioral change
	42. lowered motor coordination	concentration

Appendix A-2, page 2

		MOOS FACTORS
Factor 6:	Water retention	
	1. weight gain	water retention
	30. painful breaths	water retention
	34. swelling	water retention
Factor 7:		
	6. forgetfulness	concentration
	28. hot flashes	autonomic reactions
	35. accidents	concentration
Factor 8:		
	22. backache	pain
	36. irritability	negative affect
Factor 9:		
	2. insomnia	concentration
Factor 10:		
	16. cramps	pain
	20. avoid social activities	behavioral change
Factor 11:		
	13. affectionate	arousal
Factor 12:		
	10. skin disorders	water retention
	12. feeling of suffocation	control
Factor 13:		
	27. restlessness	negative affect

Items with no correlation weight over 0.50

highest weight on
Factor

2	9. headache	pain
13	11. loneliness	negative affect
4	21. anxiety	negative affect

Appendix A-3

Consistency/Inconsistency of Self Assessment

Debriefing Questionnaire

ID# _____ SS# _____

To what extent were you aware of the experimenter's interest in menstrually related problems during your 40 days of self assessment?

NOT AT ALL VERY MUCH
1 2 3 4 5 6 7

Are you or were you pregnant during the 40 days of self assessment?

1. Yes 2. No

Did you take birth control pills during your 40 days of self assessment?

1. Yes 2. No

Do you suffer from premenstrual syndrome or premenstrual problems?

1. Yes 2. No

please describe:

Were you taking any medication for cramps or premenstrual discomfort during your 40 days of self assessment?

1. Yes 2. No

what kinds of medication?

Appendix A-4

"Pure" Subjects Factors

(N=58; all unaware, no oral contraceptive use subjects)

		MOOS FACTORS
Factor 1:	General complaints	
	5. muscle stiffness	pain
	17. dizziness	autonomic reactions
	19. chest pains	control
	23. cold sweats	autonomic reactions
	32. ringing in the ears	control
	39. heart pounding	control
	42. lowered motor coordination	concentration
	43. numbness, tingling	control
	46. blind spots, fuzzy vision	control
Factor 2:	Depression	
	3. crying	negative affect
	7. confusion	concentration
	11. loneliness	negative affect
	38. mood swings	negative affect
	40. depression	negative affect
	45. tension	negative affect
Factor 3:	Positive Affect	
	14. orderliness	arousal
	18. excitement	arousal
	31. feeling of well being	arousal
	47. bursts of energy	arousal
Factor 4:		
	6. forgetfulness	concentration
	26. nausea, vomiting	autonomic reactions
	28. hot flashes	autonomic reactions
	35. accidents	concentration
Factor 5:		
	9. headache	pain
	36. irritability	negative affect
	41. decreased efficiency	behavioral change
Factor 6:	Water retention	
	1. weight gain	water retention
	30. painful breaths	water retention
	34. swelling	water retention

Appendix A-4, page 2

MOOS FACTORS

Factor 7:	4. lowered school or work performance	behavioral change
	29. difficulty concentrating	concentration
	33. distractible	concentration
Factor 8:	Behavior Change	
	8. take naps; stay in bed	behavioral change
	15. stay at home	behavioral change
	20. avoid social activities	behavioral change
Factor 9:		
	10. skin disorders	water retention
Factor 10:		
	2. insomnia	concentration
Factor 11:		
	27. restlessness	negative affect
Factor 12:		
	16. cramps	pain
Factor 13:		
	13. affectionate	arousal

Items with no correlation weight over 0.50

highest weight on
Factor

4	21. anxiety	negative affect
6	22. backache	pain
1	24. lowered judgement	concentration
5	25. fatigue	pain
5	37. general aches and pains	pain
1	44. change in eating habits	n/a

Appendix A-5

PMS Subjects Factors
(N=33; "pure" subjects
complaining of premenstrual symptoms)

		MOOS FACTORS
Factor 1:	General complaints	
	5. muscle stiffness	pain
	17. dizziness	autonomic reactions
	19. chestpain	control
	23. cold sweats	autonomic reactions
	32. ringing in the ears	control
	39. heart pounding	control
	43. numbness, tingling	control
	44. change in eating habits	n/a
	46. blind spots, fuzzy vision	control
Factor 2:	Depression	
	3. crying	negative affect
	7. confusion	concentration
	38. mood swings	negative affect
	40. depression	negative affect
	45. tension	negative affect
Factor 3:	Behavior Change	
	4. lowered school or work performance	behavioral change
	8. take naps; stay in bed	behavioral change
	15. stay at home	behavioral change
	25. fatigue	pain
	29. difficulty concentrating	concentration
	33. distractible	concentration
Factor 4:	Positive Affect	
	14. orderliness	arousal
	18. excitement	arousal
	31. feeling of well being	arousal
	47. bursts of energy	arousal
Factor 5:		
	24. lowered judgement	concentration
	26. nausea, vomiting	autonomic reactions
	37. general aches and pains	pain
	41. decreased efficiency	behavioral change
	42. lowered motor coordination	concentration

Appendix A-5, page 2

		MOOS FACTORS
Factor 6:	Water retention	
	1. weight gain	water retention
	30. painful breasts	water retention
	34. swelling	water retention
Factor 7:		
	6. forgetfulness	concentration
	28. hot flashes	autonomic reactions
	35. accidents	concentration
Factor 8:		
	22. backache	pain
	36. irritability	negative affect
Factor 9:		
	2. insomnia	concentration
Factor 10:		
	16. cramps	pain
	20. avoid social activities	behavioral change
Factor 11:		
	13. affectionate	arousal
Factor 12:		
	10. skin disorders	water retention
Factor 13:	no item correlation weight greater than 0.50	

Items with no correlation weight over 0.50

highest weight on

Factor		
	8	9. headache
	2	11. loneliness
	12	12. feeling of suffocation
	4	21. anxiety
	6	27. restlessness
		pain
		negative affect
		control
		negative affect
		negative affect

Appendix B

Screening Interview

The principal investigator presents, explains, and has the subject sign consent form I, and then witnesses her signature. She asks the subject:

her name:

address:

telephone number:

age:

marital status:

today's date: month: day: year:

any pregnancies?

using birth control pills?

other physician prescribed medication? for what?

to describe her premenstrual symptoms and problems:

behavior changes?

time resting?

depression?

crying episodes?

physical symptoms or changes?

symptoms and problems start how many days before menstrual onset?

how long has she had these symptoms and problems?

history of psychological problems? family history?

hospitalized?

hallucinations/delusions?

suicidal ideation?

plan?

medications?

currently under psychological/psychiatric care?

Appendix B, page 2

If eligible, have subject complete MDQ.

If eligible, explain data collection procedure and treatment program.

have subject sign consent form II, witness her signature.

collect data deposit.

If not eligible, offer subject alternative referral sources.

principal investigator signature

date

Appendix C

Menstrual Distress Questionnaire

The following is a list of symptoms which women sometimes experience. Please describe your experience of these symptoms during the week before a typical menstrual flow (for screening) or your most recent menstrual flow (for baseline, post-treatment, and follow-up measurement occasions). Rate each item on the following scale: 1 = no experience of the symptom; 2 = barely noticeable; 3 = present, mild; 4 = present, moderate; 5 = present, strong; and 6 = acute or partially disabling.

Weight Gain	_____	Lowered judgement	_____
Insomnia	_____	Fatigue	_____
Crying	_____	Nausea, vomiting	_____
Lowered school or work performance	_____	Restlessness	_____
Muscle stiffness	_____	Hot flashes	_____
Forgetfulness	_____	Difficulty concentrating	_____
Confusion	_____	Painful breasts	_____
Take naps; stay in bed	_____	Feelings of well being	_____
Headache	_____	Ring in the ears	_____
Skin disorders	_____	Distractible	_____
Loneliness	_____	Swelling	_____
Feeling of suffocation	_____	Accidents	_____
Affectionate	_____	Irritability	_____
Orderliness	_____	General aches and pains	_____
Stay at home	_____	Mood swings	_____
Cramps	_____	Heart pounding	_____
		Depression	_____
		Decreased efficiency	_____

Appendix C, page 2

Dizziness, faintness	_____	Lowered motor coordination	_____
Excitement	_____	Numbness, tingling	_____
Chest pains	_____	Change in eating habits	_____
Avoid social activities	_____	Tension	_____
Anxiety	_____	Blind spots, fuzzy vision	_____
Backache	_____	Bursts of energy, activity	_____
Cold sweats	_____		

Appendix D
Tables

Table 1
Subject Data
Screening Assessment Scores

Subject #	Age	Marital Status	MDQ neg affect	beh change
Depression Treatment Group				
101	41	m	45	22
102	35	m	31	15
103	23	m	47	26
104	31	m	35	23
105	40	d	35	21
106	24	s	40	16
107	41	m	48	25
108	38	m	32	19
109	41	d	43	20
110	43	sep	33	16
111	24	m	35	15
112	23	s	26	15
mean	33.7		37.5	19.4
range	23-41		26-48	15-26
Behavior Change Treatment Group				
201	24	s	46	20
202	28	s	33	18
203	38	m	39	23
204	27	m	39	19
205	38	m	26	15
206	33	m	37	22
207	29	m	27	23
208	27	m	36	24
209	34	m	29	25
210	36	m	34	20
211	40	m	35	19
212	27	m	37	17
mean	31.8		34.8	20.4
range	24-40		27-46	15-25

Table 1 continued

Subject #	Age	Marital Status	MDQ neg affect	beh change
Control Group				
301	32	m	25	15
302	21	s	42	23
303	31	m	38	18
304	23	m	31	23
305	38	m	34	15
306	31	m	27	16
307	33	m	33	22
308	30	m	42	19
309	38	m	27	23
310	23	s	43	23
311	38	m	28	15
312	24	s	46	21
mean	30.2		34.7	17.8
range	21-38		25-46	15-23
Overall (all groups) N = 36				
mean	31.9		35.6	19.1
range	21-41		25-48	15-26
		minimum for inclusion	24	15

Table 2
Individual Subject Data
Depression Treatment Group

Pretreatment (baseline) Scores

Subject #	101	102	103	104	105	106	107	108	109	110	111	112	Group Means
Daily Ratings													
depression	15	12	11	09	10	01	08	00	10	00	13	08	8.1
behavior change	16	11	11	10	08	14	08	11	07	00	15	03	9.5
crying episodes	00	01	03	00	01	01	01	00	01	00	01	00	.8
minutes resting	0000	0000	0750	0000	0000	1200	0330	0660	0000	0000	0030	0180	262.5
BDQ													
negative affect	39	40	46	27	39	32	33	29	24	13	34	35	32.6
behavioral change	16	18	24	19	19	10	24	26	09	07	14	10	16.3
MSQ													
congestive score	43	45	46	47	54	39	59	38	39	30	37	48	43.8

Table 2, page 2

Depression Treatment Group

Post-treatment scores

Subject #	101	102	103	104	105	106	107	108	109	110	111	112	Group Means
Daily Ratings													
depression	17	03	04	06	04	04	12	12	19	01	03	06	7.6
behavior change	20	05	04	07	06	12	12	16	17	02	03	01	8.8
crying episodes	00	01	00	01	02	06	02	01	08	00	00	00	1.8
minutes resting	0000	0480	0420	0000	0000	1200	0150	0720	0000	0000	0000	0060	252.5
MDQ													
negative affect	45	19	29	23	29	31	25	31	36	15	12	16	25.9
behavioral change	22	13	17	13	25	20	07	09	18	08	06	08	13.8
MSQ													
congestive score	52	35	38	40	46	27	57	39	43	28	21	31	38.1

Table 2, page 3
Depression Treatment Group

Follow-up Scores

Subject #	101	102	103	104	105	106	107	108	109	110	111	112	Group Means
Daily Ratings													
depression	18	05	03	02	10	00	04	14	07	06	10	03	6.8
behavior change	21	04	03	04	05	05	04	07	01	04	05	00	5.3
crying episodes	01	00	00	01	01	01	00	01	00	05	03	01	1.2
minutes resting	0000	0000	0360	0000	0480	1440	0180	0720	0000	0450	0000	0000	302.5
MDQ													
negative affect	43	14	19	11	31	09	27	37	22	32	17	26	24.0
behavioral change	14	08	10	05	23	09	14	17	11	16	08	07	11.8
MSQ													
congestive score	40	25	34	24	51	20	51	40	37	29	34	45	33.1

Table 2, page 4

Behavior Change Treatment Group

Pretreatment (baseline) Scores

Subject #	201	202	203	204	205	206	207	208	209	210	211	212	Group Means
Daily Ratings													
depression	13	10	11	07	18	13	08	05	10	04	10	12	10.1
behavior change	14	09	11	05	16	14	05	10	16	05	12	13	10.8
crying episodes	03	05	00	02	00	03	00	00	00	00	00	01	1.2
minutes resting	0120	0060	0060	0000	0000	0000	0090	0240	1680	0060	0540	035	266.7
MDQ													
negative affect	33	37	34	39	24	34	14	36	10	28	26	37	29.3
behavioral change	21	12	18	17	11	15	18	25	05	23	18	17	16.7
MSQ													
congestive score	49	34	36	40	35	44	44	49	46	38	44	40	41.6

Table 2, page 5

Behavior Change Treatment Group

Post-treatment Scores

Subject #	201	202	203	204	205	206	207	208	209	210	211	212	Group Means
Daily Ratings													
depression	11	03	11	10	09	15	04	01	00	03	05	14	7.2
behavior changes	08	04	10	08	10	15	00	05	00	04	00	09	6.1
crying episodes	00	00	00	01	00	00	00	00	00	00	00	00	.1
minutes resting	0090	0120	0030	0360	0000	0060	0075	0060	1920	0030	0060	0155	246.7
MDQ													
negative affect	30	23	32	34	13	32	17	36	16	20	31	33	26.4
behavioral change	17	10	15	15	07	18	14	26	06	15	14	15	14.3
MSQ													
congestive score	44	35	35	39	24	46	37	38	30	35	40	34	36.4

Table 2, page 6

Behavior Change Treatment Group

Follow-up Scores

Subject #	201	202	203	204	205	206	207	208	209	210	211	212	Group Means
Daily Ratings													
depression	11	13	12	14	16	16	06	08	00	01	03	13	9.4
behavior change	11	12	10	09	12	17	02	06	03	03	03	11	8.3
crying episodes	04	04	00	04	00	01	00	01	00	00	00	05	1.6
minutes resting	0420	0240	0060	0425	0000	0000	0000	0000	2280	0030	0000	0195	304.2
MDQ													
negative affect	35	34	33	46	23	29	17	39	10	14	32	34	28.8
behavioral change	15	12	15	16	09	18	12	22	05	09	11	17	13.4
MSQ													
congestive scores	42	44	34	51	35	46	40	36	25	28	39	35	37.9

Table 2, page 7

Control Group

Pretreatment (baseline) Scores

Subject #	301	302	303	304	305	306	307	308	309	310	311	312	Group Means
Daily Ratings													
depression	00	10	16	01	00	01	21	06	06	17	13	17	9.0
behavior change	05	08	10	03	14	04	19	09	11	18	06	18	10.4
crying episodes	00	00	05	00	02	01	03	00	00	11	01	04	2.3
minutes resting	0180	1740	0100	0000	0150	0000	0510	0000	0060	0320	0120	0900	340.0
MDQ													
negative affect	17	31	37	31	29	24	37	41	26	42	28	42	32.1
behavioral change	07	17	22	14	15	12	25	18	18	21	15	17	16.8
MSQ													
congestive score	30	47	37	43	37	30	45	41	27	48	39	43	38.9

Table 2, page 8

Control Group

Post-treatment Scores

Subject #	301	302	303	304	305	306	307	308	309	310	311	312	Group Means
Daily Ratings													
depression	02	14	06	00	08	14	20	15	09	09	08	10	9.6
behavior change	04	14	02	00	11	12	15	11	00	14	10	13	8.8
crying episodes	00	01	02	00	01	06	02	00	02	01	00	02	1.4
minutes resting	0120	1620	0020	0000	0240	0360	0690	0060	0000	0300	0020	0600	335.8
MDQ													
negative affect	19	36	20	18	38	25	32	38	23	29	28	41	28.9
behavioral change	13	20	09	10	21	14	16	20	11	16	15	13	14.8
MSQ													
congestive score	40	44	34	36	31	38	50	43	28	36	39	44	38.6

Table 2, page 9

Control Group													
Follow-up Scores													
Subject #	301	302	303	304	305	306	307	308	309	310	311	312	Group Means
Daily Ratings													
depression	01	14	05	06	05	09	11	12	05	12	12	02	7.8
behavior change	04	14	06	07	15	05	12	12	00	15	10	06	8.8
crying episodes	00	01	01	00	03	01	01	00	01	05	01	03	1.4
minutes resting	0120	1620	0020	0180	0240	0600	0780	0000	0000	0240	0120	0560	373.3
MDQ													
negative affect	21	39	15	29	27	23	38	42	12	41	28	42	29.8
behavioral change	06	18	11	12	19	11	23	24	06	24	15	24	16.1
MSQ													
congestive score	29	41	32	38	30	38	52	45	23	48	39	35	37.5

Table 3
Multivariate Analysis of Variance for
Depression Symptom Measures

Source	Wilks Lambda	<u>F</u>	df (hypothesis, error)
Treatment Groups	.91	.53	6,62
Time	.71	1.93	6,28
Groups X Time	.55	1.61	12,56

Note

*p < .05

Table 4

Analysis of Variance for
MDQ Negative Affect Symptom Cluster

Source	df	MS	F
Treatment Groups	2	73.64	.41
Subjects within Groups	33	180.20	
Time	2	196.46	4.79*
Groups X Time	4	54.33	1.32
Within Cells	66	41.04	

Note* $p < .05$

Table 5

Newman-Keuls Post Hoc Tests:
Means of MDQ Negative Affect Symptom Cluster
Scores for Time

Time Periods		Post- Treatment	Follow-up	Baseline	r
	Means	27.08	27.53	31.33	
Post- Treatment	27.08	-	.42	3.97*	3
Follow-up	27.53		-	3.55*	2
Baseline				-	

Note* $p < .05$

Table 6
Analysis of Variance for
Daily Depression Ratings

Source	df	MS	F
Treatment Groups	2	21.85	.39
Subjects within Groups	33	56.25	
Time	2	11.73	.69
Groups X Time	4	15.29	.89
Within Cells	66	17.11	

Note

* $p < .05$

Table 7
Analysis of Variance for
Crying Frequency Measure

Source	df	MS	F
Treatment Groups	2	5.18	1.06
Subjects within Groups	33	4.90	
Time	2	1.12	.38
Groups X Time	4	5.94	2.02
Within Cells	66	2.94	

Note

* $p < .05$

Table 8
Multivariate Analysis of Variance for
Behavior Change Symptom Measures

Source	Wilks Lambda	<u>F</u>	df (hypothesis, error)
Treatment Groups	.95	.28	6,62
Time	.59	3.21*	6,28
Groups X Time	.66	1.10	12,56

Note

*p < .05

Table 9

Analysis of Variance for
MDQ Behavioral Change Symptom Cluster

Source	df	MS	F
Treatment Groups	2	38.62	.71
Subjects within Groups	33	54.53	
Time	2	76.40	4.25*
Groups X Time	4	12.16	.68
Within Cells	66	17.98	

Note* $p < .05$

Table 10

Newman-Keuls Post Hoc Tests:

Means of MDQ Behavioral Change Symptom Cluster Measure

Scores for Time

Time Periods		Follow-up	Post- Treatment	Baseline	r
	Means	13.78	14.50	16.58	
Follow-up	13.78	-	1.01	3.94*	3
Post- Treatment	14.50		-	2.93*	2
Baseline	16.58			-	

Note* $p < .05$

Table 11

Analysis of Variance for
Daily Behavior Change Ratings

Source	df	MS	F
Treatment Groups	2	21.53	.39
Subjects within Groups	33	55.11	
Time	2	81.87	6.43*
Groups X Time	4	28.89	2.27
Within Cells	66	12.73	

Note* $p < .01$

Table 12

Newman-Keuls Post Hoc Tests:
Means of Daily Behavior Change Ratings
for Time

Time Periods	Follow-up		Post- Treatment	Baseline	r
	Means	7.44	7.89	10.25	
Follow-up	7.44	-	.76	4.76*	3
Post- Treatment	7.89		-	4.00*	2
Baseline	10.25			-	

Note

* $p < .01$

Table 13

Analysis of Variance for
Time Resting Measure

Source	df	MS	F
Treatment Groups	2	71559.25	.11
Subjects within Groups	33	661645.39	
Time	2	22984.25	1.00
Groups X Time	4	355.10	.02
Within Cells	66	22935.77	

Note* $p < .05$

Table 14

Analysis of Variance for
MSQ overall measure of PMS complaints

Source	df	MS	F
Treatment Groups	2	7.35	.06
Subjects within Groups	33	130.12	
Time	2	198.04	7.52*
Groups X Time	4	46.51	1.77
Within Cells	66	26.34	

Note

*p < .01

Table 15

Newman-Keuls Post Hoc Tests:
Means for MSQ Overall Measure of PMS Complaints
for Time

Time Periods		Follow-up	Post- Treatment	Baseline	r
	Means	37.08	37.69	41.42	
Follow-up	37.08	-	.71	5.05*	3
Post- Treatment	37.69		-	4.34*	2
Baseline	41.42			-	

Note* $p < .01$

Table 16
Correlation Matrix^a
All Dependent Measures Pretreatment Scores

	MDQ NEGAFF	MDQ BEHCHG	DAILY DEP	DAILY BEHCHG	CRYING FREQ	TIME RESTING	MSQ
MDQ NEGAFF		.55**	.42**	.34*	.47**	-.10	.37*
MDQ BEHCHG	.55**		.24	.23	.23	-.06	.43**
DAILY DEP	.42**	.24		.62**	.46**	.34*	.33*
DAILY BEHCHG	.34*	.23	.62**		.41**	.32*	.19
CRYING FREQ	.47**	.23	.46**	.41**		-.00	.14
TIME RESTING	-.10	-.06	.34*	.32*	-.00		.24
MSQ	.37*	.43**	.33*	.19	.14	.24	

Note

* $p < .05$

** $p < .01$

^a

MDQ NEGAFF = MDQ negative affect symptom cluster measure (retrospective); MDQ BEHCHG = MDQ behavioral change symptom cluster measure (retrospective); DAILY DEP = daily depression rating measure (prospective); DAILY BEHCHG = daily behavior change rating measure (prospective); CRYING FREQ = reported crying frequency measure (prospective); TIME RESTING = reported time resting measure (prospective); MSQ = MSQ overall measure of PMS (retrospective).

Table 17
Individual Subjects Change from Baseline
and Sign Tests

TO	POST-TREATMENT			FOLLOW-UP		
	+	=	-	+	=	-
MSQ overall measure of PMS						
depression treatment group	9	0	3 ~	10	0	2 *
behavior change treatment group	10	0	2 *	8	1	3 ~
control group	5	1	6	7	2	3
overall sign test	(n=35; r=11; p<.05)			(n=33; r=8; p<.01)		
MDQ negative affect cluster						
depression treatment group	8	0	4	9	0	3 ~
behavior change treatment group	8	1	3 ~	5	1	6
control group	7	1	4	6	2	4
overall sign test	(n=34; r=11; p<.10)			(n=33; r=13; p>.25)		

+ improved; = no change; - worsened; * $p < .05$; ~ $p < .25$; r number of times the less frequent sign occurs; n number of matched pairs whose differences have a sign

Table 17, page 2

TO	POST-TREATMENT			FOLLOW-UP		
	+	=	-	+	=	-
<hr/>						
MDQ behavioral change cluster						
depression treatment group	7	0	5	9	0	3 ~
behavior change treatment group	9	0	3 ~	8	3	1
control group	6	1	5	6	1	5
overall sign test	(n=35; r=13; p<.25)			(n=32; r=9; p<.05)		
<hr/>						
daily behavior change rating						
depression treatment group	7	0	5	10	0	2 *
behavior change treatment group	10	0	2 *	9	0	3
control group	8	0	4	6	0	6
overall sign test	(n=36; r=11; p<.05)			(n=36; r=11; p<.05)		

+ improved; = no change; - worsened; * p<.05; ~ p<.25; r number of times the less frequent sign occurs; n number of matched pairs whose differences have a sign

Table 17, page 3

TO	POST-TREATMENT			FOLLOW-UP		
	+	=	-	+	=	-
<hr/>						
daily depression rating						
depression treatment group	6	0	6	8	1	3 ~
behavior change treatment group	8	1	3 ~	6	0	6
control group	6	0	6	6	0	6
overall sign test	(n=35; r=15; p>.25)			(n=35; r=15; p>.25)		
<hr/>						
crying frequency						
depression treatment group	2	4	6	4	2	6
behavior change treatment group	5	7	0	2	6	4
control group	5	4	3	3	4	5
overall sign test	(n=21; r=9; p>.25)			(n=24; r=9; p>.25)		

+ improved; = no change; - worsened; * $p < .05$; ~ $p < .25$; r number of times the less frequent sign occurs; n number of matched pairs whose differences have a sign

Table 17, page 4

TO	POST-TREATMENT			FOLLOW-UP		
	+	=	-	+	=	-
time resting						
depression treatment group	4	6	2	4	4	4
behavior change treatment group	7	1	4	5	3	4
control group	7	1	4	6	2	4
overall sign test	(n=28; r=10; p<.25)			(n=27; r=12; p>.25)		

+ improved; = no change; - worsened; * $p < .05$; ~ $p < .25$; r number of times the less frequent sign occurs; n number of matched pairs whose differences have a sign

Table 18

Percentage of Total Possible on Targeted Symptom Clusters
Based on Screening Assessment Scores

Subject #	Negative Affect Cluster x/48	Behavioral Change Cluster x/30	
101	94	73	
102	65	50	
103	98	87	
104	73	77*	
105	73	70	
106	83	53	
107	100	83	
108	67	63	
109	90	67	
110	69	53	
111	73	50	
112	54	50	
201	96	67	* endorsed
202	69	60	greater
203	81	77	percentage
204	81	63	of
205	54	50	behavioral
206	77	73	change
207	56	77*	cluster
208	75	80*	than of
209	60	83*	negative
210	71	67	affect
211	73	63	cluster
212	77	57	
301	52	50	
302	88	77	
303	79	60	
304	65	77*	
305	71	50	
306	56	53	
307	69	73*	
308	89	63	
309	56	77*	
310	90	77	
311	58	77*	
312	96	70	

Appendix E
Consent Forms

Appendix E-1

Consent Form I

I understand that I am answering questions (by completing a questionnaire and being interviewed) to be used in selecting subjects for a psychological investigation involving the treatment of premenstrual syndrome and associated psychological discomfort. I have been informed that although the information I will supply will be available to the principal investigator and her supervisor, the information will be kept confidential. In addition, I have been informed that I am participating in research and alternative treatment for my problem is available through psychologists and gynecologists in clinics and private practice. I have also been informed that I may withdraw from this screening session at any time.

I understand that in order to participate in this study, I will be asked to collect assessment data (which will be kept confidential) everyday throughout my participation in this study. I understand that I will be asked to contact my therapist and turn in the data I collect once per week. I understand that the procedure for collecting these data will be explained to me in full during this screening session.

I understand that treatment will be conducted on an individual basis over a two-week period. I understand that I will be asked to attend four sessions, each of approximately 50 minutes duration during that two week period. I understand that these sessions may be audiotaped or observed by the principal investigator or her supervisor and will be kept confidential. I understand that if I am eligible, the experimental procedure will be explained to me more fully before I commence treatment and that I may withdraw from treatment at any time.

I understand that if I am eligible for treatment, I have agreed to make a \$20.00 "data deposit" during this session. I understand that I am not paying for any treatment that I may receive. If I am eligible for treatment, I have agreed to have my money refunded, gradually and fully, if I collect all the assessment data and come to all required sessions. I must also agree to forfeit the money that matches the commitments I fail to keep. Specifically, I understand that my data deposit will be refunded according to the following schedule:

Appendix E-1, page 2

If I collect all my assessment data, contact my therapist and turn in my data as scheduled and attend all scheduled treatment sessions my data deposit will be refunded as follows:

day one or within two days of the onset of
my first menstrual period.....\$5
my second menstrual period.....\$5
my third menstrual period.....\$5
upon attendance at four treatment sessions.....\$5

\$20

I understand that if I have to miss a scheduled session with my therapist, I may call in advance to reschedule the appointment within two days of the original time. I understand that I will need to contact my therapist:

- (a) once per week to turn in my data,
- (b) on the first day or within two days of the onset of my next three menstrual periods, and
- (c) by attending the four scheduled treatment sessions

in order to receive my refund.

I understand that if I become dissatisfied with this study, withdrawal can be arranged promptly and the remainder of my data deposit will be refunded.

I understand that if I am not eligible for this study, I will be given a list of referrals (medical and psychological) for treatment.

signed: _____

witnessed: _____

date: _____

Appendix E-2

Consent Form II

I, _____, hereby agree to participate in psychological research to be conducted under the supervision of Rosemary O. Nelson, Professor of Psychology, UNC-G, involving assessment and psychological treatment of premenstrual syndrome and associated psychological discomfort.

As explained to me, for the next three months I will be required to collect assessment data to be turned in on a weekly basis. In addition, I understand I will participate in four treatment sessions conducted on an individual basis.

I have been assured that all data will be kept confidential. I understand that I will be required to collect assessment data daily through my next three menstrual cycles.

I understand that in the two weeks following my third menstrual period, following three months of data collection, I will have the opportunity to meet with my therapist for four treatment sessions (for the control group).

I understand that in the two weeks following my first menstrual period I will meet with my therapist four times and receive training in a cognitive treatment for depression (for the depression treatment group) or a behavior management procedure for activity changes (for the behavior change treatment group).

I understand that my therapist will be a graduate student in psychology who has received training in the techniques employed here. Therapists will be supervised by Dr. Rosemary O. Nelson, Professor of Psychology, and Susan Leonard, principal investigator. I am aware that these supervisors will observe some of my treatment sessions through a one way mirror or listen to audio tapes of the sessions.

I understand that if I become dissatisfied with this study, withdrawal can be arranged promptly by contacting Susan Leonard and the remainder of my data deposit will be refunded.

Appendix E-2, page 2

I understand that the purpose of this investigation is to evaluate methods of treating premenstrual syndrome and associated psychological discomfort using methods which have been successful with similar problems (not PMS) in the past. I realize, however, that there is no guarantee that I will be free from premenstrual symptoms and discomfort because I participate in this research. Hopefully, my participation will contribute to the development of effective treatment of premenstrual syndrome and associated discomfort for others, as well as for myself. In addition, if at the end of this investigation I am not satisfied with my progress, then I will receive a referral for continued evaluation and treatment.

signed: _____

witness: _____

date: _____

Appendix F

Daily Self Assessment

Name _____

Date _____

	none		little		moderate		extreme
depression	0	1	2	3	4	5	6

	very	little		some		much	
behavior change	0	1	2	3	4	5	6

number of
episodes of crying _____duration of time resting
engaged in required _____
activities

menstrual bleeding _____

comments _____

Appendix G

Menstrual Symptom Questionnaire

Instructions: The first twenty-four items on this questionnaire describe symptoms associated with the menstrual period. Please indicate the degree to which you experience each symptom by selecting one of the five response choices (not at all, mild, moderate, strong, or severe experience of the symptom) and circle the number which corresponds to your choice. For item 25, please read carefully the descriptions of two types of menstrual discomfort and select the type which most closely fits your experience.

Item	not at all					severe				
	1	2	3	4	5	1	2	3	4	5

* 1. I feel irritable, easily agitated, and am impatient a few days <u>before</u> my period.	1	2	3	4	5					
2. I have cramps that <u>begin</u> on the first day of my period.	1	2	3	4	5					
* 3. I feel depressed for several days <u>before</u> my period.	1	2	3	4	5					
4. I have abdominal pain or discomfort which begins one day <u>before</u> my period.	1	2	3	4	5					
* 5. For several days <u>before</u> my period, I feel exhausted, lethargic or tired.	1	2	3	4	5					
6. I only know that my period is coming by looking at the calendar.	1	2	3	4	5					
7. I take a prescription drug for the pain <u>during</u> my period.	1	2	3	4	5					
8. I feel weak and dizzy <u>during</u> my period.	1	2	3	4	5					
* 9. I feel tense and nervous <u>before</u> my period.	1	2	3	4	5					
10. I have diarrhea <u>during</u> my period.	1	2	3	4	5					
* 11. I have backaches several days <u>before</u> my period.	1	2	3	4	5					
12. I take aspirin for pain <u>during</u> my period.	1	2	3	4	5					
* 13. My breasts feel tender and sore a few days <u>before</u> my period.	1	2	3	4	5					

Appendix G, page 2

- | | | | | | |
|---|---|---|---|---|---|
| 14. My lower back, abdomen, and the inner sides of my thighs <u>begin</u> to hurt or be tender on the first day of my period. | 1 | 2 | 3 | 4 | 5 |
| 15. <u>During</u> the first day or so of my period, I feel like curling up in bed, using a hot water bottle on my abdomen or taking a hot bath. | 1 | 2 | 3 | 4 | 5 |
| *16. I <u>gain</u> weight <u>before</u> my period. | 1 | 2 | 3 | 4 | 5 |
| *17. I am constipated <u>during</u> my period. | 1 | 2 | 3 | 4 | 5 |
| 18. <u>Beginning</u> on the first day of my period, I have pains which may diminish or disappear for several minutes and then reappear. | 1 | 2 | 3 | 4 | 5 |
| *19. The pain I have with my period is not intense but a continuous dull aching. | 1 | 2 | 3 | 4 | 5 |
| *20. I have abdominal discomfort for more than one day <u>before</u> my period. | 1 | 2 | 3 | 4 | 5 |
| 21. I have backaches which <u>begin</u> the same day as my period. | 1 | 2 | 3 | 4 | 5 |
| *22. My abdominal area feels bloated for a few days <u>before</u> my period. | 1 | 2 | 3 | 4 | 5 |
| 23. I feel nauseous <u>during</u> the first day or so of my period. | 1 | 2 | 3 | 4 | 5 |
| *24. I have headaches for a few days <u>before</u> my period. | 1 | 2 | 3 | 4 | 5 |

* denotes congestive items

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25. TYPE 1

The pain begins on the first day of menstruation, often coming within an hour of the first signs of menstruation. The pain is most severe the first day and may or may not continue on subsequent days. Felt as spasms, the pain may lessen or subside for a while and then reappear. A few women find this pain so severe as to cause vomiting, fainting or dizziness; some others report that they are most comfortable in bed or taking a hot bath. This pain is limited to the lower abdomen, back and inner sides of the thighs.

TYPE 2

There is advanced warning of the onset of menstruation, during which the woman feels an increasing heaviness, and a dull aching pain in the lower abdomen. This pain is sometimes accompanied by nausea, lack of appetite, and constipation. eadaches, backaches, and breast pain are also characteristic of this type of menstrual discomfort.

The type that most closely fits my experience is TYPE ____.

(TYPE 2 is scored +5 congestive and TYPE 1 is scored 0)

Appendix H
Depression Treatment Materials

Appendix H-1

Cognitive Therapy of Depression Protocol

Session 1: present rationale for the treatment procedure
(H-2)

explain how to identify negative interpretations/self talk:
use of evaluative words (e.g., should, ought, must),
catastrophizing (e.g., awful, terrible, can't stand it),
overgeneralization (e.g., I'll never..., I'm incompetent),
and focusing only on the negative, not seeing the positive.

give examples (H-3)

have subject imagine it's her premenstruum and generate
incidents and identify 3-5 negative self statements

instruct subject to practice identifying such thoughts and
record them for next session

Session 2: review negative self talk subject identified
in herself

explain how to dispute depression related statements: via
hypothesis testing (Beck et al., 1979; pp. 253-255):
through use of environmental data, going out into the actual
situation, checking belief against facts, e.g., negative
thought: "I'm fat," test: get on scale, check charts of
normal weights, ask M.D.

logical challenge (Beck et al., 1979; pp. 265-270):
pointing out irrational assumptions, carrying beliefs to
logical extreme, identifying positive aspects of situations,
noting discrepant criteria

give examples and complete homework form (H-4) using
subjects identified negative self talk and in session
generated disputes

have subject practice homework: imagine it's her
premenstruum, dispute negative self talk

instruct subject to practice disputing negative thoughts
daily using the homework form

Appendix H-1, page 2

Session 3: review identification and disputing of
negative self talk

give examples and have subject dispute negative talk
therapist generates

have subject practice homework: imagining it's her
premenstruum, disputing negative self talk (twice for each
statement she identified)

instruct subject to practice homework daily

Session 4: same as Session 3

instruct subject to continue daily practice and to apply the
technique during her premenstruum

Appendix H-2

Cognitive Therapy of Depression Rationale

Physical changes associated with premenstrual tension are thought to be related to mood changes and an increased tendency to feel depressed. Many women complain of feeling unhappy or depressed before their period starts. The goal of this treatment is to teach you how to control your feelings of depression. Individuals often experience depressed feelings because of the way they perceive or think about various situations. Many psychologists feel it is the person's interpretation of the situation, themselves, or the future, rather than the actual situation which causes depression. In addition, it is believed that we learn to interpret situations in certain ways and so we can learn new ways to view the same situations. When we feel depressed, it may be because the interpretation and thoughts we have learned are not reflecting the situation accurately.

The treatment procedure we will use over the next four sessions (today and our next three meetings) is designed to teach you to identify and dispute any thoughts or interpretations you have during your premenstruum which may be making you feel depressed. Through this procedure you will learn new ways to view situations which will help alleviate these depressed feelings. Thus, three steps are involved in this treatment. The first is learning to identify the interpretations and thoughts which may be causing depression. The second is learning to dispute those negative or depressing thoughts through use of logical challenge and hypothesis testing. The third step is practice in disputing negative thoughts.

We will start today learning to identify negative thoughts.

Appendix H-3

Examples of Negative Thoughts

From Beck et al., 1979; p. 246 and 261

In order to be happy, I have to be successful in whatever I undertake.

To be happy, I must be accepted by all people at all times.

If I make a mistake, it means I am inept.

I can't live without you.

If somebody disagrees with me, it means he doesn't like me.

My value as a person depends on what others think of me.

If it's true in one case, it applies in any case which is even slightly similar.

The only events that matter are failures, deprivation, etc.

I am responsible for all bad things, failures, etc.

If it has been true in the past, then it's always going to be true.

I am the center of everyone's attention - especially my bad performances.

I am the cause of misfortunes.

Always think of the worst. It's the most likely to happen to you.

Everything is either one extreme or another (black or white; good or bad).

Appendix H-4

Depression Treatment Homework Form

(to be completed with therapist during session 2 and expanded as appropriate) practice disputing your negative self talk using hypothesis testing or logical challenge responses

Automatic negative thought:

Rational disputing response:

Automatic negative thought:

Rational disputing response:

Automatic negative thought:

Rational disputing response:

Automatic negative thought:

Rational disputing response:

Appendix I
Behavior Change Treatment Materials

Appendix I-1

Operant Intervention for Behavior Control Protocol

Session 1: present rationale (I-2)

explain techniques of behavior change (Fordyce, 1976; pp. 83-88): reinforcers, selection of reinforcers, and attention as a reinforcer.

have subject imagine that it's her premenstruum and complete an activities form (I-3) identifying behavior which is required but low frequency during the premenstruum, high frequency rewarding behavior, and significant others

Session 2: explain activity planning/scheduling

have subjects schedule required activities for the days between this session and the next

make sure subject schedules rewarding events contingent upon required activities specifying duration, frequency, etc.

Session 3: review impact of and adherence to schedule from previous session

assess effectiveness of scheduled rewarding events/activities and revise as needed

have subject schedule required activities and contingent rewarding activities for the days between this session and the last session

Session 4: review the schedule from the previous session

assess the effectiveness of the scheduled rewarding events and revise if necessary

have subject schedule required activities daily/weekly

instruct subject to implement this self management technique during her next premenstruum

Appendix I-2

Operant Intervention for Behavior Control Rationale

Physical changes associated with premenstrual tension are thought to be related to a tendency to perform and behave differently than usual. Many women complain of performing inefficiently, staying at home, and avoiding normal activities. The experience of being ill, impaired, or ineffective is heightened inactivity and avoidance of responsibility. Alternatively, a sense of accomplishment and well-being is evoked through performance of required activities and receipt of rewards and approval for such performance. The goal of this treatment is to help prepare you for the negative premenstrual experiences so that you will have greater control of your behavior. Many psychologists feel that activity level can be increased by presentation of contingent positive consequences. The alteration of consequences may be achieved by planning or scheduling activities and the reward or acknowledgement of them.

The treatment procedure we will use over the next four sessions (today and our next three meetings) is designed to teach you to plan ahead for the experiences of the premenstruum by scheduling your activities and arranging for positive consequences to occur.

We will start today by identifying high frequency, potentially rewarding activities and those required activities you tend to avoid during your premenstruum.

Appendix I-3
activities form

potentially rewarding/high frequency activities

1

2

3

4

5

required activities usually avoided during the premenstruum

1

2

3

4

5

significant others who could serve as helpers

1

2

3

4

5

(at least five activities in the first two categories should be identified; more would be fine; at least three helpers should be identified)

Appendix I-4

Using a daily activity schedule
adapted from Effective Study Materials

The secret of more effective use of time and greater enjoyment of living lies in organizing and planning. Each person will, of course, plan her own 168 hours to harmonize with her unique requirements and interests.

The idea of a daily activity schedule in dealing with PMS is to plan for the premenstrual time period. Carefully arranging your time to account for required activities which premenstrual distress usually leads you to avoid and carefully including clearly pleasant activities to follow those required activities will help decrease a sense of ineffectiveness and poor performance. You will be accomplishing what you need to through planning your time and activities to help maintain or improve your normal level of functioning.

Build your schedule around your fixed time commitments. Some activities have fixed time requirements and others are flexible. FIXED: eating, work, church
FLEXIBLE: sleeping, recreation

Borrow time, don't steal it. Whenever an unexpected activity arises that takes up time you had planned to use otherwise, decide immediately where you can trade for "free" time to make up the missed time and then adjust your schedule for the week.

It is particularly important not to cheat yourself out of the pleasant activities you have scheduled to help you complete your required or somewhat undesirable activities. Try to have alternative pleasant events in case your scheduled pleasant activity is not possible.

Appendix J

Debriefing

The purpose of this study was to evaluate the treatment validity of identifying specific target behaviors within the premenstrual syndrome. This treatment validity issue is concerned with the question: does selection of target behaviors and matching treatment to them enhance treatment outcome? That is, the purpose of this study was to assess the effectiveness of treatment previously used with symptoms of depression or with behavior change symptoms when these treatments are used with women who complain of similar symptoms associated with premenstrual syndrome. The women who have participated in this study have at least mild premenstrual symptoms of depression and behavior changes. All participants recorded their symptoms for three months. Some of the women participated in the treatment which has been used with nonmenstrually related depression and some participated in the treatment which has been used with nonmenstrually related behavior changes. The reason that all the women in the study did not participate in the same or both treatments is that evidence is needed to demonstrate that the logical treatment is, in fact, the most effective or efficient treatment for the targeted symptoms. Some research has demonstrated that the presumably illogical treatment is more effective or more efficient than the logical treatment. It is important to experimentally test the impact of various treatments on specific symptoms in order to demonstrate the effectiveness, rather than assuming effectiveness based upon what seems to be logical. In addition, some of the women in the study recorded their symptoms daily for three months, but participated in neither treatment procedure. Treatment is available to these participants upon completion of their three months of data collection. The reason that some of the women in the study received the experimental treatments during their three months of data collection and some (the control subjects) did not is so that any effectiveness in symptom management can be attributed to the experimental treatment procedure and not to the data collection process or the passage of time. The use of control subjects is another way to experimentally establish the effectiveness of an untried treatment procedure.

Appendix J, page 2

Your participation in this investigation of the appropriateness of specific treatment procedures for premenstrual symptom management is greatly appreciated. If you would like to pursue further professional help for your premenstrual symptoms you will be given a list of referral sources (including psychologists and gynecologists) in your area. For any further assistance please feel free to ask the principal investigator.

Appendix K
Figures

Figure 1:
MDQ Negative Affect
Symptom Cluster Scores

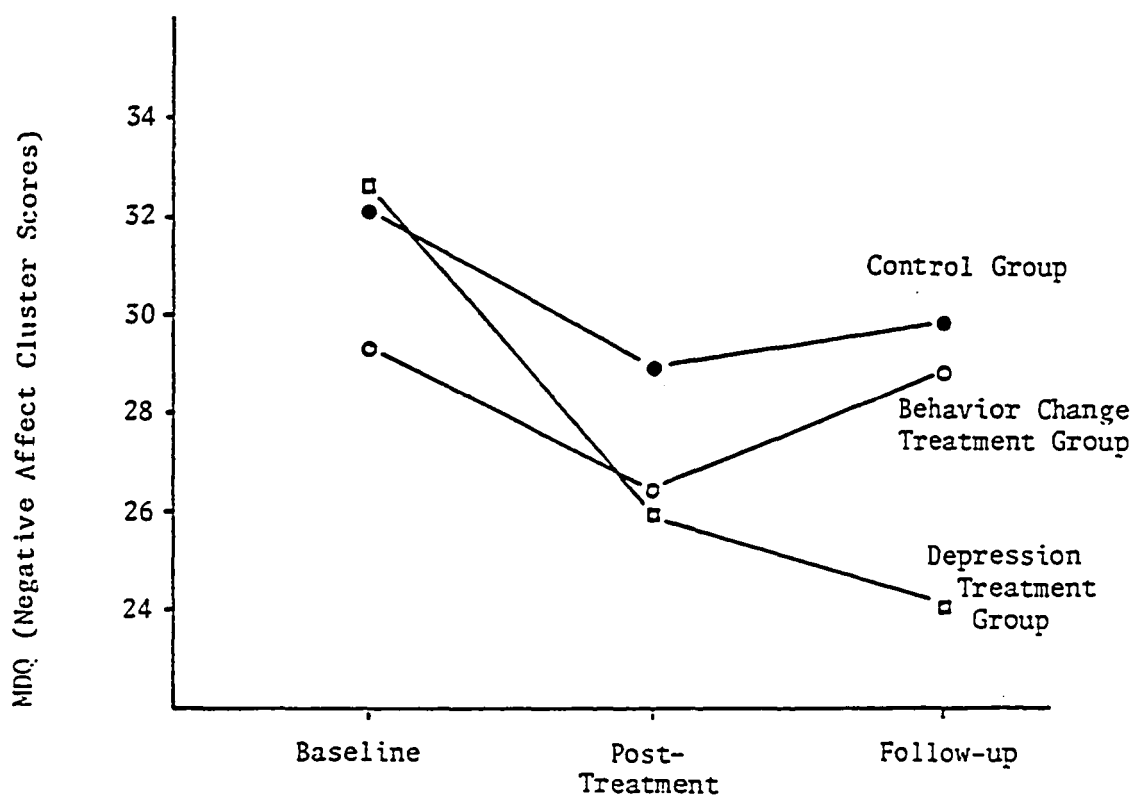


Figure 2:
Daily Depression Ratings
(Average Four Day Totals)

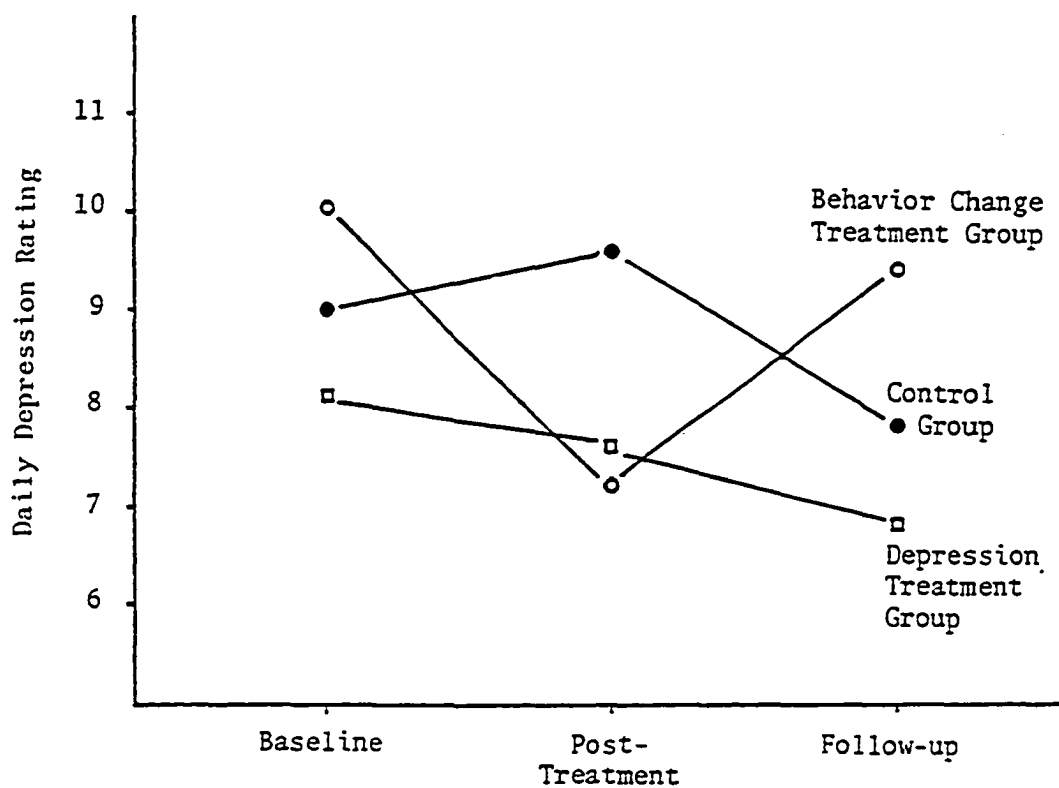


Figure 3:
Reported Crying Frequency
(Average Four Day Totals)

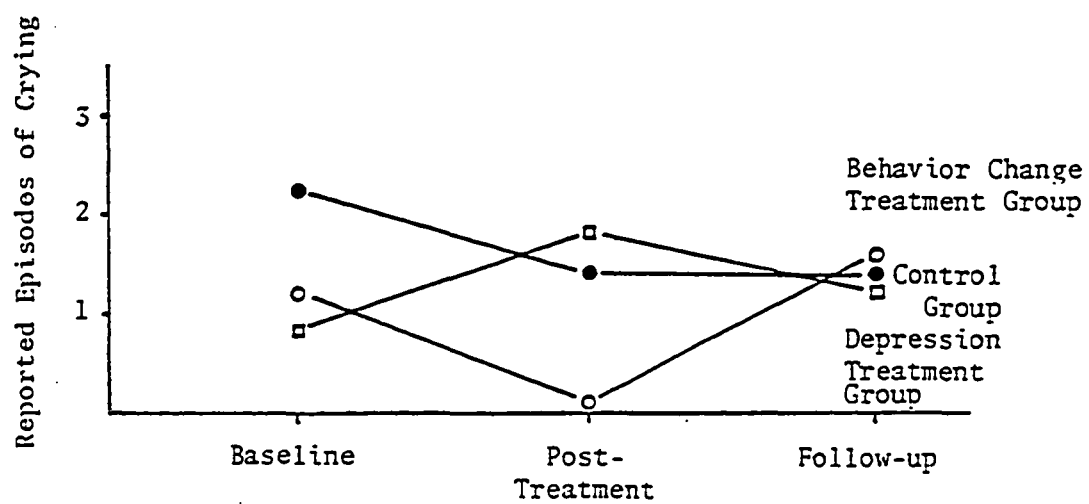


Figure 4:
MDQ Behavioral Change
Symptom Cluster Scores

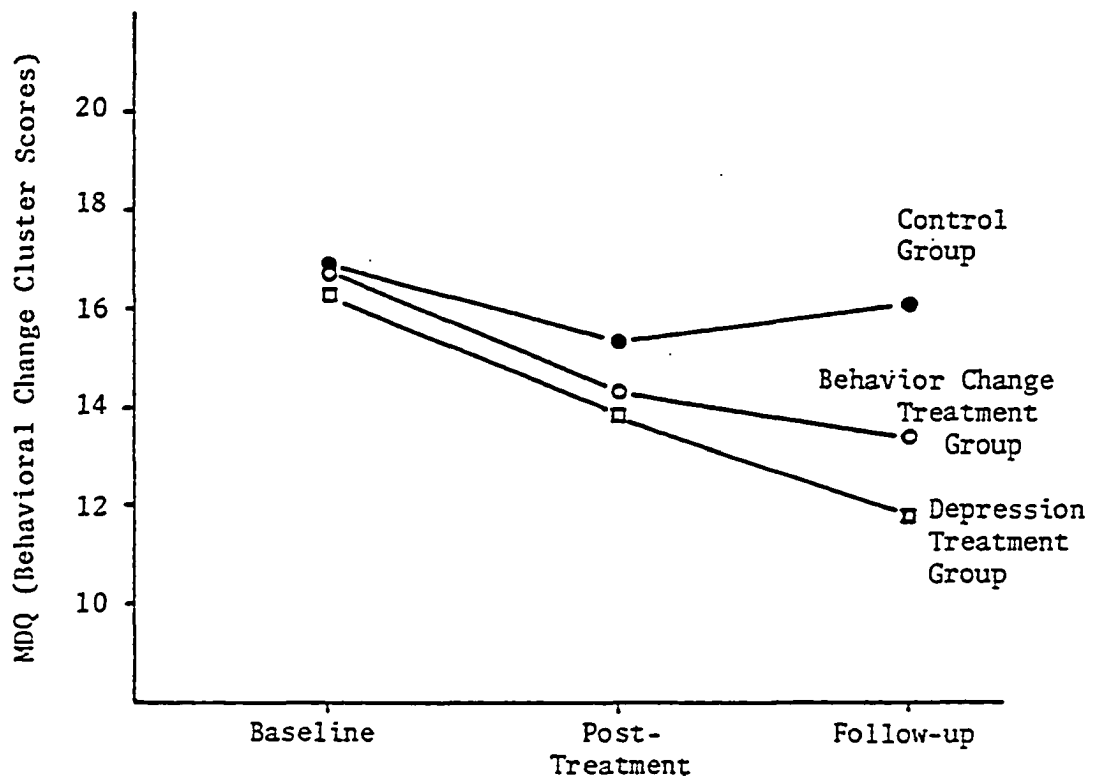


Figure 5:
Daily Behavior Change Ratings
(Average Four Day Totals)

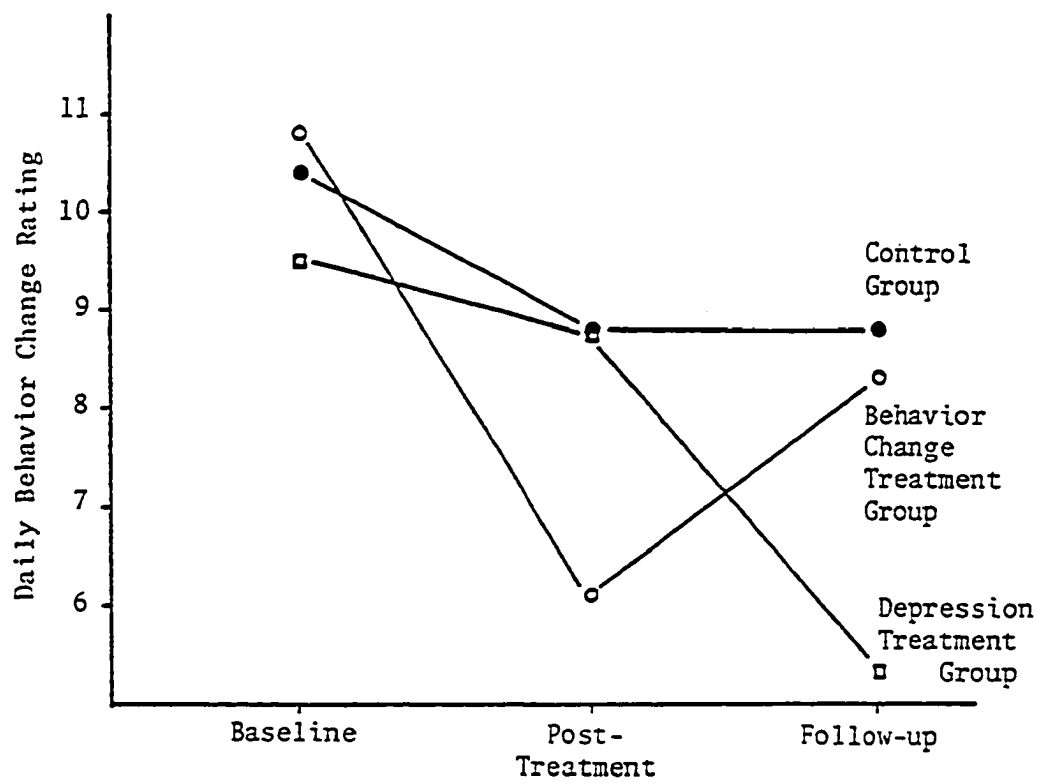


Figure 6:
Reported Time Resting in Minutes
(Average Four Day Totals)

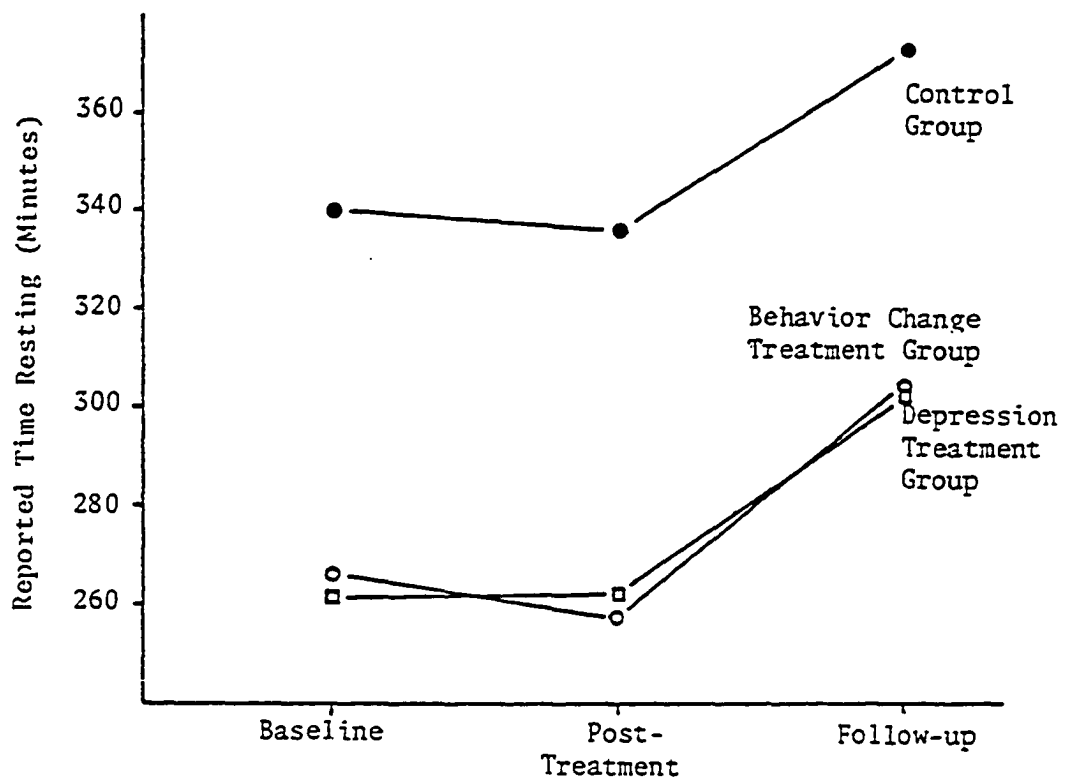


Figure 7:
MSQ Congestive Scores
(Overall Measure of PMS)

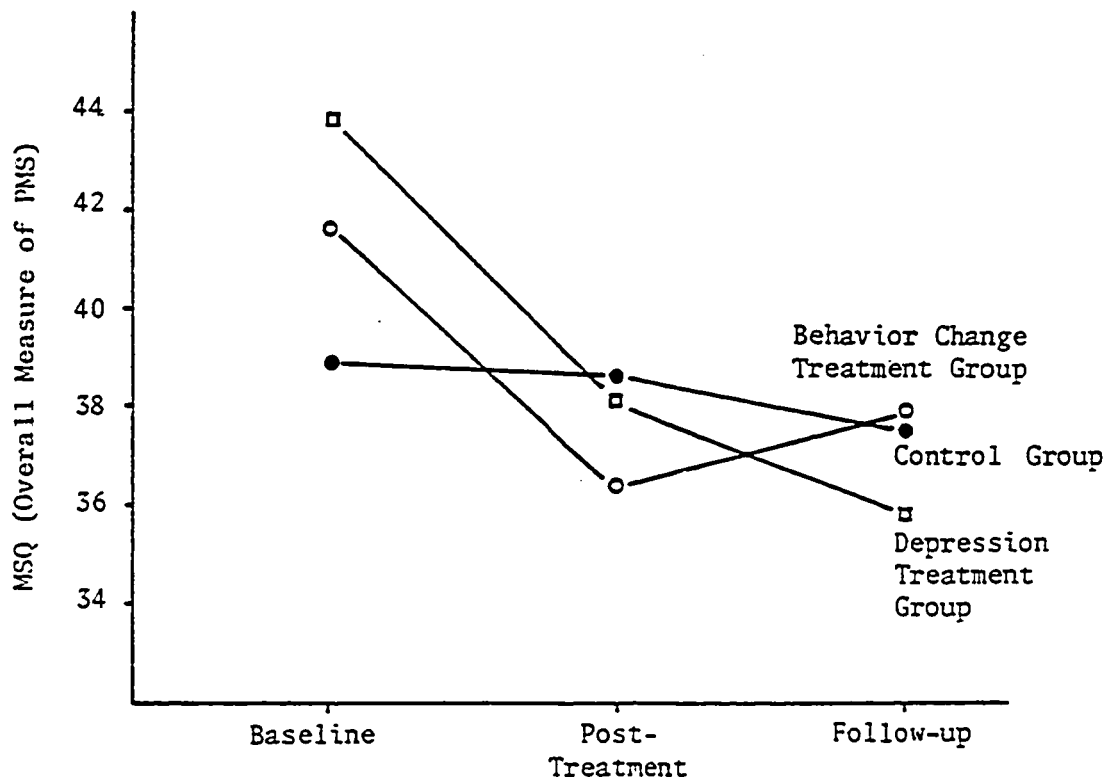


Figure 8:
Depression Treatment Group
Individual Subject MSQ Scores

MSQ Overall Measure Scores

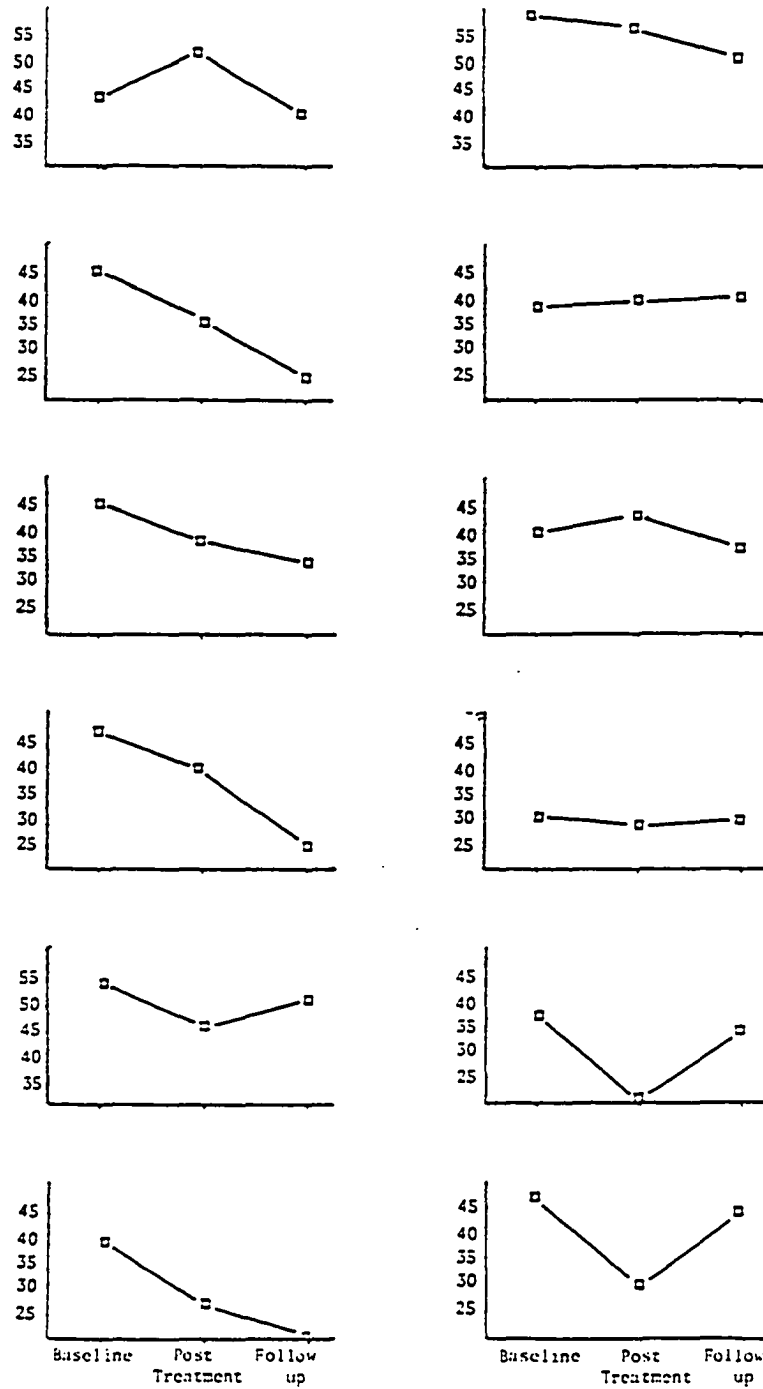


Figure 9:
Behavior Change Treatment Group
Individual Subject MSQ Scores

MSQ Overall Measure Scores

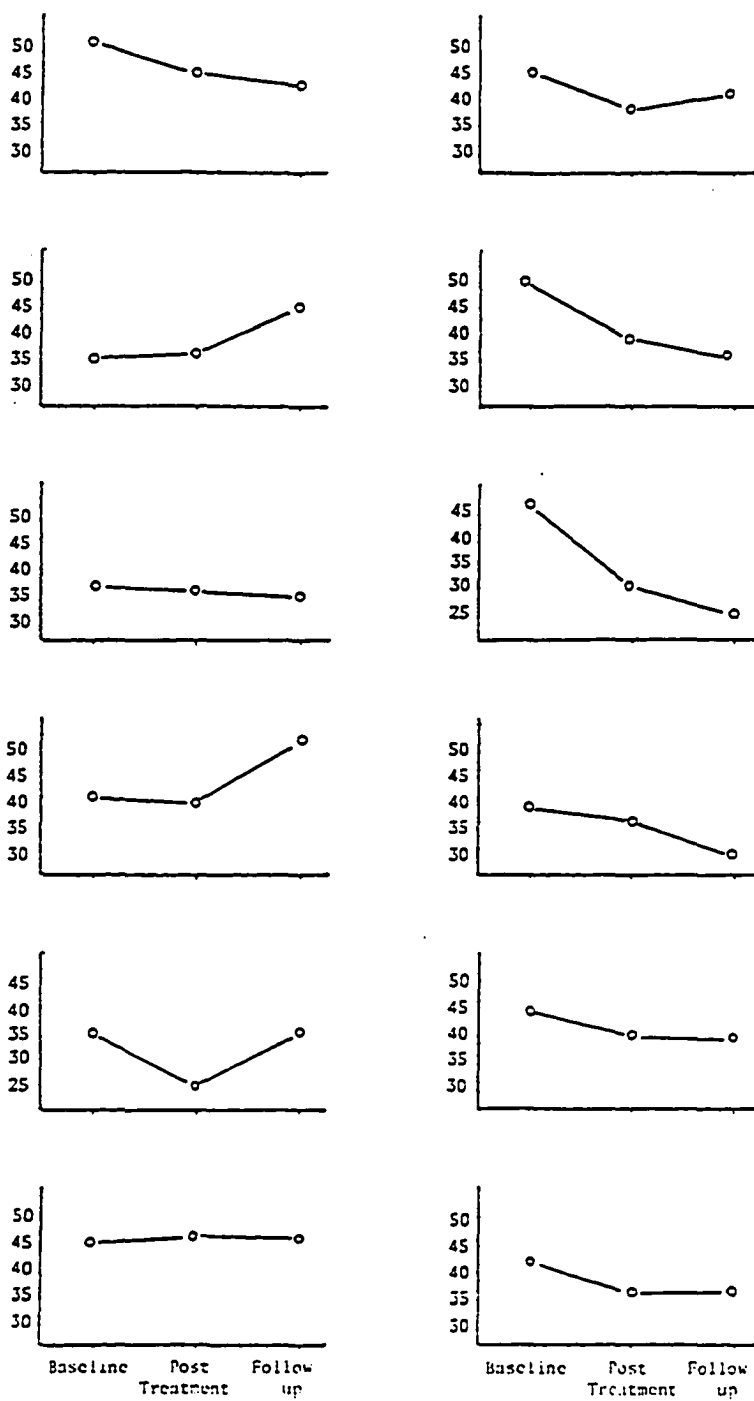


Figure 10:
Control Group
Individual Subject MSQ Scores

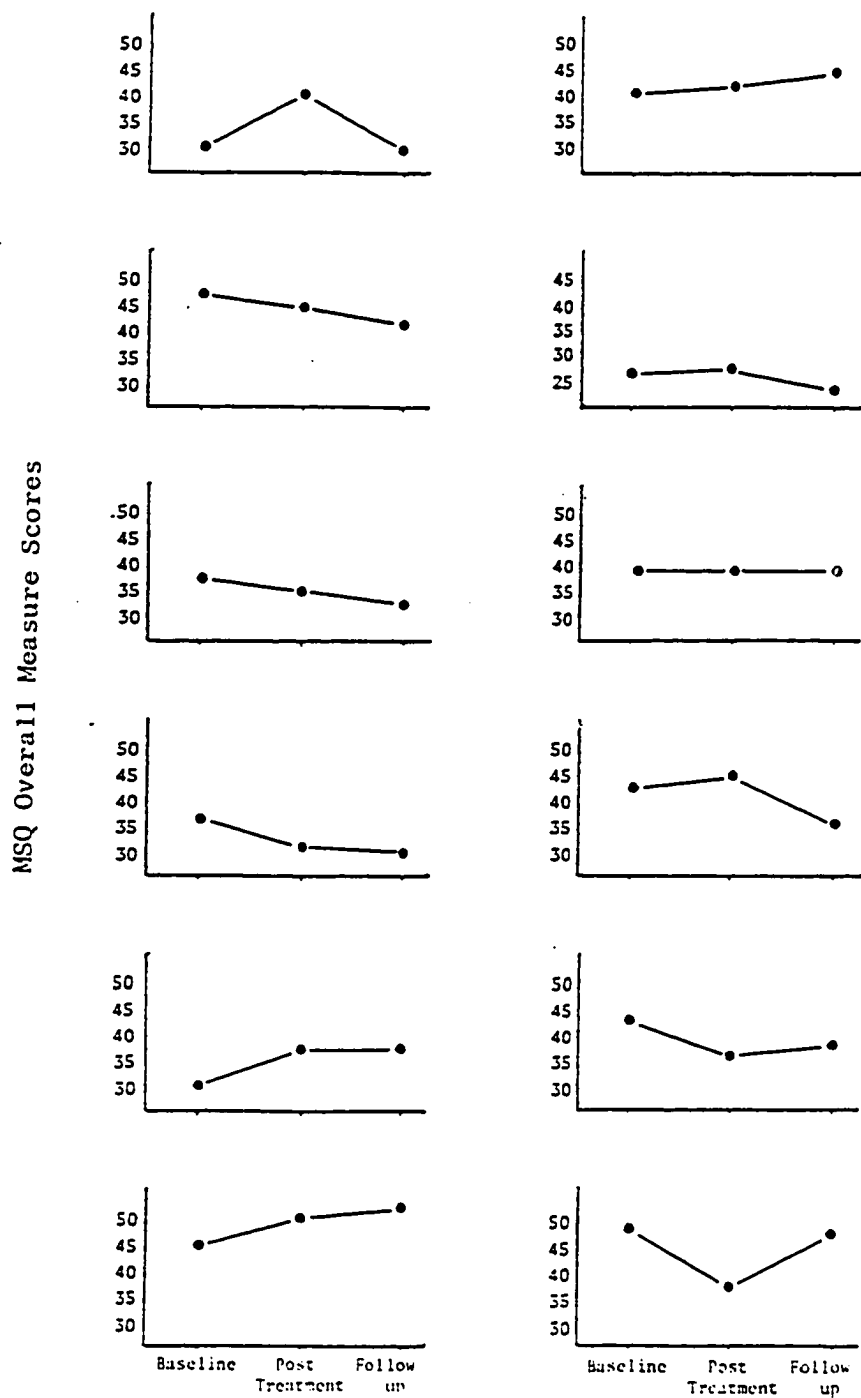


Figure 11:
Depression Treatment Group
Individual Subject MDQ Negative Affect Scores

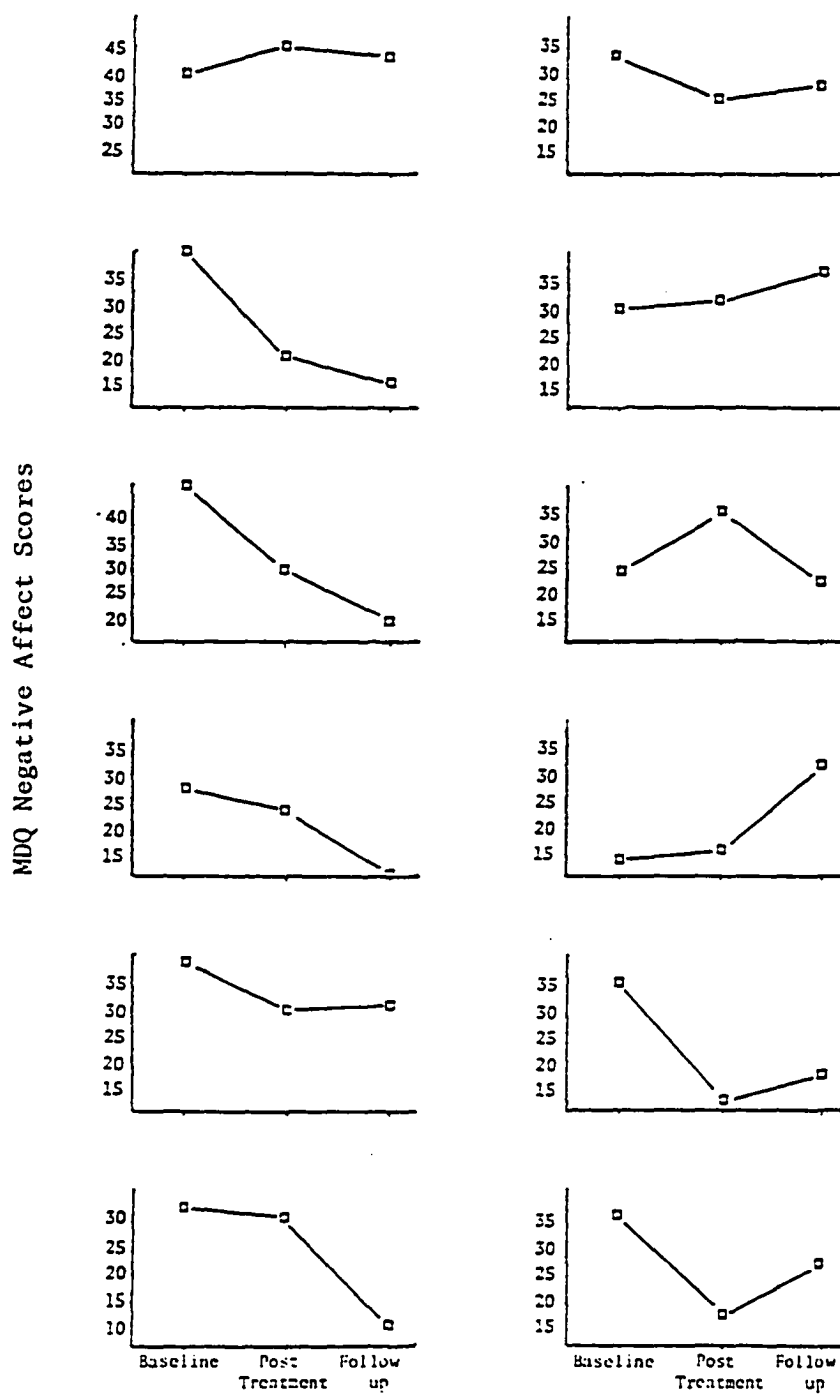


Figure 12:
Behavior Change Treatment Group
Individual Subject MDQ Negative Affect Scores

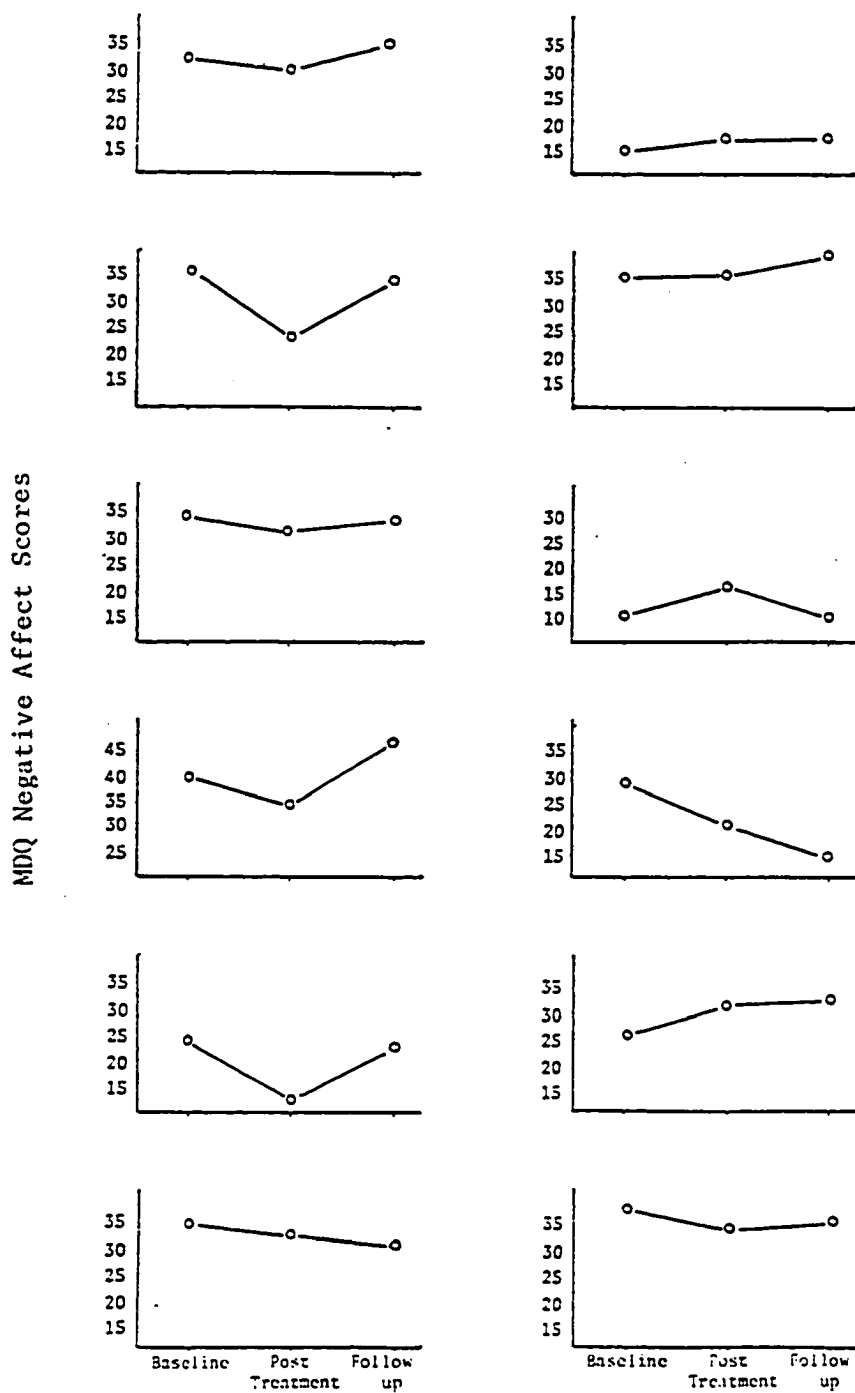


Figure 13:
Control Group
Individual Subject MDQ Negative Affect Scores

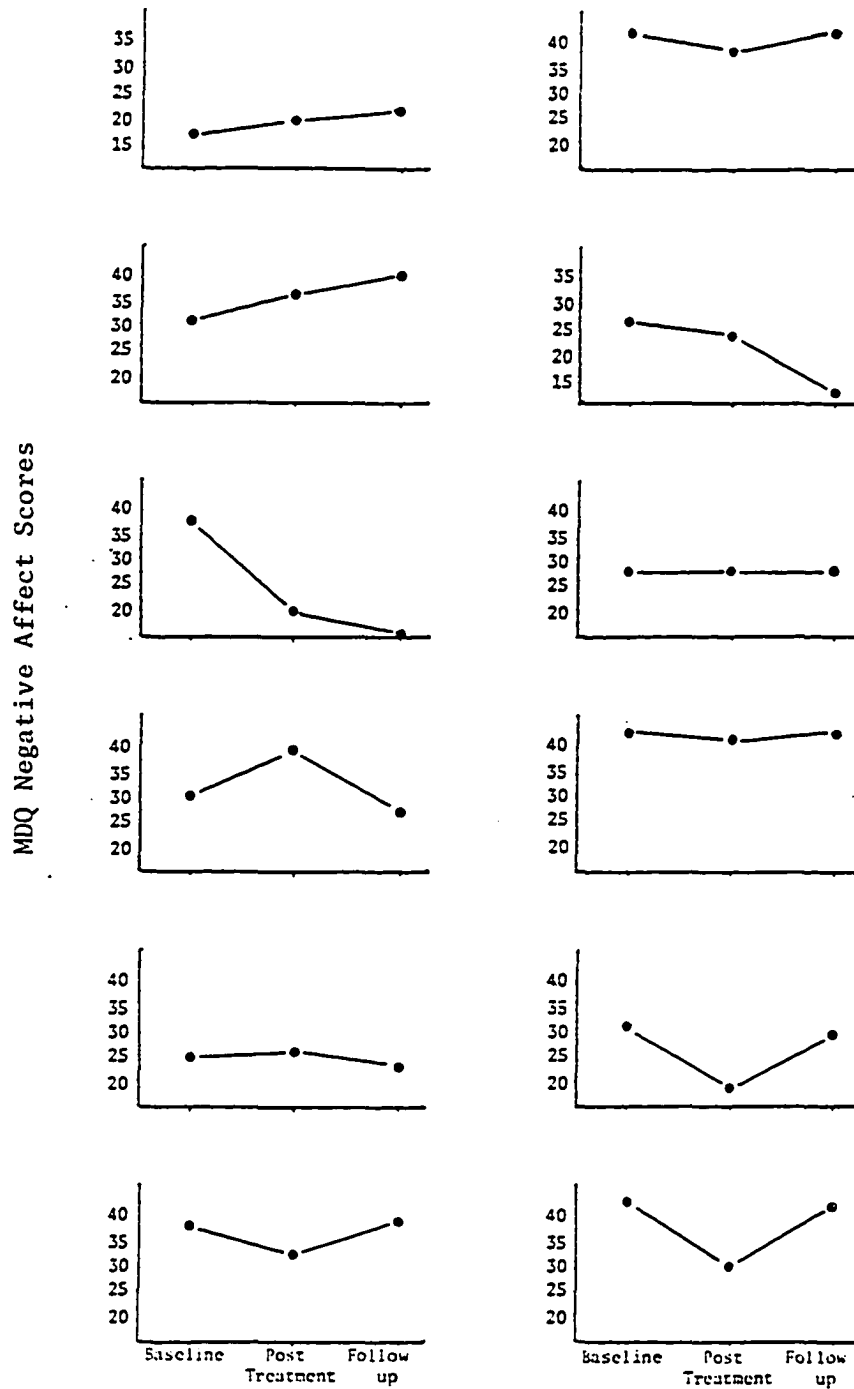


Figure 14:
Depression Treatment Group
Individual Subject MDQ Behavioral Change Scores

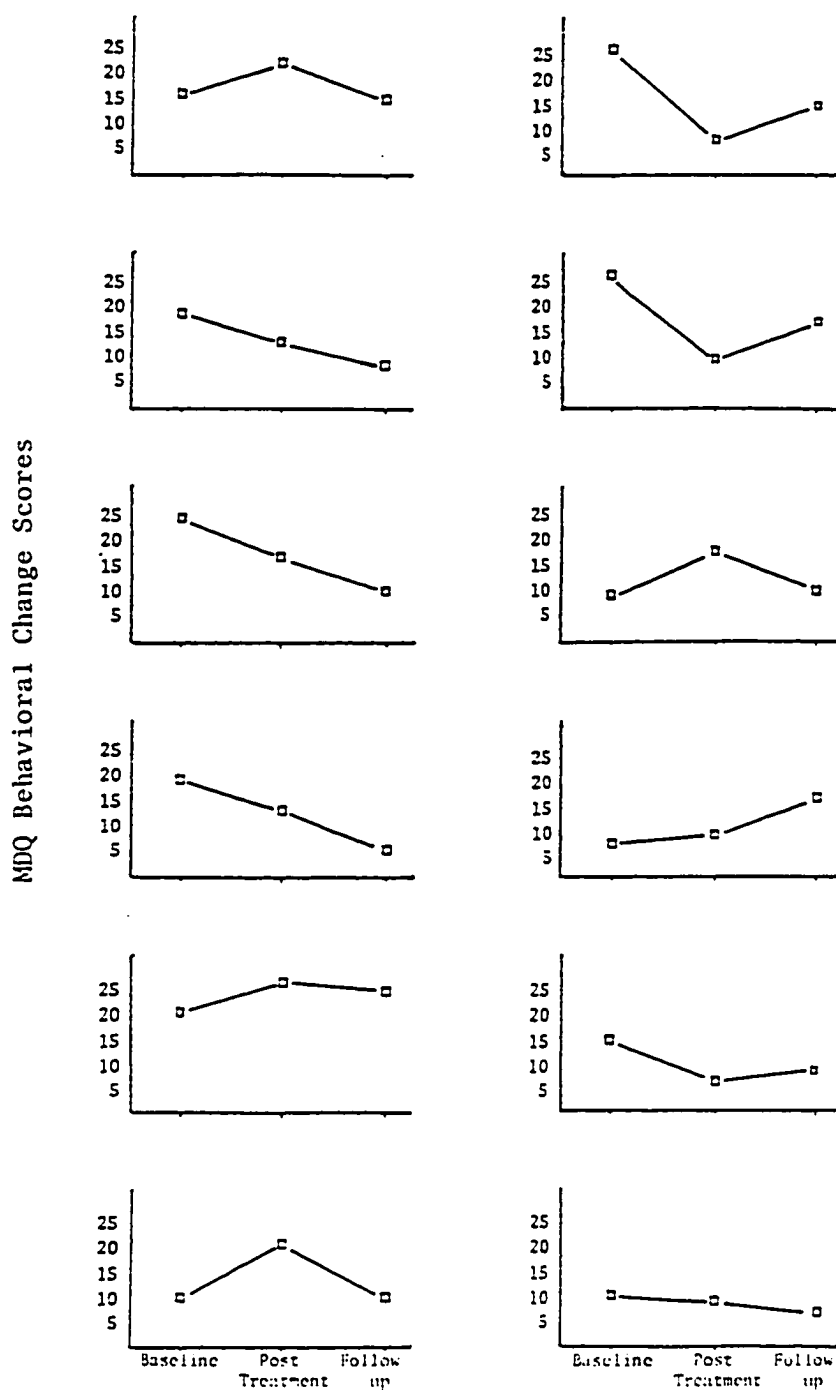


Figure 15:
 Behavior Change Treatment Group
 Individual Subject MDQ Behavioral Change Scores

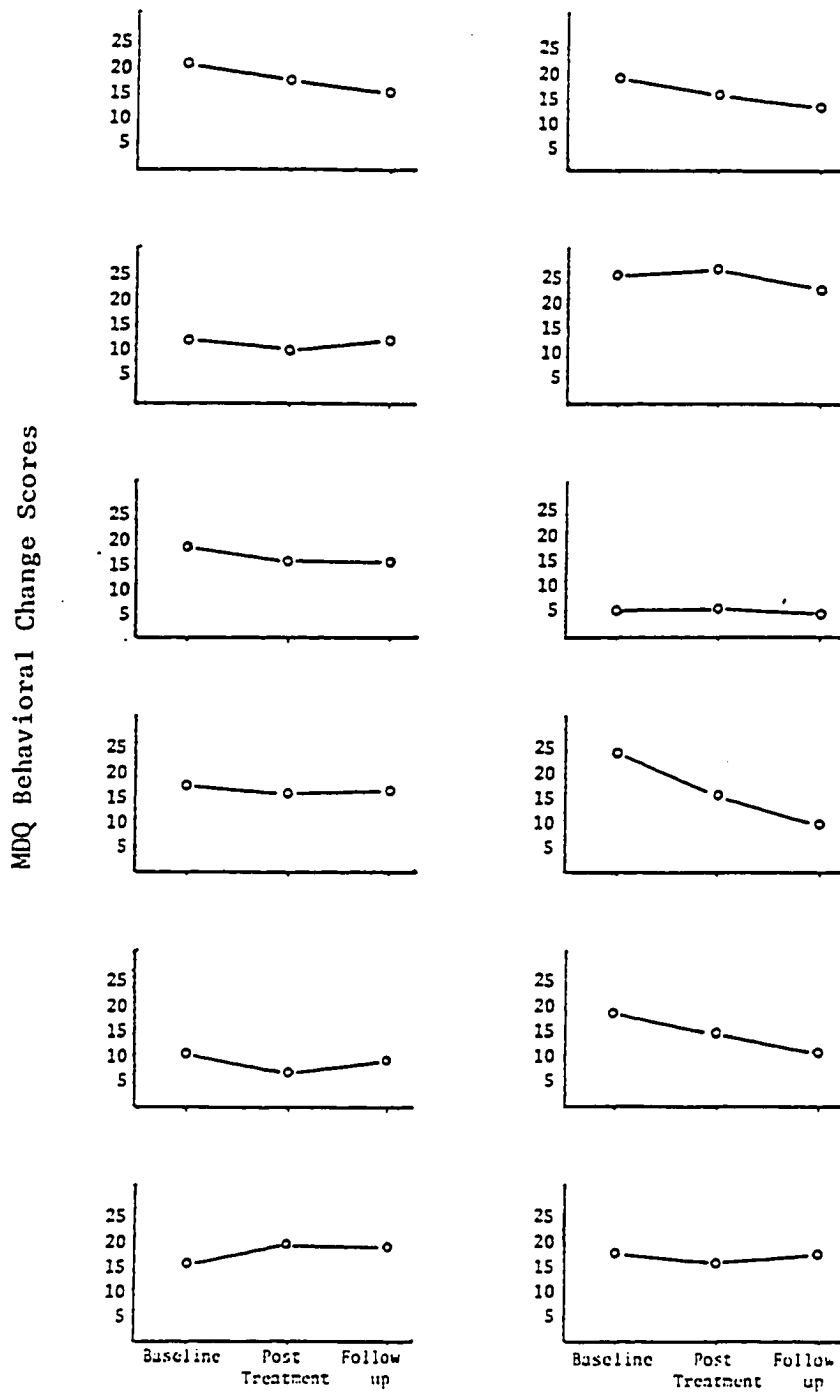


Figure 16:
Control Group
Individual Subject MDQ Behavioral Change Scores

MDQ Behavioral Change Scores

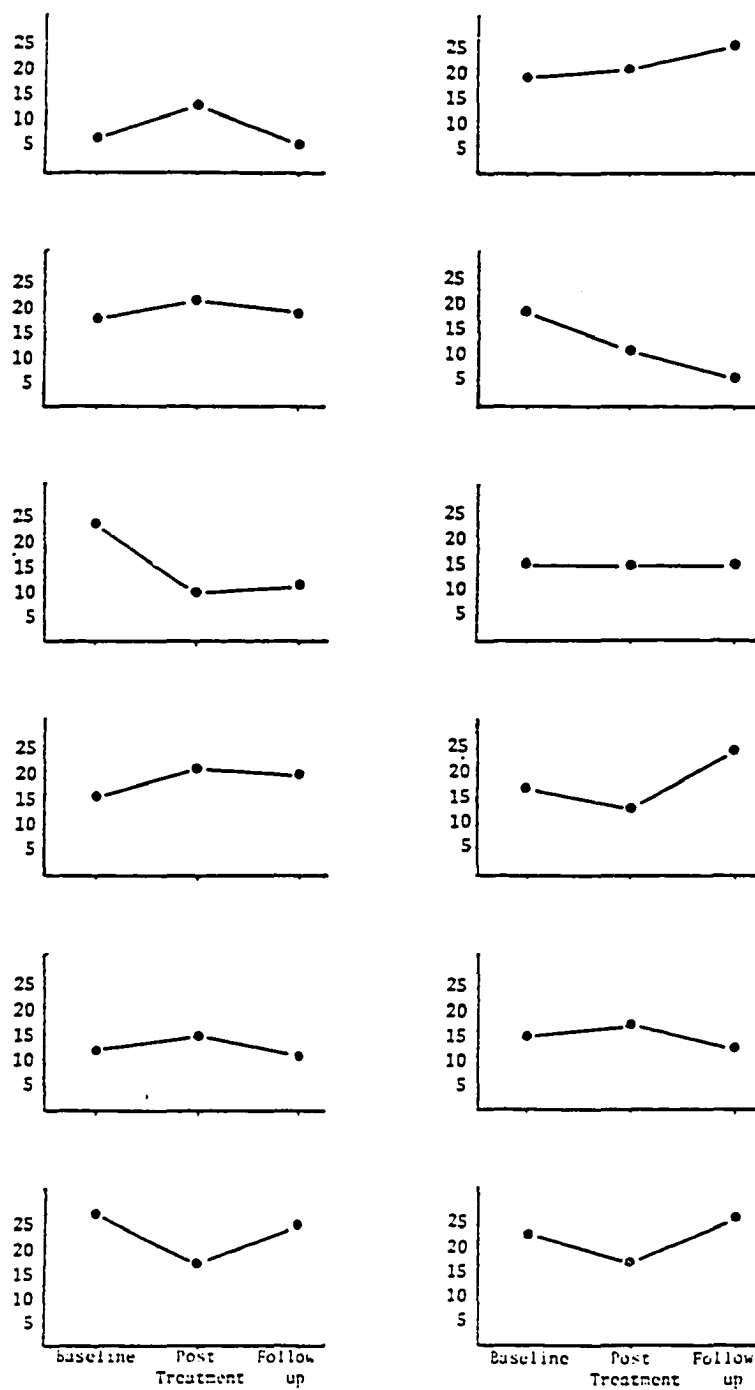


Figure 17:
Depression Treatment Group
Individual Subject Daily Behavior Change Ratings

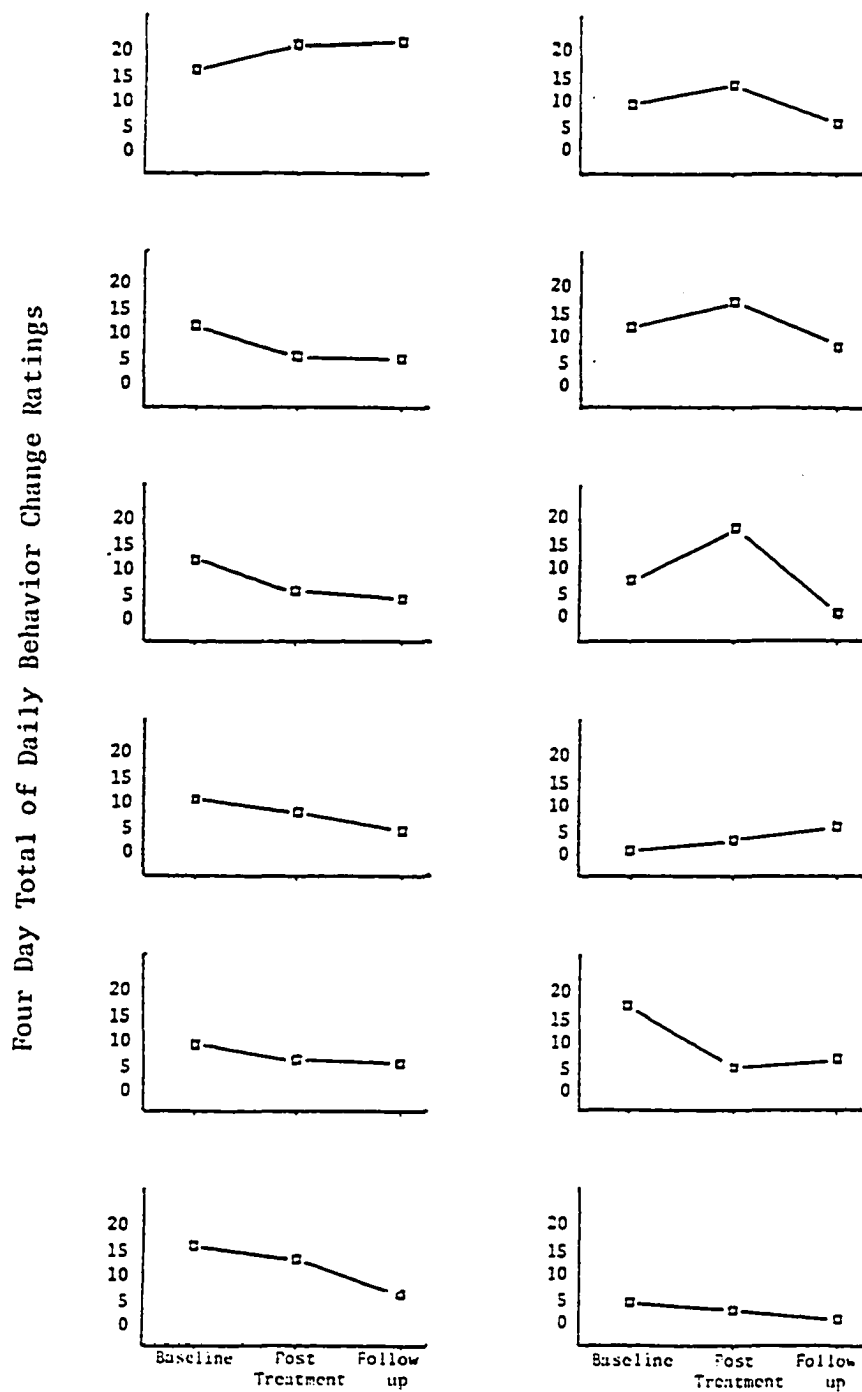


Figure 18:
 Behavior Change Treatment Group
 Individual Subject Daily Behavior Change Ratings

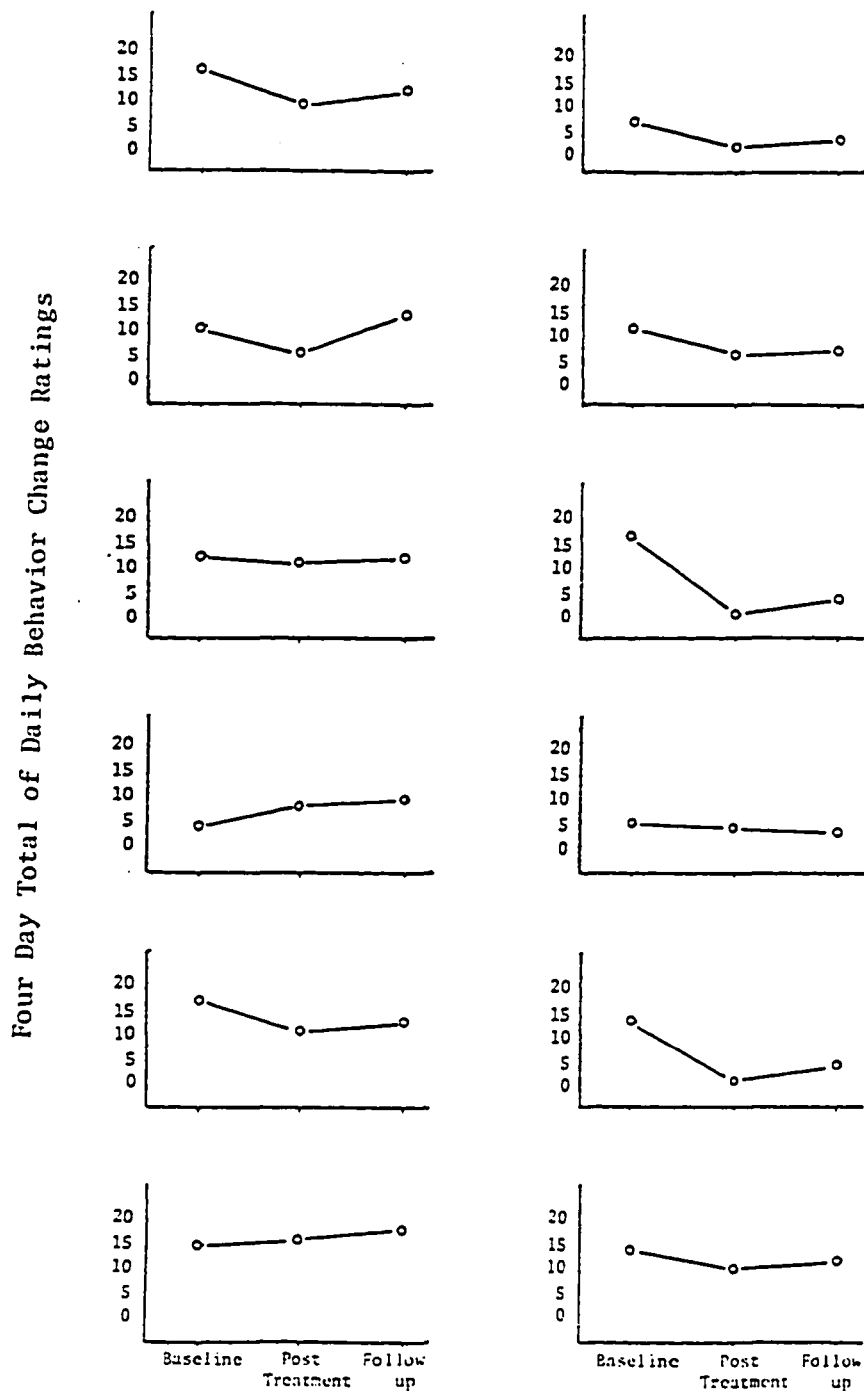


Figure 19:
Control Group
Individual Subject Daily Behavior Change Ratings

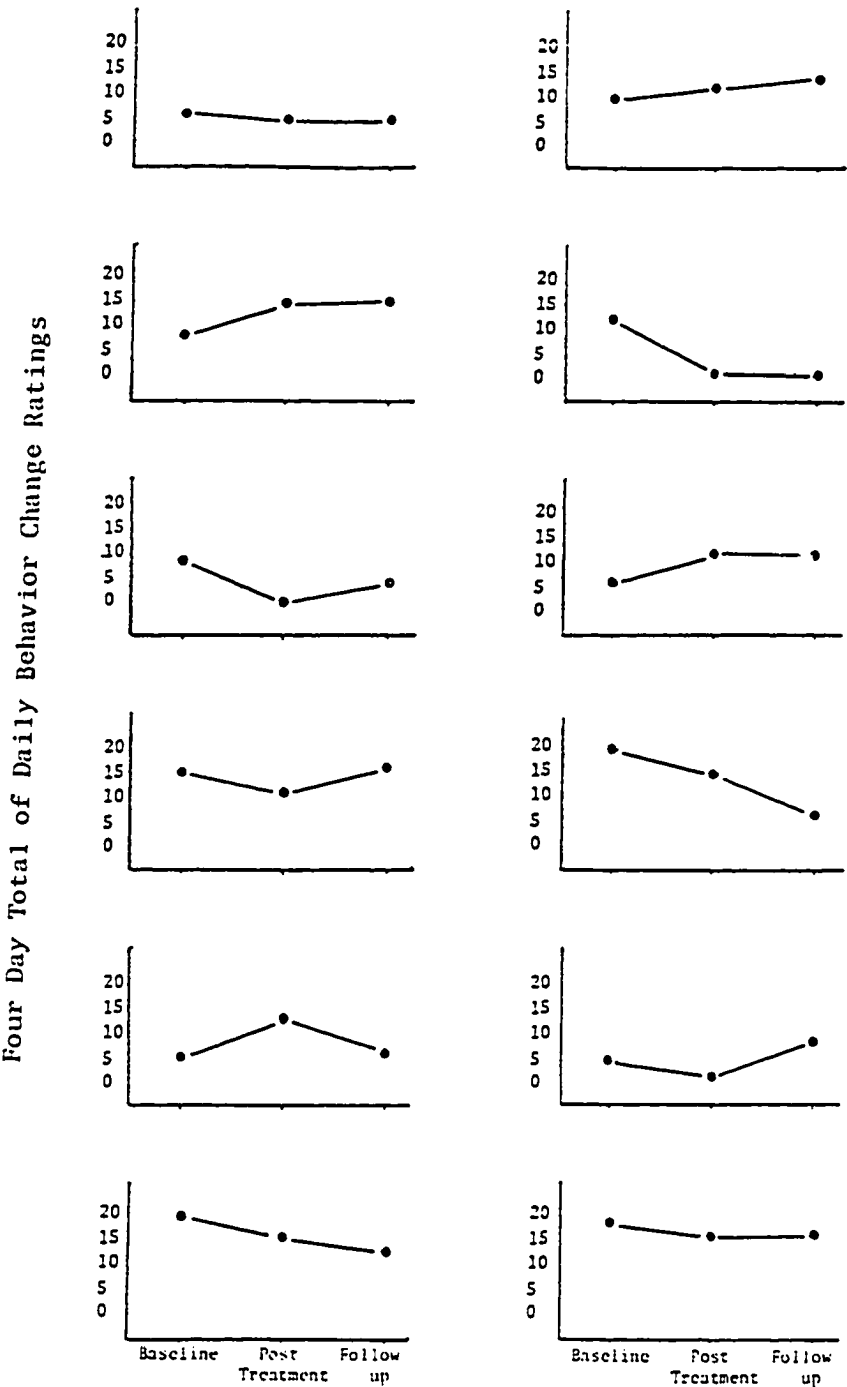


Figure 20:
Premenstrual and Postmenstrual Group Means
of Daily Depression and Behavior Change Ratings
for Baseline and Post-treatment Menses

