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Premenstrual Tension, Expectancy and Mother-Child Relations

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Thesis proposal presented to the School of Graduate Studies, University of Ottawa, as partial fulfillment of the requirements for the degree of Doctor of Philosophy.

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CURRICULUM STUDIORUM

Barbara Fraser Fradkin (née Currie) was born in Montreal, Canada on October 24, 1947. She received her Bachelor of Arts degree in Honours Psychology in 1968 from McGill University, Montreal, and her Master of Arts degree in Experimental Social Psychology in 1969 from the University of Toronto. The title of her Master's thesis was "Some Determinants of the Influence of Verbal Suggestion on Altruism".

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ABSTRACT

The role of expectancy in enhancing or mitigating premenstrual symptoms was examined in 51 mothers of preschoolers. Expectancy was manipulated by providing information either in support of a biological cause for genuine, universal mood changes or in support of a psychological cause arising out of negative societal myths. A third group were given no information. Mood, cognitive function, and mother-child interaction were assessed both at midcycle and premenstrually, and the results suggested that expectancy enhances symptoms. The Psychological group lowered their symptom expectations and reported less negative mood premenstrually as well as fewer symptoms at the end of the test month. The other groups reported no change in symptoms and greater premenstrual negative mood than the Psychological group. There were no changes in anagram solving but greater puzzle persistence and more positive interaction with the child during the premenstrual phase in all groups. The predicted premenstrual increase in negative maternal behaviour was not found, but the methodological limitations of laboratory observation may have contributed to this failure.

CHAPTER I

REVIEW OF THE LITERATURE

INTRODUCTION

Research, myth, and rumour have linked certain phases of the menstrual cycle, notably the premenstrual phase, to a vast array of evils ranging from increased irritability through poor school performance to increased suicide and violent crime. Yet controversy is still strong about the very existence of genuine cyclic fluctuations and about the mechanisms underlying them. The present paper will review the theories and the evidence, and will then report on a study designed to examine the role of symptom expectations in the alteration of moods, cognitive functioning, and behaviour. Surprisingly, no research has been done examining the effects of menstrual cycle fluctuations on maternal behaviour; thus, a second purpose of this study was to examine possible cyclic fluctuations in positive and negative maternal behaviour.

THE MENSTRUAL CYCLE

Physiology. The following exposition is drawn from Bell (1975), Vandewiele (1970), Dyrenfurth et al. (1974) and Yen et al. (1974). The menstrual cycle is the result of the delicate interplay between five main hormones whose secretion is regulated by a series of complex feed-back loops along the hypothalamic-pituitary-gonadal axis.

Gonadotropin releasing factor (GnRH) is secreted by the hypothalamus to stimulate the pituitary, which in turn secretes two gonadotropins, follicle-stimulating hormone (FSH) and luteinising hormone (LH). These act on the

ovaries to facilitate or inhibit their production of the two main ovarian hormones, estrogen and progesterone. Modulation of these cyclic hormones is thought to be the result of estrogen, whose blood level and rate of rise or fall acts on the hypothalamus and the pituitary to regulate their hormone production.

Briefly, at the onset of menstruation, both progesterone and estrogen are low, but FSH (under the action of GnRF) is at its maximum, stimulating new follicle growth in the ovary during the first week of the cycle. As the follicles mature, they begin to produce estrogen, at first gradually around the seventh day of a standard 28-day cycle and rising rapidly to a peak around the twelfth day. The increase in estrogen temporarily inhibits FSH and at a critical level around the twelfth day stimulates the pituitary to release a burst of LH, which triggers ovulation a day or so later. FSH shows a simultaneous but smaller peak.

At ovulation, LH stimulates the ovary to form the corpus luteum, a collection of cells in the ruptured follicle which is responsible for progesterone production. After ovulation, FSH and LH levels drop, estrogen levels first fall and then rise to a second plateau at about Day 20-23. Progesterone, which was almost non-existent in the preovulative phase, gradually rises to a peak around Day 22, and then both ovarian hormones begin to decline, first slowly and then rapidly from Day 25 to 28 to their low menstrual levels. This period of hormonal decline is generally referred to as the premenstrum.

The above description is representative of average trends, but even in normal, cycling women there is marked variation across women and across cycles

in the same woman, both in absolute hormone levels and in the relative patterns of hormone fluctuation. For example, the LH surge has been observed anywhere from 24 to 40 hours before ovulation, and the preovulative estrogen peak may coincide with ovulation or may precede it by as much as four days (Dyrenfurth et al., 1974). Furthermore, there are marked individual differences in total cycle length, with a length of 21 to 35 days being considered normal, as well as in the lengths of both the preovulative and post-ovulative phases. Generally, variation is greater in the preovulative phase, while the post-ovulative phase is 11 to 15 days long in over 90% of mature women (Presser, 1974). To confound matters further, however, ovulation cannot be reliably determined without direct viewing of the ovary; all indirect methods (basal body temperature, cervical mucus, midcycle pain, and assays of LH and estrogen) are subject to error of several hours in the case of LH to as much as several days in the case of cervical mucus (Vollman, 1974).

Notwithstanding these complexities, researchers have used the overall pattern of hormonal fluctuation to divide the menstrual cycle in various ways, depending on their research interest. Much of the interest, whether due to inherent negative bias or medical concern, has focussed on the paramenstrual (premenstrual-menstrual) phase.

Menstrual cycle fluctuations have been the subject of myth, superstition and scientific observation for centuries (Weideger, 1976), but "premenstrual tension" was first distinguished by Frank in 1931 as a clinical entity characterised by "indescribable tension and a desire to find relief by foolish and ill-considered actions" (Parlee, 1973, p. 454). Since then, the menstrual cycle has

been the focus of voluminous research, yet the body of fact and theory which emerges is far from solid. On the one hand, considerable evidence is accumulating which points to cyclic changes in sensory and seizure thresholds (Henkin, 1974; Messent, 1976; Vernikos-Danellis, 1972), MAO levels (Broverman et al., 1980; Klaiber et al., 1971), autonomic arousal (Asso, 1978; Little & Zahn, 1974; Wineman, 1971), emotionality, and a variety of other self-reported symptoms and complaints (Coppen & Kessel, 1963; Moos, 1968, 1969; Moos & Leiderman, 1978). On the other hand, the confusion, bias, and poor methodology found in many of the studies have led some researchers to claim that as yet, the evidence for a genuine, hormonally based Premenstrual Syndrome is scanty indeed (Parlee, 1973; Ruble, 1977, 1980; Ruble & Brooks-Gunn, 1979; Slade, 1981). Therefore, before considering the evidence concerning cyclic fluctuations in various areas, it is worth considering the major methodological issues which have muddied the waters in menstrual cycle research and contributed to the conflicting mass of data.

Methodological Issues. In 1973, Parlee presented a comprehensive critique of both the methodology and the basic conceptual biases in menstrual cycle research, and since that time a number of good critiques and reviews have appeared which offer either a broad critical perspective (Sherif, 1980; Sommer, 1973) or a critique of special issues such as hormone measurement (McClintock, 1981) and conceptual bias (Koeske, 1980; Parlee, 1978; Sommer, 1980). An overview of six major issues will be presented here.

First, the definition of the phases and the methods used to determine phase (or underlying hormonal status) have varied widely from study to study, some using broad categories (such as menstrual vs. intermenstrual) and indirect techniques for determining hormonal status (such as last reported period), while others operate with considerably more physiological precision (e.g., performing daily hormonal assays). Terminology varies widely as well, making inter-study comparisons even more difficult. For example, the premenstrum has been variously defined as the last two weeks, the last week, and the last two days. The choice of how to partition the cycle and determine phase status depends partly on the researcher's interest and partly on the technology and money available to him. For many psychologists, the need for a fairly large and usually volunteer sample, the limitations in money, and the inaccessibility of hormonal assay facilities preclude direct hormonal measurement. Although the menstrual phase is fairly obvious, the premenstrual phase is only so in retrospect, and determination of ovulation is a hit and miss affair. Basal body temperature pinpoints exact ovulation day in only 34% of cases (McClintock, 1981). Pain and mucus are equally unreliable (Vollman, 1974). Some studies arbitrarily choose the midpoint of the cycle; however, as noted earlier, the preovulative phase is the more likely to be lengthened or shortened by variation from the 28-day norm. On the other hand, the technique of counting back fourteen days from menses will still result in inaccuracies, since the post-ovulative phase can last from 10 to 15 days in the normal woman (Presser, 1974).

Even if hormonal assays are used, inaccuracies occur (McClintock, 1981). Individual differences in hormone levels and relative patterns can lead to false

estimates of cycle phase. In addition, there are differences in the sensitivity of receptor cells to circulating hormones at different times of the cycle (Yen et al., 1974) and also differences between individuals in sensitivity to and clearance of various hormones (Broverman et al., 1980). Furthermore, because LH is secreted in a pulsatile fashion, very frequent assays would be needed to detect the true peak, a requirement which is impractical for most psychological work. Koeske (1981) has even questionned whether cycle phase and hormone level should be considered as an independent variable in research design, or whether it more properly should be viewed as another dependent variable.

This problem of defining cycle phase leads directly to the second major methodological obstacle to accurate and adequate menstrual cycle research - namely, maintaining subject naivety. Keeping the subjects unaware of the interest in the menstrual cycle while at the same time adequately defining hormonal status is very difficult, particularly with existing (albeit necessary) standards of ethics. Once subjects are aware of the experimenter's interest, expectancies play a biasing role, in this case a compounded one since subjects harbour their own expectancies about menstrual cycle changes in addition to those they think the experimenter has (Koeske, 1981; Parlee, 1981; Rosenthal, 1966). Researchers have dealt with the problem either by providing false, misleading or incomplete information (Paige, 1971), by obtaining menstrual data in retrospect (Rogers & Harding, 1981), by hiding menstrual questions in a long questionnaire on general health (Swandby, 1981), or most often by simply admitting to an interest in the menstrual cycle and ignoring the possibility that expectancies may bias their results.

One of the first three tactics may be applicable for some research designs and questions, but there are occasions when none is quite adequate. If phase prediction is required, for example, so that specific testing can be done at a certain phase, the date of the last menses obtained in a casual health questionnaire is unlikely to be an adequate cycle marker.

Rogers and Harding (1981) compared the daily mood and symptom self-reports of three groups of subjects; a group of women informed of the focus on the cycle, a group of uninformed women and a group of uninformed men. Cycle-related changes occurred only in the informed women; uninformed women showed random ups and downs over their cycle, as did the men. This study highlights the importance of subject naivety, particularly when self-report measures are being used, although expectancy effects can bias any measures (Rosenthal, 1966).

The particular vulnerability of self-report underscores the third methodological issue which has muddied the field; the choice of measure. There are few standardised measurement tools for menstrual cycle research. What measure is chosen depends, of course, on what phenomena are being studied, but once a particular phenomenon has been defined, any number of measures have been used to study it, and often these measures have little comparability from one study to another. Some are sensitive, reliable, and have demonstrated validity for the phenomenon in question; others are crude and global. This problem is particularly acute in the study of cycle-related negative affect, where masses of data have been accumulated using numerous measures and yet few definitive conclusions can be made. Most studies have relied on self-report (either daily or retrospective), and of these some have used a well-standardized, multi-dimensional measure such

as the Profile of Mood States (Parlee, 1980) or the Multiple Affect Adjective Checklist (Haskett et al., 1980), while at the opposite extreme some have used a unidimensional, bipolar scale like "good vs. bad" (May, 1976; Reynolds, 1969). A handful of studies have relied on thematic analysis of free-associative talk (Gottschalk, 1962; Paige, 1971), a technique based on psychoanalytic theory and reflecting presumably unconscious as well as conscious affect. Whether or not this technique is less subject to expectancy bias is an open question (Parlee, 1978; Sherif, 1980). A review of the literature revealed only one study where an outside observer was used to rate the moods of the subjects (Silbergeld et al., 1971), although this would appear to be a useful technique. Silbergeld's study measured affect through self-reports, thematic analysis, and third-person ratings, and he found the three correlated poorly. Similarly, Dan (1980) compared mood self-reports and free-association material over one month in a small sample of husbands and wives, and found no correlation between the two measures. She suggested that we do not yet have an adequate theory of mood and emotion from which to derive meaningful measures of affect.

In addition to the choice of dependent variables and measures, differences in research design have resulted in systematic bias and lack of comparability across studies. Smolensky (1980) has distinguished seven different designs used in menstrual cycle research, but most studies use one of three basic designs, one cross-sectional, involving measures from different subjects at different phases, and two longitudinal, the first involving two to five measures on the same subject over one cycle and the second involving daily or near-daily measures on the same subject over the cycle. The cross-sectional method typically uses

large samples and focusses on general averages. The second method uses somewhat smaller samples and, although still focussing on average trends, has the added advantage of being able to observe the nature of within subject change from one phase to another. Both these methods, however, are interested in average trends and regard all other features contributing to individual variation as random noise to be cancelled out across subjects and phases. In the third model, by contrast, individual fluctuations across the cycle are a prime focus. Almost by necessity, sample sizes are small and the same subject may be studied over several cycles. The first two designs, which use group averages, tend to amplify the menstrual cycle effect by strengthening the signal: noise ratio, so that small and experientially insignificant cycle effects may be emphasized. The third design tends to weaken the signal: noise ratio, so that menstrual cycle effects which are weak but nonetheless operative may be lost within the larger fluctuations caused by daily life events and other rhythms (like circadian or social week). Typically, therefore, the group averages design yields significant cycle effects while the individual longitudinal design does not (Parlee, 1980; Swandby, 1981). All three models are useful in clarifying the full picture and are needed to counteract each other's biases. The small samples of the longitudinal studies limit the generality of their findings, particularly in a field where individual variation is great.

Before leaving the discussion of research design, a further methodological note should be made about retrospective, one-time measurement. Now that methodology in this field has become increasingly sophisticated, this design is used less frequently, but it still occurs, most often in correlational studies between

menstrual symptoms and personality or attitudinal variables (Good & Smith, 1980; Gruba & Rohrbaugh, 1975; Hain et al., 1970). It has the advantage of being easy to use with large samples, but the validity of retrospective data about mood and symptom changes over the previous month has been called into serious question. Besides being vulnerable to the problems of self-report, it is additionally vulnerable to bias in recall which may result from the influence of culturally stereotyped expectations. Thus, Parlee (1980) found none of the usual cycle-related mood changes in the daily self-reports of her seven experimentally naive subjects, yet retrospective symptoms self-ratings at the end of the test month followed the culturally expected pattern. May (1976) and Alplanalp (1979b) also found no correlation between prospective and retrospective mood ratings. On the other hand, a recent study (Maitland-Shilling, 1981) found moderate to strong correlations between daily mood and retrospective symptom ratings when both are referring to the same phenomenon (e.g., negative affect, as opposed to overall symptom score).

The fifth methodological issue which contributes to the present confusion in the field is the nature and size of the sample. As noted above, design practicalities influence sample size, but the bias introduced by the kind of sample used is much more subtle. Researchers tend to use that which is most readily available; hence the majority of the menstrual cycle work done by psychologists uses undergraduate students whose ages range around eighteen to twenty-two years. Quite apart from the biological differences between twenty and thirty-five year-olds, striking cultural, attitudinal, and personality differences

might be expected as well. Although this will be discussed in a later section, there is some suggestion that premenstrual tension increases with age and/or parity. Thus generalising from an essentially asymptomatic population to a potentially symptomatic one may result in serious distortion. At the other extreme, medical researchers tend to use their own patients, either those who report to a clinic seeking relief of symptoms or at least the general outpatient clinic population, who may be on the average older and parous. Generalization from this selected population to all of womankind may result in equivalent distortion. Similarly, psychiatric researchers may see more subjects in whom psychiatric complaints are more prominent, thus their conclusions regarding the relationship of premenstrual symptoms to overall psychopathology, for example, may be biased.

In addition to this unintended bias, some studies deliberately screen subjects so that only those with particular symptoms will be included, while others make a random selection of healthy normal volunteers. In the former studies, the chance of finding cycle effects is augmented, in the latter true cycle effects which occur in a smaller subgroup may be lost in the overall averages.

These sampling biases are further compounded by the most fundamental methodological problem of all; the bias introduced by the researcher's own model regarding the etiology (or even the existence) of menstrual cycle changes. Etiological models will be discussed in a later section but they fall into four main categories - biological, psychosocial, psychiatric, and psycho-socio-biological (i.e., combination) models. As an oversimplified generalization, medical

researchers tend to espouse the first, psychologists the second or fourth, and psychiatrists the third. This conceptual bias influences the sample selection and the choice of dependent variables which are singled out for study, as well as those which are ignored (Sommer, 1980).

Even beyond the scientific bias of the individual researcher may lie his or her personal bias. Not surprisingly, the field is replete with preconceived notions, stereotypes, and political implications for the ongoing battle of the sexes, and everyone is able to find proof of their view in the confusion of cloudy definitions, inadequate measures, biased designs and samples, and contradictory data.

Review of Cycle Effects. A complete review of research on menstrual cycle changes is neither possible nor appropriate here, but the reader is directed to general reviews by Parlee (1973), Smith (1975) and Friedman et al. (1980). Research can be divided into four broad categories - physical, affective, behavioural, and cognitive. Relevant reviews will be noted as each category is discussed.

a) Physical changes. Of all the areas, the argument for genuine cycle changes in physical variables is the most solidly grounded. From self-report data, Moos (1968) determined that about 35-45% of women complained of at least mild menstrual pain and about 30% complained of menstrual water retention. The figures for the premenstrual phase were 25% and 34% respectively. By contrast, similar complaints at other phases of the cycle ranged around 5% (except for headache, which was 14%). These findings are based on the Menstrual Distress Questionnaire (Moos, 1968), a checklist of 47 symptoms which

have been factor-analysed into eight symptom clusters - pain, concentration, behaviour change, autonomic reactions, water retention, negative affect, arousal (positive), and control (non-cycle-related symptoms). The MDQ has been widely used over the last ten years and remains one of the rare standardized psychometric tools in the field. Moos based his standardization norms on a large (839) but restricted sample (wives of graduate students, between twenty and thirty years old), and therefore his percentages may be biased. Using another self-report questionnaire developed in Britain, Coppen and Kessel (1963) found incidences of menstrual and premenstrual physical complaints even higher (e.g., menstrual pain in 45% and premenstrual swelling in 72%). Their sample, 465 patients at outpatient family planning clinics, covered a wider and probably older age group.

At this point, it is worth noting that estimating the incidence of menstrual cycle symptoms depends on what measures are used and what cut-off point is chosen. Thus incidence estimates vary widely across studies; one recent British survey using the MDQ reported an incidence of 92% for premenstrual pain and 87% for water retention (Rouse, 1978). The cut-off point is not specified but is presumably different from the one used by Moos.

Based on these findings, it seems clear that a substantial proportion of women report physical changes in their bodies during the premenstrual and/or menstrual phases, including headaches, swelling, weight gain, cramps and breast pain. The experience of pain is difficult to measure objectively, but swelling and weight gain can and have been objectively studied, yielding conflicting results. In a careful longitudinal study of twenty female college students

tested daily over one cycle, Voda (1980) failed to find any systematic cyclic change in weight or swelling. On the other hand, Freedman et al., (1974) in a massive longitudinal study on the effects of birth control pills, demonstrated two monthly peaks in weight - at ovulation and again premenstrually - in subjects not using birth control pills. This group comprised on the average 639 women with a mean age of 35 years, considerably older than the Voda sample. Average weight change was 2½ to 4 pounds.

To complicate matters further, however, cyclic changes in food preferences (with a preference for sweets in the latter half of the cycle) and in the amount of food eaten have been demonstrated in animals, including primates (Messent, 1976), so the cause of premenstrual weight gain remains unclear. Neither of the above studies controlled for diet, but Janowsky et al. (1973) did so and found a weight gain during the latter half of the cycle, reaching a peak in the premenstrum and dropping rapidly at menses. He attributed this rise to an increase in the K/Na ratio with resultant water retention.

Apart from possible water retention and weight gain, other studies have demonstrated cyclic changes in almost every bodily system. Using special time-series analysis techniques, Smolensky (1980) has reanalysed the data from a large number of studies on variables ranging from metabolic and physiological to affective, and his results serve as a wide-ranging review of the area. He concluded that there were significant monthly fluctuations in reaction time (other studies have not confirmed this; see Hutt et al., 1980; Zimmerman & Parlee, 1973) taste and pain thresholds, and visual and acoustic acuity, in addition to changes in a great many bodily substances and processes such as

renin, aldosterone, urinary excretion, sweat gland activity, basic metabolic rate, basal body temperature, pulse rate, capillary permeability to fluid and protein, serontonin, etc. Many of these peak in the luteal phase.

Messent (1976), in a review of physical and physiological changes, noted that changes in olfactory and tactile sensitivity also occur, with greatest sensitivity at ovulation and a marked decrease in the luteal phase. Zimmerman and Parlee (1973) found no change in GSR or reaction time, but a decrease in arm-hand steadiness from the luteal to the premenstrual phase.

The question of cyclic changes in arousal is of particular interest because of its possible relationship to the jitteriness, irritability and emotionality of premenstrual tension (Asso, 1978). Thus, time estimation, skin conductance, GSR, reaction time, critical flash-fusion, MAO levels, EEG driving responses, EEG changes in alpha frequency, GSR habituation, heart rate, blood pressure and respiration rate have all been studied, and the resulting picture is contradictory. Physiological indices which purport to measure the same thing correlate poorly (Kopell et al., 1969), phase definitions vary from study to study, and theoretical interpretations differ. Asso (1978), in a recent review of twenty-four menstrual cycle studies on arousal, points out that the concept of arousal is not a unitary one, and that there is inadequate understanding of how a particular physiological index (such as MAO level) relates to arousal level. She divides the indices used in the studies into three categories according to the dimension of arousal each is assumed to measure: 1) behavioural/psychological arousal, 2) autonomic arousal, and, 3) cortical arousal (activation). The first involves mainly self-report studies of anxiety, whereas the

latter two involve more objective physical measures. She concludes that, taken as a whole, the evidence points to a premenstrual increase in behavioural/psychological and autonomic arousal, but that the evidence on cortical arousal is inconclusive, suggesting either no change or somewhat lower CNS arousal premenstrually. However, she phrases her conclusions in very tentative terms, pointing out that indices of cortical arousal in particular relate poorly to one another, that many of the studies showed a shift through the entire second (luteal) half of the cycle (e.g., Creutzfeldt et al., 1976; Klaiber et al., 1971; Little & Zahn, 1974) which does not explain disturbances in the premenstrum alone, and that we do not know how the three dimensions of arousal relate to one another.

In a later study, Friedman and Meares (1979), using GSR habituation to an auditory stimulus as a measure of CNS arousal, concluded that the preovulative phase is one of heightened sensory sensitivity and cortical arousal, but that there is an abrupt shift to lower CNS arousal and reduced sensitivity around ovulation which continues through to the onset of menses. This supports Asso's tentative conclusion of lowered CNS arousal in the premenstrum. Friedman and Meares suggested that the abrupt shift from low (luteal) to high (menstrual) arousal is problematic for some women and may be a factor in irritability at this time. While interesting, this does not account for the earlier onset of premenstrual tension which some women report a week before menses.

It has been suggested that arousability, rather than baseline levels of arousal, is important in premenstrual tension. Marinari et al. (1976) found that although baseline levels of adrenocorticoids (hormones which are highly

reactive to psychological stress) do not vary across the cycle, premenstrual subjects showed a significantly greater adrenocorticoid response to stress, and concluded that women are more physiologically reactive to stress in the premenstrum than at other times. Interestingly, their subjects did not report feeling emotionally reactive to that same stress.

Whatever the final verdict on arousal and the menstrual cycle, it seems clear that there are cycle-related changes in several physiological processes which are presumed to relate to overall levels of activation and reactivity. However, it should be noted in conclusion that most of these physiological processes also show equivalent (or even greater, as in the case of arousal) circadian and ultradian fluctuations in both men and women (Colquhoun, 1971; Ferin et al., 1974). Indeed, as Parlee (1978) pointed out: "rhythmicity may in fact be the norm in biological and psychological phenomena" (p. 197).

b) Affective changes. As noted earlier, affective changes in the menstrual cycle have been measured mainly by self-report checklists and rating scales, occasionally by thematic analysis of free-associations, and even more rarely by third-person ratings (Silbergeld et al., 1971). Most self-report studies have found that a substantial percentage of women (31% in Coppen & Kessel, 1963; 40% in Moos, 1968) complain of some form of negative affect in the premenstrual and/or menstrual phase, variously defined as depression, mood swings, irritability, tension, anxiety, etc. (e.g., Dan, 1980; Golub, 1976a, 1976b; Maitland-Shilling, 1981; Reynolds, 1969; Silbergeld et al., 1971). Increases are small, compared to psychiatric mood disturbance (Golub, 1976a) and individual variability is marked. Some studies have found no increase at all (Alplanalp

et al., 1979a, 1979b, 1980; Marinari et al., 1976; Persky, 1974; Slade, 1981; Swandby, 1981; Zimmerman & Parlee, 1973). Others have found mood swings due to environmental factors greater than those attributable to menstrual phase (Parlee, 1980; Wilcoxin et al., 1976). Furthermore, Moos and Leiderman (1978) found that 13% of their sample reported only positive changes at the premenstrum, such as bursts of energy and excitement. Parlee (1980) also found a premenstrual increase in positive activation in a small sample of healthy, well-adjusted women.

Using psychoanalytic theory, Gottschalk (1962) devised a system of scoring verbal free-association material along two dimensions - hostility (inwards and outwards) and anxiety. Using this technique he failed to find consistent cycle fluctuations but used only five subjects. Three later studies did find significant effects for both variables (Ivey & Bardwick, 1968; Paige, 1971; Silbergeld et al., 1971), and one found no cycle effects (Dan, 1980). Silbergeld, however, found that hostility was highest at the mid-follicular stage, low at ovulation, and high again premenstrually. Paige found no such biphasic relationship. However, the theoretical basis for this scoring system lacks sound empirical confirmation and, at the very least, the relationship between unconscious affect and self-reported affect is uncertain. In Silbergeld's study, premenstrual subjects were also rated higher on tension and aggression by trained observers in a double-blind set-up, thus providing rare objective evidence of visible affect changes during the menstrual cycle.

Slade (1981) reviewed the evidence for cycle-related affective changes and found it far less solid than most researchers assumed. Bias in design and

sampling has exaggerated the changes, and as she pointed out, attitudes and expectations (and therefore perhaps the severity of premenstrual symptoms) have changed in the fifty years since the early students of premenstrual tension did their pioneering work.

On the other hand, Dennerstein and Burrows (1979) reviewed twenty-four prospective studies of affect (measured by self-report, physiological indices and behavioural measures) and although they found many inconsistencies, they felt it safe to make some "cautious generalizations", namely, that most studies found a premenstrual increase in self-reported negative affect and a peak in positive moods at the follicular or ovulative phase.

Thus, it seems that even reviewers cannot agree on the evidence they review. Slade's criticisms are well taken, and as pointed out in the methodology section, sampling biases can augment or decrease the likelihood of finding cycle effects, group designs instead of longitudinal designs favour the detection of weak cycle effects, and the use of naive subjects reduces the chance of finding cycle effects. Beyond this, one of the major reasons for the contradictory findings is the use of different sampling procedures. Some studies preselect symptomatic women, while most psychological studies use random samples. Sommer (1980) pointed out that these two sampling procedures reflect different underlying models of menstrual stress. The random-sample approach assumes that menstrual cycle effects (or lack of them) are universal, whereas the selected-sample approach assumes that there are individual differences, that the menstrual cycle may or may not produce menstrual stress, and further that different groups of women may experience different types of menstrual stress. Since it has already

been observed that only about 40% of women complain of premenstrual negative affect, it seems on the face of it unwise to take a random sample of women in order to demonstrate cycle effects, when 60% of the sample is likely to disprove the hypothesis. As Sommer pointed out, it seems unprofitable to continue trying to demonstrate the universality of an effect (or non-effect); instead, we ought to be examining sub-groups of women who seem to have features in common in order to untangle the variables involved in their particular menstrual stress (or equally important, their lack of stress).

c) Behavioural changes. The evidence concerning cyclic changes in behaviour comes from three sources: 1) self-report, which yields a measure of perceived rather than actual behaviour change, 2) the incidence of actual, unusual events, such as suicides, crimes, and illness, and, 3) changes in the frequency of everyday events or performance. General reviews of behaviour change can be found in Sommer (1973) and Redgrove (1971). Moos (1968) found that about 25% of women report at least mild changes in behaviour, such as decreased efficiency, at menstruation, and about 15% report a similar change in the premenstrum. However, as will be discussed in the section on cognitive changes, women often perceive themselves as being more impaired than they actually are. Alplanalp (1979a, 1979b, 1980) has recently designed a long-term study using an individualized, daily Social-Sexual Activities Log which subjects used to record their activities and which may prove to be a promising compromise between objective (and often impractical) observation and impressionistic self-report. At latest report (Alplanalp, 1979b), no significant cycle effects had been found in subjects' enjoyment of daily activities.

Suicide, attempted suicide (Tonks et al., 1968), threatened suicide, acute psychiatric admissions (Glass et al., 1971), crime and especially violent crime (Dalton, 1961; Morton et al., 1953; d'Orban & Dalton, 1980) have all been reported in the literature as occurring more frequently in the premenstrual phase. Wetzel and McClure (1972) reviewed 23 studies made of suicide (completed, attempted, and threatened) since 1900 and they found many contradictions. However, they concluded that there is an increased risk of all three kinds of suicidal behaviour in the premenstrum and a reduced risk of completed suicides in the menstrual phase, although attempts remain high. They hinted that the premenstrual phase may be associated with more violent and lethal methods. Sommer (1973) reviewed the suicide literature and concluded that the only consistent finding was a decrease in the follicular phase.

The foregoing studies on crime, suicide and psychiatric relapse have been criticised by Parlee (1973) on several grounds: 1) that the behaviour of a select group of pathological women says nothing about the behaviour of normal women, 2) that the act committed during one premenstrual phase says nothing about how the woman dealt with the several hundred premenstrual phases she lived through before and after the one act, and, 3) that in any case the trauma of the arrest or suicide attempt or hospitalisation may have triggered premature menstruation, thus creating a spurious link between the premenstrum and the act. Nonetheless, the findings do lend support to the possibility of severe premenstrual affective disturbance in at least a small percentage of women.

Besides these striking negative acts, a number of other behaviours have been linked to the premenstrum in various studies. Dalton (1970) found that the

accident rate for women and also for the children of women in the paramenstrual phase was higher than expected by chance, and also that mothers sought help for their children's illnesses more often in the paramenstrum. Tuch (1975) made a similar finding. Four prospective studies correlating aggressive behaviour with the menstrual cycle have yielded mixed results; three found a premenstrual incarese (Ellis & Austin, 1971; Schonberg et al., 1976; Teitler, 1979) while one found no cycle effect (Herlihy, 1977). These will be discussed further in the section on maternal behaviour. It is interesting to note that the data used to demonstrate a premenstrual peak in aggressive behaviour can be alternatively interpreted as showing a striking luteal decrease (Ellis & Austin, 1971).

Studies on more benign behaviours such as production decline, worker absenteeism and athletic performance are reviewed in Sommer (1973) and Redgrove (1971). Sommer noted that cycle effects in athletic performance may depend on the sport - for example, swimmers are less confident of their performance during menses while volleyball players are not, a difference which may reflect cultural attitudes. Redgrove (1971) noted that two studies on athletes revealed pre-menstrual decrement followed by menstrual improvement. However, the menstrual cycle may be affected by rigourous physical training (resulting in amenorrhea, for example), and thus evidence from top young athletes may not generalize to women in general.

After reviewing the evidence, Sommer (1973) concluded that "those instances in which cycle effect occurs are those in which responses are mediated by social and psychological factors: subjects express feelings and interact with

their social environment in ways consonant with their expectations about themselves and about the demands and expectations of (their) social milieu. When those expectations were altered ... or controlled for ... the effect vanished." (p. 531).

Redgrove (1971) conducted a series of longitudinal studies on the daily work performance of small samples of British working women and was able to demonstrate no cycle effects. She pointed out that in most jobs poor efficiency can be offset by increased effort or by greater efficiency the following day. Jobs where a worker has to perform at top capacity continuously may be affected by the cycle, but she did not have any data to support this possibility and it seems likely that such a demanding job would be adversely affected by any number of other disruptive factors (such as lack of sleep and personal stress) that affect everyone, male and female. Other studies relevant to the issue of performance will be discussed in the following section.

In summary, the evidence suggests that there is a premenstrual increase in negative behaviours, such as crimes and suicides, in a small proportion of abnormal women, and that biological factors may interact with socio-cultural expectations to yield a premenstrual decrement in some athletes and workers, while having no effect or the opposite effect on others. But hard data pointing to clear-cut performance decrements at any phase of the cycle is non-existent. It is important to note that all the studies focussing on the premenstrum have been looking at negative features - disturbances, deficits, etc. - while none have examined positive behaviours with the exception of sexual arousal.

Friedman et al. (1980) and others, on reviewing the evidence on sexual behaviour, found a midcycle increase in sexual arousal in both primate and human females and a second peak in the premenstrum (Bardwick, 1971; Michael, 1969; Moos et al., 1969). One study measuring activity level by use of a pedometer found an activity peak at midcycle and two lesser peaks premenstrually and menstrually (Morris & Udry, 1970).

d) Cognitive changes. Studies on paramenstrual deficits in intellectual and cognitive functioning have yielded the least positive results. According to Moos (1968), a small proportion of women (about 14%) report difficulty concentrating during the premenstrum, but this is not generally supported by objective tests. Sommer (1973) reviewed the evidence for perceptual-cognitive cycle effects and concluded, as noted above, that when performance is examined under carefully controlled conditions, no cycle effect is noted. The results of her own study (Sommer, 1972) support this notion, in that no cycle effects were found on the complex Watson-Glaiser Critical Thinking Appraisal.

A great deal of research has been done on this issue since Sommer's review. Studies of reaction time, either simple or choice, have revealed no deficit (Hutt et al., 1980; Slade & Jenner, 1980a; Zimmerman & Parlee, 1973). Aware that the presence of asymptomatic subjects may mask genuine cycle effects which may occur in premenstrual sufferers, a number of studies have selected only women complaining of negative affect and/or poor concentration. Thus, Walsh et al. (1981) failed to find a paramenstrual decrement in examination performance in 244 female medical and paramedical students, even in those who complained of quite severe symptoms. Using symptomatic subjects, Slade and Jenner (1980a)

found a small premenstrual decrement in performance of a secondary task only when the task was extremely difficult. They suggest that in ordinary daily functioning, so many other factors play a role in performance that the cycle effect is negligible. In another carefully controlled prospective study, Golub (1976b, 1980) administered a battery of 15 cognitive tests known to be sensitive to physiological and emotional factors to a group of older, high-symptom women. She found no consistent cycle effects and concluded that even in symptomatic women the mood changes associated with the menstrual cycle are so small (compared to psychiatric outpatients or highly stressed experimental subjects) that they exert no effect on intellectual efficiency.

Some authors have suggested that women expect poorer performance and increase their efforts to compensate (Munchel, 1979; Redgrove, 1971; Rodin, 1976), and therefore decrements are only evident in the most complex tasks, as in the Slade and Jenner study above. Rodin (1976) found that when women were able to re-attribute task-arousal to their menstrual cycle, they performed better compared to low-symptom subjects who could not re-attribute. It has been demonstrated that intellectual impairment produced by alcohol can be offset if the subject is aware of his condition and expects to perform poorly (Williams et al., 1981), although this compensation is more effective with simpler tasks.

A group of researchers (Broverman et al., 1968, 1980; Klaiber et al., 1971), studying the effects of sex hormones on brain processes and cognitive functioning, have suggested that estrogen increases CNS activation, probably by depressing MAO levels, while progesterone, as an antagonist to estrogen, increases CNS

inhibition. They argue that some tasks are facilitated by CNS activation (notably psychomotor speed and accuracy; rapid attentional shifts and strongly automated habits) while others require greater CNS inhibition (notably perceptual restructuring tasks, and complex judgment tasks requiring suppression or delay of obvious responses). Most of their research has focussed on sex differences in activation-inhibition (females being the more activated), as an explanation for the observed differences in cognitive function. However, they also postulate that MAO fluctuations across the menstrual cycle affect tasks requiring different CNS activation-inhibition balance (Broverman et al., 1980). Specifically, estrogen levels in the preovulative phase ought to facilitate automated and speed tasks, whereas progesterone dominance in the luteal phase ought to facilitate perceptual-restructuring tasks.

Three recent studies set out to confirm these hypotheses, with mixed success. Komnenich et al. (1978) administered three automated tasks and two restructuring tasks to a small sample (nine no-pill, nine pill females, and nine males) four times over one month and found cycle differences only in the 'no-pill' group (i.e., naturally cycling women). The only significant effect was lower performance in the preovulative phases on the two restructuring tasks (inhibition-facilitated). They claim this provides support for the Broverman theory, although as Hutt et al., (1980) pointed out, there were no differences across the cycle in automated tasks, despite the prediction of a luteal drop. Hutt et al. (1980) tested choice reaction time (an activation-facilitated task) and found no cycle effects and therefore no support for Broverman's theory.

Graham (1980) measured estrogen levels and obtained 15 cognitive scores from 48 naturally-cycling young women at three points in the cycle (early follicular, ovulative and luteal). Her battery was chosen to include inhibition- and activation-facilitated tasks and the results do not give clear-cut support to the Broverman theory. She found that estrogen levels were not related to a specific cognitive style. She did find significant cycle effects on some of the cognitive tasks, but these were not related to the activation-inhibition dimension; her main finding was that cognitive performance on most of the tasks which showed cycle effects seemed to be better in the luteal phase. Since the premenstrum is not assessed in this study, it is not known whether this luteal improvement is sustained through the premenstrum.

In summary, the studies on overall cognitive performance generally reveal no cycle effects, even in high-symptom women, except possibly in the most difficult tasks of concentration. However, more work is needed to clarify the possible relationship of estrogen and progesterone to the performance of specific types of cognitive tasks.

Correlates of Premenstrual Tension. Age, parity, general neuroticism, and specific difficulties with femininity have all been reported to be correlated with premenstrual symptomatology, yet the evidence for each is scanty, muddled, or both.

a) Age and parity. Dalton (1977, 1979) and other clinicians have noted an increase in severity and incidence of premenstrual tension as women get older

and/or have children (these two factors often go together). It has in fact been dubbed the "mid-thirties" syndrome (Golub, 1976b). However, Moos (1968) found premenstrual complaints only weakly correlated with age ($r = .12$, but he notes the limited age range of his sample). Only one study (Rouse, 1978) could be found in which the sample was large enough (392) and the age range wide enough (15 to 55 years) to permit an adequate test of this relationship. Rouse did find a significant age effect; women over thirty years old reported more premenstrual and menstrual symptoms on the MDQ than did younger women, and this was particularly true of naturally-cycling women. No mention was made of parity. Since Rouse's was a cross-sectional study, one cannot say whether symptoms actually increase with age within the same woman or whether changes in cultural conditioning affects different generations of women in ways which alter their experience of symptoms. A review of the literature revealed only one study which investigated parity (Banks & Beresford, 1979) and its findings were inconclusive. They found that generally, among non-pilltakers, premenstrual symptoms tended to increase with number of children, but this effect depends on age, marital status, and type of symptom.

Dalton (1977) noted that premenstrual tension sometimes begins following the birth of a child. She also observed a relationship between suffering from pre-eclampsia and/or post-partum depression and the subsequent development of premenstrual tension. According to her figures, 86% of pre-eclampsia victims later developed premenstrual symptoms, as measured by personal interview. Similarly, 90% of post-partum depressives subsequently developed premenstrual

tension. Dalton feels that the cause of all three disorders is progesterone deficiency, and although the figures and the theory are provocative, further replication under more rigourous scientific control is needed.

b) Neuroticism. The belief that premenstrual tension is a manifestation of a woman's general neuroticism, and/or conflicts over her sexuality stems in part from original psychoanalytic formulations concerning women, sexuality, and symptom formation. The early psychiatric literature on premenstrual tension is replete with stereotyped and sexist theorising based on meager anecdotal evidence. Shader and Ohly (1970) provide an interesting review of the diverse explanations advanced by psychiatrists.

These early explanations have inspired a large number of studies measuring various aspects of personality, femininity, acceptance of the "female role", sexual attitudes, and the like. Diverse personality measures such as the MMPI, CPI, MPI, STAI, 16PF, and Taylor MAS make inter-study comparison difficult. Golub (1976b) found that women suffering from premenstrual negative affect were no more anxiety-prone than the general population (as measured by the STAI), and Walsh et al. (1981) found no correlation between Eysenck's neuroticism score and premenstrual symptoms although neuroticism was correlated with menstrual symptoms.

Apart from these two studies, most researchers (Coppen & Kessel, 1963; Good & Smith, 1980; Gruba & Rohrbaugh, 1975; Halbreich et al., 1977; Slade & Jenner, 1980b; Watts et al., 1980), have reported some correlation between self-reported premenstrual symptoms (either somatic, emotional, or both) and general neuroticism, notably anxiety. However, many of these studies did not correct for general level of complaining by subtracting intermenstrual scores

from premenstrual scores. Furthermore, the correlations, although statistically significant because of the large samples used, tend to be weak, ranging from $r = .20$ to .35. This suggests that even if generalized neuroticism and anxiety are factors, they may not play a large role.

Assuming that premenstrual tension is correlated with general anxiety, psychological distress, symptom-complaining, or whatever, several possible explanations come to mind. First, neurotic, anxious people may be inclined to exaggerate or focus excessively in minute changes in bodily cues which other people simply ignore. Second, the experience of recurrent emotional upset which affects a quarter of every month may make them more tense and anxious in general, or at least make them check off such items on a personality questionnaire (this explanation is rarely considered, but seems plausible). Third, both premenstrual tension and generalized emotional distress may result from some underlying physiological sensitivity or instability which may be exacerbated by different hormone levels.

The picture is further complicated by the increasingly evident fact that the premenstrual syndrome is not a unitary concept. Recently, Moos and Leiderman (1978) factor-analyzed the scores of 579 women on the eight scales of the MDQ, and found not one or two major clusters but about twenty different clusters which represent unique patterns of symptom complaints. Thus, some women complain only of negative affect in the premenstrual phase, others of negative affect, pain and poor concentration in both menstrual and premenstrual phases, etc. Moos concluded that one cannot therefore talk of a "Premenstrual Syndrome" as a cohesive, unitary set of symptoms, and that much of the confusion

in research and treatment reflects the underlying heterogeniety of cycle changes. Some studies have found neuroticism related to affective but not to somatic complaints, others have found the reverse.

Recently, Haskett et al. (1980) studied 42 women complaining of severe premenstrual tension, and they concluded that there were two different subgroups of women, who present different symptom profiles. The first group reported low-grade discomfort (both physical and mood) throughout the cycle on the MDQ, even in the intermenstrual phase when they claimed to be symptom free. During the premenstrum these women tended to have physical complaints and depressive mood changes. The second group reported virtually no complaints in the intermenstrum, and their premenstrual symptoms were more psychological than physical, and irritability, mood swings, restlessness, and tension were more prominent than depression. The authors concluded that the former group represents a premenstrual worsening of a general neurotic tendency towards symptom-complaining, whereas the latter (larger) group is essentially free of neurotic tendencies and represents the true example of premenstrual tension. If this is the case, and if these two groups are undifferentiated in other studies, it would explain the inconsistencies found and the weakness of the correlations between neuroticism and premenstrual symptomatology.

c) Femininity conflicts. The question of premenstrual tension and femininity, acceptance of female role, conflicts over sexuality, etc. is even more inconclusive. Again, measures and definitions have varied widely, particularly between earlier studies using traditional measures of femininity (e.g., acceptance of motherhood and male dominance) and newer studies with a feminist

perspective. Shader and Ohly (1970) surveyed 2000 women and found no relationship between feminine role conflicts and premenstrual tension. They did not specify the tests used nor the numerical data obtained. Berry and McGuire (1972) also found no correlation between premenstrual negative affect and acceptance of the traditional female role in their sample of 100 women. They found that women who complained of physical discomfort accepted the female role less, but argue that the discomfort causes the rejection and not vice versa, as psychoanalytic theory would predict. Slade and Jenner (1980b), in a study with a similar sample, found much the same results; femininity scores were not correlated with premenstrual symptoms, but the attitude towards the female role was weakly correlated with menstrual symptoms.

Watts et al. (1980) studied an older sample of women and found that premenstrual tension sufferers (mean age 37 years) had more anxieties about their bodies and genitals and about sex and masturbation than did nonsufferers (mean age 34 years). They found no difference between the groups on acceptance of traditional female roles and childbearing attitudes, and they suggested that it is not conflict over the female role per se that relates to symptoms but conflict over sexuality. However, because the age difference between the groups is substantial, although not significant, this finding must be viewed with caution, and in any case it is not known whether it applies to younger women. Chernovetz et al. (1979) found symptoms (they do not specify whether premenstrual or menstrual) positively correlated with femininity, as measured by the Bem Sex-Role Inventory.

Two researchers have added the variable of religion, and their results reveal the extreme complexity of the socio-cultural-attitudinal picture. Paige (1973) found femininity positively correlated with severity of menstrual symptoms (unspecified) in Catholic women but not in Protestants or Jews. Good and Smith (1980) compared femininity, religion, contraceptive pill use, and menstrual symptoms (premenstrual and menstrual combined) and their results almost defy interpretation. In Catholic pill-users, for example, masculinity was positively correlated with symptomatology, whereas in Catholics who did not use the pill, femininity was positively correlated with symptoms. There was no relationships between femininity, symptomatology and pill use among Protestants and Jews, but the number of subjects in some subgroups was small.

It appears, then, that a great many variables must be considered simultaneously before the relationships (if any) between specific menstrual cycle symptoms and attitudes towards femininity, sex, and sex roles can be clarified. However, the available evidence does contradict the psychoanalytic contention that premenstrual tension results from a denial of or conflict over feminine identity.

Before leaving the question of cultural influences, a brief note should be made of the cross-cultural survey of premenstrual symptoms done by Janiger et al. (1972). Most of the research has been done on European, North American and Australian women, and it has been argued that premenstrual tension is a spoiled rich girl's syndrome. Janiger and his coworkers surveyed Greek, Japanese, Nigerian, Turkish, Apache Indian, and American women and found that although

the predominant symptoms and symptom groupings varied from culture to culture, premenstrual complaints were universal, and that individual variability within cultures was more marked than inter-culture variability. Interestingly, Turkish and Nigerian women tended to show the most complaints and Japanese the least, with Americans in the middle.

In summary, there is some evidence to suggest that premenstrual tension is mildly correlated with age and generalized anxiety and neuroticism, although the reason for the relationship is unknown. Evidence for an increase with parity is scanty but positive. However, the relationship between sex role attitudes, femininity-masculinity, and religion remains indecipherable.

Etiological Mechanisms. Not surprisingly, the preconceptions and contradictory evidence have allowed the birth of a wide range of theories regarding the etiology of menstrual changes, particularly those occurring in the premenstrum. The theories fall into three main categories:

a) Physiological theories. Among the oldest and most researched, these link affective changes to some action of the fluctuating levels of estrogen and progesterone. The action may be direct, with estrogen and/or progesterone acting directly on certain receptive centres of the hypothalamus or limbic system, or it may be indirect, with hormones causing fluctuations in other substances which acts on the brain. Gonadotropins, prolactin, testosterone, renin, angiotensin, aldosterone, and MAO all show phasic changes, many with a drop beginning six or so days before menstruation.

Recent reviews of physiological theories presented by Smith (1975), Steiner and Carroll (1977), Warnes (1980) and Reid and Yen (1981) all conclude that "no one hypothesis has adequately explained the constellation of symptoms comprising the premenstrual syndrome" (Reid & Yen, 1981, p. 97). This failure notwithstanding, the evidence pointing to some kind of physiological-endocrinological basis is strong. In addition to cyclic changes noted earlier (higher autonomic arousal levels, weight gain, higher MAO levels, higher sensory thresholds, etc.), many other physical changes have been recorded in humans and animals over the years (Bell, 1975; Messent, 1976; Smolensky, 1980; Vernikos-Danellis, 1972). One of the major obstacles to interpretation is the complex tangle of interrelationships and facilitatory-antagonistic influences between the various hormones and hormones systems through the body.

Two indirect-action theories have dominated interest in the last decade: i) the renin-angiotensin-aldosterone hypothesis (Janowsky et al., 1967, 1973), which proposes that progesterone depletes sodium and causes a homeostatic increase in renin and angiotensin, which leads to a rise in aldosterone in the luteal phase. Aldosterone functions to retain sodium in the kidneys, thus increasing the K/Na ratio which produces premenstrual edema. Delay in feedback mechanisms result in renin, angiotensin and aldosterone remaining high once progesterone drops, thus aggravating symptomatology in the premenstrum. Proponents of this line of inquiry have argued that premenstrual mood changes may be secondary to fluid retention in the brain or may be the result of direct action of angiotensin on neurotransmitters in the brain. Angiotensin is thought to increase cholinergic activity in the brain (Warnes, 1980). However,

diuretics have proved no better than placebo in alleviating premenstrual mood changes, although they reduce the edema (Reid & Yen, 1981), and recent studies suggest that the three hormones drop six days before menstruation rather than at its onset as Janowsky had thought (Steiner & Carroll, 1977).

ii) The MAO hypothesis (Broverman et al., 1980; Grant & Pryse-Davies, 1968; Klaiber et al., 1971) postulates that estrogen inhibits MAO, while progesterone causes an increase in MAO levels in the plasma, endometrium and (hypothetically) hypothalamus, which upsets the brain adrenergic-cholinergic balance in favour of the latter. Cholinergic dominance is believed to have a depressant effect and MAO has been implicated in other affective disorders where MAO inhibitors are an effective treatment. There is some literature attempting to link the premenstrual syndrome to primary affective illness, which is believed to have a physiological origin, but there has been no conclusive success (Haskett et al., 1980; Schuckit, 1975). Similarly, lithium has proved to be disappointing as a treatment (Steiner & Carroll, 1977) and, although depression consistent with cholinergic dominance is a feature of the premenstrual syndrome in some patients, irritability and restlessness are more common features (Haskett et al., 1980; Moos, 1977).

A variety of other substances have been proposed as causes, but have not as yet been born out in treatment or further research, including vitamin A or vitamin B6 deficiency, abnormal prolactin levels, endogenous hormone allergy, hypoglycemia, and vasopressin (Reid & Yen, 1981; Warnes, 1980). In addition Reid and Yen have implicated neurointermediate lobe peptides, including endophins (endogenous opiates), which have been shown to exert a great many regulatory functions throughout the endocrine system and which appear to show cyclic effects.

Those arguing for a direct hormonal-brain action point to the numerous effects of the hormones on the brain; progesterone and its metabolites have been found to exert a sedating effect (Gyermek et al., 1967) and estrogen an excitatory effect. Seizure thresholds are lower in the premenstrum, when both hormones are falling (Vernikos-Danellis, 1972). Theorists argue that the rapid fall and the potential for imbalance might trigger psychological disturbances in individuals whose metabolism of hormones is inadequate. They point to the existence of post-partum and menopausal affective disturbances which also occur at a time of rapid hormone fall (Hamburg et al., 1968).

Dalton (1977, 1979) is a major proponent of the "progesterone deficiency" explanation of all premenstrual symptomatology. She argues that premenstrual tension sufferers have low levels of progesterone in the luteal phase, either in absolute terms or relative to estrogen levels, although she is unsure of the mechanism by which progesterone production is impeded. She presents clinical case studies demonstrating very good treatment success with high doses of natural progesterone. However, independent attempts to replicate this success using controlled, double-blind procedures have found progesterone no more effective than placebo (Sampson, 1979; Smith, 1975). Furthermore, Smith (1975) has pointed out that the progesterone deficiency hypothesis by itself does not explain why premenstrual symptoms do not occur in the follicular phase, when progesterone levels are almost non-existent.

As usual, contradictory results have been obtained from oral contraceptive research, in which hormone levels are artificially controlled and normal fluctuations are assumed not to occur. Many studies (Campos & Thurow, 1978; Paige,

1971; Silbergeld et al., 1971) found less complaints of depression and irritability in pill users, but some have found more (Grant & Pryse-Davies, 1968; Wilcoxin et al., 1976). Some women find that oral contraceptives alleviate their premenstrual and menstrual distress, but some discontinue because they cause it. Grant and Pryse-Davies (1968) found that the estrogen-progesterone make-up of a particular brand influences the amount of negative symtomatology; progesterone content, endometrial MAO level, and negative symptoms were all positively correlated. Highly estrogenic pills, by contrast, produced fewer complaints. However, Steiner and Carroll (1977) point out that other studies have found the opposite effect, and they argue that there may be at least two potentially distinct premenstrual syndromes, one in which irritability predominates and another in which depression does. Progesterone treatment and high-progesterone contraceptives may alleviate symptoms in the former but exacerbate them in the latter. As noted in the previous section, their later study (Haskett et al., 1980) lends some support to this distinction.

In summary, a great many physiological factors have been implicated in the etiology of premenstrual tension at one time or another; and a great many treatments have been proposed, but as yet, no definitive mechanism has been able to account for the variable affective and somatic changes which comprise the premenstrual syndrome. Two main obstacles are the complexity of hormone interrelationships within the endocrine system and the strength of the placebo response observed in this condition (Reid & Yen, 1981), which underscores the need for careful scientific controls.

b) Psychological theories. Psychological theories of premenstrual tension derive from two sources - psychoanalysis and the more recent cognitive psychology. As noted earlier, early psychoanalytic formulations regarding menstrual symptomatology centered around the woman's denial of her femininity, difficulties with her mother, or other psychosexual conflicts which express themselves in somatic form (Shader & Ohly, 1970). These conflicts, anxieties and fears of mutilation are thought to be activated each month as a woman nears her menses (Ivey & Bardwick, 1968). It is felt that the low and fluctuating hormone levels in the premenstrum may lead to a weakening of ego controls and a regression in psychosexual integration (Benedek, 1973). Attempts to link premenstrual symptoms to difficulties or conflicts regarding femininity have failed, as noted earlier, revealing evidence which tends to run counter to psychoanalytic predictions. Similarly, premenstrual symptomatology and neuroticism are only weakly correlated, with the majority of premenstrual sufferers falling within the normal range on personality measures.

Of more relevance to current research endeavours is the growing body of opinion and evidence supporting a cognitive component to at least some menstrual cycle changes, especially those concerning mood and social behaviour. Critical of the traditional medical model, cognitive theorists point to the fact that despite voluminous research over decades, all that has emerged is a variety of divergent physiological hypotheses with no clear-cut explanation of how hormones affect behaviour and mood in the premenstrum (Paige, 1973; Parlee, 1973; Ruble & Brooks-Gunn, 1979). While not ruling out possible biological contributions, they emphasize instead the large cognitive component which derives from culturally-mediated expectations.

Several psycho-social factors have been suggested as contributing in large measure to premenstrual and menstrual distress; negative cultural myths which engender negative expectancies, anxiety about pregnancy or menstrual flow, pain and inconvenience of menstrual flow (Frieze & Ruble, 1978; Paige, 1971) and biased perceptions which lead to noticing and recalling negative moods but not positive ones (Koeske & Koeske, 1975; Ruble & Brooks-Gunn, 1979).

Historically, menstruation has been viewed in a very negative light and menstrual blood has been imbued with a magical power dangerous to men (Weideger, 1976). In many cultures, menstruating women were seen as unclean or cursed, and were often isolated so as to prevent them from contaminating others. Some of this negative aura has carried over to today, particularly in traditional religious teaching (Paige, 1973), so that the paramenstrual period continues to be viewed as negative and debilitating, and menstrual blood as shameful and unclean. Recent research has shown that there is a large body of myth and stereotype regarding negative symptoms during the premenstrum (e.g., women are irrational, unreliable, and intellectually less competent), and that this stereotyped view is well known to women, men and even preadolescents (Clarke & Ruble, 1978; Parlee, 1974). Men, in fact, are found to believe the myths more than women, perhaps because they have no personal disconfirming experience. Cognitive theorists argue that women report, and even experience, negative feelings because they have been conditioned to expect them (Paige, 1973; Parlee, 1978; Ruble, 1977). Ruble (1977), for example, found that when women were falsely induced to believe they were premenstrual, they reported more symptoms than those in the same cycle phase who were induced to believe they were intermenstrual.

Paige (1971) argued that because of the historical aversion to menstrual blood, shame, anxiety and fear of discovery may be aroused in women at the time of menses and just before. She studied oral contraceptive users, claiming that hormonal variations were held constant by the artificial hormones (a questionable assumption), and found that anxiety was positively correlated to intensity of menstrual flow.

Parlee (1978), Koeske (1980), and many others have pointed to the problems of self-report data because of the bias introduced by expectancies. The MDQ has been criticized on the grounds that it merely measures a woman's knowledge and acceptance of cultural stereotypes regarding female cyclicity (Alplanalp, 1979b; Parlee, 1974, 1980; Ruble & Brooks-Gunn, 1979). This contention is supported by the finding that daily self-ratings lead to less negative symptom reporting than retrospective monthly ratings (Alplanalp, 1979b; Parlee, 1980) and also by the finding that women perceive themselves as performing more poorly during the premenstrum when in fact they are not. It is argued that this results from a bias in perception and recall stemming from the woman's knowledge of cultural myths. Negative moods are noticed more than positive, and all negative moods are blamed on biology. Bias and expectancy are especially likely to operate when a subject is aware of the researcher's focus on the menstrual cycle, a condition in most of the studies done.

It is worth noting, however, that despite wide public knowledge of these negative myths and knowledge of the purpose of the study, a large percentage of women do not report premenstrual symptoms and are selective in those they do

report. Thus, as noted earlier, only 40% of Moos' subjects (1968) complained of premenstrual negative affect and only 12-15% complained of any intellectual impairment. By subtraction, the majority of women do not respond to the questionnaires according to the prevalent cultural stereotype. Furthermore, Brooks, Ruble and Clarke (1977) found that their sample of young, unmarried women had more positive views of menstruation than had been expected. Positive ideas about menstruation were endorsed by 77%, and 68% disagreed that it was debilitating. In general, menstruation was seen as a minor, routine event with both bothersome and positive aspects to it. In addition, contrary to Paige's finding (1971), length and intensity of menstrual flow was related only to physical distress and not to psychological.

Recently, Ruble and Brooks-Gunn (1979) put forward an information-processing model demonstrating how a woman comes to attribute negative moods to the paramenstrum independently of any cultural expectancies or attitudes she might have. Based on current information-processing theory concerning the cognitive acquisition and strengthening of beliefs, they point to the built-in bias in human information processing which leads to negative moods being linked to a salient and negative event like menstruation.

In summary, cognitive theorists argue that although there is probably a physiological basis for some physical symptoms such as pain and water retention, actual mood and behaviour fluctuations are far less pronounced that women think, influenced as they are by the still-prevalent negative aura surrounding menstruation. Those fluctuations which do occur are largely due to self-fulfilling prophesy and to various anxieties and annoyances about pregnancy, bleeding, and pain.

c) Psychobiological theories: A third point of view acknowledges the valid findings made by both the physiological and cognitive schools, and arrives at a merger of the two. Most researchers do not specify the nature of the merger, but recently some have implicated an interaction between expectancy and arousal. Taking the recent work on emotion in general (Mandler, 1975; Schacter, 1964), they postulate that through some as yet unknown physiological mechanism, there is a premenstrual rise in autonomic arousal, and that this arousal interacts with cognitive expectations and interpretations to produce an experience of mood. Two different theories have been advanced as to the nature of the interaction, however.

i) Synergistic model: Asso (1978) proposed an interactional model to account for premenstrual negative affect based on Mandler's theory of emotion (1975), which is in part an information-processing elaboration of Schacter and Singer's coggitive formulation (Schacter, 1964). Asso postulates that the premenstrum is characterized by increased autonomic arousability (which means that the woman is more physiologically reactive, not that she is constantly more aroused, an idea in keeping with Marinari's finding (1976) on adrenocorticoid reactivity), and that this arousability primes the woman for more intense emotional experience, both negative and positive. Cognitive factors, notably expectancy and past experience, will determine the way in which the arousal is labelled and experienced as an emotion. The expectation of negative premenstrual mood, combined with any personal negative connotations the menstrual period might have for that woman, would act to increase further the physiological arousal in a synergistic spiral. This model predicts that the greater the expectancy of negative mood, the greater the experience of it.

A variant of this hypothesis was put forward by Koeske and Koeske (1975), who used attribution theory to account for women's belief in premenstrual tension. They found that negative moods tended to be attributed to a woman's premenstrual state, even when plausible external explanations existed; positive moods, on the other hand, were attributed to external factors. They argued that because of its salience and the myths surrounding it, women tend to attribute all negative moods to the premenstrual phase while overlooking the less salient but often correct environment causes. Positive moods occurring during the premenstrual phase are not linked to the cycle, however, but are explained by environmental factors, as are negative moods occurring at other phases. In retrospect, because of the frequency of the pairing, women will recall more readily the negative moods linked to the premenstrual phase than any of the other, presumably random, moods.

ii) Stress-reduction model: A very different model of interaction has been advanced by Rodin (1976). Basing her reasoning on the attributional research of Nisbett, Storms, Valins, and others (Nisbett & Valins, 1971), she argues that an expectation which is confirmed by subsequent experience helps to counteract and perhaps even reduce the physiological arousal experienced. Thus, far from heightening the negative affect, expectancy may reduce it. A woman who knows that her tension and irritability are due to premenstrual changes will be less bothered by them. Rodin compared the performance of three groups of women on some frustrating cognitive tasks. One group was intermenstrual, the other two were menstruating but half consisted of high-symptom subjects and half low. Before the tasks, menstruating women were given

information on the menstrual symptoms they typically experienced (which for the high-symptom group would resemble the arousal they would experience due to the tasks, but which for the low symptom group would not). She found that the high-symptom menstruating women performed significantly better and she concluded that they had been able to attribute their arousal to menstruation while the other groups had no such attribution available. Because part of her cognitive tasks was a set of insoluble puzzles which were inherently frustrating, she viewed the relevant-attribution group as showing better tolerance for frustration that the no-attribution groups.

While Rodin was unable to conclude from her data whether this effect was due to the direct stress-reducing potential of attribution or due to self-preparation and extra effort in the high arousal group, she favours the former hypothesis. However, a recent finding by Munchel (1979) that women who expected to do poorly on a cognitive task during the premenstrual phase showed greater persistence than those with no such expectation, suggests that in fact compensatory extra effort may have been involved in the better performance noted by Rodin in her high-symptom group. A similar finding by Williams et al. (1981) that drunk subjects who expected to be drunk compensated for their impairment supports this view.

A study on attempted suicide by Tonks et al. (1968) provides some support for Rodin's view that expectancy may mitigate the detrimental effects of arousal. Methodological problems in the study make its conclusions tentative, but the authors found that parous women who complained of premenstrual tension

made significantly fewer suicide attempts than those who reported no tension. They conclude that "perhaps being aware that the premenstrual dysphoria will end in a few days with the onset of menstruation helps to prevent such drastic behaviour as attempting suicide" (p. 322). Some other studies, however, have failed to find this inverse relationship between premenstrual complaints and suicide attempts (Wetzel & McClure, 1972).

Expanding on Schacter and Singer's original work, attributional research postulates that, given autonomic arousal, people will seek an explanation within themselves and their environment and respond with the appropriate emotion. If arousal is attributed to a neutral or an emotional but predictable, normal or expected source (like menstrual cycle changes), then arousal is reduced. But if the actual experience of arousal contradicts expectations or judgements of what is normal, then arousal and emotionality may be increased. When Storms and Nisbett (1970) told a group of anxious insomniacs that a placebo would reduce their arousal and allow them to sleep, they took even longer than before. They expected arousal reduction and when they found themselves as aroused as ever, they apparently concluded that they must really be upset if even with the pill they felt this aroused, and that thought increased their arousal. By contrast, when told the placebo would cause arousal symptoms, they were able to sleep more quickly, because their expectancy (of arousal due to the pill) was congruent with their experience (of arousal due to intrinsic anxiety), and the neutral attribution removed the emotional meaning of the physiological arousal symptoms.

These findings have implications for the menstrual cycle, in that not only could the experience of negative affect and emotionality be reduced by attributing it to the normal menstrual fluctuations which most women experience, as Rodin found, but also the experience of negative affect and its possible detrimental effects on behaviour could be increased by informing the woman that there is no such thing as real, physiological mood changes across the menstrual cycle. If indeed there is a change in physiological arousal or some other genuine biological cause of mood fluctuations, women without the normal and predictable attribution of menstrual changes would have to search elsewhere for an explanation and would perhaps have to conclude that they really are depressed or anxious and that something in the environment was upsetting them far more than was actually the case. One study reports findings which suggest that women may in fact perceive more stress and tension in their environment during the premenstrual phase (Englander-Golden et al., 1977). Without the 'excuse' of premenstrual tension, women might perceive even more irritants in their environment (in the form of husbands and children, for example).

In summary, there is evidence that both physiological and psychological factors contribute to the experience of premenstrual tension. One possibility is that emotionally ambiguous physiological arousal or arousability interacts with cognitive expectations. Two interactional models have been advanced. In Asso's synergistic model, expectancy contributes to increase the experience of negative affect, whereas a lack of expectancy ought to reduce negative affect.

In Rodin's stress-reduction model, expectancy serves to reduce the emotional tone of the arousal and therefore reduces the experience of negative affect, whereas a lack of expectancy adds to the emotional tone.

MATERNAL BEHAVIOUR

In recent years, psychologists have become increasing involved in both menstrual cycle and child abuse research. It is surprising, therefore, that a review of the literature failed to find one study relating the two, or even examining the broader question of menstrual cycle effects on the quality of maternal care. Dalton (1975, 1977, 1979) has repeatedly pointed out the possible connection between child abuse and premenstrual tension based on her own clinical experience, but no systematic investigation has ever followed this up. The only studies which came close were two doctoral dissertations done by Herlihy (1977) and Teitler (1979), who both examined menstrual cycle effects on the behaviour of school teachers towards their young pupils. Their findings will be discussed further on.

In attempting to find material which might bear indirectly on the question of how the menstrual cycle affects maternal behaviour, the writer found large gaps in our understanding of the genesis and maintenance of maternal behaviour, particularly in humans. Students of maternal behaviour have mainly been interested in its effects on child development, particularly abnormal development, and not in the mechanisms by which various features of maternal (or more broadly, parental) behaviour are developed, sustained, and elicited. There are exceptions

to this - the growing research on child abuse and mother-infant bonding are two - but as a rule, little is known about why parents behave as they do.

When considering the question of maternal behaviour, at least two distinctions must be made between 1) the factors which affect the basic capacity for and quality of mothering in a given individual (i.e., predisposes him or her towards certain maternal behaviours) and 2) the factors which elicit and modulate maternal behaviour in a given situation. Early childhood relationships, socialisation and role modelling experiences, personality and temperament are likely to affect the former, whereas stress, illness, mood, daily experiences and possibly hormone fluctuations affect the latter. The human research that exists relates more to the former question than to the latter (Chodorow, 1978; Mussen & Eisenberg-Berg, 1977), and thus is not directly relevant to the question of menstrual cycle effects on maternal behaviour.

Besides making the distinction between capacity and performance, the concept of maternal behaviour must be broken down into various dimensions through which individual behaviours can be related. Schaefer (1971), after reviewing several factor-analytic studies of maternal behaviour including his own, postulated a circumplex model of maternal behaviour with two, possibly three, dimensions, the most clear-cut and replicable being an affective dimension variously labelled in the different studies as Love-Hostility. Warmth, or Acceptance-Rejection. Other dimensions relate to discipline, control, and involvement. In his own research, Schaefer found that the Love-Hostility dimension was quite stable over time; that is, the mother's affective stance towards her preschool child was predictive of her affective stance towards the same child in adolescence. Control and Involvement behaviours, by contrast, were less stable (Schaefer & Bayley, 1976).

The long-term stability of the Love-Hostility dimension does not, however, preclude a great deal of day to day fluctuation. The long-term stability probably reflects the basic capacity and maternal predisposition of the parent, which is relatively (but not completely) impervious to change, whereas short-term fluctuation reflects daily changes in both the internal and external environment.

In the discussion to follow, the research evidence and theory relating to short-term fluctuations in the Love-Hostility dimension will be examined with a view to possible menstrual cycle contributions. Positive maternal behaviour (accepting, loving, helping, etc.) and Negative maternal behaviour (rejecting, critical, punitive, aggressive) will be examined separately.

Negative Maternal Behaviour. Information on the possible antecedents of negative maternal behaviour comes from two divergent sources: 1) the literature on hormones and aggression, and, 2) the literature on child abuse.

a) Hormonal evidence: animal. There has been considerable interest in the effect of sex hormones on aggressive behaviour in animals (see Crabtree & Moyer, 1977), although most of this has centred on male aggression and the male hormone testosterone. One of the more consistent findings in the literature on human and animal aggression is that males are more aggressive than females, at least when the aggression is of the violent, physical kind (Conner, 1972; Frieze & Ruble, 1978; Parsons, 1980). Testosterone is generally believed to underlie this difference. It is postulated that the differential action of the male and female sex hormones during the critical pre- and early post-natal period serves to sensitise certain areas of the brain and facilitate the

development of certain nerve pathways which determine the pattern of later sexual and aggressive response. Testosterone in this period is believed to result in male-like sexuality and aggressivity. At maturity, male sexual and aggressive responsivity is thought to be related to testosterone levels, at least in animals (Moyer, 1976).

By contrast, the absence of testosterone priming at birth and/or the presence of estrogen at maturity is thought to inhibit aggression in animals. Testosterone is also secreted by the adrenals and ovaries of women, in far lower concentrations than in men, and it has been implicated as a possible determinant of sexual and aggressive behaviour in women as well (Bardwick, 1971, 1974; Frieze et al., 1978; Persky, 1974). Persky found that testosterone levels in young women vary slightly across the menstrual cycle, being low in the early follicular stage, peaking at ovulation, and remaining fairly high through the premenstrum. It has been suggested that the premenstrual rise in aggression in women may be due to the fall of aggression-inhibiting estrogen, which allows the more "male-like" violent behaviours to occur under the still high levels of testosterone (Frieze et al., 1978).

However, the results of hormone manipulations on animals reveal that the picture may be more complicated than that. Sex differences in hormone effects were marked. Testosterone injected at infancy and/or maturity facilitates most types of aggressive behaviour in male mice (Davis & Gandelman, 1972; Erpino & Chapelle, 1971) and male monkeys (Dixson & Herbert, 1974). Both estrogen and progesterone have been found to inhibit aggressive behaviour in male animals

(Bronson & Desjardins, 1968; Erpino & Chapelle, 1971). Similarly, the administration of testosterone seems to facilitate aggressive behaviour in female mice, but the effect varies; for some types of aggression, testosterone priming in infancy is necessary to produce aggression in adulthood, whereas the killing of young requires no prior priming but simply testosterone at maturity (Davis & Gandelman, 1972; Gandelman, 1972). The researchers concluded that the female mouse brain is normally organized at birth to permit pup-killing and only requires testosterone to elicit it.

The evidence of estrogen and progesterone effects on female animals is contradictory. Several correlational studies on primates have observed that females are more aggressive during the follicular-ovulative (high estrogen) phase than at other times (Mallow, 1981; Michael, 1969), although this is true only for the milder forms of aggression. Dixson and Herbert (1974) found females somewhat but not significantly more aggressive when injected with estrogen. Similarly, Bronson and Desjardins (1968) found an increase in aggression in female mice injected with estrogen in infancy. On the other hand, Scruton and Herbert (1974) observed no cycle-related changes in aggressive behaviours in monkeys, and Svare and Gandelman (1975) found that post-partum injections of estrogen significantly reduced aggressive attacks by mice on intruders into the maternal nest. Progesterone has been studied less than estrogen; Michael (1969) found that exogenous administration of progesterone decreased aggressive behaviour (attacks on mates) in monkeys, but Svare and Gandelman did not find that it decreased attacks on intruders in post-partum mice, and Michael (1969) observed that aggression increased greatly as pregnancy advanced, when progesterone levels were also rising.

Thus, the animal evidence is complex and general principles are difficult to formulate. It appears that at least in the case of female aggression, distinctions must be made between the kinds of aggression (pup-killing, attacks on mates, attacks on intruders into the maternal nest) and also between species (talapoin vs. rhesus monkeys, for example). Because of the marked differences between mice and humans, and because of the infinitely more complex nature of human aggression compared to animal, it seems very difficult to make any generalisations from these hormonal studies to the possible effects of menstrual hormonal fluctuations on maternal aggression. The complex interplay between estrogen, progesterone and testosterone is as yet poorly understood.

b) Hormonal evidence: human. Human research on hormonal effects must usually be correlational rather than experimental in nature, and therefore subject to ambiguities in interpretation. It is often assumed that physiology causes behaviour and not vice versa, but the evidence demonstrating that stress and shock can affect ovulation and menstruation (Sommer, 1978) and that changes in dominance status can change testosterone levels in monkeys (Frieze et al., 1978) puts this assumption in doubt. In addition, because hormones cannot be manipulated independently of social, cultural and psychological influences (such as expectations) which may covary with them, observed correlations between a particular hormonal or menstrual status and aggressive behaviour cannot be attributed directly to the hormones. Ellis and Austin's (1971) hypothesis that aggression may increase because it is condoned more in the premenstrual phase is an example of other explanations.

The evidence from clinical studies of patients born with hormonal abnormalities demonstrates this confounding of psychosocial and hormonal factors. Fetally androgenised females (excess androgen during pregnancy) are more "tomboyish" and physically active than controls: however, they are not more aggressive and they do not reject "feminine" interests such as marriage and motherhood (Ehrhardt, 1973; Kaplan, 1980). Females born with insufficient female hormones are excessively feminine and passive in their behaviour, and estrogen given prenatally to boys results in less "masculine" interests and abilities. In all these examples, however, a number of critics (Chodorow, 1978, Kaplan, 1980; Parsons, 1980) have pointed out that awareness of their condition, both by the parents and the patients, may produce subtle changes in socialisation processes and self-definition.

The human studies relating aggressive behaviour to the menstrual cycle provide the most direct evidence for a possible cycle effect on negative maternal behaviour. As noted earlier, crime (especially violent crime) and suicide behaviour both show a premenstrual peak, or alternatively a luteal dip. Whether normal women exhibit a similar but lesser peak remains a moot point, however (Parlee, 1973). In addition, these studies were based on retrospective data with considerable room for error.

There are five prospective studies which attempted to study aggression in relation to the menstrual cycle. The first (Ellis & Austin, 1971) used an admittedly deviant sample of 45 female prisoners. They obtained daily reports on negative mood and aggressive incidents (as recorded by the guards) and found

a luteal decrease and paramenstrual increase in both. They suggested that aggression may be secondary to irritability or pain, or perhaps that it is instrumental in that prisoners knew they could get away with more in their premenstrual phase.

The second (Bardwick, 1974) did not study overt aggression but rather self-ratings and game-playing style. It is of interest because the use of oral contraceptives approximates the exogenous administration and control of hormones found in animal research. Naturally cycling women were compared with women taking estrogen-dominant and progestin-dominant combination brands. The competitive vs. cooperative game-playing style of these women was compared at midcycle and premenstrually. The non-pill users showed themselves to be more competitive, assertive, and self-confident at midcycle than at the premenstrum, while the combination pill users (with a constant hormone level) maintained the same playing style throughout. But the estrogen-dominant users described themselves as higher in aggression, assertiveness, and hostility, while the progestin-dominant group scored higher on deference, nurturance, and affiliation. Bardwick proposed a model of the combined effects of testosterone, estrogen, and progesterone to account for cyclic changes in affect and behaviour. The high testosterone-high estrogen profile at midcycle results in assertive, active, self-confident behaviour at midcycle. During the luteal phase the sedating, passive progesterone inhibits testosterone, whereas in the premenstrum testosterone in the absence of estrogen and progesterone results in more overt hostility and aggression. Because of the complex hormonal relationships found in animals, however, this model remains highly speculative, although provocative as a methodology for future research.

Among the three controlled studies investigating the link between the premenstrum and overt aggressive behaviour in normal women, two found a positive relationship and one found none. Herlihy (1977) observed eight preschool teachers for twenty minutes each day for two months, rated them on thirteen behaviour categories of nurturance, control and punishment, and obtained daily mood self-ratings from them. She found no cycle changes in verbal or physical punishment, but did find that positive self-rated affect was positively correlated with the use of forceful and imposing control techniques, while negative self-rated affect was correlated with nurturance.

In contrast, Teitler (1979) examined ten measures of verbal aggression (Whining, threats, sarcasm, etc.) scored from tape-recorded segments of regular classroom teaching and found that these subtle measures of aggression increased during the premenstrual phase. She attributed this to greater irritability and lower frustration tolerance. Both studies have small samples, and the contradictions in findings could be related to a difference in the measures used to define aggression or to the difference in the age of the children in the classes.

In the only experimental, laboratory study of aggression and the menstrual cycle, Schonberg (1976) compared premenstrual and intermenstrual women on the amount of shock they were willing to administer in an attempt to train a 'stupid' learner in a remote control situation. He found premenstrual women gave significantly higher shocks and interpreted this to be an indication of increased aggressivity in the premenstrual phase.

In summary, there appears to be some evidence for a luteal decrease and a premenstrual increase in aggressive behaviour, although it is by no means a universal phenomenon. The role of hormones in this fluctuation is unclear; testosterone, implicated in aggressivity in both males and females, peaks at midcycle and remains fairly high through the premenstrum. Estrogen may inhibit, facilitate, or modulate aggressivity, whereas progesterone appears to inhibit it. However, it is unknown whether these effects are dose-related, or indeed if the changes in hormone levels observed over a normal menstrual cycle are sufficient to produce changes in behaviour. Furthermore, even if hormones do alter aggressivity, the mechanism by which this operates is unknown (Moyer, 1976); is there a direct CNS lowering of aggressivity thresholds by testosterone, for example, or does heightened arousal or pain, both of which increase aggressivity in animals (Conner, 1972; Marler, 1976), reduce stress tolerance and facilitate aggression?

The only studies directly relating menstrual phase to some aspect of maternal behaviour have been those of Dalton (1970) and Tuch (1975), who found that a higher proportion of children are brought to emergency or admitted to hospital as emergencies when the mother is in the paramenstrual phase. Tuch does not differentiate illness from accident admissions and postulated that paramenstrual mothers were more anxious, less able to cope with the extra demands or possibly more empathically sensitive to the child's suffering. Dalton differentiated accidents from illness and found the proportion even higher (62%) when only accidents were considered. She suggested that the mothers

were less vigilant, clumsier, and less able to judge danger, all unlikely in view of the negative findings on perceptual-motor impairment in the premenstrual phase (Sommer, 1973).

Perhaps the mothers were less patient, more harassed, and more distracted; research on domestic accidents show that the majority occur between the hectic hours of five and seven p.m. But another possibility is that some of the accidents were not accidents at all, but incidents of child abuse. An estimated 10% of accidents to children are thought to be unconfirmed incidents of abuse, and the percentage may be higher. A brief review of other findings and theories from the child abuse literature is worth presenting here, as they form the bulk of current knowledge and theory about the antecedents of parental aggression.

c) Child abuse. The child abuse literature is fraught with seemingly contradictory viewpoints, some highlighting personality deficits in the abusers, some the characteristics of the abused child (Bell & Harper, 1980), others environmental stresses (Egeland et al., 1980) and still others the aggressive nature of our society as a whole. Belsky (1980), in a recent good critique, demonstrated how these differing views can be integrated into a multi-causal, multi-level model of child abuse which highlights the many questions which have not yet been answered.

Until recently, the child abuse literature was full of broad, vague, largely common-sense observations, and few methodologically sound, well controlled studies had been done (Spinetta & Rigler, 1972). Abusers were thought to be

defective in character, having been deprived or abused children themselves, or unable to cope with the stresses and responsibilities of parenthood because of immaturity and egocentricity (Bennie et al., 1969; Melnick & Hurley, 1969). Often they were described simply as having poor impulse control or uncontrolled hostility. Stresses such as unemployment, marital strife, poverty and illness were viewed as possible contributors but neither necessary nor sufficient cause in themselves.

Then Gil (1971), analyzing the results of a massive demographic survey of child abuse in the U.S., reported that child abuse was largely concentrated among the economically deprived lower classes where cultural sanctions of physical discipline were more widespread and ingrained, and where poverty, lack of education and lack of available community health care resources made families less able to cope with the many stresses that faced them. He took the responsibility for child abuse out of the hands of the abusers and placed it onto the society and culture.

Critics pointed out that economic deprivation is neither a necessary nor sufficient cause either. Gelles (1973) disputed the uni-dimensional cause of abuse, be it sick society or sick abuser, and argued for a multi-dimensional model which incorporates social, situational and personal factors such as the abuser's background and habits learned in childhood - including his habitual mode of handling stress - together with any emotional deviance resulting from this background. The personal factors interact with the situational stresses which the family experiences, including unemployment, too many children, unwanted pregnancy, problem child, marital strife, etc. These stresses in turn are

influenced by the socioeconomic status of the family. Against the backdrop of these stresses, the personality of the abuser, and the violence-endorsing values of his community, there is the immediately precipitating event which leads to the actual abusive act. Gelles lists these as "misbehaviour of the child, argument, etc." (p. 619). Anything which either increases stress to the overload point or reduces stress tolerance below a critical level could be a precipitating factor.

Gil (1971) has pointed out that the act of child abuse itself is not a single phenomenon but a whole group of phenomena, each of which may have a different constellation of causal factors. Some involve sexual assault, others deliberate sadistic gratification, while others are an angry over-reaction to misbehaviour. He factor-analyzed the components of 1380 child abuse incidents and derived seven categories. In nearly 73% of these cases, however, the element of "inadequately controlled anger of perpetrator" was present. Hence, most child abuse has as one of its features poor impulse control, whatever the reasons for this may be.

After twenty years of formal and informal research in child abuse, Kempe and Kempe (1978) concluded that the distribution of parenting skills in the general population approximates the normal curve, with a small percentage at the extreme of excellence, most falling into the middle range of "good to good enough", and 20-30% in the lower range of "poor to abusive". They regard this 20-30% as a high-risk group all capable of child abuse if placed under the right stress. Of the smaller group of confirmed abusers, only 10% suffer from some gross deficiency such as mental retardation or florid psychosis, and

another 10% have some intractible personality deficit such as alcoholism or psychopathy. But 80% (in their experience at least - other researchers are less optimistic) have no gross psychopathology and can be helped with treatment to become adequate parents.

When (and if) this 30% of parents actually performs an abusive act may depend on an overload of stress and a precipitating factor. Egeland et al. (1980), in a prospective study of 267 high-risk mothers, found that abusive mothers were experiencing higher levels of stress in their lives. Personality factors (isolation, defensiveness, and anxiety) as well as faulty parent-child interaction were found to differentiate those abusive mothers from non-abusive mothers under similar stress.

This finding of the facilitating role of stress is further supported by one of the rare laboratory studies done on child abuse (Passman & Mulhern, 1977). Mothers were required to perform a key-punching task with ambiguous instructions (high stress) or unambiguous instructions (low stress), while simultaneously monitoring their child's puzzle-solving telemetrically. The child's errors produced a buzz in the mother's console which required an answer from her. In actual fact, the buzzes were programmed to be frequent (high interference) or infrequent (low interference). Because of ethical considerations, punishment was non-corporal (deprivation of candies), but the results did demonstrate that mothers under stress did punish more severely, regardless of whether the source of stress was an irritating child or a difficult task.

The importance of a precipitating factor, although specified by Gelles (1973), has tended to be overlooked in child abuse research, possibly because it is

considered to be so idiosyncratic and unpredictable. However, abusive incidents do not occur continously, in fact they may only occur once a week or month, or less. There is thus more time when abuse is not occurring than when it is. While many precipitating factors may not be predictable or controllable, some, like premenstrual tension, can be.

Although physical child abuse is often discussed as a dichotomous variable - one either is or isn't an abuser - it would be more realistically viewed as a continuum (or set of continua) from the occasional slap to sadistic batterings. Somewhere short of this latter extreme fall most of the abusers classified as such, those whose periodic loss of control results in damage severe enough to be brought to the attention of the authorities. As Kempe and Kempe (1978) have found, they may not differ in type from the average parent, perhaps only in degree. Any attempt to elucidate the factors contributing to 'normal' parental aggression has valid implications for the understanding of extreme cases as well.

In summary, physical child abuse is a complex group of related but distinct phenomena whose causes are multidimensional. One factor common to most types of abuse is poor impulse control on the part of the perpetrator. Any factor which weakens impulse control or increases stress beyond the coping point can precipitate an abusive act. The evidence discussed in the preceding sections suggests that the premenstrual phase may be a time of increased irritability, decreased stress tolerance, and increased aggression in some women. It may thus be one factor which contributes to stress overload and helps to precipitate an aggressive or in extreme cases abusive act.

Positive Maternal Behaviour. Positive maternal behaviour falls towards the other extreme of the Love-Hostility dimension and is described by labels such as empathic, accepting, loving helpful, encouraging, kindly, nurturant, affectionate, etc. As noted earlier, most research has been interested in it only as an independent variable affecting child outcome, and not as a dependent variable itself. Those studies which do examine the antecedents of positive maternal behaviour (often simply called maternal behaviour), usually focus on the factors influencing capacity or predisposition and not those influencing its day to day performance. There has been no systematic research relating the menstrual cycle to the performance of positive maternal behaviour in either humans or primates, as far as could be discerned. This reflects the more general lack of research relating positive mood and behaviour to the menstrual cycle (the exception being sexual behaviour). Whether this bias results from the prevalent negative connotations of menstruation in our society or from the medical concern with pathology, it remains that there is a marked asymetry in our understanding and exploration of cycle-related changes.

From the broader perspective, however, a variety of factors have been implicated in the genesis and maintenance of positive maternal behaviour and these may be considered under three headings: 1) hormonal (or other biological) influences, 2) personality traits, predispositions or needs based on early childhood relationships, socialization, etc., and, 3) situational factors within the familial, marital or mother-child relationship or within the child himself.

a) Hormonal influences. Extensive hormone studies with animals have demonstrated the importance of female sex hormones such as prolactin, estrogen, and oxytocin on the execution of various maternal tasks like nest-building, nursing, licking and retrieving the young (Klopfer et al., 1973; Zarrow, 1972). However the picture is complex and the effect often species-specific. For example, nest-building in rabbits and rats is elicited by falling progesterone levels at the end of pregnancy, whereas in mice and hamsters it is related to rising progesterone at the beginning of pregnancy (Klopfer et al., 1973). Hormones may affect one aspect of maternal behaviour but not another, or may change the latency to onset but not the quality (Ehrhardt, 1973). The relative importance of hormonal, environmental, and experiential factors varies between species and even between first-time and experienced mothers in the same species (Klopfer et al., 1973). Furthermore, maternal behaviour can be elicited in the males of many species, even in rats, although the latency to onset is longer than for hormonally primed females.

Despite this complexity, some tentative observations on the role of estrogen and progesterone deserve mention. There is evidence to suggest that, in rats at least, exogenous estrogen facilitates the performance of maternal behaviour (as measured by decreased latencies of retrieving pups to the nest and attempting to nurse) in pregnancy-terminated, hysterectomized females (Numan, 1978; Siegal & Rosenblatt, 1978), virgin females (Davis & Gandelman, 1972) and even in males (Gandelman, 1972). By contrast, progesterone administered to pregnancy-terminated rats inhibits maternal behaviour (Numan, 1978; Siegal & Rosenblatt, 1978) and these authors postulate that the hormone changes of late pregnancy and parturition - namely the rise in estrogen and prolactin and the fall in progesterone -

are the trigger for the onset of maternal behaviour. Other factors such as stimulation from the pups may be needed to maintain it.

This evidence, together with the evidence for a possible physiologically-based bonding mechanism between human mother and child in the critical period just after birth (Magnus, 1980), suggests that the female hormonal state at birth serves to prime and sensitize the mother and thus facilitates the performance of maternal behaviour. Progesterone and androgens (e.g., testosterone) may desensitize the mother (or father) and increase the latency of maternal response.

The clinical studies on human sex-hormone abnormalities reported earlier yield mixed conclusions on the role of hormones in mothering-orientation or mothering-readiness. Fetally androgenized females were reportedly less interested than controls in doll-play and other "feminine role-rehearsal" and less interested in marriage and motherhood as their primary life goal (Ehrhardt, 1973). Similarly, boys exposed to prenatal estrogen were reported to be less masculine in interests and athletic ability. Genetic males with androgen insensitivity who are reared as females were feminine in interests and domestic orientation in an almost stereotypic, excessive fashion (Magnus, 1980). Thus, it would appear that estrogen may sensitize the individual to mothering concerns while androgen reduces sensitivity. However, this hormonal hypothesis cannot explain why Turner's syndrome females (who lack ovaries and adequate female hormones) are also feminine and domestically oriented to excess, nor why fetally androgenized males are gentle, shy, and less aggressive, sexually or otherwise, than normal males who have not been exposed to excess prenatal androgen (Magnus, 1980).

Parsons (1980) and Magnus (1980) have suggested that these clinical populations must be regarded as differing from normals in several socio-psychological ways (for example, awareness of their abnormality and parental reaction to it), and that these factors may influence their self-image and the type of sex-role definition they choose to follow. It seems safe to say, at the very least, that the hormonal influence on mothering is more complicated than the estrogen vs. androgen, male vs. female dichotomy, and that there may be optimal levels or optimal ratios between hormones which facilitate maternal orientation and which may be different for men and women because of their differing hormonal sensitivities. In addition, it is quite plausible that hormone levels are influenced by environmental stimuli or other psychological factors as well as vice versa.

The studies on animal maternal behaviour and human mother-child bonding all deal with maternal behaviour in the immediately pre- and post-natal period when the young are being nursed and hence the hormones of parturition and lactation are still of prime importance. Indeed, in the animal kingdom, maternal behaviour usually ceases, often with aggressive rejection by the mother, once the young have been weaned and the mother resumes her normal reproductive cycle (Klopfer et al., 1973). In the human, we find the unique kind of maternal caretaking which extends far beyond the period of early physical dependency and far beyond any of the supposedly maternal hormone states.

As noted earlier, no attempt has been made to relate maternal behaviour in primates or humans to the hormonal fluctuations of the menstrual cycle. But there are a few studies which bear indirectly on the issue. Scruton and Hurbert (1968) found no change in social interaction (grooming, cuddling or

sitting together) over the menstrual cycle in adult talapoin monkeys. In a study not directly concerned with hormones Redmond et al. (1979) found a negative correlation between MAO levels and both aggressive and affiliative behaviour in rhesus monkeys, and a positive correlation between MAO levels and solitary activities. The researchers were examining between-monkey and not within-monkey differences, but it would be interesting to see whether fluctuations in MAO levels across the menstrual cycle affected affiliative vs. solitary behaviour in the same monkey. MAO levels are high in the luteal phase and low in the follicular; thus, if this theory were correct, the follicular phase would be characterized by sociability and the luteal-premenstrual phase of withdrawal.

The relationship between sociability and maternalism is not necessarily a positive one, however, and within the human literature, the scanty evidence that exists seems to suggest a luteal or premenstrual increase in maternalism. In their classic 1942 psychoanalytic study correlating hormonal status and psychic processes in a group of psychoanalysis patients, Benedek and Rubenstein (reported in Benedek, 1973) described the estrogen-dominated follicular stage as one of active heterosexual interest with increasing sexual tension until ovulation, after which there is relief from tension and a "passive-receptive tendency" during the progesterone-dominated luteal phase as the woman prepares for the patient, giving role of mother. As noted earlier, the fall in both hormones in the premenstrual phase results in tension, anxiety, and fearfulness. However, hormone levels fluctuate a good deal in this phase and Benedek found variable, unpredictable behaviour ranging from the active, aggressive, estrogen style to the passive, nurturant, progesterone style.

Using Gottschalk's psychoanalytically derived scoring system (1962), Ivey and Bardwick (1968) analyzed the content of free-association material made by college students at midcycle and again premenstrually. The study can be seriously criticized on methodological grounds because all of the subjects lived in the same college house along with one of the experimenters. This problem aside, Ivey and Bardwick found more themes of anxiety, hostility and lack of self-confidence in the premenstrum, but also found themes of "yearning for love" and anxiety about being separated from love. And as noted earlier, Bardwick (1974) found progestin-dominant pill users higher on self-rated measures of deference, nurturance and affiliation.

Another piece of very indirect evidence linking the premenstrual phase to increased nurturance comes from Tuch's (1975) findings on children's illness and mother's menstrual phase. He noted that more of the premenstrual mothers made spontaneous comments about their child's suffering, which suggested that their excessive concern about the illness might be due to an increase in maternal empathy.

The only controlled correlational evidence linking the premenstrum to increase nurturance is that of Herlihy (1977). She found that the only behaviour which varied significantly across phases in pre-school teachers was physical affection, which peaked at the late follicular phase and again at the premenstrum. This pattern is contrary to that predicted by Benedek, for whom the follicular-ovulative phase was related to low maternalism, the luteal phase to highermaternalism and the premenstrum to unpredictable but lower maternalism.

An interesting, but unrelated and as yet inexplicable finding is that of Levy (1942) that the length of menstrual flow is positively correlated with a maternal nature. Those women who were independently rated as being highly maternal had significantly longer menstrual flows than those rated as low maternal. It seems possible that length of menstrual flow is related to hormone levels or ratios, but this remains to be seen.

In summary, while there is considerable evidence that hormones, notably estrogen and prolactin, facilitate post-partum maternal behaviour, probably by sensitizing the parent to the presence and needs of the young, there is little consistent evidence that hormone fluctuations associated with the normal menstrual cycle, as opposed to the post-partum period, have a predictable effect of positive maternal behaviour. Because of its sedating qualities and its role in maintaining pregnancy, progesterone has been labelled the "maternal hormone" (Bardwick, 1971) and is believed to be associated with passive, patient, nurturant tendencies. On the other hand, however, no cycle-related changes in social behaviour were observed in monkeys, and progesterone is found to inhibit maternal behaviour in post-partum rats. Furthermore, Herlihy (1977) found an increase in physical affection in the premenstrum, when progesterone levels are dropping. Her findings are more consistent with the animal evidence that estrogen facilitates maternalism while progesterone inhibits it.

b) Personality factors. Most of the theory and evidence on personality predisposition, role modelling and socialization forces is not directly relevant here, since they address the question of the capacity to perform maternal

behaviour or long-term influences on its performance, rather than day to day fluctuations in performance. Thus, there is evidence that deprivation, abuse, neglect or rejection in early childhood, and lack of parenting experience or role models (Belsky, 1980) interfere with the development of the empathy, maturity and skills needed to perform positive maternal behaviour.

Chodorow (1978), in a thought-provoking, although highly abstract, psychoanalytic account, traces how the empathic, relationship-oriented personality characteristics needed for mothering evolve in the female child through her unique constellation of relationships with a same-sexed primary caretaker (mother) and more remote, opposite-sexed heterosexual choice. Furthermore, she postulates that women want to mother and develop a strong mother-child bond in an attempt to recreate the early intense and intimate relationship with their own mothers, which cannot be adequately recaptured with men. Thus she provides not only an explanation for the development of mothering abilities but also a motive for their performance. Of interest in the present context is her linking of mothering desires with relationship or succorance needs in the mother. This is reminiscent of findings in the child abuse literature that abusive parents, often immature and deprived in their own childhood, seek the love and care from their children that they never received themselves (Belsky, 1980).

The wish to be mothered or taken care of may be a more or less permanent trait in some individuals, but even most healthy individuals experience the need at particular times of stress, illness or unhappiness. Thus succorance needs in the normal individual may fluctuate day to day and month to month

depending on one's general feeling of competence and well-being. If the premenstrual phase is accompanied by pain, discomfort and especially a depressive, hopeless feeling, succorance needs may be higher than at other times of the cycle when a woman is feeling energetic and at her best. She may derive some satisfaction from close and loving contact with her child.

Whether or not the elaborate and highly speculative psychoanalytic derivation of this hypothesis is accurate, the notion of seeking love and comfort from a child in times of ill-health seems plausible. Indeed, Ivey and Bardwick's (1968) finding about the anxieties and yearning for love in the premenstrual phase seem suggestive of this view. Additionally, Herlihy (1977) found that the nurturant behaviour of her pre-school teachers (measured by direct classroom observation) was positively correlated with self-rated negative mood, a finding also consistent with the succorance hypothesis.

This view, then, predicts a premenstrual increase in positive maternal behaviour independent of any hormonal influence on "maternal impulses". If it were accurate, one might expect to find positive maternal behaviour (particularly physical affection) closely linked to negative affect, and particularly to depression rather than to irritability.

c) Situational factors. Life stress (Egeland et al., 1980), low socio-economic status, poor physical or emotional health, marital conflict (Schaefer & Bayley, 1976), particular characteristics of the child (Bell & Harper, 1980; Gelles, 1973) or of the mother-child interaction (Magnus, 1980; Patterson, 1980) have all been found to interfere with positive maternal behaviour. On the

whole, these factors affect the performance of and not the capacity for mothering, and most can and do exhibit short-term fluctuation as well as long-term. Daily stresses, marital quarrels, illnesses, and child misbehaviour may all reduce positive maternal behaviour on that day, whereas the absence of conflict, stress, or illness will allow optimal mothering.

It is possible for daily stress (or at least perceived stress, cf. Englander-Golden et al., 1977), marital quarrels, and illness in the mother or child (Dalton, 1970, 1977; Tuch, 1975) to covary with the menstrual cycle in such a way that there is maximum interference with positive maternal behaviour in the premenstrual phase. Indeed, it was predicted in a previous section that negative maternal behaviour would increase in the premenstrual phase. However, it is not necessary for negative maternal behaviour to be inversely related to positive. Aggressive and nurturant behaviour may increase simultaneously and findings from the study of altruistic and helping behaviour suggest that they may do so.

Cialdini et al. (1973) have pointed out that inflicting or witnessing harm to another person makes people more likely to exhibit helping behaviour, whether the harm was inflicted by the subject or merely witnessed and whether the retribution is directed at the victim or at an unharmed third person. Cialdini reviewed three hypotheses which have been advanced to account for the phenomenon: 1) guilt; 2) self-esteem bolstering; and, 3) social justice. All account for some but not all of the findings, and the authors propose a more general model whereby they argue that harm done to another (inflicted or witnesses) makes

people feel bad and they then seek to find ways to make themselves feel good again. Altruism (or more generally kindness) is one way to restore good feelings.

These authors argue that any bad mood increases prosocial behaviour and they cite evidence to support their claim. They present a general model describing a U-shaped relationship between mood and prosocial behaviour; both negative and positive moods increase altruistic, helping behaviour, whereas neutral moods do not.

Other researchers have replicated these findings (Mussen & Eisenberg-Berg, 1977), and it seems profitable to apply them to a discussion of mood and positive maternal behaviour, which may be similar to general prosocial behaviour. The altruism-mood model may be applied in three ways: a) premenstrual depressive mood in itself may facilitate positive maternal behaviour as a means of restoring good mood. This is a similar but less explicit formulation than the succorance hypothesis put forth earlier. b) Premenstrual irritability may make the mother more impatient and punitive with her child (thereby inflicting harm). She then feels guilty and overcompensates by lavishing extra love in an attempt to atone and make herself feel good again. (One of the weaknesses of Cialdini's model is that mood seems to be viewed as unidimensional. Thus, the term "negative mood" makes no distinction between anger and unhappiness, for example. Most of the studies, however, seem to manipulate mood towards sadness or guilt rather than anger. It seems useful, therefore, to distinguish two hypotheses, on the assumption that depressive negative affect may lead directly to maternal

kindness, whereas irritability will not unless mediated by guilt). c) If Asso's (1978) model of premenstrual affect is correct, then premenstrual arousability enhances both negative and positive moods; therefore positive maternal behaviour will be greater in the premenstrum simply because positive mood is greater.

In summary, the existing scanty evidence linking positive maternal behaviour to the menstrual cycle suggests that there may be a premenstrual increase in positive maternal behaviour as well as negative. The evidence for a hormonal contribution is equivocal, for the evidence from post-partum nursing animals does not apply to humans, where caretaking is more complex and prolonged far beyond the nursing period. Estrogen and prolactin may facilitate maternal behaviour by sensitizing the mother, while progesterone and androgens may inhibit it. This relationship would predict a midcycle peak (which Herlihy did find) and a luteal dip, but the pattern in the premenstrum would be difficult to decipher.

Environmental and experiential factors play a great role in the genesis and maintenance of positive maternal behaviour, even in animals, and three psychological explanations can be advanced to account for the possible premenstrual increase in positive maternal behaviour without reference to hormones at all. The first emphasizes the succorance needs in the woman suffering from premenstrual symptoms, particularly depression. The second emphasizes her guilt over her irritability and her attempt to overcompensate, and the third derives from the arousal theory of premenstrual affect postulated by Asso

(1978). Both positive and negative emotions are felt more intensely because of premenstrual arousability, and stronger positive mood (including stronger loving feelings) leads to more positive maternal behaviour.

THE PRESENT STUDY

The evidence reviewed so far suggests that although many, perhaps most, normal, healthy women do not report any significant cyclic changes in mood, there are a substantial number who do report suffering from irritability, tension, depression and poor concentration during the premenstrual phase. Both physiological and psychological factors have been implicated in the etiology of these changes.

The present study was designed with two purposes: 1) to determine the role of expectancy in the experience of premenstrual symptoms of mood and concentration, and 2) to examine the possibility of cycle-related changes in maternal behaviour, both positive and negative. Two opposing models, both assuming a premenstrual increase in physiological arousal, have been advanced to explain the interaction between this arousal and the expectaion of premenstrual symptoms. The synergistic model postulated by Asso (1978) predicts that the greater the expectancy, the greater the symptoms. The Stress-reduction model suggested by Rodin (1976) predicts, on the other hand, that the greater the expectancy, the less the distress.

The relative predictive accuracy of these two models was examined by experimentally manipulating expectancy in women who had initially reported premenstrual symptoms of mood and concentration. Volunteer mothers of

preschoolers were divided into three matched groups and their expectancy of symptoms was manipulated by, 1) providing information strongly supportive of universal, biologically-caused menstrual cycle fluctuations (Biological Group), 2) providing information refuting the existence of genuine, biologically-caused cyclic changes and claiming that premenstrual tension is a psychological phenomenon arising out of the myths and expectations of our society (Psychological Group) or, 3) providing no etiological information (Control group). Because the experiment was actively focussing the subjects' attention on premenstrual beliefs, it was not possible to maintain subject naivety and no attempt was made. Subjects were not told of any specific hypotheses, however, nor of the focus on maternal behaviour.

Cycle phase was determined by basal body temperature, the date of the last menstrual period and the usual cycle length. Cognitive performance, maternal behaviour, and the moods of both the mother and her child were measured at both the preovulative and the premenstrual phase. Premenstrual symptoms were assessed by self-report three times, first prior to the experimental manipulation to obtain a measure of usual symptomatology, second after the manipulation to obtain a measure of symptoms expected in the month to come, and thirdly at the end of the month to measure the amount of symptoms actually experienced in the test month.

It was predicted that the group given a psychological explanation would lower their expectation of subsequent symptoms whereas those given a biological explanation would expect more symptoms. Under these circumstances, Asso's synergistic model would predict that, relative to their initial symptom level,

the Biological group would experience more symptoms as a result of the manipulation whereas the Psychological group would experience fewer. Furthermore, the Biological group would report the most premenstrual negative affect and the Psychological group the least. Additionally, the Psychological group ought to manifest an increase in positive mood. In contrast, Rodin's stress-reduction model would predict that the Biological group would experience less premenstrual distress than previously and that they would manifest less premenstrual negative affect than the Psychological group.

In accordance with previous findings, there should be no marked differences between menstrual phases in cognitive functioning. However, Rodin's model predicts that the Biological group, being able to reattribute task-arousal to the menstrual cycle, ought to perform best in the premenstrual phase, whereas the Psychological group, having no such reattribution mechanism, ought to perform worse. Asso's model predicts the reverse effect. Similarly, if Rodin's model is correct, the Psychological group ought to perceive their child as more moody and troublesome in the premenstrual phase, whereas the Biological group ought to perceive their child as even less troublesome than usual because they might attribute the moodiness to themselves rather than to their child. Asso's model does not make any predictions about the perception of environmental tensions.

Maternal behaviour was assessed by direct observation of mother-child interaction in the laboratory, and it was predicted that both positive and negative maternal behaviour would increase in the premenstrum relative to midcycle. According to Rodin's model, the increase in negative behaviour would

be greatest in the Psychological group and least in the Biological, whereas Asso's model would predict the reverse. Three hypotheses predicting an increase in positive maternal behaviour were examined for their relative predictive accuracy: 1) if guilt over irritability enhances positive maternal behaviour, the latter ought to be correlated with negative maternal behaviour and with mood measures of anger and hostility. 2) If succorance needs in the mother motivate her positive maternal behaviour, the latter ought to be correlated with premenstrual symptoms and particularly with depression, and , 3) if Asso's model of premenstrual affect is correct, premenstrual positive mood ought to enhance positive maternal behaviour; in this case the two ought to be correlated and positive maternal behaviour ought to be greatest in the Psychological group.

CHAPTER II

METHODOLOGY

DESIGN

The study utilized a 3 x 2 repeated measures design with two independent variables. Matched groups of subjects were assigned to one of three information manipulations - Biological, Psychological, and Control - designed to raise, lower, or maintain expectations of symptoms respectively. All subjects were then assessed at each of two phases of the menstrual cycle - the Preovulative Phase (about Day 9 to 12 of a standard 28-day cycle) and the Premenstrual Phase (about Day 25 to 28).

Seven dependent variables were examined, four pertaining to the mother - positive and negative maternal behaviour, mood, and problem-solving skills - and three pertaining to the child - positive and negative behaviour, and troublesomeness as rated by both parents.

SUBJECTS

The subjects were 51 normal, volunteer mothers recruited from the general Ottawa population through the preschools and through advertisements. Initial applicants were screened for the following characteristics:

1. Married and between 25 to 40 years old.
2. Having at least one child between 2 and 4 years who is physically healthy and who has never been referred to a mental health specialist.

3. Reporting normal, regular menstrual cycles predictable within two or three days.
4. Not on mood-altering drugs, birth control pills, migraine medication, or hormone treatments, not pregnant either now or in the last six months, not breastfeeding.
5. Reporting at least some moderate symptoms of premenstrual negative affect. This criterion was evaluated by a procedure described below in the Procedure section.

MEASURES

a) The Response-Class Matrix designed by Mash et al. (1973, 1979) was used to systematize the observations made of the mother-child interaction in the laboratory. Developed out of a need for a reliable, objective method for assessing therapeutic change, it is one of the few observational coding systems in existence which has been shown by several researchers (Cunningham & Barkley, 1979) to be sensitive to change following treatment intervention, and which has a detailed manual including category definitions, reliability data, a computer program, and an observer training procedure (Mash et al., 1979).

The Response-Class Matrix organizes the discreet behaviour units into sixteen response classes, which are more global groupings of behaviours assumed to have features in common and to be maintained by similar contingencies in the environment. There are eight response classes for the child - compliance, independent play, competing behaviours (noncompliance), question, negative, verbal interaction, nonverbal interaction, and no response - and eight for the

mother - command, command-question, question, praise, negative, verbal interaction, nonverbal interaction, and no response.

These two sets of classes are organized as rows and columns of two matrices, each scored simultaneously by a separate observer. One matrix codes the mother's behaviour as a response to the child's and is referred to as the Child Antecedent/Mother consequent (C/M) form. This form allows one to see at a glance how the mother responds to different classes of behaviour from her child. The second matrix, the Mother Antecedent/Child Consequent (M/C) form, codes the child's behaviour as a response to the mother's. By analyzing both matrices, and S-R-S picture is obtained depicting not only how a particular maternal behaviour functions as a stimulus to the child, but also how the child's response affects the mother in return.

Rather than using a continuous recording technique, the observation period (in the present study, 16 to 17 minutes) is divided into ten-second recording intervals, each followed by a five-second rest period. In each ten-second interval, only the first behaviour unit to occur is scored. A behaviour unit consists of an S-R-S chain yielding three pieces of information, for example, mother's initial interaction, child's response, and mother's reaction to that response. Behaviour is scored by placing a hash mark (or the interval number, as was done in the present study) in the appropriate box of each matrix. Thus at the beginning of a ten-second interval mother says 'Don't touch that', child obeys, and mother says 'Good boy'. The observer using the M/C matrix would place a mark in the Mother Negative - Child Compliance box, and the C/M observer would mark the Child Compliance - Mother Praise box.

Appendix A presents sample forms of both matrices. The boxes have been numbered in order to permit identification and explanation of the summary measures discussed below. By counting the scores in a particular box, the number of specific mother-child behaviour combinations can be obtained. In addition, any number of summary measures can be calculated by combining the boxes in different ways. For example, by summing rows or columns, the total number of behaviours in a particular response class may be obtained, and ratios or percentages calculated.

Mash et al. (1979) provide a list of forty-four summary measures, and others can be derived to suit the needs of a particular study. In the present study, the following measures were used:

Measures of positive maternal behaviour

I. M/C form: Mother's Interaction % - sum of question, praise, verbal and nonverbal interaction rows.

$(\Sigma\ 15 -- 28) + (\Sigma\ 36 -- 49)$/total no. of observations.

II. C/M form: Mother's positive control of child's negative and competing behaviours %.

$17 \rightarrow 20 + 22 + 23 + 33 \rightarrow 36 + 38 + 39 / (\Sigma\ 17 -- 24) + (\Sigma\ 33 -- 40)$

Measures of negative maternal behaviour

III. M/C form: Mother's Negative %.

$(\Sigma\ 29 -- 35)$ / total.

Measure of Child's Positive behaviour

IV. C/M form: Child's Compliance, Question, and Interaction %.

(Σ 1 -- 8) + (Σ 25 -- 32) + (Σ 41 -- 56) / total.

Measure of Child's Negative behaviour

V. C/M form: Child's Negative and Competing Behaviours %.

(Σ 17 -- 24) + (Σ 33 -- 40) / total.

Scoring of the matrices was done from videotapes by two volunteer undergraduate students who had no knowledge of the subjects' experimental condition. They were trained according to the guidelines set down by Mash (1979), first using the lesson plans provided in the manual and later using a test tape which simulated the actual experimental procedure. Although each student initially learned both matrices, they were later assigned to score only one of the two matrices in order to reduce confusion and increase reliability. Individual practice and group discussions continued for several weeks until reliability (no. of agreements / total no. of scores) between each student and the experimenter was over .80. The actual reliability of any one tape depended on its difficulty and ranged from .80 to 1.

Once training was complete, the raters were told that the experimenter would score randomly selected tapes at irregular intervals in order to check reliability. This technique of irregular spotcheck threats was suggested by Talpin and Reid (1973, reported in Quay & Werry, 1979) as a mean of counteracting the documented fade in interrater reliability over time.

When half of the tapes had been scored, the experimenter performed reliability checks on randomly selected tapes and obtained a reliability score of .82 for the M/C form and .80 for the C/M form. Feedback on this check and on the nature of disputed scores was provided to the rater.

b) The Conners' Parent-Teacher Questionnaire (PTQ, Conners, 1973) was used to assess the child's perceived troublesomeness, as rated by both the mother and the father.

No well-standardized, commonly used measure could be found which entirely met the needs of the present study; that is, a parent-rated scale which measured daily fluctuations in their child's mood and behaviour within the normal population. The Conners' PTQ is a clinical scale which grew out of Conners' work in psychopharmacology, and as such it is intended for use with deviant or disturbed children. It has, however, been standardized against normal children, and because it focusses on activity level, impulsivity, and aggressivity, and has proved to be sensitive to changes in these variables as a result of pharmacological treatment (Conners, 1972), it seemed appropriate for use in this study. In order to circumvent the tendency for normal children to score low or not at all on the scale, certain modifications were made which will be described below.

The PTQ consists of ten questions from the Conners Teacher Questionnaire (36 items) and the Conners Parent Questionnaire (93 items), selected because they were among the items most often checked by parents and teachers in response to drug changes. They were found to be sensitive to drug change and also to

correlate well with the longer scales (Werry, Sprague, & Cohen, 1975). The longer scales yield four different symptom clusters and hence are more useful for differential diagnosis, while the PTQ yields only one Disturbance score and because of its brevity and ease of administration, it is recommended for repeated assessment of progress (Werry, 1977).

All three questionnaires were selected for inclusion in the test battery compiled by the Early Clinical Drug Evaluation Unit of the NIMH, and hence are being used more and more extensively. To suit the needs of this study, the instructions and the wording of the PTQ have been changed, and the modified version is presented in Appendix B. Briefly, 1) the instructions have been changed from "Rate the presence and severity of symptoms in your child as he is currently functioning" to "Rate your child's behaviour today, compared to what it is usually like". 2) To increase variability, a seven-point ipsative scale has been substituted for the usual four-point scale, with the midpoint labelled "as usual", the lowest point labelled "much less than usual" and the highest point "much more than usual". 3) All verbs describing behaviour were changed from present to past tense, to emphasize the temporary and past nature of the behaviour. The words "drastically" in item 9 and "explosive and unpredictable behaviour" in item 10 were deleted. These changes lessen the pathological implications of the scale in the parents' eyes.

These changes, of course, make the use of the previously established norms invalid, but since it was not the child's deviance but the mother's percpetion of him during different menstrual phases that was of interest, scores were not compared against externally set norms but against the same child's score at the other menstrual phase.

c) Moos' Menstrual Distress Questionnaire (MDQ) was used to assess both recollection of and expectation of menstrual cycle symptoms. The MDQ is a list of 47 symptoms, most of them commonly reported by women at some phase of their cycle, although six control symptoms are included to assess the tendency to report symptoms in general. It was developed by Moos in 1968 and is widely used as a standard self-report tool for assessing perceived behavioural, affective, and somatic changes during the menstrual cycle.

Factor analysis of the 47 symptoms yielded eight symptom clusters labelled Pain, Water Retention, Autonomic Reactions, Negative Affect, Arousal (pleasant), Concentration, Behavioural Change, and Control (the group of non-menstrual symptoms included to check response bias). A sample of the MDQ is found in Appendix C.

Instructions can be and have been varied to suit the needs of a particular study (Clarke & Ruble, 1978; Parlee, 1974; Wilcoxin et al., 1976), but in the general form subjects are instructed to rate the presence and severity of each symptom on a six-point scale, as it is experienced at three different phases of their last cycle - once for the menstrual week, once for the premenstrual week, and once for the remaining intermenstrual weeks. Comparisons can then be made between symptoms and across phases in the same subject. In the present study, three different sets of instructions were used, the first asking subjects to rate the symptoms they usually experienced, the second the symptoms they expected to experience in the coming month, and the third the symptoms they actually experienced over the last month.

The following two questions were added to the standard MDQ form, to be answered on a four-point scale from "Not at all", to "Somewhat", to "Quite a lot", to "Very Much".

1. What do you believe is the main cause of your premenstrual symptoms?

   Hormones _ _ _ _
   Water Retention _ _ _ _
   Worry or dislike of coming period _ _ _ _
   Other psychological reasons _ _ _ _

2. What do you believe is the main cause of your menstrual symptoms?

   Hormones _ _ _ _
   Water Retention _ _ _ _
   Worry or dislike of your period _ _ _ _
   Other psychological reasons _ _ _ _

The MDQ is subject to the usual weaknesses of self-report measures (selective perception and recall, social desireability bias, attitudinal and personality biases) and, as discussed earlier, it has recently been criticized as measuring little more that culturally stereotyped expectations. It was used in this study not as a measure of genuine behaviour change, but as a measure of the subject's perceptions, beliefs, and expectations about her menstrual fluctuations, which are hypothesized to influence at least what she experiences and how she perceives it, if not how she actually behaves.

d) Two problem-solving tasks were used to assess the subject's concentration, efficiency, and ability to screen out environmental interference in the laboratory setting: 1) anagram solution (10 minutes), and, 2) line-puzzle solution (7 minutes). Both of these were included in the battery Rodin (1976) provided for her attribution testing.

Mayzner and Tresselt (1966) provide norms for 72 five-letter anagrams, giving the Thorndike-Loge frequency of the solution word and the median solution time required for each anagram. From these 72 words, two equivalent lists of 20 words each were drawn up, matching Thorndike-Loge frequencies and solution times as closely as possible for each word (see Appendix D). Each of these lists was typed on a single page in ascending order of solution time (and therefore of assumed difficulty) with a blank space beside each anagram for the solution. Subjects were instructed to make no other marks on the page. The total median solution time for each list is 21 minutes, 18.5 seconds, according to Mayzner's and Tresselt's norms, and thus it was expected that very few subjects would get near the end in the ten-minute time limit. This ought to have increased the feeling of pressure on the subject.

Anagram-solution is a complex verbal problem-solving task requiring memory, cognitive flexibility, and successive hypothesis testing. As such, it ought to be impaired to the extent that the subject is distractible, and unable to concentrate because of high arousal, excessive environmental interference, or an interaction of the two. It was also assumed that attempts to solve anagrams under distracting conditions would produce added frustration which would facilitate the expression of maternal aggression and irritability (Schonberg, 1976).

Using Feather's four original line puzzles (1961) and adding four of their own, Glass and Singer (1971) used a series of five soluble and three insoluble line puzzles to test subjects' frustration tolerance under conditions of

environmental stress (noise). Rodin used four of these in her study to measure stress tolerance and persistence as well. Two sets of two puzzles each, the first insoluble, the second soluble, were selected from the Glass and Singer group for use in the present study (see Appendix D). To solve the puzzle, the subject had to trace over all the lines of the puzzle without lifting her pen or going over any line twice. The number of trials a subject persisted on the insoluble puzzle before going to the soluble one was taken as a measure of stress tolerance. Each puzzle was printed on a card 4" x 4 3/4", and a one-inch pile of cards for each puzzle was placed face down in front of the subject, with instructions that each solution attempt be done on a new card. This task was also chosen partly to enhance maternal irritability.

Each pair of puzzles was grouped with a particular anagram list, and the two resulting test batteries were labelled A and B. Each subject received each test battery once, in an order balanced across Expectancy manipulations and menstrual phase of first testing.

e) The Profile of Mood States (POMS - McNair, Lorr, & Droppleman, 1971) was used by the subject and her husband to rate the subject's moods. The POMS is a standardized mood adjective checklist published by the above authors after years of work on adjectives and mood description (e.g., Lorr et al., 1967). It has very good reliability and validity data, is well recommended by its two reviewers in Buros (1978) and is recommended by NIMH for assessing psychotherapy change (Waslow & Parloff, 1975). It has been standardized for both psychiatric outpatients and college students, and its authors recommend its use for anyone with a high school education.

Because it is fairly recent in its present form, it has only recently been used in menstrual cycle research (Alplanalp, 1980; Parlee, 1980), but it has demonstrated sensitivity to mood change due to psychotherapeutic and pharmacological intervention, due to sleep deprivation, and due to experimental manipulation of emotions (McNair et al., 1971). In addition, many of its adjectives and factors are similar to the Nowlis Mood Adjective Checklist (Nowlis, 1965), a research tool which has been successfully used as a menstrual cycle mood measure.

The POMS is a list of 65 adjectives describing feelings and moods to which the subject is asked to respond on a five-point scale from "not at all" to "extremely". By repeated factor-analysis, six oblique, moderately correlated factors have been derived: Tension-Anxiety (tense, shaky, etc.), Depression-Dejection (blue, worthless, etc.), Anger-Hostility (angry, grouchy, etc.), Vigour-Activity (lively, active, etc.) Fatigue-Inertia (worn-out, listless, etc.) and Confusion-Bewilderment (confused, forgetful, etc.).

Norms are provided for male and female outpatients and for college students of both sexes combined. The authors recommend its use with the college norms for research purposes, provided the subjects are over 17 years old and have some high school education. In the usual form the instruction direct the subject to report his moods "during the past week including today". This has been modified to "right now" for some kinds of research and in the present study, the subject and her husband were instructed to report how the subject "has felt in general during the past day".

EXPECTANCY MANIPULATION

The subjects, once selected, were divided into three equal groups and given one of the following "explanations" of the cause of premenstrual tension: Biological information, Psychological information, and No information. The latter group acted as an experimental control and were told simply that the study was examining moods and mental ability as they relate to the menstrual cycle.

For the two experimental groups, the information manipulation was composed of three parts:

1. Reading of an article published in a reputable journal and written by a bonafide professional, discussing premenstrual tension.
2. Viewing of a 20-minute videotape of a prominent Ottawa gynaecologist*, also discussing premenstrual tension.
3. An informal, forty-minute discussion group led by the Experimenter, dealing with the contents and implications of the foregoing material.

In the Biological group, the videotape and the article strongly endorsed the notion of physiological etiology for genuine, universal fluctuations in mood and concentration, leading subjects to confirm their own experience and enhance their expectations of symptoms in those areas. The transcript of the

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* The author is indebted to Dr. Norman Barwin, B.Sc., M.B., Ch.B., B.A.O., M.D., M.R.C.O.G., F.A.C.O.G., for his assistance and participation in this matter.

videotape (actually written by the experimenter and acted out by the doctor) is presented in Appendix E, and the article was an edited and abridged version of one published in the Journal of Clinical Endocrinology and Metabolism, by Klaiber et al. (1971).

In the Psychological group, both the videotape and the journal article presented the view that premenstrual tension is not due to any biological fluctuations but rather to a wide-spread and long-held negative myth which has several psychological consequences; it engenders a self-fulfilling expectation of symptoms, it induces a bias in perception and recall which gives an illusion of premenstrual tension where none exists, and it results in the labelling of ambiguous psychological arousal as negative emotion. This refutation of genuine, biologically based mood changes was designed to lead subjects to reevaluate their past experience and to reduce their expectation of premenstrual problems in affect and concentration.

The journal article was an edited and abridged version of 'Women learn to sing the menstrual blues' by Karen Paige (1973). Appendix E presents the transcript of the videotape, also done by the same doctor. To accompany this videotape, subjects were presented with a chart summarizing in four points the ways in which the myth augments premenstrual tension. The Biological tape was accompanied by an explanatory graph outlining the rise and fall of estrogen and progesterone over the cycle.

A preliminary pilot study was carried out to determine whether these two information manipulations were effective in altering expectations in the desired direction. Using mothers who had volunteered but who did not qualify for the

main study, two groups of eight subjects were formed. The journal article was mailed to them about a week in advance, together with an abbreviated symptom questionnaire presented in Appendix F, which measured their symptomatology in five areas (pain, swelling, depression, irritability, and poor concentration) and assessed their preliminary attributional beliefs about the etiology of these symptoms, using the same attribution questions which were added to the end of the MDQ. They were instructed to fill out the questionnaire before reading the article.

The eight subjects in each expectancy group were then gathered together and subjected to the manipulation as described above. At the end of the hour they were given a second brief questionnaire with the same questions on symptomatology and etiology, together with questions on how helpful and applicable the information had been to them (see Appendix F). They were asked to rate the symptoms they would now expect in the future, and once the questionnaire had been filled in, the subjects were debriefed.

Table 1 presents the results of the pilot study. Comparison of the pre and post-manipulation symptom scores revealed that in the Psychological group, the mean symptom score was significantly lower after the manipulation than before ($t = 3.00$, $df = 7$, $p < .02$). In fact, six of the eight subjects expected fewer symptoms, while the other two showed no change. None of the subjects expected more symptoms.

In the Psychological group there was also a significant decrease in biological attribution ($t = 3.91$, $df = 7$, $p < .01$) and a significant increase in psychological attribution ($t = 2.57$, $df = 7$, $p < .02$). Seven of the eight subjects reduced

Table 1

Mean Symptom and Attribution Ratings Before and After the Information Manipulations in the Pilot Study

	Pre-measure	Post-measure	SD	df	t
Symptoms					
Biological Group	8.63	9.63	2.53	7	1.12
Psychological Group	9.25	6.13	2.94	7	3.00*
Biological Attribution Ratings					
Biological Group	6.13	5.88	1.75	7	.40
Psychological Group	5.63	3.75	1.36	7	3.91**
Psychological Attribution Ratings					
Biological Group	2.13	2.38	1.28	7	.56
Psychological Group	2.75	3.88	1.25	7	2.57*

*$p < .025$
**$p < .01$

their biological attribution and six increased their psychological. In addition, six of the eight found the information they had gained sensible and helpful to them.

The Biological manipulation produced less clear-cut results. Although there was an increase in the mean symptom score from the pre- to post-measures this difference was not significant ($t = 1.12, df = 7, p = n.s.$). Four subjects increased their expectation of symptoms, two remained the same (one of these had an initial symptom score of 0 and maintained it), and two decreased theirs slightly. One of these had an abnormally high initial score. If the two extreme scorers are eliminated, four of the remaining six showed shifts in the desired direction.

There were no significant changes in attributional beliefs in the Biological group. This was due in large part to floor and ceiling effects. Even prior to the manipulation, seven of the eight attributed their symptoms to hormones 'very much' or 'quite a lot', whereas seven of the eight blamed psychological factors 'not at all'. Thus it was difficult to raise biological attribution significantly, and almost impossible to lower psychological attribution.

On the basis of the pilot study, the information manipulations were judged to be effective in altering expectations in the desired direction, although the Psychological manipulation seemed to be the more powerful.

A further check on the manipulations was accomplished in the main study by measuring each subject's expectations of symptoms by means of a second MDQ after the treatment manipulation. This will be discussed in more detail in subsequent sections.

EQUIPMENT AND SETTING

The testing room was an 11 x 9 foot room carpeted in green and painted white, with an unused one-way mirror along one side. The following set-up remained the same for each testing. There was a small table and chair in one corner facing the wall, at which the mother was seated. On the table was a copy of the anagram list, face-down, two piles of puzzle cards, face-down, an index card with instructions for the puzzles, a red pen, and a simple kitchen timer with a loud tick. Beside the table was an empty waste-paper basket.

Behind the mother's chair was a large, attractively painted wooden doll's house furnished with dolls, cars, furniture and tiny dishes designed to interest a preschooler. On the floor was a box of Playskool wooden blocks, a drum with two sticks, a bell, and a toy flute with a loud, off-key sound. In the middle of the room was a large, rather ricketty easel with fresh paper, two large paint brushes and two full pots of water paints.

On a tripod in the opposite corner of the room, positioned for maximal observation of the mother, was a Sony black and white video camera, model CVC-2100A. On the floor against the wall beside the mother's table was a Sony cartioid microphone. Both the microphone and the camera were connected by extension cord through a hole in the wall to a Sony-matic Solid State video-recorder, model AV-3600, and a nine-inch Sony transistor TV receiver, model TV-960, which were both located in the adjoining observation room. One-half inch Sony and Scotch videotapes were used to record the mother-child interactions for later scoring.

Also attached by auxiliary input to the video-recorder was a Bell and Howell cassette tape recorder, model 3191C, containing an audiotape on which the observational intervals were premarked for scoring the Response-Class matrix.

The start of each ten-second observation interval was denoted by its number, from one to seventy, and the end of the ten seconds marked by two rapid clicks. After five seconds had elapsed, the next number sounded. This tape was activated simultaneously with the video-recorder, so that each subject's tape was marked with permanent auditory scoring intervals.

PROCEDURE

Once the subjects had been screened according to the criteria listed above (except for degree of symptomatology), they received an MDQ in the mail, asking them to rate their symptoms as they usually experienced them during a typical month. Completed MDQs were mailed back to the experimenter for scoring and subject selection.

MDQ symptoms scores were calculated not from the absolute scores but from the difference between the premenstrual and intermenstrual rating for each symptom, which produced a clearer picture of symptom change independent of base-line levels. Since only the Negative Affect and Concentration scales were being directly manipulated by the experimental treatment, only those two symptom scales were used in the calculation. Thus, the MDQ score referred to in this study is the sum of the premenstrual-intermenstrual differences of all the symptoms in the two scales.

A cut-off point of six was selected after careful study of the responses, and any subject with an MDQ score below six was excluded from the study. Six was chosen because all the scorers at the lower end of the spectrum complained far more of negative affect than of concentration problems, and of the negative affect items, 'irritability' was the most often reported. Even among low scorers between 6 and 10, irritability was rated as 'moderate' or more often 'strong'. Since irritability was a major focus of the study, these subjects were judged acceptable.

Once the final selection had been made, subjects were assigned to one of the three experimental groups. Assignment was random except that an attempt was made to maintain equivalence between the groups on the four variables of initial MDQ score, age, age of child and sex of child.

Each subject was then visited at home and given a brief outline of the nature of their participation. It was explained that the study focussed on mothers with small children at home because no one had ever tried to investigate this, despite the unique frustrations and extraordinary demands of motherhood. They were told that the study would be measuring moods and mental ability at two different times in the cycle, and that mental ability would be measured by two brain-teaser tests done at the university. In order to approximate real-life, where they had to accomplish their work with a child constantly underfoot, they were to bring their preschool child with them to provide the built-in distraction. They were given a sample temperature chart for reference and a chart on which to record their morning temperatures, with instructions on how to obtain a basal temperature.

Also included in the home visit was the administration of the short form of the MMPI (399 questions). Information concerning the subject's educational, employment, and family income status was also obtained.

In the case of the control group, recent menstrual information was obtained and, when possible, an appointement for the first testing was made, based on the date of the last menstrual period and the usual length of the subject's cycle. Control subjects were also given a second MDQ asking them to indicate the presence and severity of the symptoms they expected to feel during the month to come (the test month).

Subjects in either of the experimental groups were given the appropriate article to read and the date of the discussion group. Menstrual information was taken at the end of the discussion group or in a subsequent phone call.

Subjects in the same Expectancy group were gathered together in groups of five to eight, and subjected to the manipulations described earlier, as in the pilot study. At the end of the forty-minute discussion, the second MDQ was given to each subject, with instructions to fill it out in the next day or two. The difference between the second MDQ (expected symptoms) and the initial MDQ (usual) was a measure of the shift in expectations and hence a further check on the effectiveness of the manipulations.

All subjects were requested to avoid discussing the study with anyone, particularly their husbands, until participation was completed. They were also requested not to tell their husbands about their menstrual status, in order that he could be as objective an observer as possible.

As far as possible, subjects were given their premenstrual and preovulative assessments during the month immediately following the discussion group. The menstrual status of each subject was carefully monitored by the experimenter over the phone, and ovulation dates determined by the experimenter from the temperatures provided by the subjects.

To control for order effects, half the subjects in each group were tested first at the preovulative phase, and half of these were given test battery A, the other half battery B. The other half of each group were tested first at the premenstrual phase, with similar balancing of test batteries. To control for the effects of fatigue and diurnal variation, subjects were tested between 9:30 and 11:30 a.m. whenever possible. Eighty percent of the assessments took place between these times.

On arrival at the University laboratory for the first testing session, the subject and her child were met by the experimenter and the MDQ-2 collected. They then proceeded to the testing room and received the following instructions:

> If you recall, this session lasts seventeen minutes. You're to sit here, and here are the two tests (E indicates). Start with this one (points to anagram sheet lying face down), read the instructions at the top and then fill in the answers. I'll set this timer for ten minutes, and when it rings, turn over the sheet again and start on this task. These are two puzzles, marked 1 and 2. They are geometrical designs which you have to solve by tracing your pen over all the lines of the design without lifting your pen or going over any line twice. You can take as many tries as you like, there are plenty of cards here, and for each new try take a new card and throw the old one in the basket here. All the cards in this pile are copies of puzzle 1, and these are puzzle 2. You have seven minutes to do both puzzles combined, and you can divide the time any way you like, but if you decide to give up on the first to try the second, then you can't go back to the first for another try. I'll be back at the end of seventeen minutes, and in case you've forgotten any of the instructions for the puzzles, they are written for you on this card.

> Your child (name) can play with any of these toys here, except please don't let him touch the doll's house. There are some very valuable, breakable things in it, so he can look at it, but not touch it. Thanks a lot. Now I'll set the timer and leave you to it.

The experimenter then left the room and activated the video-recorder in the room next door. After the testing session the subject was thanked and given two copies each of the POMS and PTQ, with instructions that they be filled out by herself and her husband that evening, rating her feelings and behaviour of the last day. She was reminded that they were to fill out the questionnaires without consultation. These were to be placed in a manilla envelope and returned to the experimenter at the next session.

Before the subject left, her menstrual status was reassessed by studying the temperature charts and the date of the last period. Status was reassessed again a week later by phone and the next appointment set. Procedures for the second testing session were identical to the first, and instructions were such as to refresh the subject's memory about the tests and the doll's house.

After the second, final testing, the subject was given a third MDQ in addition to the two POMS and PTQs. She was asked to wait three or four days (or until her period came, in the case of premenstrual testing) and then to rate her symptoms as they occurred in the last month (i.e., the test month), according to the usual rating method. She was also asked to continue her temperature chart for a week, or until her period came, to note down the date of onset, and to mail the chart to the experimenter along with the completed MDQ, POMS, and PTQs in a stamped, self-addressed envelope provided by the

experimenter. She was then thanked for her cooperation and told that she would be contacted once the questionnaires were received in the mail. The false information was rectified at that time and the subject given feedback on her participation.

Two of the 51 subjects were found to have been tested at grossly inaccurate times (in both cases, two days or so after ovulation, not before), and they were retested the next month.

The videotapes were given a code number by which only the experimenter knew the expectancy condition and menstrual phase of the subject, and then were scored by the student raters.

The above procedure resulted in three MDQ scores for each subject:

1. A measure of pre-experiment symptomatology - the extent to which the subject perceived herself to be affected by menstrual fluctuations.
2. A measure of post-manipulation expectations - the amount of symptomatology the subject expected to experience during the next cycle, after having been subjected to one of three experimental conditions.
3. A measure of post-experiment symptomatology - the extent to which the subject perceived herself as having symptoms in the test month.

The attribution questions in the MDQ resulted in three measures of biological attribution and three measures of psychological attribution for each subject:

1. A measure of the subject's original beliefs about etiology.
2. A measure of her beliefs after being subjected to one of the three experimental conditions.
3. A measure of her beliefs at the end of a month's experience and reevualuation.

The cognitive tests generated two scores per session for each subject, replicated twice:

1. A problem-solving efficiency score - no. of anagrams solved.
2. A frustration tolerance score - no. of trials spent on the insoluble puzzle.

The two mood checklists resulted in four sets of scores per phase for each subject, replicated twice:

1. Six mood scores derived from the self-rated POMS.
2. Six mood scores derived from the husband-rated POMS.
3. A Conners PTQ score on the child as perceived by the mother.
4. A Conners PTQ on the child as perceived by the father.

Direct observation resulted in five scores per phase for each subject, replicated twice:

1. Mother's Interaction %.
2. Mother's Positive Control %.
3. Mother's Negative %.
4. Child's Negative and Competing Behaviours %.
5. Child's Compliance, Question and Interaction %.

CHAPTER III

RESULTS

All parametric statistical analyses were conducted using the SPSS programs (Hull & Nie, 1981; Nie et al., 1975) unless otherwise indicated. The .10 level of significance for multivariate effects was adopted throughout, as recommended by Stevens (1980) because of the test's poor power with small to moderate effects and sample sizes. If multivariate effects were found to be significant at the .10 level, univariate analyses were conducted, and the .05 level of significance was adopted for both these univariate analyses of variance and the "a posteriori" means comparisons, which were calculated by hand according to the Newman-Keuls formula.

Before any analyses were performed, the within-cells variances were examined for homogeneity and in doubtful cases were tested with both Cochran's C and Hartley's Fmax (Kirk, 1968, pp. 61-62). Unless otherwise specified, the variances were found to be homogeneous at the .05 level.

GROUP AND SAMPLE CHARACTERISTICS

Table 2 presents the sample and group means on eight background variables and the thirteen scales of the MMPI. One-way analyses of variance were performed on each of these variables and none reached significance, suggesting comparability between the experimental groups on a wide variety of possible concomitant variables.

Table 2

Comparison of Group Means on Background and Personality Measures

	Sample Mean	Psychological Group	Control Group	Biological Group
MDQ	22.53	21.29	22.18	24.12
Age	32.41	33.47	31.59	32.18
Age of Child	3.39	3.55	3.12	3.49
Education[a]	2.33	2.18	2.12	2.71
Family Income[b]	2.88	2.71	2.71	3.24
Cycle Length	27.84	27.65	28.29	27.59
Days preovalutive at testing	3.16	2.67	3.67	3.13
Days premenstrual at testing	2.78	2.94	2.65	2.76
MMPI: L	46.73	46.71	45.41	48.03
F	52.67	53.76	52.53	51.71
K	53.29	51.82	52.76	55.29
Hs	50.25	49.24	48.94	52.59
D	55.84	58.24	54.29	55.00
Hy	56.45	55.00	55.53	58.82
Pd	53.94	54.18	50.71	56.94
Mf	43.86	44.06	42.29	45.24
Pa	53.90	50.65	55.47	55.59
Pt	54.41	53.59	52.94	56.71
Sc	53.94	52.94	52.35	56.53
Ma	53.08	51.41	53.76	54.06
Si	52.67	55.06	52.00	50.94

[a]Education: 1) high school; 2) post-secondary diploma or some college; 3) college degree; 4) post-graduate.

[b]Family Income: 1) less than $15,000; 2) $15,000 - 25,000; 3) $25,000 - 35,000; 4) above $35,000.

Table 2 indicates that the average ages of the subject and of her child were 32.4 and 3.4 years respectively. The level of education was fairly high: 78.5% of the sample had some diploma or coursework beyond a secondary-school certificate, compared to about 55% of comparably-aged women in the metropolitan Ottawa area as a whole (Census of Canada, 1976). The average income per family in the sample was about $25,000; this figure is comparable to the average for Ottawa, which was $28,290 in 1979 (Survey of Consumer Finance, 1980).

Examination of the MMPI scales reveals that the sample as a whole had scores above the 50th percentile on quite a few of the scales, most notably on the D, Hy, and Pt scales. The average score on the Mf scale was 43.9 probably reflecting the homemaking and mothering role of the subjects. Only 2 of the 51 subjects had a full-time job outside the home, while about 30% held part-time jobs.

Menstrual cycle length ranged from 22 to 35 days, with a mean of 27.84 days. On the average, subjects were tested 3.16 days before ovulation (range 0 to 9) and 2.78 days before-menstruation (range .5 to 7).

The distribution of birth control methods across the three groups was fairly even (see Appendix G); in the whole sample, 10 subjects used physical barriers, 12 had an IUD, 11 tubal ligations, while the husbands of 8 had had vasectomies. Birth control method was nonexistent or unspecified in 10 subjects, although initial screening had, of course, eliminated birth control pill users.

MOOS MENSTRUAL DISTRESS QUESTIONNAIRE

Preliminary one-way analyses of variance of the MDQ, biological attribution and psychological attribution pre-measures revealed no significant differences in

baseline values between the groups on any of the variables (see Appendix H). Pearson-product-moment correlation coefficients among the three pre-measures are reported in Table 3 and reveal a significant correlation of .38 between the amount of symptoms and biological attribution, and an even stronger correlation of .57 between amount of symptoms and psychological attribution. These three variables were therefore analysed together in a 3 x 3 multivariate analysis of variance with repeated measures, the results of which are summarized in Appendix I. There were significant main effects for both Expectancy group and Time of Testing as well as a highly significant interaction.

Because the multivariate analysis yielded significant effects, each variable was then subjected to a univariate analysis of variance with repeated measures. The MDQ variable did not exhibit a significant Group effect, but the repeated measures effect (Time of Testing) and the interaction effect were both significant: $F(2,96) = 3.38$, $p = .04$ and $F(4,96) = 4.06$, $p = .004$ respectively (see Appendix J). The mean MDQ scores are presented in Table 4 and the interaction is graphically depicted in Figure 1.

As Kirk (1968) recommends in the case of a significant interaction, tests of simple effects were done, revealing a significant Expectancy group effect at follow-up (i.e., in symptoms actually experienced during the test month), $F(2,66) = 4.21$, $p < .05$. Newman-Keuls comparisons of follow-up scores revealed that the Psychological group was significantly lower than the Biological and Control groups, who did not differ from each other. A highly significant repeated measures effect was also found within the Psychological group,

Table 3

Pearson Product-Moment Correlation Coefficients
Between Initial MDQ and Attribution Ratings

	MDQ	Biological attribution	Psychological attribution
MDQ	-	.38**	.57**
Biological attribution		-	.20
Psychological attribution			-

*p < .05
**p < .01

Table 4

Mean MDQ Score for Each Expectancy Group in the Pre-, Post-, and Follow-up Assessment

	Psychological Group	Control Group	Biological Group
Pre-measure	21.29	22.18	24.12
Post-measure	14.18	21.53	26.29
Follow-up measure	10.06	23.12	23.24

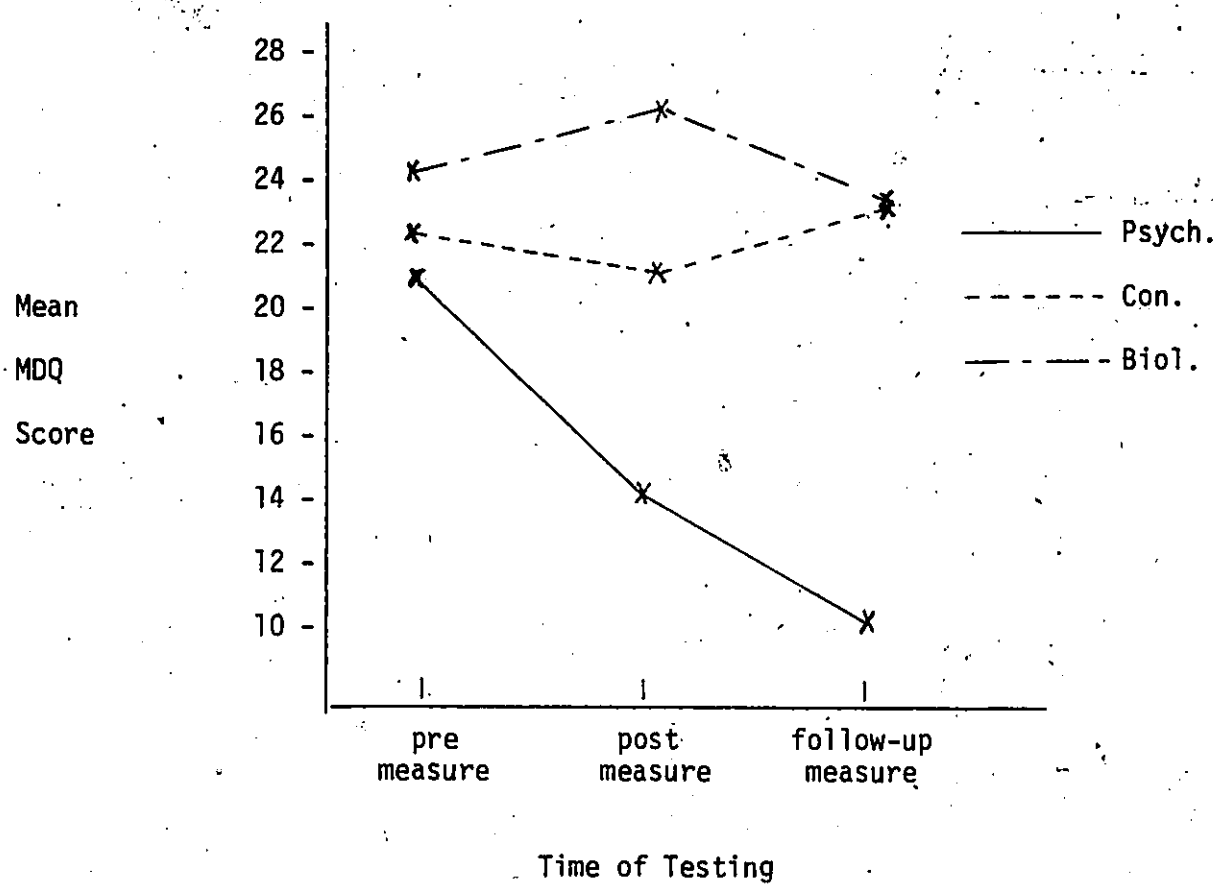

Figure 1. Mean MDQ score for each Expectancy group in the pre-, post-, and follow-up assessment.

$F(2,96) = 10.48$, $p < .001$, and Newman-Keuls comparisons revealed that the post-and follow-up measures were both significantly lower than the pre-measure but not significantly different from each other ($p < .10$). A similar decrease over time did not occur in the other two groups.

The analysis of variance of the biological attribution ratings, reported in Appendix K, yielded significant main effects; $F(2,48) = 7.71$, $p = .001$ for the Expectancy factor and $F(2,96) = 3.42$, $p = .04$ for the repeated measures factor. A highly significant interaction effect was also found, $F(4,96) = 3.61$, $p = .009$, which is illustrated in Figure 2. Means for each group are presented in Table 5.

Simple main effects testing showed significant Expectancy group effects at both the post- and follow-up assessments, $F(2,93) = 7.59$, $p < .01$ and $F(2,93) = 11.00$, $p < .001$ respectively. In addition, a significant repeated measures effect was found within the Psychological group, $F(2,96) = 10.39$, $p < .001$. Newman-Keuls comparisons revealed that the Psychological group was significantly lower than the other two at both the post- and follow-up assessments ($p < .01$ in all cases). As well, within the Psychological group, both the post- and follow-up measures were significantly lower than the pre-measure, ($p < .01$ in both cases), although they do not differ from each other.

Mean psychological attribution ratings are presented in Appendix L and the analysis of variance is presented in Appendix M. There were no significant differences found between groups or repeated measures, nor in the interaction.

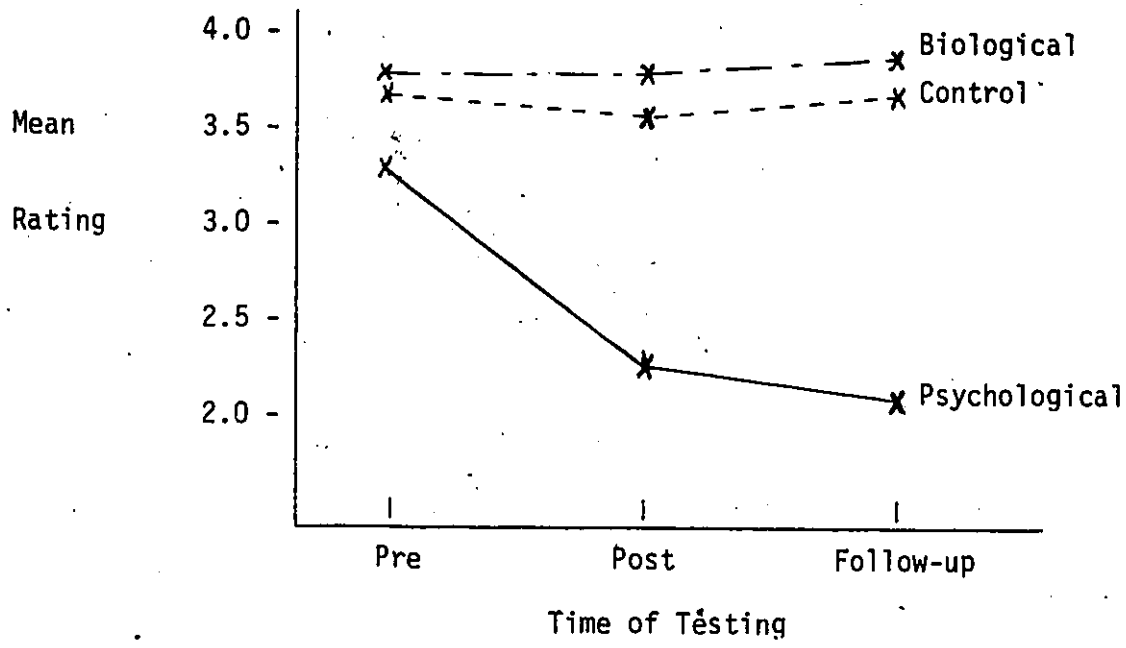

Figure 2. Mean biological attribution rating for each Expectancy group in the pre-, post-, and follow-up assessment.

Table 5

Mean Ratings on Biological Attribution in Each Condition

	Psychological Group	Control Group	Biological Group
Pre-measure	3.29	3.65	3.76
Post-measure	2.24	3.53	3.76
Follow-up	2.06	3.65	3.88

In summary, the Psychological group expected and reported actually experiencing significantly fewer symptoms relative to the other groups and to its initially reported amount, whereas the Biological and Control groups showed no change. As well, the Psychological group attributed their symptoms significantly less to biology after being exposed to the manipulation, and this change remained even after the test month. The Biological and Control groups showed no attributional shifts. No significant changes in psychological attribution were found in any of the groups.

Pearson product-moment correlation coefficients between the MDQ pre-measure and six of the background variables (Appendix N) revealed no significant correlations between initial level of symptomatology reported and age, education, family income level, child's age, child's sex, or menstrual cycle length.

The thirteen scales of the MMPI were correlated with the pre-, post-, and follow-up MDQ scores, and the results are presented in Table 6. There were statistically significant but weak correlations between the MDQ pre-measure and seven of the MMPI scales; Lie (-.28), F(.34), K(-.33), Pd(.37), Mf(-.29), Sc(.39), and Ma(.33). Examination of the post- and follow-up correlations reveals an overall tendency for the correlations to weaken, so that only four MMPI scales (L, K, Sc, and Ma) remain weakly but significantly correlated with the follow-up MDQ (amount of symptoms actually experienced).

AFFECTIVE, COGNITIVE, AND BEHAVIOURAL MEASURES

Inspection of the cell means of the six POMS mood variables (see Appendices O and P for wife- and husband-rated means) revealed that all the moods tended to

Table 6

Pearson Product-Moment Correlation Coefficients
Between the MMPI Scales and the MDQ Scores
at the Pre-, Post-, and Follow-up Assessment

	Initial MDQ (pre-measure)	Expected MDQ (post-measure)	Actual MDQ (follow-up)
Lie	-.28*	-.27*	-.27*
F	.34**	.27*	.20
K	-.33**	-.31*	-.27*
Hs	-.07	-.10	-.03
D	.20	.18	.09
Hy	.02	-.02	-.14
Pd	.37**	.27*	.16
Mf	-.29*	-.24*	-.22
Pa	.18	.16	.22
Pt	.21	.16	.06
Sc	.39**	.32*	.24*
Ma	.33**	.22	.29*
Si	.01	-.02	-.13

*$p < .05$
**$p < .01$

follow a similar pattern across cells, with Vigour following an inverse pattern. Therefore, in accordance with McNair's et al. suggestion (1971), a Total Mood Disturbance Score (TMDS) was calculated for each subject by summing the six POMS scores, weighting Vigour negatively. Cell means for the wife-rated TMDS are given in Table 7 and for the husband-rated TMDS in Table 8.

Correlation matrices were calculated for the six POMS variables and the TMDS, and the results are presented in Tables 9 (wife-rated) and 10 (husband-rated). For the wife-rated POMS, the correlation of TMDS with vigour was -.61, and with the other five moods was over .80. Correlations in the husband-rated POMS were even higher. Therefore, in further analysis of the data, the TMDS was adopted as a measure of overall negative mood. The Anger-hostility POMS mood was also retained in further analysis because of its possible relationship to maternal aggression.

There were then eleven dependent measures to be analyzed, wife- and husband-rated TMDS, wife- and husband-rated anger, wife- and husband-rated PTQ, anagrams score, puzzles score, Mother's Interaction %, Mother's Negative %, and Child's Compliance and Interaction %. The latter three were derived from the Mash Response-Class Matrix. The two other Mash measures - Child's Negative and Competing Behaviours % and Mother's Positive Response to Child's Negative and Competing Behaviours - could not be analyzed by parametric methods because of the large number of zero scores and missing values in the data. Only about half the children exhibited any scoreable negative and competing behaviour. These measures were analyzed by chi-square.

Table 7

Mean Total Mood Disturbance (TMDS)

as Rated by the Wife

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	155.59	149.18	157.76
Phase 2 (premenstrual)	172.53	212.12	208.29

Table 8

Mean Total Mood Disturbance Score (TMDS)

as Rated by the Husband

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	147.00	164.53	163.18
Phase 2 (premenstrual)	145.29	194.71	178.88

Table 9

Pearson Product-Moment Intercorrelations Among the Six Wife-Rated POMS Variables and TMDS

	Tension	Depression	Anger	Vigour	Fatigue	Concentration
Tension	1.0					
Depression	.77**	1.0				
Anger	.70**	.78**	1.0			
Vigour	-.27*	-.25*	-.27*	1.0		
Fatigue	.53**	.54**	.53**	-.58**	1.0	
Concentration	.72**	.71**	.59**	-.36**	.60**	1.0
TMDS	.81**	.83**	.81**	-.61**	.83**	.83**

*p < .05
**p < .01

Table 10

Pearson Product-Moment Intercorrelations Among the Six Husband-Rated POMS Variables and TMDS

	Tension	Depression	Anger	Vigour	Fatigue	Concentration
Tension	1.0					
Depression	.79**	1.0				
Anger	.78**	.87**	1.0			
Vigour	-.56**	-.54**	-.53**	1.0		
Fatigue	.66**	.63**	.58**	-.67**	1.0	
Concentration	.78**	.74**	.71**	-.47**	.67**	1.0
TMDS	.89**	.89**	.87**	-.75**	.84**	.85**

*p < .05
**p < .01

As a preliminary step, one-way analyses of variance were done on the midcycle scores (considered as baseline values) of all eleven variables, and no significant differences were found between the groups on any of the measures (see Appendix Q).

Pearson correlation coefficients were calculated between the midcycle scores of all eleven variables, and the resulting matrix is presented in Table 11. The variables were then grouped into two clusters for multivariate analysis on the basis of a minimum correlation of .30 with at least one other variable in the cluster. The first cluster consisted of the wife- and husband-rated scores of TMDS, PTQ, and anger. The second cluster combined Mother's Interaction % and Child's Compliance and Interaction %. The remaining three variables - anagrams, puzzles, and Mother's Negative % - did not correlate with any others and were each analyzed in separate univariate analyses of variance.

The multivariate analysis of variance of the first six variables is summarized in Appendix R. There was a highly significant Phase effect as well as a significant Group by Phase interaction. The Phase and interaction effects for each variable were therefore considered in separate univariate analyses.

TMDS and Anger. Appendices S and T summarize the analyses of variance of the wife- and husband-rated TMDS respectively. The Phase effect was seen to be highly significant in the wife-rated scores, $F(1,48) = 30.2$, $p = .000$, and showed a similar, marginally significant trend in the husband-rated scores, $F(1,48) = 3,64$, $p = .06$. The interaction was not significant in the husband-rated scores but there was a trend towards a significant wife-rated interaction effect,

Table 11

Pearson Product-Moment Correlation Coefficients Among Eleven Dependent Variables at the Midcycle Phase

	Wife TMDS	Husb. TMDS	Wife anger	Husb. anger	Wife PTQ	Husb. PTQ
Wife-TMDS	1.0					
Husb-TMDS	.49**	1.0				
Wife-anger	.75**	.30*	1.0			
Husb-anger	.31*	.84**	.29*	1.0		
Wife-PTQ	.39**	.21	.41**	.11	1.0	
Husb-PTQ	.42**	.32*	.38**	.21	.45**	1.0
Anagrams	-.14	-.07	-.03	-.08	-.05	-.22
Puzzles	.09	-.22	.02	-.18	-.12	-.09
Mother's Neg. %	-.10	-.18	-.05	-.12	.01	.04
Mother's Int. %	-.10	.13	-.02	.24*	-.04	-.06
Ch. Comp. + Int. %	-.15	.12	-.03	.12	-.09	-.29*

	Anagr.	Puzzles	Mother's Neg. %	Mother's Int. %	Child's Comp. + Int. %
Anagrams	1.0				
Puzzles	-.18	1.0			
Mother Neg. %	.04	-.07	1.0		
Mother's Int. %	.19	-.28*	.05	1.0	
Ch. Comp. + Int. %	.15	-.06	.22	.39**	1.0

*p < .05

**p < .01

$F(2,48) = 3.02$, $p = .06$. Because comparisons between expectancy groups had been planned and "a priori" predictions had been made, Newman-Keuls comparisons were carried out despite the marginal significance of the interaction effect. It was found that the premenstrual TMDS was significantly higher than the midcycle TMDS in the Biological and Control groups ($p < .01$ in both cases), but not in the Psychological group. As well, the Psychological group had a significantly lower premenstrual TMDS than the other two groups ($p < .05$ in both cases), who did not differ from each other. Figure 3 illustrates these relationships graphically.

Tables 12 and 13 present the cell means of the Anger-Hostility variable as rated by the wife and the husband respectively. The analyses of variance on the two measures yielded no significant interaction effects but a significant Phase effect for both variables (see Appendices U and V). For the wife-rated anger, $F(1,48) = 23,3$, $p = .000$, and for the husband's, $F(1,48) = 5.99$, $p = .02$, Inspection of the means reveals that anger is higher premenstrually than midcycle according to both the wife and the husband.

In summary, the wife-rated TMDS showed a significant midcycle to premenstrual increase in the Biological and Control Groups but not in the Psychological group. None of the husband-rated TMDS differences reached significance. Both husband- and wife-rated anger scores were significantly higher premenstrually than midcycle, but the differences between Expectancy groups did not reach significance.

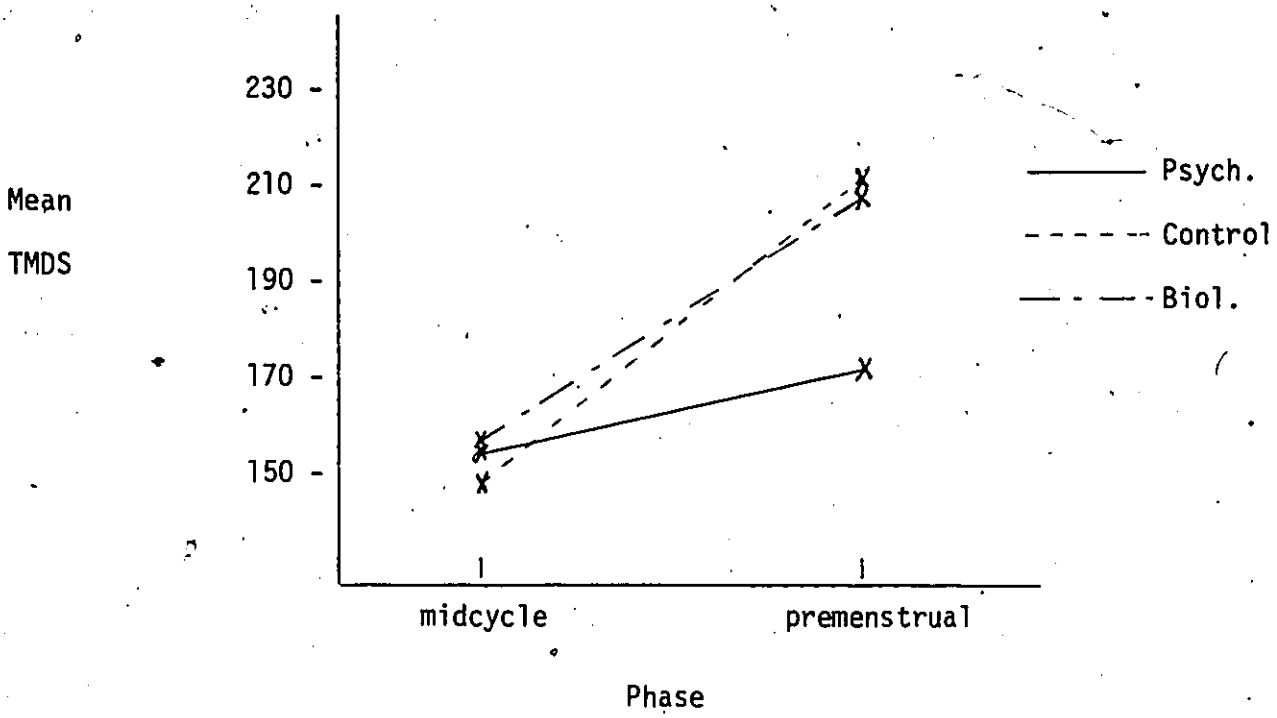

Figure 3. Mean wife-rated TMDS for each Expectancy group at each phase.

Table 12

Mean Wife-Rated POMS Anger-Hostility Score in Each Condition

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	43.24	44.00	42.82
Phase 2 (premenstrual)	48.47	56.59	53.29

Table 13

Mean Husband-Rated POMS Anger-Hostility Score in Each Condition

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	41.12	47.35	42.71
Phase 2 (premenstrual)	42.94	50.76	48.18

Conners' PTQ. The mean scores of the PTQ as rated by the wife and the husband are presented in Appendices W and X, and the respective analyses of variances are summarized in Appendices Y and Z. No significant effects were found for either variable.

Anagrams and Puzzles. The mean number of anagrams solved in each condition is presented in Appendix AA and the analysis of variance in Appendix BB. No significant effects were found for the anagrams measure, but the analysis of variance of the number of puzzles attempted in each condition yielded a significant Phase effect, $F(1,48) = 4.25$, $p = .04$ (see Appendix CC). Inspection of the means in Table 14 reveals that subjects in all Expectancy groups attempted more puzzle cards in the premenstrual phase than they did at midcycle.

Behavioural Measures. Mother's Interaction % and Child's Compliance and Interaction % were submitted to a 3 x 2 multivariate analysis of variance which yielded a significant menstrual Phase effect (see Appendix DD). Subsequent univariate analyses revealed no significant effects for the Child's Compliance and Interaction % (see Appendix EE for mean scores and Appendix FF for the analysis of variance), but a significant Phase effect for Mother's Interaction % (Appendix GG); $F(1,48) = 4.42$, $p = .04$. Examination of the means in Table 15 reveals that all groups had a higher Mother's Interaction % premenstrually than midcycle.

Inspection of the raw data for the Mother's Negative % measure showed marked heterogeneity of within-cells variances as well as positive skewedness. The scores were therefore subjected to a variety of transformations as recommended

Table 14

Mean Number of Puzzle Cards Attempted in Each Condition

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	7.65	6.94	6.88
Phase 2 (premenstrual)	7.94	8.71	7.88

Table 15

Mean Percentage of Mother's Total Scored Behaviour

Which was Either Praise, Question, or Interaction

	Psychological Group	Control Group	Biological Group
Phase 1 (midcycle)	9.68	16.32	12.19
Phase 2 (premenstrual)	13.46	16.51	17.91

by Winer (1971) and Kirk (1968), and the logarithm transformation was found to produce the most homogeneous variances. A separate univariate analysis of variance was performed on the log-transformed scores, and it yielded no significant effects. The cell means of the Mother's Negative % are presented in Appendix HH and the analysis of variance summary in Appendix II.

The total frequency of Child's Negative and Competing Behaviours, summed across all subjects in each cell, is presented in Appendix JJ. Because the total number of observations in any one observational period was not always 65 but sometimes two or three more or less (because of failed equipment, subject leaving the room early, or equipment not turned off, etc.) each subject's Negative and Competing Behaviours frequency count was divided by her total number of observations and then multiplied by 65 to equalize the differences. Total frequency counts across subjects were then calculated on the basis of these adjusted frequencies. A chi-square test of the differences between the three groups and two phases failed to reach significance, χ^2 (2) = 1.69, $p < .25$.

Appendix KK presents the frequency of Mother's Positive vs. Negative Response to her Child's Negative and Competing Behaviours in each of the three Expectancy Groups. Frequencies were again adjusted for inequalities in matrix totals. Yates' correction for small expected frequencies (Ferguson, 1966) was made in the Negative and Positive Expectancy groups, and chi-square values calculated on the 2 x 2 contingency table (type of response x phase) for each

Expectancy group. None of the chi-squares reached significance. For the Psychological group, χ^2 (1) = 3.26, p = .07. For the Control group, χ^2 (1) = 1.26, p = .26, and for the Biological group, χ^2 (1) = .28, p = .60.

In summary, there were no significant differences between groups or phases in either the mother's or the child's negative behaviour, nor in the mother's response to the latter. The only behavioural change that emerged was that mothers in all Expectancy groups interacted more with their child in the premenstrual phase than they did at midcycle.

CORRELATIONS BETWEEN DEPENDENT MEASURES

Pearson correlation coefficients between each of the eleven dependent measures at the premenstrual phase are presented in Table 16. Comparison with the midcycle values in Table 11 reveals much the same pattern of intercorrelations. There is no correlation between the premenstrual behavioural measures and mood scores of either the mother or the child, as rated by either the subject or her husband. That is, the subject's TMDS and anger scores show no relationship to her Negative % or Interaction %, and the child's mood scores (PTQs) bear no relationship to his/her Compliance and Interaction %. The scores on the cognitive tasks show no significant correlation with any of the subject's mood scores, except wife-rated anger with puzzles (r = -.29, p = .02).

In general, the TMDS and anger intercorrelations are stronger at the premenstrual phase than at midcycle, probably because the range of scores is larger. The Mother's Interaction % - Child's Compliance and Interaction % correlation is also stronger. But there are two notable differences between the midcycle and premenstrual correlation matrices.

Table 16

Pearson Product-Moment Correlation Coefficients Among Eleven Dependent Variables at the Premenstrual Phase

	Wife TMDS	Husb. TMDS	Wife anger	Husb. anger	Wife PTQ	Husb. PTQ
Wife-TMDS	1.0					
Husb-TMDS	.57**	1.0				
Wife-anger	.87**	.42**	1.0			
Husb-anger	.49**	.90**	.39**	1.0		
Wife-PTQ	.17	.12	.11	.17	1.0	
Husb-PTQ	.24*	.20	.23	.16	.40**	1.0
Anagrams	-.03	.04	.07	.03	.16	-.07
Puzzles	-.22	-.06	-.29*	-.05	-.18	-.11
Mother Neg. %	.01	.11	.02	.14	.11	.00
Mother Int. %	.06	.20	.11	.17	.00	-.19
Ch. Comp.+Int. %	-.16	-.01	-.07	-.04	-.02	-.20

	Anagr.	Puzzles	Mother's Neg. %	Mother's Int. %	Child's Comp.+Int. %
Anagrams	1.0				
Puzzles	-.14*	1.0			
Mother Neg. %	.32*	-.12	1.0		
Mother Int. %	.39**	-.14	-.06	1.0	
Ch. Comp.+Int. %	.32*	.08	.09	.51**	1.0

*p < .05
**p < .01

Firstly, anagram solving and the three behavioural measures are positively correlated in the premenstrual phase (r = .32 to .39); more anagrams were solved in situations in which there was simultaneously more interaction between mother and child and more negative behaviour on the mother's part. This relationship is non-existent in the midcycle testing.

Secondly, the correlations between PTQ scores and TMDS and anger scores are consistently lower in the premenstrual phase, suggesting less relationship between the mother's and the child's mood premenstrually than midcycle. However, if this relationship is examined separately for each Expectancy group, marked differences appear. Table 17 presents the correlation matrix between PTQ scores and TDMS and anger scores for each Expectancy group. In the Psychological Group, there are significant positive correlations between the subject's mood scores and PTQ ratings (r = .48 to .83) at midcycle, but significant negative correlations in the premenstrual phase (r = -.47 and -.57). The Control Group, on the other hand, shows little consistent correlation at midcycle, but some moderate positive correlations in the premenstrum (r = .49 and .62). The Biological group demonstrate no consistent trends. These findings will be discussed further in the next chapter.

Table 18 presents the correlations of the eleven measures of premenstrual mood, performance, and behaviour with the three MDQ symptom scores (usual, expected, and actual), as well as with the number of days premenstrual at which the assessment was made. As expected, the three MDQ scores correlated significantly with measures of the subject's negative mood, the highest correlation

Table 17

Pearson Product-Moment Correlation Coefficients Between Mood Scores and PTQ Scores for Each Expectancy Group

	Midcycle		Premenstrual	
	Wife-PTQ	Husb-PTQ	Wife-PTQ	Husb-PTQ
Psychological Group				
Wife-TMDS	.70**	.48*	-.57*	.10
Husb-TMDS	.51*	.33	.02	.17
Wife-anger	.83**	.53*	-.47*	.05
Husb-anger	.62**	.29	-.01	.17
Control Group				
Wife-TMDS	.23	.51*	.62**	.18
Husb-TMDS	.06	.08	-.05	.07
Wife-anger	.16	.34	.49*	.27
Husb-anger	.00	-.11	-.02	.11
Biological Group				
Wife-TMDS	-.18	.23	-.12	.35
Husb-TMDS	.30	.58*	.13	.15
Wife-anger	-.12	.20	-.25	.24
Husb-anger	.06	.80**	.33	.03

*$p < .05$

**$p < .01$

Table 18

Pearson Product-Moment Correlation Coefficients Between the Usual, Expected, and Actual Amount of Symptoms and Eleven Measures of Premenstrual Mood, Performance and Behaviour

	MDQ1 (usual)	MDQ2 (expect)	MDQ3 (actual)	Days premenstrual at testing
Wife-TMDS	.58**	.61**	.79**	.08
Husb-TMDS	.32*	.39**	.48**	-.08
Wife-anger	.46**	.47**	.70**	.02
Husb-anger	.26*	.32*	.38**	-.05
Wife-PTQ	.21	.18	.20	-.10
Husb-PTQ	.25*	.24*	.30*	-.12
Anagrams	-.14	-.11	-.07	-.03
Puzzles	-.09	-.18	-.12	-.04
Mother's Neg. %	.01	-.04	-.06	-.17
Mother's Int. %	.03	.06	.09	-.08
Ch. Comp.+ Int. %	-.13	-.19	-.14	-.17

*p < .05
**p < .01

being with the follow-up MDQ, which represents the actual amount of negative symptoms experienced duringthe test month. Mild but significant correlations between MDQ and husband-rated PTQ scores are evident as well, but there are no significant correlations between MDQ scores and any of the behavioural or cognitive measures. None of the eleven dependent variables showed any relationship with the number of days before menstruation at which the variable was measured.

CHAPTER IV

DISCUSSION

This study was designed to examine two major issues, the first being the role of expectations in the experience of negative premenstrual symptoms and the second being the possibility of premenstrual changes in maternal behaviour. Before the experimental hypotheses relating to either of these issues can be evaluated, however, two methodological issues which may have influenced the results must be considered - the efficacy of the expectancy manipulation and the role of experimental artifact.

THE EFFICACY OF THE EXPERIMENTAL MANIPULATION

From the results of both the pilot study and the main experiment, it is clear that the expectancy manipulations used in the study were not equally effective in altering expectations. Subjects exposed to the psychological information lowered their subsequent expectation of symptoms significantly relative to their initial level and to that of the control group. However, subjects given biological information did not raise their expectations at all. The level of symptoms reported initially, after the manipulation, and at follow-up remained essentially constant for both the Biological group and the Control group. Therefore, it is possible to evaluate whether lowering expectations has any effect on subsequent symptoms but not whether raising expectations has any effect.

This is not to say that the biological manipulation had no effect; it provided subjects with an explanation for the bodily and emotional changes they experienced and provided the kind of 'normative validation' which Rodin (1976) considered so important if attributional processes were to be effective in reducing anxiety. In fact, the kind of information provided in the biological manipulation was very similar to that provided in many attribution studies; that is, subjects were usually told what physiological and psychological changes to expect from a given drug. The difference is that the women in this study already "knew" what symptoms to expect and what caused them, as evidenced by their high biological attribution ratings at the beginning of the study. The subjects in the present study were older, more sophisticated, and on the whole, well-read. The biological information may merely have confirmed and validated their own beliefs and given them a more detailed, cohesive understanding. But this, Rodin suggests, is important in itself and may be a factor not in changing actual symptom severity but in weathering it with more equanimity.

THE ROLE OF EXPERIMENTAL ARTIFACT

There were two major findings in the study which involved differences between the two experimental groups. Firstly, the Control group and the Biological group reported essentially the same level of symptomatology before and after the manipulation, whereas the Psychological group expected and reported actually experiencing fewer symptoms following the manipulation. Secondly, the Biological and Control groups reported significantly greater negative affect premenstrually compared to midcycle, while the Psychological group

showed no increase. Before it can be argued that there was a genuine lowering of expectations in the Psychological group which produced a genuine lowering of symptomatology, two other possible explanations must be eliminated: 1) Subjects wished to be "good subjects" (Orne, 1962; Parlee, 1980) and confirm what they perceived to be the experimenter's expectancies, and related to this, 2) Subjects may have been reluctant to admit to symptoms once they had been told it was all in their heads, for fear of appearing stupid or neurotic. As noted in Chapter 1, one of the major methodological difficulties in menstrual cycle research is that of maintaining subject naivety. Once subjects are aware of the experimenter's focus on the menstrual cycle, then subject expectancies and subjects' expectancies about experimenter expectancies automatically confound the results. Since in the present study the experimenter was trying to modify those very beliefs and expectancies, it was by definition impossible to hide her interest in the menstrual cycle.

Over the years, researchers into the social psychology of the experiment (Orne, 1962; Rosenthal, 1966; Rosenthal, & Rosnow, 1975) have generated considerable evidence that the outcome of a particular psychological experiment can be markedly affected by factors apart from the experimental variables under study. Specifically, characteristics of the experimenter, the subject sample and the experimental situation can increase or decrease the likelihood of confirming the experimental hypothesis.

Orne (1962) and others have pointed out that human subjects are not passive reactors in the experiment, but rather have their own motives and goals in

participating, one of which is to figure out the experimenter's hypothesis (i.e., what the study is "really about"). Reasons for this include simple curiosity, the desire to advance science, and the wish to be a "good subject" by confirming the experimenter's hypothesis, so that the subject's participation will seem worthwhile and/or the experimenter will think well of him.

Rosenthal and Rosnow (1975) have developed a model which conceptualizes the combined influence of experimenter, subject, and situational effects on the outcome of the experiment. They postulate that, with regard to the fulfillment of experimenter expectancy, there are three possible outcomes - compliance, noncompliance, and countercompliance. Which outcome the subject chooses depends on: 1) the subject's receptivity (awareness) to the cues given by the experimenter and the situation as to what the experimenter's expectancy is. This receptivity depends on how obvious the cues are; if they are conflicting or nonexistent, subjects will not be able to figure out the hypothesis and will be unable to confirm or disconfirm it. 2) The subject's motivation; having figured out the hypothesis, the subject will be predisposed to confirm it, ignore it, or disconfirm it, in essence to conform to the experimenter's hopes, to ignore them, or to rebel against them. It is at this stage that the characteristics of the experimenter and of the subject affect outcome, by influencing motivation. 3) Capability; assuming awareness and motivation, the subject must be physically and/or psychologically capable of the response he desires. As Rosenthal and Rosnow point out, this factor is rarely a problem in psychological research, as experiments are usually designed to be within the subjects' capabilities.

Rosenthal and Rosnow suggest that receptivity and motivation combine to determine the extent of experimental artifact in the following manner. Receptivity increases as the experimenter expectancies become clearer, but motivation to comply decreases. When there are almost no clues as to experimenter expectancy, motivation may be high but awareness or receptivity negligible, so compliance (or countercompliance) will not occur. When there are subtle but clear-cut hints, receptivity will be high and motivation still high, so that compliance will be at its maximum. When the clues are blatant, however, awareness will be at its maximum but motivation to comply will be seriously decreased; in this case either noncompliance or countercompliance will occur. Thus, the authors postulate an inverted U-shaped relationship between the clarity of experimenter expectancy and the probability of compliance. Although they cite some studies confirming this U-shaped relationship and particularly the drop in motivation to comply as blatancy increases, the reasons for this effect are speculative. Some which have been proposed include "leaning over backwards to be honest" (Orne, 1962), rebellion against perceived coercion or manipulation, and a desire not to appear too gullible.

An application of the foregoing model to the present study should prove helpful in trying to determine the extent to which the mood and symptom effect is due to experimental artifact rather than to bona fide manipulation of expectancies. Receptivity and motivation will each be examined.

On the face of it, receptivity ought to have been very high, because experimenter expectancy (as perceived by the subject, that is) was very clear, perhaps

even blatant. Subjects given the psychological information ought to have inferred that they were expected to report fewer symptoms, while subjects told that premenstrual symptoms were a universal, unavoidable result of chemical changes ought to have deduced that they were expected to report as many if not more symptoms than before. For the Psychological group at least, the blatancy of the attempt at attitude-change ought to have reduced their motivation to comply.

However, anecdotal feedback from subjects upon debriefing suggested that many, if not most, felt that there was more to the study than met the eye. When told of the deception, they responded with comments like "Oh, it's alright, I had a feeling there might be more going on", or "I knew when I volunteered for a psychology experiment that there would be something like that". This suggests that some of the subjects, suspicious of the obvious clues, may have searched beyond for some ulterior design on the experimenter's part. There is no way of knowing what alternative hypotheses they came up with, but, for these subjects at least, experimenter expectancies were neither clear-cut nor consistent, thus again making compliance less likely. In this regard, it is worth noting that the subjects did not seem to doubt the authenticity of the films themselves, nor the experimenter's belief in them (at least, none dared express the doubt openly); therefore their awareness of the actual ulterior design of the experimenter was probably minimal.

The question of motivation to comply is far more complex, for here experimenter characteristics interact with subject characteristics and with situational

variables within the experiment. Research into experimenter effects (Rosenthal, 1966) suggests that if the experimenter is of high status, pleasant but business-like, known to the subject, and commands an air of authority, compliance is maximized. Conversely, an experimenter who is of lower status, likeable, friendly, personal and relaxed, or actually unpleasant, obtains more noncompliance, or even countercompliance. Most work in this field has been done with undergraduates, hence extrapolation to the present sample is tentative. In the present study, subjects viewed themselves as equal in many respects to the experimenter, a woman of similar age with preschool children of her own. The relationship between experimenter and subject tended to be friendly, relaxed and personal because of the prolonged contact and intimate material of the study. These qualities might be seen to facilitate noncompliance or countercompliance. Noncompliance in this case would probably reflect a greater feeling of nonjudgmental acceptance, with corresponding freedom to respond as they chose to, without pressure to please or to conform. Countercompliance would probably reflect a "leaning over backwards to be honest" rather than a rebellious rejection, although the wish not to appear too gullible might also play a part.

On the other hand, however, it could be argued that since the subjects liked the experimenter and identified with her as a fellow woman and mother, they would be anxious to help her succeed by being "good subjects". If this motive were operating, compliance could be very strong.

Rosenthal and Rosnow (1975) reviewed the evidence from hundreds of studies and concluded that volunteer subjects differed from those who did not volunteer

in several systematic ways which could bias the results of a study, mainly by operating on the motivation variable. As a rule, relative to nonvolunteers, volunteers were more highly educated, more intelligent, more sociable, more approval-motivated, of higher social class, less authoritarian, less conforming and less conventional. They were also more interested in the topic and felt it to be more important that did those who did not volunteer. Finally, they considered that the experimenter would evaluate them favourably; those who feared that the experiment might reveal some weakness or deficit were less likely to volunteer. Although based largely on college students, this composite picture seems to fit the prototypical volunteer subject in the present study. The recruitment process, during which over a thousand pamphlets were distributed and about 5% returned, favoured selection of the unusual woman, not the usual one. She would probably have been more adventuresome, self-assured, non-conforming and independent. She tended to have a strong interest in the topic and saw it as important. Motives which subjects gave for volunteering included simple curiosity, the hope that they would derive some benifit, either short-run or long-run, for their own symptoms, the wish to advance knowledge in an area of women's suffering which they felt was not taken seriously enough, and the desire to help a fellow woman accomplish a difficult goal.

It has generally been found that volunteer subjects show greater compliance with experimenter expectancies than do nonvolunteers (Rosenthal & Rosnow, 1975). This is attributed to their greater commitment to the topic and their eagerness to help science, to please the experimenter, and to be favourably evaluated as good subjects. In the present study, most subjects were by their own admission

eager to advance science and to help the experimenter, and it is likely that they were personally involved in the study. They were not receiving any payment, yet were willing to invest considerable time and effort for the sake of science or the experimenter or both, and they would probably have wanted to make their contribution to science worthwhile. Given this constellation of factors, motivation to comply with experimenter expectancies might have been at its maximum.

But research into experimental artifact has dealt mainly with more innocuous topics such as verbal conditioning, simulated attitude-change, and a host of artificial social psychology situations where the subject has little prior opinion or vested interest in the theories being tested. In the present study, premenstrual tension was a topic of serious and long-standing personal interest. It affected, often disrupted, the subject's everyday life. It seems logical to assume that her wish to advance knowledge in the area and to derive benefit for her own symptoms would have been a stronger motive governing her participation that the wish to help the experimenter or even to look good in her eyes.

In a study designed to place two motives in conflict, Rosnow et al. (1973, reported in Rosenthal & Rosnow, 1975) placed subjects in a position where they believed compliance with experimenter expectations would lead to their being judged stupid. The researchers found that subjects abandoned compliance in favour of promoting a favourable self-image. It seems that compliance may occur only when there is no more powerful motive against complying.

In the present study, such a motive might well have existed. Some of the subjects had been suffering from premenstrual tension for a long time, strongly

believed in a hormonal cause, and hoped that someday a drug would be found to help them. To be told that premenstrual tension was nothing but an elaborate psychological illusion ("all in their heads"?) was probably not pleasant. The implication was that there is no miracle drug, that what the mind creates, the mind must overcome. Subjects often reacted to this with dismay, at times anger and often even guilt. In the discussion following the film, some subjects said that now they would have to hold themselves responsible for their behaviour if they could no longer blame their hormones. It would seem to be more in the subjects' interests to disregard and in fact to disprove the experimenter's absurd theory in the hope of putting science back on the right track again.

In summary, the Rosenthal-Rosnow model presented above suggests that some of the characteristics of the experiment, namely the subjects' personal involvement with the topic and identification with the experimenter would favour compliance with experimenter expectancies (as perceived by the subject), while other characteristics such as the nonauthoritarian, friendly relationship with the experimenter, the sophisticated, relatively self-assured type of subject, the blatant (or suspect) experimenter expectancy, and the possible conflict of motives would favour noncompliance or countercompliance. (Countercompliance is less problematic than compliance in this case; if it were operating, then the true treatment effects would be even larger than those observed.) Thus from the model alone it is not possible to determine how much, if any, of the observed group differences were due to compliance with experimenter expectancies, although the evidence seems to be weighted against its being a significant factor.

Apart from the blend of common sense, artifact theory, and anecdotal feedback presented above, inspection of the data themselves reveals three features which collectively argue against the "good subject" explanation.

First, although the effect is not significant, the mean husband-rated negative mood scores (TDMS) and anger scores tended to follow a similar pattern to the wife-rated scores; that is, the husbands in the Psychological group tended to see their wives as less bad-tempered and miserable in the premenstrual phase than did the husbands in the other groups. It appears that not only did the subjects in the Psychological group report themselves to be better-tempered, but they may also have appeared better-tempered to an outside observer. The strength of this argument rests partly on the assumption that the subjects did not tell their husbands about the film or about their menstrual status, as they were instructed, but the validity of this assumption is unknown.

Secondly, in the Psychological group, the amount of symptoms reported at follow-up (symptoms actually experienced during the test month) was even lower than the amount of symptoms expected after the manipulation. Although the differences in the means did not reach significance($p < .10$), examination of the raw scores reveals that 12 of the 17 subjects reported even fewer symptoms than they had expected, despite the intervention of at least one month's time and experience. If the subjects had considered their post-manipulation scores to be a respectable reduction in symptoms needed to fulfill experimenter expectancy or to avoid looking stupid, why reduce them still further a month later, when the strength of the persuasive attempt had faded and they had had

disconfirming evidence from their own bodies to bolster their confidence? It seems much more likely that the follow-up symptom score was lower because subjects had confirmed by their own experience the validity of some of the psychological information they had learned.

This possibility is given further credence by the biological attribution ratings of the Psychological group. These were significantly lower following the manipulation; that is, subjects lowered their ratings of 'hormones' and 'water retention' as main causes of their premenstrual symptoms as a result of the manipulation. However, these ratings were even lower at follow-up, again not significantly, but suggestively.

At this point it is worthwhile to digress briefly to note the weakness of both the biological and the psychological attribution measures as designed in this study. The wording of the question refers to premenstrual symptoms in general, not to the specific disturbances in concentration and affect being manipulated in this study. This was done to avoid drawing too much attention to any particular symptom or to the experimenter's interest in attribution. Some of the subjects complained of appreciable swelling or pain as well as negative affect. If these subjects were in the Psychological group, their negative affect and concentration scores dropped significantly, often close to zero, after the manipulation, whereas their swelling and pain scores remained elevated. When these women were asked to rate the main causes of their premenstrual symptoms, the only symptoms which remained salient and which would therefore be used as the basis for the ratings were swelling and pain, the physiological

basis of which had never been disputed. By the same token, they would not give significant psychological attribution ratings for their symptoms of negative affect and poor concentration because these had in many cases virtually disappeared.

Problems occurred within the Biological group as well. Information provided in the film specified hormones and not water retention as the underlying cause of premenstrual tension; therefore, some subjects who had initially rated both hormones and water retention as high now retained the high score for hormones but scored water retention at zero, thus lowering their overall biological attribution score (which is the sum of scores on both these variables). If the hormone score alone had been taken as a measure of biological attribution, the control group, many of whom believed water retention was a major cause, would have been affected. In view of the inadequacies of the attribution measures, it is surprising that the inter-group differences in biological attribution were as significant as they were, attesting to the strength of the manipulation and the underlying shift in beliefs.

The third feature of the data which argues against the "good subject" explanation is the contradiction between the cognitive and behavioural data on the one hand and the mood and symptom self-reports on the other. The Biological group, and possibly even the Control group, would probably have assumed that the experimenter expected them to do worse on the cognitive tasks in the premenstrum. Yet all groups persisted longer at the puzzles in the premenstrum, striving to do better.

Similarly, the subjects probably knew from their consent forms and the apparatus in the corner that their behaviour, and that of their child, was being videotaped, and they might fairly easily have guessed the hypothesis in question - namely that the experimenter expected them to have more trouble concentrating and dealing with their child in the premenstrum. Irritability with the children was a topic frequently raised in the discussion group and in general. The "good subject" would probably have supplied the experimenter with negative behaviour to suit the hypothesis; these did not. Their behaviour is more in keeping with the notion of "leaning over backwards to be honest" or with some of the hypotheses under investigation.

It should be emphasized that this last feature is based on statistically significant differences in the data, but the first two are not. Several hypotheses can be, and will be, advanced to explain these features, including random error. However, of importance to the present discussion is the fact that they all run counter to a compliance explanation of group differences. Although the foregoing analysis of the data and the demand characteristics of the experiment is highly speculative, it seems safe to conclude that experimental artifact was not the sole determinant of the effects found in this study. It seems likely that genuine changes in expectancy and symptoms occurred.

THE ROLE OF EXPECTANCY

Moods and Symptoms. The study was designed to determine whether expectancy operates to enhance negative affect and symptoms, as predicted by Asso, or to

mitigate them by reducing their emotional tone, as predicted by Rodin. Rodin's stress-reduction formulation predicted that if a perceived arousal state can be attributed to a predictable, expected, and 'normal' source, emotionality will be reduced, whereas if the perceived arousal state is contrary to expectancy or to standards of normalcy, emotionality will be augmented. Assuming an increase in basic physiological arousal in the premenstrum, the first state of affairs would apply to the Biological group and the second to the Psychological. Rodin would predict, therefore, an increase in negative mood and symptoms in the Psychological group and a decrease in the Biological group.

The results do not confirm these predictions; the Psychological group manifested a significant decrease in symptoms and negative mood. In contrast, the Biological group manifested no change either in relation to their initial symptom level or to the Control group. Furthermore, their level of premenstrual negative mood was equivalent to that of the Control group, who had no clear-cut attributional mechanism.

It can be argued, of course, that the Control group was so highly informed and familiar with the physiology of their bodies that they were equivalent to the Biological group, and their belief in a biological cause, as evidenced by high biological attribution ratings, supports this view. But Rodin (1976) found that the positive effects of attributing arousal to the menstrual cycle accrued only when subjects were given normative validation of their symptoms. Her control group of high-symptom women showed no reduction in arousal or improvement in performance. However, she was dealing with cognitive performance and not moods and symptoms, which are more readily connected to the menstrual

cycle, and it is debatable whether normative validation applies to the emotional impact of moods and symptoms. In any case, two things are suggested from the data; providing women with detailed information on the normal, predictable, and universal chemical changes underlying their perverse moods and symptoms does not seem to affect the severity of their experience of them. Additionally, removing the 'normal', predictable attributional source does not seem to enhance the emotional tone or severity of the symptoms.

Asso's formulation predicted that expectancy and arousal act synergistically so that the greater the expectancy, the greater the negative symptoms. This formulation appears to fit the data more closely, although as pointed out at the beginning of the chapter, failure to raise expectancy beyond initial levels in the Biological group meant that only half the proposition could be empirically tested. In actual fact, the study found that lowering expectancy lowers symptomatology. This finding holds whether retrospective checklists, daily mood self-reports, or husband-rated mood scores are used, although the latter measure was not significant. In fact, the correlation between self-rated and husband-rated mood scores was .57, between self-rated daily and retrospective checklists .79, and between husband-rated daily moods and self-rated retrospective checklists .48, indicating a fair degree of consistency between measures, particularly between those that are self-rated. This consistency is greater than that found in some studies but comparable to recent work by Maitland-Shilling (1981).

This positive relationship between symptoms and expectancy is given further support by the correlation of .74 between expected and actual symptoms (post-measure and follow-up MDQs) and the correlation of .61 between expected symptoms

and daily self-rated negative mood. Parenthetically, it is interesting to note that there were low positive correlations between midcycle negative mood scores and symptom scores (MDQ), which together with the low correlations found between MDQ and some of the MMPI scales (notably the validity scales, which reflect general ego strength and a tendency towards pessimistic complaining) suggest some relationship between premenstrual symptoms and generalized psychological distress.

To date, the concept of expectancy has not been examined in any detail in menstrual cycle research; it has been bandied about as if it explained something in and of itself, without much attempt to define what exactly it is or how it operates. In fact, expectancy as a hypothetical mediating variable has been used in psychology to explain everything from a bird's startle response to a rat's learning of a maze to a man's suicide after a business failure. It is doubtful, therefore, that it is even a unitary variable, let alone that it operates in the same way in all circumstances.

Both Rodin and Asso, working from within the interactive, arousal-plus-cognitive-interpretation theory of emotion, suggest that expectancy operates on the cognitive element, by giving meaning to the arousal. This meaning then defines the emotion (or nonemotion), and may even increase or decrease the actual arousal. But the final picture, when all the pieces are in place, is likely to be a good deal more complicated than Schacter and Singer (Schacter, 1964) or even Mandler (1975) imagined, as Mandler himself admits.

As researchers have frequently pointed out (e.g., McClintock, 1981; Parlee, 1981) there are major gaps in our understanding of how psychic and even social

events influence bodily processes and even of how emotion is linked to physiology. The more rigourous and reductionist areas of experimental psychology such as learning, motivation, and perception have chipped away at the question as it applies to their own domains, but in the fields of clinical and social psychology more research needs to be aimed at dismantling the notion of expectancy, which has been shown to be a powerful agent in successful response to medication, placebos, and psychotherapy (Franks, 1973; Jospe, 1978).

As a first step, the process of change has to be broken down into manageable components. Expectancy may produce its effect at any, or all, points along the chain from raising sensory thresholds to modifying central attention to biasing recall to stimulating the hypothalamus to alter hormone output. Research (mainly animal) in more circumscribed, traditional psychology has shown that expectancy can alter sensory thresholds and produce selective attention and recall (Hinde, 1966). Whether these findings generalize to the more complicated forms of expectancy has yet to be shown. Studies with placebos have demonstrated that a patient's faith and expectancy alone can produce sometimes dramatic physiological changes in the body, even to the extent of reversing the effects of a pharmacologically active drug (Franks, 1973; Shapiro, 1964).

With reference to the present study, expectancy can be viewed as influencing emotion (and psychological symptoms of mood and concentration) in one or both of the following ways: 1) by altering the sensitivity to and the perception, interpretation and/or meaning of a given physiological state (i.e., by operating at the information-processing level), with or without the subject's awareness,

and 2) by altering the physiological state itself. That psychological processes can alter physiological states has been demonstrated in both placebo and biofeedback research, as well as by certain highly trained specialists in meditation. Both biofeedback and meditation suggest a deliberate act of the mind, whereas the mechanism(s) by which placebos influence bodily processes is unknown, although it is believed to involve some ill-defined, nondeliberate process known as suggestion (Jospe, 1978; Frank, 1973; Shapiro, 1964).

Without autonomic measures, there is no way to determine whether the lowered expectancy in the present study reduced negative affect at the perceptual and cognitive (information-processing) level or at the physiological level, or at both. Nor can it be determined whether, within the information-processing chain, changes occurred primarily in sensory-perceptual or central-interpretive processes. It is possible that all these processes were involved and that there were individual differences in the predominance of one process over another. However, the nature of the psychological manipulation and the pattern of symptom reduction suggests that deliberate cognitive reappraisal may have played a large role. Thus, changes occurred predominantly at the information-processing level and resulted from deliberate reevaluation, not unconscious suggestion.

Most of the information provided in the psychological manipulation directed the subjects' attention to the ways in which cognitive, information-processing factors can augment symptoms; it was pointed out that negative menstrual myths facilitate blaming all bad moods on biology in the premenstrum, while at other times bad moods are attributed to a whole host of possible causes, like too

much caffeine, too little sleep, hunger, etc. It was pointed out that this salient, repeated causal connection biases recall in favour of bad moods, and in addition, that ambiguous physiological arousal may be labelled negatively in the premenstrum but labelled as a burst of energy at other times. All this information provided subjects with a new perspective and more concrete strategies for self-analysis. It is likely that the subjects considered the arguments plausible, based on the vagueness and capriciousness of their symptoms, and that they were subsequently put on guard against exaggerating symptoms through cognitive bias. The post-measure scores revealed that subjects expected a reduction in symptoms, not an elimination.

The next month proved a test of the validity of the arguments in the psychological manipulation, and the fact that at follow-up one month later twelve of the seventeen subjects reported even fewer symptoms than they had expected suggests that they may have found considerable validity in the arguments. This further reduction in symptomatology at follow-up is not statistically significant ($p < .10$ using the Newman-Keuls formula), but it seems plausible to suggest that subjects accepted some of the psychological manipulations on a conditional basis, then deliberately reappraised their symptoms in the light of these arguments before they were prepared to endorse them more completely. Researchers into attributional processes have observed that often a subject's original attribution concerning his physiological state in the experiment resembles a hypothesis, which he has to test further before believing it (Nisbett & Valins, 1972).

Compliance with therapist expectancy and deliberate cognitive reappraisal resulting from changed expectancies no doubt play a role in placebo reactions as

well (Jospe, 1978), but it is generally assumed that some kind of unknown, unconscious suggestion process is operating as well. Placebo reactions are very complex phenomena which depend a great deal on the immediate situational variables, the motivation of the subject, the personality and status of the therapist, and the meaning of the interchange (Jospe, 1978). They occur most often in patients who have feelings of social inadequacy and insecurity and who tend not to be analytical or discriminating, who are anxious, acquiescent, trusting and eager to be helped (Frank, 1973). Effects are stronger when the "healer" is of high status, trusted, and embued with power. Faith in the "healer" seems to be very important, and if the "healer" has faith in the medication, that faith is transmitted to the patient (Shapiro, 1964). In addition, the placebo, being a pill (or symbolic equivalent), is a symbol of healing in our society, and this symbolic meaning adds to its power.

It is unlikely that these factors were operating in the present study. No symbol of healing was ingested by the subjects, nor was there a high-status, trusted, powerful "healer" in whom they had faith and from whom they had hopes of a cure. Nor were the subjects as a whole socially inadequate, anxiety-ridden, and actively seeking help; indices of maladjustment and anxiety showed only minor elevations. As noted earlier, many of the subjects volunteered in the hope of deriving benefit for their own symptoms, but they signed up for a psychology experiment - they were not seeking relief from a medical clinic. As Jospe (1978) has pointed out, there is a world of difference between a sufferer seeking a cure and a person volunteering for a psychology experiment. And even

had some subjects been motivated to receive a cure, the cure they received was far from a placebo; it was being told there was no illness!

Suggestion requires faith and belief in the healer, as well as an acceptance of the truth of the healer's opinion. The women in the present study were by and large sophistocated, informed, and familiar with gynaecologists and with the uncertainties of medical and scientific knowledge. The doctor's credentials probably added weight to his opinion, but it seems unlikely that subjects would simply accept his claims unquestionningly. 'It is more likely that, despite their skepticism and wishes to the contrary, his arguments had a ring of truth within their own experience.

Before concluding the discussion on the effect of expectancy on mood and psychological symptoms, a note must be made of the pattern of positive mood found in the three groups. Asso's formulation is based on the notion of pre-menstrual arousal or arousability, which enhances both positive and negative mood, although negative mood is more often experienced and/or noticed because of prior expectations. It was predicted, therefore, that both positive and negative mood would rise in the premenstrum if Asso's model were correct, but that the positive mood change would be greater in the Psychological group, who would be freer of expectations to the contrary.' No such increase in positive mood was observed in the present study. The positive mood measure (the POMS vigour score) tended to vary inversely with the negative mood scores. There are two possible reasons which may explain this without implying an error in the theoretical model. Firstly, the Vigour scale consists of the following

adjectives: elated, lively, full of pep, energetic, cheerful, alert, carefree, and vigourous. Though undeniably positive, these refer primarily to a sense of energy rather than of enthusiasm, warmth, or sociability. It may be that the POMS taps one dimension of positive mood while leaving untouched other dimensions which may show a premenstrual increase. Secondly, a halo effect in the measure itself probably operated to dampen positive mood responses. Those eight adjectives, along with seven other positive ones not included in the scoring, are interspersed in a list of fifty negative adjectives, producing an overall set towards focussing on negative moods and minimizing positive ones. Most other mood checklists possess the same bias, and more research is needed to develop a multi-dimensional measure of positive mood.

In summary, the findings support Asso's view that expectancy interacts with arousal in a synergistic manner, so that when expectancy is lowered, symptoms and negative mood are lessened. It seems likely that most of the symptom reduction was due to deliberate cognitive reappraisal rather than to unconscious suggestion. Both the symptom and mood measures referred to subjectively perceived states and were based on self-reports. There were no significant differences found between expectancy groups on any of the behavioural or cognitive measures, although the husband's ratings of the wife's mood, which was presumably based on observable behaviour, showed a pattern consistent with the self-reports. Nor were there any significant differences linking expectancy groups to the perception of child's troublesomeness. There were, however, two interesting trends which merit footnotes in the discussion to come.

Cognitive Tasks. Rodin's model predicted that subjects in the Psychological group, being unable to reattribute task-arousal to the menstrual cycle, would persist least on the insoluble puzzles and solve the least anagrams, and that those in the Biological group, having a clear-cut reattribution mechanism would persist longest and solve the most anagrams. The Control group ought to fall somewhere in between. Asso's model predicted the reverse.

None of these predictions were confirmed by significant findings. Consistent with most findings in the literature (e.g., Golub, 1976b; Sommer, 1972), there was no premenstrual deficit in problem-solving ability as measured by anagram solving. In addition, all groups persisted significantly longer on the insoluble puzzles during the premenstrum than at midcycle. Thus, the present study did not replicate Rodin's (1976) finding that high-arousal subjects who could reattribute their symptoms to the menstrual cycle persisted better than subjects who could not reattribute. However, an interesting trend in the anagram results does lend some tentative support to Rodin's view. The Expectancy group by Phase interaction approached significance ($p < .09$), and inspection of the means in Appendix AA suggests that the Psychological group may have solved less anagrams in the premenstrum than at midcycle, whereas the other two groups solved more. As in Rodin's study it is not possible to determine whether these differences, if they exist, are due to the Biological and Control groups' exerting more effort to compensate (which the Psychological group had been led to believe was unnecessary), or whether they were able to reduce task and menstrual arousal by attributing it to the menstrual cycle (which the Psychological group could not).

This effect remains highly speculative, however, because the differences are small, unreliable, and not significant. It should be noted that in the sample as a whole, there was no correlation between negative mood and either anagram scores or puzzle scores (although puzzles correlated -.29 with self-rated anger, $p < .05$). Golub (1980) has pointed out that many studies have shown the detrimental effects of negative mood in general on cognitive functioning. She argues that the findings in menstrual cycle research are generally negative because the magnitude of mood change in premenstrual subjects (compared to psychiatric patients, for example) is too small to affect cognitive function. This depends, of course, on the severity of the mood changes suffered by any one woman; in the sample used in the present study, at least, it appears that mood changes are too small to affect cognitive function. However, it would be important to study cycle-related changes in women who complain of marked intellectual deficit in the premenstrum.

The finding that the sample as a whole persisted significantly longer on the insoluble puzzle in the premenstrum is difficult to explain. It runs contrary to what would be expected if puzzle-persistence were a measure of frustration tolerance, as it was designed to be (Glass & Singer, 1971), since arousal is hypothesized to be higher in the premenstrum and high arousal is supposed to reduce frustration tolerance. Perhaps the arousal experienced in the premenstrual phase - an internal physiological state which is familiar, expected, and understood by the subject - has a different effect or evokes different coping strategies than the arousal typically used in studies of frustration tolerance, that is, some experimentally manipulated external stress.

It may be, for example, that the subjects exerted more effort to counteract the premenstrual inefficiency they expected to experience, as in the case of the Williams et al. (1981) study, where subjects who expected impairment due to alcohol were able to compensate for its effect to some degree. Alternatively, it may be that the reattribution of general task-arousal to premenstrual tension reduced its detrimental effects. Both these possibilities were discussed above. A third possibility was a methodological artifact which merited further examination. There was a possibility that experience with the puzzles during the first assessment influenced the subjects' response to them in the second assessment in a way that resulted in systematic bias in favour of the premenstrum. This would occur if the brighter, quicker midcycle subjects solved the second (soluble) puzzle very quickly and therefore resolved to spend more time on the first (insoluble) puzzle in the next assessment (which for them would be premenstrual). Similarly, the slower, duller premenstrual subjects might have taken so long to solve the second puzzle, or did not solve it at all, so they resolved to spend less time on the first puzzle next time (which for them would be midcycle). To rule out this possibility, phase by order-of-testing means were examined, and these disconfirmed the hypothesis. In fact, the premenstrual superiority in puzzle persistence was only evident at first testing; at second testing both premenstrual and midcycle scores were the same. Going even further, the midcycle group, despite having spent less time on the first puzzle, solved the second puzzle less often than the premenstrual group did. Although this latter finding was not significant, it runs counter to the notion that the premenstrual group were slower and

duller. Not only did the premenstrual group spend more time on the first puzzle while still managing to solve the second, but they were able to do so within the context of increased interaction with their child (a finding to be discussed later).

It seems possible, therefore, that something more than increased persistence and reattributed arousal may have been operating to improve performance in the premenstrual phase. It has been pointed out (Graham, 1980; Broverman et al., 1968, 1980) that although cognitive abilities tend to be correlated together with some general factor called intelligence, there is great individual variation in the ability to perform different tasks and that the optimum level of arousal may be different for different tasks. As noted in Chapter 1, Broverman et al. (1968, 1980) contend that observed sex differences in cognitive function result from differing degrees of central nervous system activation, which in turn are caused by differential action of estrogen, progesterone, and testosterone. They contend that estrogen increases activation and thus facilitates those kinds of tasks thought to be superior in females - psychomotor speed, automatic, over-practiced behaviour requiring speed and accuracy, rapid attention shifting, etc. Testosterone increases activation to a lesser degree so that males are thought to be inferior at the above tasks but superior in tasks requiring suppression or delay of response, complex problem solving requiring less obvious responses and perceptual restructuring, etc. Progesterone (antagonistic to estrogen) reduces activation, perhaps by increasing MAO levels in the brain.

In an independent attempt to assess this theory, Graham (1980) correlated hormone levels of subjects in different cycle phases with performance on a wide range of cognitive tasks, some of which required CNS activation and some CNS inhibition. Her results suggested that cognitive-hormonal interactions within women, at least, are more complex than the simple activation-inhibition dimension would imply. She found no consistent correlation between cognitive performance and estrogen levels within subjects across phases or across subjects with differing levels of estrogen. She did find that, by and large, subjects performed better during the luteal phase on both activation and inhibition-type tasks. She hypothesized that estrogen provides a "background of excitement" in the brain during the high-estrogen phase. Generally, this background is distracting and impairs performance, but this depends on the optimum level of internal arousal desirable for each task. Progesterone counteracts estrogen and reduces distraction, thus improving performance, subject to the qualification noted above.

It appears, then, that the issue is quite complex and that the term "cognitive function" is too broad and insensitive to be used as a variable in menstrual cycle or hormonal research. Findings which contradict each other and confuse the issue will continue to be made as long as closer attention is not paid to the dimension of activation-inhibition, to optimal levels of arousal, etc. Task-arousal, general life anxiety, menstrual cycle arousal, and individual arousal baselines all interact in a given situation with the optimal level of arousal needed for a given task. Only when all these factors are accounted for will it be possible to predict whether a specific task in a specific situation will show improvement or impairment.

Looking at the specific tasks in the present study in this light, they seem to fall more into the category of inhibition-facilitated tasks. Anagrams require rejection of obvious letter combinations, perceptual restructuring, flexibility, and delay of immediate impulses (much like hidden figures, an inhibition-facilitated task). Puzzle-solving too requires flexibility and the perception of hidden clues. The premenstrual phase has been hypothesized to be one of high autonomic arousal, but the relationship between autonomic arousal and CNS activation is unclear (see Chapter I). The Broverman-Klaiber group (Klaiber et al., 1971; Broverman et al., 1980) hypothesize that MAO, a CNS inhibitor, builds up during the luteal phase under the action of progesterone and is still high in the premenstrual phase; therefore, the premenstrum would be characterized by high CNS inhibition relative to the high-estrogen, high-activation midcycle phase. It seems possible, therefore, that some of the premenstrual superiority in puzzle solving and persistence results from the lower levels of internal distraction and other possible facilitating effects of CNS inhibition. Anagram-solving, however, did not improve premenstrually as one would expect if this theory were correct. Indeed, anagram solving and puzzle persistence were not even correlated.

In summary, the finding that persistence in puzzle solving increased significantly in the premenstrual phase may be explained by one or more of three reasons: 1) subjects expected to do worse and therefore worked harder to compensate, 2) task-arousal could be attributed to premenstrual tension and thereby reduced, with a resulting improvement in frustration tolerance and

performance, or, 3) CNS inhibition, dominant in the premenstrual phase, facilitated this type of cognitive task, whereas the activation produced by estrogen at midcycle disrupted the task through distraction, impulsivity, etc. However, at the present time there are too many inconsistencies and unknowns in the field of cognitive functioning and physiological processes to permit any more than speculation.

MATERNAL BEHAVIOUR

The study's predictions relating to maternal behaviour were that there would be an increase in both positive and negative behaviour in the premenstrual phase, compared to midcycle. The results confirm the increase in positive behaviour but not in negative. These findings are consistent with those of Herljhy (1977), who noted a premenstrual increase in physical affection in a small sample of preschool teachers but no systematic changes in any punitive or control behaviours.

Negative Behaviour. Before discussing the findings in detail, it is worthwhile to consider the context in which they were made. Subjects were brought with their children into a novel and presumably exciting situation which might well have been the highlight of their day. They were aware that they were being not only observed, but videotaped, so that their behaviour would be available for minute analysis at any time. They knew that no matter how terrible the tasks or their children became, it would all be over in seventeen minutes. Most of the time it was the middle of the morning; both they and

their children were rested, well-fed, and unhurried. Under these circumstances one must question how accurately the results reflected real life.

The limited validity and generalizability of findings from contrived laboratory observation has been a continuing concern in psychological research, particularly in clinical research with children who lack the skills for accurate self-reporting (Bell, 1964; Honig et al., 1973; Lytton, 1971). Nowhere is the problem more acute than in the observation of negative parental behaviour. Bell (1964) has suggested various techniques for structuring the interaction set-up so as to maximize the possibility of eliciting the behaviour under study. This is particularly necessary when that behaviour usually occurs rarely under ordinary conditions. Attempts were made in the present study to maximize the possibility that the children would disrupt and provoke the mother. Nonetheless, Bell admits that it is very difficult to elicit negative behaviour from a parent in a laboratory set-up, especially when that parent knows that he is being observed. Furthermore, when a behaviour sampling procedure is being used, as in this study, low-frequency negative behaviours may be missed altogether, if they occur during the rest interval or as the second behaviour in the observation interval (only first behaviours are scored). Generally, less than six negative responses were scored per subject per observation period. This occurred despite the fact that on the average some form of interaction took place between mother and child in over 50% of the intervals scored. The majority of these interactions were initiated by the child, since the mother's main goal was to be left alone to work in peace.

Thus when evaluating the study's finding on negative behaviour, it should first be acknowledged that compounded methodological weaknesses made it highly unlikely that significant differences between phases or groups could be found. To obtain an accurate picture of a mother's negative behaviour during the premenstrum, it would be necessary to install an invisible observer in a corner of the kitchen at five-thirty in the afternoon of a typical mother's day. It has already been noted that household accidents occur most often in the hectic time between five and seven p.m. In addition, research into circadian rhythms has demonstrated that people on a normal wake-sleep cycle show a gradual increase in arousal throughout the day, peaking at around eight o'clock in the evening and then dropping fairly rapidly to the lowest point just before dawn (Colquhoun, 1971). Dalton (1977, 1979) points out that premenstrual irritability and shakiness increases markedly as the blood sugar level drops, particularly if no food has been taken for five hours or so. (In fact, she considers transient hypoglycemia to be a major factor in abrupt mood swings and sudden rages during the premenstrum.)

Considering these two pieces of information, it can be seen that the interval in the late afternoon, when arousal is nearing its peak and blood sugar levels are low, would be optimal for the occurrence of maternal aggression and in extreme cases child abuse. By contrast, the time of day when most of the assessments took place was far from optimal; arousal was just beginning to rise and blood sugar levels were high from breakfast.

In view of all these factors, the behavioural evidence concerning the link between maternal aggression and premenstrual tension must be regarded as too weak to provide an adequate test of hypothesis. With the ethical sanctions against deception and observation without consent, and with the difficulty in getting busy mothers to volunteer, particularly at five o'clock in the afternoon, an adequate test of the hypothesis may be hard to come by. However, there are two pieces of indirect evidence which bear on the question. First of all, self-rated anger-hostility scores were higher in the premenstrum than at midcycle. More importantly, however, husband-rated anger scores were also significantly higher in the premenstrum, suggesting that the subjects may have been acting more hostile. Of all the POMS mood scales, anger showed the greatest increase from midcycle to premenstrum, both when rated by the subject and by her husband.

Secondly, although the Conner's ratings of child's troublesomeness showed no significant effects, there was a trend ($p < .07$) towards an expectancy group by phase interaction. The Psychological group perceived their children as less troublesome in the premenstrum than at midcycle, whereas the other two groups and particularly the Control group, perceived them as more troublesome. This is directly contrary to Rodin's prediction that if premenstrual arousal could not be attributed to the menstrual cycle, it would be attributed to some irritant in the environment. Since the Psychological group also experienced fewer symptoms, it was the subjects who experienced greater premenstrual tension who perceived their children as more troublesome.

These interpretations are merely tentative, of course, because the interaction effect did not reach significance. However, the pattern of correlations between moods and child's perceived troublesomeness adds further support. It was found, as one would expect, that in the sample as a whole, the mother's moods and the child's troublesomeness were weakly but significantly correlated at midcycle. Because in most cases, the mother and the child are together all day, they react to each other and influence each other in many subtle ways, so that if one has a bad day, the other will suffer too. However, in the premenstrual phase, where one would expect the correlations to be even greater because the range of moods is greater, peculiar differences between groups emerge. Since the correlations to be discussed now are based on only seventeen subjects, they must be viewed with caution; nonetheless, significant correlations occurred.

In the Psychological group, again contrary to Rodin's prediction, the correlation between the mother's negative mood and her rating of her child changed from .70 at midcycle to -.57 at the premenstrum. That is, the worse the mother felt premenstrually, the better she thought her child behaved. Husbands' ratings of mother and child showed no such relationship. No logical explanation for this reverse effect comes to mind and perhaps it is merely a chance distribution of the scores. However, it does imply that subjects in the Psychological group did not project their negative feelings on their child.

In the Biological group, no consistent correlations occurred between mood and troublesomeness at either phase of the cycle. The Control group, however,

showed a much stronger positive relationship between the mother's mood and her rating of her child at the premenstrum ($r = .62$) than at midcycle ($r = .23$). The Control group had no information on premenstrual tension other than the hodge-podge of fact and myth they had acquired on their own, and perhaps even more importantly they had not participated in a discussion group in which this topic had been raised (i.e., reacting badly to the children when it isn't their fault). The two experimental groups, on the other hand, had both been given detailed explanations which may have helped them to separate their symptoms from their ongoing life and to appraise themselves more objectively. Hence they may have been less likely to confound their own moods and their child's behaviour. Since the Control group most closely resembles the average woman, considerable confounding may occur in the average household.

Before concluding the discussion of premenstrual negative maternal behaviour, it is worth noting that many of the subjects spontaneously alluded to difficulties with their children and/or husbands in the premenstrual phase. Irritability, impatience and intolerance of noise were frequently mentioned, and many women warned their children to leave them alone. Most commonly, subjects reported screaming at or lashing out at their children for relatively trivial reasons which they would easily ignore at other times, and then feeling extremely guilty afterwards. Even if premenstrual tension does not cause the broken limbs and internal injuries most often seen in emergency rooms, one wonders how many bruises and battered spirits never find their way onto any official record.

To summarize, no direct behavioural evidence of increased maternal aggression in the premenstrum was found, but methodological limitations made this almost impossible. Indirect and anecdotal evidence, although very tentative, does suggest more overt hostility and a jaundiced view of the child's behaviour in the premenstrual phase, the latter mainly in uninformed subjects.

Positive Behaviour. Three hypotheses advanced at the beginning of the study predicted an increase in positive maternal behaviour during the premenstrum: 1) Asso predicted that because premenstrual arousal intensifies all emotional experience, both negative and positive, positive maternal behaviour will be greater during the premenstrual phase than at midcycle, especially in the Psychological group who will be less prone to negative labelling. 2) Guilt over irritable and punitive behaviour causes an attempt to apologize and atone by lavishing extra love. If this were the case, aggressive and positive maternal behaviour ought to be positively correlated, and, to a lesser extent, anger and positive behaviour. 3) Depressive negative affect produces a succorant need which the mother tries to fulfill by affectionate contact with her child. In this case, depressive mood and positive maternal behaviour ought to be positively correlated.

Before discussing the relative merits of these hypotheses, several reservations must be pointed out. All the methodological and contextual factors which contributed to the lack of significant findings on negative behaviour apply here in reverse. Thus, the context, the time of day, and the artificial observational set-up (particularly the awareness of being observed) all made it more likely

that positive maternal behaviour would be observed. In addition, the subject's expectancies and the subject's perception of experimenter expectancies probably played a major role. Subjects were aware that they were being observed and aware that they were premenstrual, and they may also have assumed that the experimenter expected them to be more irritable and distractible. They may even have warned themselves beforehand "I'd better watch myself today". Unlike checking off words on a checklist in the privacy of one's home, no one wants to be recorded on tape yelling at their child, let alone slapping him. Thus they went into the experimental situation on guard against negative behaviour, perhaps even overcompensating in the opposite direction. On the other hand, at midcycle they had no such expectations and fears; their guard would be lower and their behaviour more natural (although not completely, of course). Therefore, it is very likely that experimental artifact contributed largely to this premenstrual increase in positive behaviour.

The final reservation that must be mentioned is that the positive maternal behaviour as measured by the Mash matrix refers mainly to verbal interaction of a friendly, nondirective sort. Rarely did the mothers praise their child and even more rarely did they show any physical affection that wasn't initiated by the child's climbing into their lap. A few times during the session they helped them with the paints or the blocks, but most of the time they remained in their seats and spoke from there. This is a rather limited measure of positive behaviour which may not permit adequate differentiation of the three hypotheses. The third hypothesis, for example, refers to a more physical, loving type of affection than that observed here.

Nonetheless, these reservations aside, it is possible to draw some tentative conclusions on the explanatory power of the three hypotheses. It should be noted, of course, that positive maternal interaction increased in the premenstrual phase without a corresponding increase in the amount of interaction initiated by the child (Child's Compliance, Question and Interaction % showed no phase effect). Thus the observed increase was not in response to the child's becoming more verbal or demanding.

1) If Asso's hypothesis were correct, positive mood and positive maternal behaviour ought to be correlated. However, the correlation between Mother's Interaction % and the POMS Vigour score was zero. Asso further predicted that positive interaction would increase in all groups but most markedly in the Psychological group, which would be less hampered by negative bias, and least in the Biological group. There were no significant differences between groups, but inspection of the means in Table 15 shows that the largest increase occurred in the Biological group, closely followed by the Psychological group, while the Control group showed virtually no increase at all. The two experimental groups had in common the fact that they had viewed a film and participated in a discussion which had focussed their attention on their symptoms and behaviour, and perhaps made them more self-conscious and on guard, as noted earlier. In addition, they were more familiar with the experimenter and perhaps more sensitive to her expectancies and evaluation. These explanations seem more in keeping with the findings that Asso's model does.

One interesting trend deserves a footnote, however. Although none of the chi-square analyses comparing the frequency of mother's positive vs. negative responses to disobediance were significant, there was a trend ($p < .07$) in the Psychological group towards more negative and less positive responses in the premenstrum compared to midcycle. Because disobediance (Child's Negative and Competing Behaviours) was a very low-frequency behaviour and therefore subject to the weakness in reliability pointed out earlier, this finding is no more than suggestive. It could be that the Psychological group, having been told to expect less premenstrual trouble, were less on guard and thus let their true colours show more than the other two groups. This would imply that, although they were aware of (or reported) less premenstrual tension, the irritability and reactivity were still there. This, however, is pure speculation until the effect can be significantly demonstrated.

2) The guilt hypothesis would be implicated if there were a positive correlation between Mother's Negative % and Mother's Interaction %, or between self-rated anger and Mother's Interaction %. However, both correlations were close to zero.

3) As noted earlier, evaluation of the third hypothesis linking maternal affection to succorance needs is weakened by the non-physical nature of the positive interaction measure. Contrary to Herlihy's finding, the correlation between self-rated negative mood and positive interaction was virtually zero. In addition, the correlation between the depression scale of the POMS and Mother's Interaction % was also zero. However, besides the difference in

affection measures, it should be noted that Herlihy's correlations were intra-subject across phases, whereas those in the present study were inter-subject, which introduces much more error variability.

In summary, despite there being no differences in the behaviour of the children, subjects were found to engage in significantly more positive interaction in the premenstrum than at midcycle. It seems plausible to explain at least part of these findings in terms of increased vigilance against negative behaviour, resulting in being overly nice. Among the three other explanations explored, no conclusions can be drawn as to their relative merits, since none seems to fit the data.

SUMMARY AND CONCLUSIONS

In summary, the attempt to manipulate symptom expectancies by providing different information as to their cause and incidence appears to have been successful in lowering expectations but not in raising them. The group who were told that premenstrual tension is largely a psychological illusion expected fewer symptoms and reported actually experiencing fewer symptoms at the end of the test month. In contrast, the group who were told that it was a universal, inevitable result of chemical changes in the brain did not change their expectation nor their experience of symptoms, as measured by the post-measure and follow-up MDQs. Their scores were equivalent to those of the Control group who received no information.

Additionally, the Psychological group reported less premenstrual negative mood than did the other two groups; in fact, their premenstrual self-ratings did not differ from their midcycle ratings. The other two groups showed a marked premenstrual increase.

It is possible that experimental artifact played a role in this difference, in that the subjects were not naive as to the focus on the menstrual cycle and they might reasonably have guessed what the experimenter expected of them. However, the pattern of the data and the importance of the topic for most of the subjects make it unlikely that the "good subject" phenomenon was the sole determinant of the effect. It appears that expectancy does play a role in symptom experience and/or reporting consistent with Asso's synergistic formulation. That is, when expectancy is lowered, symptoms and negative mood are lessened. Although suggestion or some other mechanism may play a varying part in individual subjects, it seems likely that the information provided in the psychological manipulation provided subjects with a new perspective and strategies for self-analysis, so that they were put on guard against biased or exaggerated perception, attribution and recall of negative moods. Thus, much of the symptom reduction was probably due to deliberate cognitive reappraisal. This view is supported by the finding that the Psychological group believed significantly less in a biological cause of their symptoms as a result of the manipulation and maintained this shift even after a month's experience. This suggests that they were able to confirm by their own experience the validity of some of the psychological arguments.

There was, however, a great deal of individual variability both in the experience of symptoms and in the reaction to the information manipulations. The resulting large variances made it difficult to demonstrate significant effects. Thus, although the mean scores of the husband-rated mood and both the husband- and self-rated anger followed a similar pattern to the self-rated mood scores, none of the interaction effects were significant.

No significant differences were found between Expectancy groups on any of the cognitive or behavioural measures, nor in the perception of the child's troublesomeness by the husband or the subject. All groups performed equally well at both menstrual phases on the anagram-solving and persisted longer on the insoluble puzzle in the premenstrum. This latter finding may be due to the subjects' reducing task arousal by reattributing it to premenstrual tension as Rodin proposes, or to their exerting more effort to compensate for expected impairment, or to the luteal-premenstrual increase in CNS inhibition, which may facilitate this type of task.

The predicted premenstrual increase in negative maternal behaviour was not confirmed by direct observation of mother-child interaction, but compounded methodological obstacles may make this difficult to demonstrate. Informal discussion with the subjects suggested that premenstrual irritability does present a problem for them in dealing with their children. Additionally, of all the moods measured, anger-hostility showed the greatest increase in the premenstrual phase from both the subject's and her husband's point of view. Contrary to Rodin's prediction, those who could not blame their irritability on premenstrual tension did not then blame their children. Only the Control group showed any tendency to confound their moods and their children's.

Positive maternal behaviour, on the other hand, was greater in the premenstrual phase in all groups, as predicted. However, none of the proposed hypotheses - guilt, succorance needs or increased positive mood - seems adequate as an explanation. Contrary to Asso's prediction, positive mood did not increase in the premenstrum, nor was positive mood related to positive maternal behaviour. Similarly, neither depression, anger nor rejecting behaviour was related to positive maternal behaviour. It seems likely that part of the increase may result from the subjects' being on guard and overcompensating for expected irritability, or from some other methodological artifact.

In conclusion, the results of the study point to the value of educating women as to the complex psychological and physiological factors involved in premenstrual tension, not only in order to reduce cognitive bias and yield a more realistic appraisal, but also to help reduce the possibility that their own irritability may colour their perception of their environment. In further research, it would be important to see whether providing women with a more complete explanation of both psychological and physiological factors combined alters their experience of symptoms. Additionally, it would be valuable to know whether providing the information is sufficient in itself, or whether the opportunity for discussion is a necessary component. Further research is also needed on the relationship between premenstrual tension and maternal care, which has practical implications for a substantial, but often neglected, proportion of our population.

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A P P E N D I C E S

RESPONSE CLASS MATRIX - M/C FORM

Subject code _____ Coded by _____ Date _____

CHILD'S CONSEQUENT BEHAVIOUR

MOTHER'S ANTECEDENT BEHAVIOUR

	Compl.	Ind. Play	Quest.	Neg.	Verbal Int'n	Non-verb. Int'n	No Resp.
Command	1	2	3	4	5	6	7
Question-command	8	9	10	11	12	13	14
Question	15	16	17	18	19	20	21
Praise	22	23	24	25	26	27	28
Negative	29	30	31	32	33	34	35
Verbal Interaction	36	37	38	39	40	41	42
Non-verbal Interaction	43	44	45	46	47	48	49
No Response	50	51	52	53	54	55	56

RESPONSE CLASS MATRIX - C/M FORM

Subject code _____ Coded by _____ Date _____

MOTHER'S CONSEQUENT BEHAVIOUR

CHILD'S ANTECEDENT BEHAVIOUR	Comm.	Quest.-command	Quest.	Praise	Neg.	Verbal Int'n	Non-verb. Int'n	No Resp.
Compliance	1	2	3	4	5	6	7	8
Independent Play	9	10	11	12	13	14	15	16
Competing Behaviour	17	18	19	20	21	22	23	24
Question	25	26	27	28	29	30	31	32
Negative	33	34	35	36	37	38	39	40
Verbal Interaction	41	42	43	44	45	46	47	48
Non-verbal Interaction	49	50	51	52	53	54	55	56
No Response	57	58	59	60	61	62	63	64

CONNERS P-T QUESTIONNAIRE

Family name_____ Rated by _____ Date _____

Rate your child's behaviour as it is today, compared to what it is usually like.

	much less than usual	somewhat less than usual	a little less than usual	as usual	a little more than usual	somewhat more than usual	much more than usual
1. Restless or overactive							
2. Excitable, impulsive							
3. Disturbed other children							
4. Failed to finish things he started, short attention span							
5. Constantly fidgeting							
6. Inattentive, easily distracted							
7. Demands had to be met immediately, easily frustrated							
8. Cried often and easily							
9. Mood changed quickly							
10. Temper outbursts							

MOOS MENSTRUAL DISTRESS QUESTIONNAIRE

Name _____

Today's Date _____

Date of most recent menstrual flow: from_____to_____

Date of flow before that: from_____to_____

Below is a list of symptoms which women sometimes experience. Please describe your experience of each of these symptoms during the three different times of the month listed below:

Column 1 during your menstrual flow itself

Column 2 during the week before menstrual flow

Column 3, during the rest of the menstrual cycle

Note: Please describe your symptoms as you usually experience them during a typical month, and report any of the symptoms regardless of whether they seem to be related to the menstrual cycle.

For each answer choose the descriptive category below which best describes your experience of that symptom at that time of the month. Write the number in the space provided. Even if none of the descriptions seem exactly correct, choose the one which best describes your experience. Do not leave any spaces blank.

Descriptive categories:

1 - no experience of symptom
2 - barely noticeable
3 - present, mild
4 - present, moderate
5 - present, strong
6 - acute or partially disabling

SYMPTOM	1. MENSTRUAL FLOW	2. WEEK BEFORE	3. REST OF CYCLE
1. Weight gain	____	____	____
2. Insomnia	____	____	____
3. Crying	____	____	____
4. Lowered performance	____	____	____
5. Muscle stiffness	____	____	____
6. Forgetfulness	____	____	____
7. Confusion	____	____	____
8. Take naps or stay in bed	____	____	____
9. Headache	____	____	____
10. Skin disorders	____	____	____
11. Loneliness	____	____	____
12. Feelings of suffocation	____	____	____
13. Affectionate	____	____	____
14. Orderliness	____	____	____
15. Stay home from work or school	____	____	____
16. Cramps (uterine or pelvic)	____	____	____
17. Dizziness or faintness	____	____	____
18. Excitement	____	____	____
19. Chest pains	____	____	____
20. Avoid social activities	____	____	____
21. Anxiety	____	____	____
22. Backache	____	____	____
23. Cold sweats	____	____	____
24. Lowered judgment	____	____	____
25. Fatigue	____	____	____
26. Nausea or vomiting	____	____	____
27. Restlesness	____	____	____
28. Hot flashes	____	____	____
29. Difficulty in concentration	____	____	____
30. Painful or tender breasts	____	____	____
31. Feelings of well-being	____	____	____
32. Buzzing or ringing in the ears	____	____	____
33. Distractible	____	____	____
34. Swelling (abdomen, breasts, ankles)	____	____	____

	1.	2.	3.
35. Accidents(cut finger,break dish)	___	___	___
36. Irritability	___	___	___
37. General aches and pains	___	___	___
38. Mood swings	___	___	___
39. Heart pounding	___	___	___
40. Depression(feeling sad or blue)	___	___	___
41. Decreased efficiency	___	___	___
42. Lowered motor coordination (clumsiness)	___	___	___
43. Numbness or tingling in hands or feet	___	___	___
44. Change in eating habits	___	___	___
45. Tension	___	___	___
46. Blind spots or fuzzy vision	___	___	___
47. Bursts of energy or activity	___	___	___

48. What do you think is the main cause of your <u>menstrual</u> symptoms?

	not at all	some-what	quite a lot	very much
Hormones	___	___	___	___
Water retention	___	___	___	___
Worry or dislike of your period	___	___	___	___
Other Psychological reasons	___	___	___	___

49. What do you think is the main cause of your <u>premenstrual</u> symptoms?

Hormones	___	___	___	___
Water retention	___	___	___	___
Worry or dislike of your coming period	___	___	___	___
Other psychological reasons	___	___	___	___

I hereby grant permission for the Moos Menstrual Distress Questionnaire, copyrighted by myself in 1968 and 1977, to be reproduced in the doctoral dissertation entitled 'Premenstrual Tension and Aggression in Mother-Child Relations'. This thesis is being submitted by Barbara Fraser Fradkin to the School of Graduate Studies of the University of Ottawa as part of the requirements of her Ph.D. degree in clinical psychology.

Rudolf H. Moos

Rudolf H. Moos

2/26/82

Appendix D

Cognitive Tasks

1. Anagram lists A and B, matched for Thorndike-Loge frequency of use and median solution time.

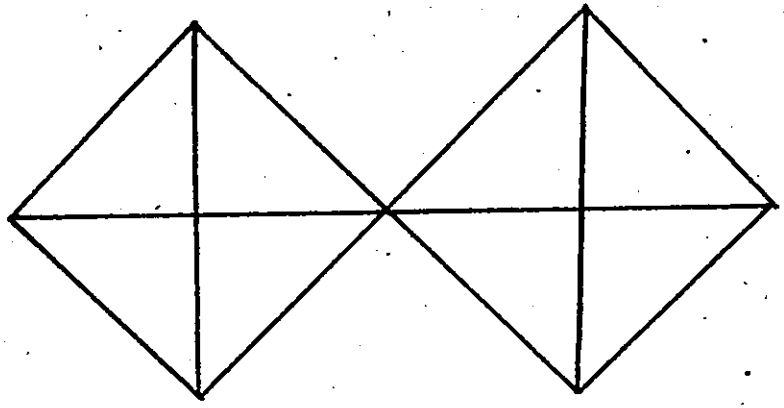
LIST A

	Word	Anagram	T-L Frequency	Time (sec)
1.	JUDGE	EUGJD	AA	3.0
2.	HOUSE	EUOHS	AA	6.0
3.	TRAIN	IRTNA	AA	12.0
4.	SUGAR	UGARS	AA	10.5
5.	FRUIT	IUFTR	AA	15.0
6.	WAGON	GAWNO	A	26.0
7.	BIRTH	RHTIB	A	28.5
8.	BEACH	HECAB	A	37.5
9.	ADOPT	DPAOT	A	42.0
10.	SCALE	ELCSA	A	60.0
11.	BRAWL	AWRLB	5	10.0
12.	SCRUB	RBSCU	14	21.0
13.	ROACH	HOCRA	1	19.5
14.	NYMPH	PHNMY	13	39.0
15.	TONIC	CIOTN	4	31.0
16.	PANIC	PNCIA	19	65.0
17.	AUDIT	DTUAI	1	159.0
18.	CIDER	RDCEI	5	223.5
19.	JAUNT	JUTAN	1	240.0
20.	PEONY	YENPO	1	240.0
			List total:	1288.5
			Half-list total:	241.5

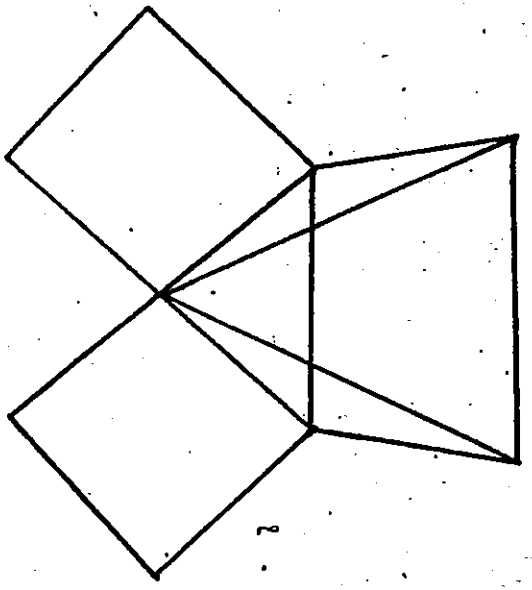
LIST B

1.	WATER	AEWTR	AA	3.0
2.	GUIDE	IUEGD	AA	7.0
3.	PAINT	IPTNA	AA	13.0
4.	CHAIR	CHARI	AA	10.0
5.	HUMAN	MHNUA	AA	15.0
6.	HABIT	TAIBH	A	25.0
7.	GIANT	IGTNA	A	29.0
8.	FAULT	UFLAT	A	36.0
9.	VALUE	AEUVL	AA	46.0
10.	MODEL	DMLOE	A	62.5
11.	CRAMP	RMCAP	8	12.0
12.	BATCH	CAHTB	3	16.0
13.	PATIO	PATOI	1	22.0
14.	FLIRT	LTIFR	7	32.0
15.	GROIN	ROING	1	31.0
16.	BACON	OCBNA	12	92.5
17.	APRON	OAPNR	17	132.0
18.	BLARE	AEBRL	2	224.5
19.	COBRA	AOCRB	1	240.0
20.	TANGO	GATON	1	240.0
			List total:	1288.5
			Half-list total:	246.5

Test A

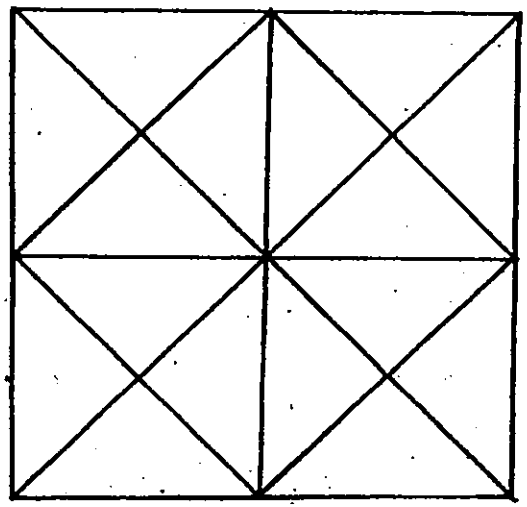


Puzzle 1 - Insoluble

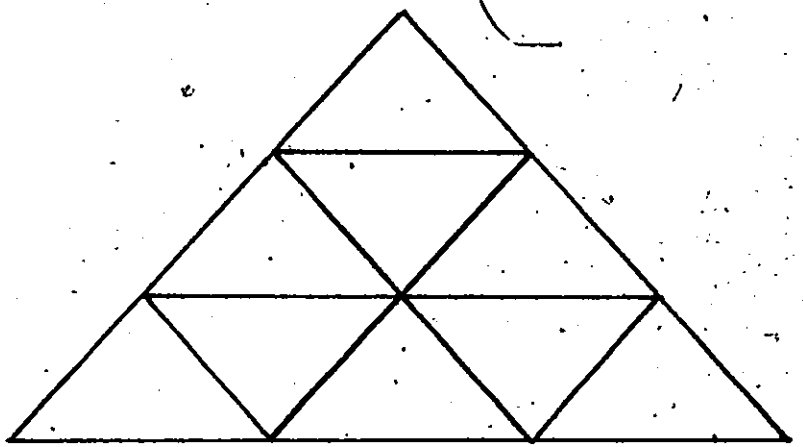


Puzzle 2 - Soluble

Test B



Puzzle 1 - Insoluble



Puzzle 2 - Soluble

Appendix E

Videotape Transcripts

BIOLOGICAL INFORMATION

P.C: Good evening and welcome to Science Today. I'm Pam Cullum and tonight we're going to be discussing premenstrual tension. That's the strange, jittery state just before menstruation that some doctors have called the commonest of all endocrine disorders.

My guest tonight is Dr. Norman Barwin, obstetrician and gynaecologist. He's a specialist in endocrinology and Director of the Reproductive Biology Unit at the new Ottawa General Hospital. For the past fifteen years Dr. Barwin has been involved in both research and practice. He holds an M.D. and a Ph.D. in physiology and he is President of the Canadian Fertility Association. He recently completed an extensive study on premenstrual tension. Tonight he hopes to be able to provide us with some of the latest scientific knowledge about hormones and the menstrual cycle.

Dr. Barwin, to begin, what exactly is premenstrual tension?

N.B: About two to five days before their period, some women experience a variety of uncomfotable or negative symptoms. Some feel more irritable or edgy, others feel more listless and depressed. They can't concentrate as well or perform their usual tasks as efficiently. Swelling, headaches and other pain may also occur. This whole group of negative complaints - poor mood, poor concentration, and pain - this is what is generally referred to as premenstrual tension.

P.C: Two to five days before the period. So we're not talking about the headaches and cramps that occur during menstruation itself.

N.B: No. That is menstrual dysmenorrhea, which is clinically quite separate from premenstrual tension and has quite a different cause.

P.C: How many women experience premenstrual tension?

N.B: That varies with different groups but it seems that roughly 40% of women have some type of premenstrual complaint.

P.C: Forty percent. That's quite a lot.

N.B: Yes, it is, and I think the number is even higher. I think most women experience some sort of premenstrual change, but they may not notice it, or if they do, they don't realize that their symptoms are connected to a particular phase of their cycle. Generally, the women who report premenstrual tension are older and have had children, so that they are more aware of their bodily changes.

P.C: You say bodily changes. But the symptoms you describe sound more like psychological changes.

N.B: They are, but they are psychological changes caused by biological changes in the brain, just as drugs can change our moods. Stimulants, for example, make us feel happy and excited, tranquillizers make us calm and content.

P.C: Do scientists know what biochemical changes cause premenstrual tension?

N.B: Yes. As a result of more than two decades of highly sophistocated research analysing the blood, tissue, and urine of women at different times of the cycle, we have come up with a fairly solid theory. But before I get into that, I'd like to outline the basic hormonal changes that make up the menstrual cycle.

P.C: Yes, that would be a good idea.

N.B: Briefly, the cycle lasts about twenty-eight days and begins with the onset of bleeding. In the first two weeks an egg is being ripened in the ovary and on about the fourteenth day it bursts out and begins to travel down the fallopian tube towards the uterus. As it is travelling, the uterus is being prepared to receive it; its walls are thickening and becoming congested with blood to nourish the fertilised egg. If the egg is not fertilised, it passes out of the vagina and the extra blood and tissue in the uterus begins to break down, leading again to menstrual bleeding.

There are a number of hormones which are important in regulating this cycle, but for our purposes the two main ones to consider are estrogen and progesterone. Estrogen is responsible for the ripening of the egg. It is low in the first week of the cycle, begins to rise

around the seventh day, and reaches a peak at ovulation around the fourteenth day. Then it drops down and rises again to a lesser extent around the twenty-second day. After that, it begins to fall so that it is very low again by menstruation. The two important things to remember about estrogen are 1) it is at its highest and is the single dominant hormone at midcycle, and 2) it is low and falling in the premenstrual phase.

Progesterone has been dubbed the pregnancy hormone; it is responsible for preparing the uterus for pregnancy in the third week of the cycle. It is almost non-existent in the first two weeks and begins to rise only at ovulation. From the fourteenth to the twenty-second day it rises quickly and becomes the dominant hormone, but if there is no pregnancy it then begins to fall, slowly for a couple of days and then quickly so that by menstruation it has reached its former, almost non-existent level. So the important things to remember about progesterone are 1) it has a short life - rising to dominate the system in the third week of the cycle, and 2) it then falls to low levels again during the premenstrual phase.

Now, putting these two hormone patterns together, we see that in the premenstrual phase, both estrogen and progesterone are low and getting lower rapidly. This is the key to premenstrual tension.

P.C: If I understand you so far, Dr. Barwin, we can picture the estrogen levels as two mountains, one with a peak at Day 14, then a valley and a smaller peak a week later (GESTURE). Progesterone has just one big mountain overshadowing estrogen's second peak. And then they both decline to almost nothing in the premenstrual phase.

N.B: That's right. Both hormones are also low in the menstrual phase itself, but the crucial difference is that during the premenstrual phase the hormones are dropping quickly and the body is struggling to adjust. By the time menstruation itself arrives, the change is no longer new. That's why many women notice an improvement in their symptoms once the period comes.

P.C: Are you saying, then, that it is the estrogen and progesterone that changes the woman's moods?

N.B: Indirectly, yes. Animal and human research has shown that estrogen is a stimulant - it acts much like caffeine or mild amphetamine; it causes the body to be more alert and energetic, quicker to act and react. Physiologists and psychologists refer to this state as one of high arousal. Estrogen makes both animals and humans more aroused, and there is a good biological reason for this. Estrogen is the mating hormone; it is at its highest

during the fertile ovulative period so that females are more sensitive to the smells, sounds and sight of their potential mate, more alert and reactive to cues, and more eager for action. Natural selection developed this pattern millions of years ago and today's women still experience vestiges of it. The arousal is not high enough to be unpleasant during this estrogen-dominant phase. Research has shown that arousal in mild degrees produces a pleasant feeling of alertness and energy, whereas in higher degrees it produces an unpleasant, restless feeling where the hands are sometimes shaky, sounds are too acute, palms are sweaty, and so on.

P.C: As is the case when you're writing an exam or waiting for the dentist.

N.B: That's right. Progesterone, on the other hand, is exactly the opposite. Progesterone is a sedative, and in high doses has been used as an anaesthetic to put animals to sleep. It causes the opposite of arousal - it slows the brain waves, the heartrate and the respiration rate, and it deadens the senses so that pain and many other stimuli are less acute. In doing this, it produces a placid, patient, homey feeling in the woman. She may not feel so energetic but she is less likely to be bothered by things that might ordinarily worry or annoy her. From the point of view of biological survival, this sedative effect is also useful. Progesterone is the hormone dominant during pregnancy and you can see the advantage of having the senses dulled against pain and discomfort. When the internal workings of the body are slowed and calmed, the woman conserves energy and maintains an even emotional keel through her pregnancy. Patience and placidity are assets in the preparation for motherhood. In fact, many pregnant women are so sedated they find themselves dropping off to sleep all the time, although there are several other reasons for this as well.

P.C: Yes, I've noticed that myself! Especially in the first three months.

N.B: That's an important point too. The body adapts to progesterone, as to an addictive drug, and you need more and more of it to produce the same effect. I'll be discussing that later. First I'd like to talk about what happens in the premenstrual phase. Biologically, progesterone inhibits estrogen. That is, its sedative effect overrides the stimulating effect of estrogen. In the first two weeks of the cycle, the stimulating effect of estrogen can operate unhampered and most women feel quite alert and active. But after ovulation, progesterone takes over. Even though estrogen rises again, progesterone suppresses its effects and most women feel rather calm and content.

But what happens when both hormones begin to drop off rapidly in the premenstrual phase? We get a classic rebound effect, similar to that which occurs in withdrawal from addictive drugs like valium. The central nervous system has been suppressed at an abnormally low level by progesterone for so long that when the progesterone effect is removed, it rebounds, jumping to a much higher than normal level for a few days. As I said, progesterone is like an addictive tranquillizer; the body struggles hard to adapt itself to this new sedation and works harder to get itself properly aroused. When the sedative effect is removed, Bang, suddenly the system finds itself overaroused and takes a few days to make its way back to normal levels.

P.C: It's like walking along pulling a wagon. You lean forward to compensate, then if the rope breaks you fall on your face.

N.B: Exactly. And add to this the fact that progesterone falls lower than estrogen in the premenstrual phase, so that although you have no more sedating progesterone, you're still left with some stimulating estrogen to add to your arousal and make you feel even more jittery and irritable than ever. Suddenly you've lost all your concentration and all your patience and tolerance, and you wonder what's come over you.

P.C: The way you describe it, all women ought to experience some change in mood and concentration.

N.B: Yes, and they probably do, although they may not notice or understand what is happening to them. And there are individual variations from one woman to another, and even from one month to another in the same woman. Hormone levels and changes vary in different women, and so do the arousal levels of their nervous systems so hormones affect them differently. That's one reason why premenstrual tension is worse in times of stress - it adds further arousal to your already anxious state.

P.C: This is fascinating. It explains all the things so many women have been feeling and never really understood. Now I'm curious to know how these hormones work biochemically in the brain. Could you explain that briefly for us.

N.B: I'll certainly give it a try. We can picture the brain as a mass of electrical circuits, made up of millions of brain cells connected together. Electricity is conducted along these circuits by chemical changes.

Some of these chemicals make it easy for electricity to pass and therefore help the brain to be alert and active. But other chemicals make it more difficult for the brain cells to fire, and if there are too many of them, the brain is sluggish, depressed, or asleep. One of these chemicals is called monoamine oxidase, or MAO. What it does is eat up the chemicals which transmit the electricity. Normally there is a nice, happy balance between all of these chemicals which allows the brain to be properly aroused, not too excited and not too sedated. Research has shown that progesterone increases the amount of MAO, so the woman has more MAO in her brain in the third week of her cycle than at other times. This accounts for her feeling slower and less excitable at this time. But as I said, the brain puts forth extra effort to adapt to this, and it increasss its output of the chemicals that transmit electricity.

Then suddenly the progesterone and the MAO excess disappears, and the brain is now putting out a surplus of the chemicals that make the brain active, so it is too aroused and reacts too eagerly to tiny stimuli. It takes a few days to restore the happy balance, and those few days are the time of premenstrual tension.

P.C: I see. So this MAO is the villain. It steals the chemicals needed for the brain's activity and makes it work harder to stay alert. Then when the progesterone and MAO suddenly disappear, the brain has a surplus of active chemicals. Have I followed you?

N.B: Yes, you have. These effects are not drastic, I might add. For most women the mood changes and the changes in concentration and mental efficiency are not so extreme as to be pathological.

P.C: Dr. Barwin, I'm afraid we're running out of time, but I'm sure you've given our viewers a good idea of what goes on in a woman's brain and body when she feels miserable and edgy for what seems like no reason at all. There is in fact a very good reason. Again, Doctor, thank you, and from Science Today, goodnight.

PSYCHOLOGICAL INFORMATION

P.C: Good evening and welcome to Science Today. I'm Pam Cullum and tonight we're going to be discussing premenstrual tension. That's the strange, jittery state just before menstruation that some doctors have called the commonest of all endocrine disorders.

My guest tonight is Dr. Norman Barwin, obstetrician and gynaecologist. He's a specialist in endocrinology and Director of the Reproductive Biology Unit at the new Ottawa General Hospital. For the past fifteen years Dr. Barwin has been actively involved in research as well as practice. He holds both an M.D. and a Ph.D. in physiology, and he is President of the Canadian Fertility Association. He recently completed an extensive study on premenstrual tension. Tonight he hopes to be able to dispel some of the old wive's tales surrounding the premenstrual period and to provide us instead with some of the latest scientific knowledge about hormones and the menstrual cycle.

Dr. Barwin, to begin, what exactly is premenstrual tension?

N.B: About two to five days before their period, some women report a variety of uncomfortable or negative symptoms; some say they feel more irritable and edgy, others report feeling more listless and depressed. They say they can't concentrate as well or perform their usual tasks as efficiently. Swelling, headaches and other pain may also be reported. This whole group of negative complaints - poor mood, poor concentration, and pain - this is what is generally referred to as premenstrual tension.

P.C: Two to five days before the period. So we're not talking about the headaches, cramps, and other discomforts that occur during menstruation itself.

N.B: No. That's menstrual dysmenorrhea, which is clinically quite separate from premenstrual tension and has quite a different cause.

P.C: How many women experience premenstrual tension?

N.B: That's extremely difficult to estimate, as you'll see when I discuss the causes. It depends on how you measure it. Many women, roughly 40%, believe they suffer from some negative premenstrual symptoms, but when you actually look closely at their moods and efficiency on a daily basis over the month, you find that they are not in fact more depressed or irritable or disorganized

than at other times of the month. Their moods go up and down, of course - we all have our good and bad days - but these ups and downs have little relationship to the menstrual cycle.

P.C: Are you saying there is no such thing as premenstrual tension?

N.B: In essence, yes. That is the most recent conclusion of medical and psychological research - that there is no such thing as a genuine, biological disorder called premenstrual tension.

P.C: But how do you explain the 40% of women who claim to suffer from it? Are they making it up or imagining things?

N.B: Oh no. I'm just saying there is no biological reason for premenstrual symptoms. Premenstrual tension is a psychological condition, not a biological one. It is partly illusion and partly the result of a woman's beliefs and expectations.

P.C: Before we go any further, could you explain the biological cycle for us?

N.B: The menstrual cycle normally lasts about twenty-eight days and begins with the onset of bleeding. This lasts four to six days, after which one of the eggs in the ovary begins to ripen. Around the fourteenth day, the ripe egg breaks out of the ovary and travels down the fallopian tube to the uterus. As it travels, the uterus is being prepared to receive it; its walls thicken and extra blood collects to nourish the egg if it is fertilized. If it is not fertilized, it passes out of the vagina and the thickened walls of the uterus begin to break down, resulting in the next bleeding. There are two major female hormones produced in the ovary which are responsible for this cycle. The first, estrogen, is responsible for the egg's ripening, and it is dominant in the first half of the cycle. The second, progesterone, stimulates the uterus to prepare for the egg, and it is dominant in the second two weeks.

P.C: Surely, with all these hormone changes through the cycle, they could affect a woman's moods and concentration.

N.B: I think that's one of the reasons why the idea of premenstrual tension is so popular - because it seems so reasonable to assume that hormones can change one's moods. But careful research has shown that they don't, not in the small amounts in which they occur in the

normal menstrual cycle. It has been one of the most energetic areas of research in the last two decades. Gynaecologists like myself and other specialists have all been looking for the link between female hormones and moods or mental ability, but we haven't been able to come up with a single solid theory that wasn't disproved by the next experiment.

P.C: But just because you haven't found it doesn't mean it isn't there.

N.B: But we've found strong evidence to the contrary. First of all, the hormone changes in the menstrual cycle are too small to be significant psychologically. For example, progesterone is high during the last two weeks of the cycle, but it is ten times higher still by the end of pregnancy, with no ill-effects to most women. Estrogen is low during the first and last week, but it is much lower in women after menopause. Yet most sixty-year old women function efficiently and happily. The problems that some pregnant and post-menopausal women have are much more readily explained by the psychological stresses that face them.

P.C: A lot of women experience bloating and weight gain in the premenstrual phase. What about that?

N.B: Yes, this is a real phenomenon, and they can be successfully treated with diuretics. However, while the diuretics help the swelling and breast pain, they have no effect on the mood problems.

P.C: Are you saying, then, that all the so-called hormonal disorders of women are caused by psychological stress?

N.B: No, there are two conditions in which drastic changes in hormones and other substances can produce problems for a small percentage of women. The first is the birth process - in a matter of days, even hours, the progesterone level drops from ten times the normal down to almost non-existent levels, and in some women this causes temporary emotional and physical upheaval. But even here there is a strong psychological component, the sudden reality of motherhood, which is bound to produce upheaval in itself.

The second condition is menopause itself - here a few women experience anxiety, depression or physical symptoms which are partly biological and partly psychological. But as for premenstrual tension, I'm merely saying that the hormone levels and changes that occur in the normal menstrual cycle are not great enough to influence mood. It is a pervasive and popular myth, which I think does a great injustice to women by portraying

them as unstable victims of wild mood swings.

P.C: But how would this myth have got started?

N.B: In fear and superstition. Historically the woman's body, and particularly her bleeding, have always been a source of mystery and fear. Menstruation was viewed as shameful and unclean, as a punishment or a curse. It is still called the curse today. Primitive tribes had rituals and taboos about the menstruating woman; she was banished to the hills or confined to her tent, she was not allowed contact with others for fear of contaminating them. In our society today, reason has replaced most of the superstition, but the emotional connotations have stayed with us. Menstruation is still embarrassing and shameful to many women and most couples avoid sex during this time. Women are no longer seen as unclean or cursed, but they are still thought to be at their least capable and happy.

It is a myth that is passed on from mother to daughter and from father to son, that just before and during the period is an unmentionable time, that a girl is going to become moody and unreasonable, break into tears or rages, and so on. The myth is so wide-spread today that researchers have found that even pre-adolescent boys and girls know all about premenstrual tension.

P.C: I see. So the whole menstrual-premenstrual period has always been viewed in such a negative light that all kinds of bad things have been associated with it, including bitchy, blue feelings and poor mental functioning. But you're saying that it's all a myth, that women are not bitchier or less capable?

N.B: Yes, it's a myth, and I'm saying that women need not feel bitchier or less capable if they stopped believing in the myth.

P.C: That's an interesting idea, but I find it hard to believe. I'm a woman, and like that 40% you mentioned earlier, I've noticed some premenstrual tension in myself from time to time. You're saying that I don't feel it, that I only think I feel it because I believe this myth?

N.B: No, I'm not saying you don't feel it, I'm saying you don't feel it as much as you think you do. There are two psychological reasons why a woman may feel slightly worse in the premenstrual phase, even though there's no biological reason for it. First of all, to some extent, because a woman expects to feel poorly, she does. Secondly, she may be more depressed or anxious for a lot of personal reasons like nervousness or shame about the upcoming period, worry about the pain, pregnancy, or inconvenience, and so on. But in addition, a woman tends to notice her bad

moods more during the premenstrual phase, and not the good moods. Then she ends up believing that she felt much worse because of premenstrual tension than at other times of the month.

P.C: So first of all, you're saying that some women do feel slightly worse but only because they expect to.

N.B: That's right. Psychologists working on theories of emotion have found that we are greatly influenced by what we expect to feel or believe we are supposed to feel in a situation. The strength of the mind is shown by how people can be successfully treated with placebos, for example. It is not a question of being naive or gullible, but because women belive these myths, they experience them to some extent. And for some women, as I said, anticipating the discomforts and worries of the menstrual period can cause edginess and anxiety.

P.C: Yes, that makes sense, Dr. Barwin, but often I'm not even aware of the time of the month. I'm certainly not thinking "Oh, I'm premenstrual so I should be feeling poorly". I just notice that I'm feeling poorly and <u>then</u> I realize I'm premenstrual. So there's no expectation, even subconsciously.

N.B: And that brings me to my third main factor - that women notice premenstrual bad moods and blame them on their cycle, but they don't notice good moods. It has to do with what psychologists call attribution - how we attribute causes to our emotions and behaviour. If your husband comes home from work one night and snaps at you, you immediately ask yourself why he's so grumpy. You might think he had a hard day at work, or he has a hangover, or didn't sleep well last night, or he's hungry. Then you choose what to you seems the most plausible explanation, based on the facts you have. If you know he had a poor night's sleep, you'd be apt to blame that, even though the real reason might be quite different. Similarly when you find yourself jittery or depressed, you might pause and say to yourself "Now why do I feel like that?". It might be any of the reasons I gave for your husband. But you might also think, because you're a woman, "maybe it's my period". If it's midcycle for you, you say "No, it can't be" and you look for another reason. But if when you stop to think about it, you realize your period is probably due in a few days, then with all you know about your past symptoms and the menstrual myths, you say "Ahah, it's premenstrual tension, of course", and you dismiss it as that. Each time you notice a bad mood in your premenstrual period, you attribute it to premenstrual tension and you strengthen your belief in the myth. The fact is that you experience just as many good and bad moods at other times of the cycle, but you don't attribute them to your cycle. They

are attributed, usually correctly, to other reasons and then they are forgotten. When someone asks you whether you have noticed any mood changes with the menstrual cycle, you can readily recall the bad moods linked to the premenstrual phase, but not those caused by quarrels, stress at work, or poor sleep.

P.C: In other words, Dr. Barwin, we have just as many bad moods at other times, but of course we don't attribute them to biology.

N.B: You find the real cause, whereas in the premenstrual phase you're quick to attribute all negative feelings to premenstrual tension rather than to other causes.

P.C: And good moods are never attributed to biology.

N.B: No, because the myth only covers bad moods, not good ones. If you do notice you're feeling happy in the premenstrual phase, you're likely to say it's because of something nice that happened, or the weather. And you won't link the premenstrual phase to happiness in your mind.

P.C: Then when you look back over the month, the only thing that stands out in your mind as being connected to the menstrual cycle is bad.

N.B: Exactly. And that is how women see themselves as being more afflicted by premenstrual tension than they really are. And research has shown this. When you ask a woman to rate her moods, or ask someone else to rate her moods, as they occur on a daily basis over a month, and you don't mention that you're studying the menstrual cycle, then both the woman rating herself and the other person rating her report ups and downs which have little to do with the time of the month. Women are not notably more depressed or anxious in the premenstrum. But these same women, asked to look back over the previous month and rate their mood changes, report feeling quite a lot worse during the premenstrum. So in retrospect, the women think they felt worse than they did. Another interesting finding is consistently made in the research. Although many women believe they concentrate poorly and function less efficiently during the premenstrum, careful psychological tests have shown they do just as well, regardless of the time of the month. There is no impairment of concentration, speed, or alertness.

P.C: So we're not the unstable victims of hormones that myth would have us believe.

N.B: Not at all. And I feel it's important that women know that, that they not go on being shortchanged by a

mythology which can influence their feelings and their perceptions of themselves and lead them to think less and expect less of themselves during almost one quarter of every month. Quite a frightening thought when you think of the whole reproductive life. They are capable of being as happy and as productive during the premenstrual phase as at any other time. And while we're on the subject of good moods, I'd like to make one last comment if there's time.

P.C: Yes, of course.

N.B: Good moods and bad moods are very closely related. Psychologists have shown that we often have to rely on circumstances to decide what emotion we are feeling. It's the old notion of "I didn't know whether to laugh or cry". We feel this state of excitation, an aroused, restless feeling that we can interpret as either good or bad - either euphoric or jittery. A woman interpreting this feeling in her premenstrual phase would probably label it negatively, whereas at another time, the same feeling might give her a burst of energy or enthusiasm.

P.C: I see. So just to sum up quickly, the myth of premenstrual tension works four ways to make women believe they suffer from it. First it creates a state of expectancy, so that we feel how we expect to feel. It makes us notice our bad moods more in the premenstrual phase and makes us blame all bad moods on biology, regardless of their real cause. And last...

N.B: It makes us label ambiguous moods as negative, whereas they could just as easily be positive.

P.C: Dr. Barwin, thank you very much. This has been extremely informative and helpful. From Science Today, goodnight.

Appendix F

Pilot Study Questionnaires

I Pre-manipulation measure

Name:_____

1. What do you understand by the term "Premenstrual Tension"?

2. Do you, or have you ever in the past, experienced any symptoms of premenstrual tension?

3. If so, rate the severity of each symptom as follows:

	none	barely noticeable	mild	mod-erate	strong	acute
Irritability.....	__	—	—	—	—	—
Depression.....	__	—	—	—	—	—
Headache, other pain.....	__	—	—	—	—	—
Swelling, bloating..	__	—	—	—	—	—
Poor concentration, inefficiency.....	__	—	—	—	—	—

4. What do you think are the main causes of these symptoms?

	not at all	some-what	quite a lot	very much
Hormones.....	—	—	—	—
Water Retention.....	—	—	—	—
Worry or dislike of your period.....	—	—	—	—
Other psychological reasons.....	—	—	—	—

II Post-Manipulation Measure

Name:_____

1. Was the information on premenstrual tension learned in this session sensible and helpful to you?

   not at all__ a little__ quite a lot__ very__

2. Do you think that the reasons explained in the film and the article apply to you and your symptoms (or lack of them) in particular?

   not at all__ a little__ quite a lot__ very__

3. Knowing what you do now about premenstrual tension, do you think this will make any difference to the feelings and symptoms you notice?

   If so, what changes do you expect?

4. Which of these symptoms would you now expect in the future?

	none	barely noticeable	mild	mod-erate	strong	acute
Irritability.....	__	__	__	__	__	__
Depression.....	__	__	__	__	__	__
Headache, other pain.....	__	__	__	__	__	__
Swelling,bloating.	__	__	__	__	__	__
Poor concentration, inefficiency.....	__	__	__	__	__	__

5. What do you think are the main causes of these symptoms?

	not at all	some-what	quite a lot	very much
Hormones.....	__	__	__	__
Water retention.....	__	__	__	__
Worry or dislike of your period.....	__	__	__	__
Other psychological reasons.....	__	__	__	__

Appendix G

The number of subjects using each birth control method in each group.

	Psychological Group	Control Group	Biological Group	Total
Mechanical Barrier	2	5	3	10
IUD	2	5	5	12
Tubal ligation	4	2	5	11
Vasectomy	3	3	2	8
None or unspecified	6	2	2	10

Appendix H

Summary of the one-way analyses of variance of pre-measure MDQ scores and biological and psychological attribution ratings.

Variable	Source	SS	df	MS	F	p
MDQ	Betw.grp.	70.9	2	35.5	.17	.85
	With.grp.	10127.8	48	211.0		
Biol.Att.	Betw.grp.	2.0	2	1.0	.69	.50
	With.grp.	70.5	48	1.5		
Psych.Att.	Betw.grp.	7.7	2	3.8	2.13	.13
	With.grp.	86.8	48	1.8		

Appendix I

Summary of 3 x 3 multivariate analysis of variance of MDQ scores and attribution ratings.

Source	Hotelling T	Approx.F	df	p.
Expectancy Group (E)	.3829	2.87	6/90	.013
Time of Testing (T)	.2790	2.00	6/43	.087
E x T	.8990	3.15	12/84	.001

Appendix J

Summary of the 3 x 3 analysis of variance of the MDQ scores.

Source	SS	df	MS	F	p.
Between subjects					
Expectancy (E)	2437.80	2	1218.90	2.07	.14
Error (Ss w.gr.)	28302.86	48	589.64		
Within subjects					
Time (T)	353.92	2	176.96	3.38	.04
E x T	850.51	4	212.63	4.06	.004
Error(TxSs w.gr.)	5030.90	96	52.41		
Simple Main Effects					
E at T1	70.95	2	35.47	.15	
E at T2	1267.10	2	633.55	2.74	.08
E at T3	1950.28	2	975.14	4.21	.05
T at E1	1098.47	2	549.24	10.48	.001
T at E2	21.71	2	10.86	.21	
T at E3	84.28	2	42.14	.80	
Pooled error(Ss w.cells)[a]		66	231.48		

T1: Premeasure
T2: Post-measure
T3: Follow-up

E1: Psychological group
E2: Control group
E3: Biological group

[a]Calculated by combining the between- and within-subjects error terms according to the formula suggested by Satterwaithe, as reported in Winer (1971, p.530). This pooled error is used in testing the simple main effects of factor E. The within-subjects error term is used for Factor T, in accordance with Winer.

Appendix K

Summary of the 3 x 3 analysis of variance of biological attribution ratings.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	48.04	2	24.02	7.71	.001
Error (Ss w.gr.)	149.57	48	3.12		
Within subjects					
Time (T)	4.98	2	2.49	3.42	.04
E x T	10.51	4	2.63	3.61	.009
Error(TxSsw.gr.)	69.84	96	.73		
Simple main effects					
E at T1	2.04	2	1.02	.67	
E at T2	23.06	2	11.53	7.59	.01
E at T3	33.45	2	16.73	11.00	.001
T at E1	15.17	2	7.59	10.39	.001
T at E2	.16	2	.08	.11	
T at E3	.16	2	.08	.11	
Pooled error(Ss w.cells)[a]		93	1.52		

T1: Pre-measure
T2: Post-measure
T3: Follow-up

E1: Psychological group
E2: Control group
E3: Biological group

[a] Satterwaithe formula (see Appendix J)

Appendix L

Summary of the 3 x 3 analysis of variance of the psychological attribution ratings.

Source	SS	df	MS	F	p.
Between subjects					
Expectancy (E)	2.52	2	1.26	.60	.55
Error(Ss w.gr.)	100.16	48	2.09		
Within subjects					
Time (T)	1.58	2	.79	1.14	.32
E x T	6.11	4	1.53	2.21	.07
Error(TxSs w.gr.)	66.31	96	.69		

Appendix M

Mean psychological attribution rating in each condition.

	Psychological group	Control group	Biological group
Pre-measure	.82	1.65	.82
Post-measure	1.06	.82	.76
Follow-up	.94	.88	.82

Appendix N

Pearson product-moment correlation coefficients between the pre-measure MDQ (usual amount of symptoms) and six background variables.

	Age	Educ.	Income	Cycle length	Child's age	Child's sex
MDQ	.17	.12	.05	.00	.05	-.06

Appendix O

Mean scores of the wife-rated POMS variables in each condition.

PHASE		Psychological Group	Control Group	Biological Group
Mid-cycle	Tension	40.88	39.53	38.76
	Depress.	40.65	40.12	39.88
	Anger	43.24	44.00	42.82
	Vigour	51.12	56.12	48.76
	Fatigue	42.53	43.82	46.82
	Concent.	39.41	37.82	38.24
Premenstrual	Tension	44.35	48.65	49.88
	Depress.	42.47	48.53	47.18
	Anger	48.47	56.59	53.29
	Vigour	48.41	42.00	43.35
	Fatigue	45.18	55.00	51.82
	Concent.	40.47	45.35	49.47

Appendix P

Mean scores of the husband-rated POMS variables in each condition.

PHASE		Psychological Group	Control Group	Biological Group
Midcycle	Tension	40.71	42.53	40.24
	Depress.	39.76	43.53	41.12
	Anger	41.12	47.35	42.71
	Vigour	52.88	52.12	47.76
	Fatigue	41.06	44.59	48.53
	Concent.	37.24	38.65	38.35
Premenstrual	Tension	39.59	47.12	43.24
	Depress.	40.24	47.71	44.12
	Anger	42.94	50.76	48.18
	Vigour	53.12	46.47	45.29
	Fatigue	40.41	52.88	47.06
	Concent.	35.24	42.71	41.59

Appendix Q

One-way analyses of variance of midcycle scores on eleven dependent variables.

Variable	Source	SS	df	MS	F	p
Wife-TMDS	Betw.grp.	677.72	2	338.86	.30	.74
	With.grp.	54327.60	48	1131.83		
Husb-TMDS	Betw.grp.	3234.47	2	1617.23	1.21	.31
	With.grp.	64066.64	48	1334.72		
Wife-Anger	Betw.grp.	12.12	2	6.06	.09	.92
	With.grp.	3435.53	48	71.57		
Husb-Anger	Betw.grp.	356.99	2	178.50	2.89	.07
	With.grp.	2963.17	48	61.73		
Wife-PTQ	Betw.grp.	45.96	2	22.98	1.08	.35
	With.grp.	1000.11	48	21.28		
Husb-PTQ	Betw.grp.	133.34	2	66.67	1.27	.29
	With.grp.	2360.57	48	52.46		
Anagrams	Betw.grp.	12.82	2	6.41	.33	.72
	With.grp.	937.76	48	19.54		
Puzzles	Betw.grp.	5.56	2	2.78	.32	.73
	With.grp.	410.46	48	8.73		
Mother's Negative%[a]	Betw.grp.	.53	2	.26	1.72	.19
	With.grp.	7.33	48	.15		
Mother's Inter'n%	Betw.grp.	381.31	2	190.66	2.17	.13
	With.grp.	4214.03	48	87.79		
Child Comp. & Inter'n%	Betw.grp.	303.56	2	151.78	.36	.70
	With.grp.	20425.73	48	425.54		

[a] log transformed

Appendix R

Summary of the 3 x 2 multivariate analysis of variance of the six mood scores for mother and child (TMDS, Anger-Hostility, and PTQ as rated by both the husband and the wife).

Source	Hotelling T	Approx.F	df	p
Expectancy group (E)	.3394	1.19	12/84	.31
Phase (P)	.8153	5.84	6/43	.000
E x P	.5551	1.94	12/84	.04

Appendix S

Summary of the 3 x 2 univariate analysis of variance of the wife-rated Total Mood Disturbance Scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	7261.59	2	3630.79	1.56	.22
Error (Ss w.gr.)	111590.12	48	2324.79		
Within subjects					
Phase (P)	48187.15	1	48187.15	30.20	.000
E x P	9628.29	2	4814.15	3.02	.058
Error(PxSs w.gr.)	76525.06	48	1594.27		

Appendix T

Summary of the 3 x 2 univariate analysis of variance of the husband-rated Total Mood Disturbance Scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	20549.25	2	10274.63	4.54	.016
Error(Ss w.gr.)	108506.76	48	2260.56		
Within subjects					
Phase (P)	5529.42	1	5529.42	3.64	.062
E x P	4332.31	2	2166.16	1.43	.250
Error(PxSs w.gr.)	72902.76	48	1518.81		

Appendix U

Summary of the 3 x 2 univariate analysis of variance of the wife-rated POMS anger-hostility scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	335.31	2	167.66	1.18	.316
Error (Ss w.gr.)	6814.71	48	141.97		
Within subjects					
Phase (P)	2268.25	1	2268.25	23.25	.000
E x P	243.55	2	121.77	1.25	.296
Error(PxSs w.gr.)	4682.71	48	97.56		

Appendix V

Summary of the 3 x 2 univariate analysis of variance of the husband-rated POMS anger-hostility scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	840.26	2	420.13	3.60	.035
Error (Ss w.gr.)	5606.24	48	116.80		
Within subjects					
Phase (P)	324.75	1	324.75	5.99	.018
E x P	56.84	2	28.42	.52	.595
Error(PxSs w.gr.)	2603.41	48	54.24		

Appendix W

Mean child's mood score (PTQ) in each condition, as rated by the wife.

	Psychological group	Control group	Biological group
Midcycle	30.24	27.94	29.59
Premenstrual	28.82	33.00	30.94

Appendix X

Mean child's mood score (PTQ) in each condition, as rated by the husband.

	Psychological group	Control group	Biological group
Midcycle	27.47	28.82	31.41
Premenstrual	29.71	32.53	30.59

Appendix Y

Summary of the 3 x 2 univariate analysis of variance of the wife-rated PTQ scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	16.65	2	8.32	.31	.732
Error (Ss w.gr.)	1272.06	48	26.50		
Within subjects					
Phase (P)	70.83	1	70.83	2.26	.139
E x P	179.20	2	89.60	2.86	.067
Error(PxSs w.gr.)	1505.47	48	31.36		

Appendix Z

Summary of the 3 x 2 univariate analysis of variance of the husband-rated PTQ scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	116.53	2	58.26	1.27	.290
Error (Ss w.gr.)	2200.18	48	45.84		
Within subjects					
Phase (P)	74.21	1	74.21	2.57	.115
E x P	90.76	2	45.38	1.57	.218
Error(PxSs w.gr.)	1384.53	48	28.84		

Appendix AA

Mean number of anagrams solved in each condition.

	Psychological group	Control group	Biological group
Midcycle	10.00	9.00	10.12
Premenstrual	8.76	10.59	10.24

Appendix BB

Summary of the 3 x 2 univariate analysis of variance of the anagram scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	10.73	2	5.36	.16	.849
Error (Ss w.gr.)	1562.53	48	32.55		
Within subjects					
Phase (P)	.63	1	.63	.09	.760
E x P	33.90	2	16.95	2.55	.089
Error(PxSs w.gr.)	319.47	48	6.66		

Appendix CC

Summary of the 3 x 2 univariate analysis of variance of the puzzle scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	4.14	2	2.07	.17	.844
Error (Ss w.gr.)	581.53	48	12.12		
Within subjects					
Phase (P)	26.51	1	26.51	4.25	.045
E x P	9.20	2	4.60	.74	.484
Error(PxSs w.gr.)	299.29	48	6.23		

Appendix DD

Summary of the 3 x 2 multivariate analysis of variance of the interaction scores for both mother and child.

Source	Hotelling T	Approx.F	df	p
Expectancy group (E)	.0435	.50	4/92	.736
Phase (P)	.1403	3.30	2/47	.046
E x P	.0569	.65	4/92	.625

Appendix EE

Mean percentage of child's total scored behaviour which was either compliance, question, or interaction.

	Psychological group	Control group	Biological group
Midcycle	45.35	51.11	46.85
Premenstrual	45.41	44.46	49.96

Appendix FF

Summary of the 3 x 2 univariate analysis of variance of the Child's Compliance & Interaction % scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	173.58	2	86.79	.15	.862
Error (Ss w.gr.)	28021.17	48	583.77		
Within subjects					
Phase (P)	34.20	1	34.20	.14	.710
E x P	424.28	2	212.14	.87	.426
Error(PxSs w.gr.)	11738.51	48	244.55		

Appendix GG

Summary of the 3 x 2 univariate analysis of variance of the Mother's Interaction % scores.

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	423.81	2	211.91	.99	.378
Error (Ss w.gr.)	10237.91	48	213.29		
Within subjects					
Phase (P)	265.81	1	265.81	4.42	.041
E x P	133.10	2	66.10	1.11	.336
Error(PxSs w.gr.)	2884.99	48	60.10		

Appendix HH

Mean percentage of mother's total scored behaviour which was negative.

	Psychological group	Control group	Biological group
Midcycle	2.99	6.72	3.39
Premenstrual	4.40	6.77	3.65

Appendix II

Summary of the 3 x 2 univariate analysis of variance of the Mother's Negative % scores.[a]

Source	SS	df	MS	F	p
Between subjects					
Expectancy (E)	.616	2	.308	1.17	.317
Error (Ss w.gr.)	12.561	48	.262		
Within subjects					
Phase (P)	.022	1	.022	.40	.528
E x P	.065	2	.032	.59	.560
Error(PxSs w.gr.)	2.659	48	.055		

[a] log transformed

Appendix JJ

The total frequency of Child's Negative and Competing Behaviours*, summed across all subjects in each condition.

	Psychological group	Control group	Biological group
Midcycle	14.30	52.49	7.53
Premenstrual	20.55	52.21	12.73

* Divided by the total number of observations in each subject's matrix and multiplied by 65 before summing across subjects, to adjust for unequal numbers of observations (matrix totals) between subjects and/or phases.

Appendix KK

The total frequency of mother's positive and negative responses to Child's Negative and Competing Behaviours[a], summed across all subjects in each condition.

	Positive response	Negative response
PSYCHOLOGICAL GROUP		
Midcycle	7.16	7.14
Premenstrual	3.22	17.34
CONTROL GROUP		
Midcycle	23.22	29.27
Premenstrual	17.50	34.71
BIOLOGICAL GROUP		
Midcycle	3.22	4.31
Premenstrual	8.33	4.40

[a] adjusted for unequal matrix totals (see Appendix JJ)