

University of Manitoba

EXERCISE AND THE REPORTED
CHANGES IN SYMPTOMS OF THE
PREMENSTRUAL SYNDROME IN SEDENTARY WOMEN

by

Lynn M. Smith

Submitted to

The Faculty of Graduate Studies

In Partial Fulfillment

of the Requirements for the Degree

Master of Physical Education

Faculty of Physical Education and Recreation Studies

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LYNN M. SMITH

A thesis submitted to the Faculty of Graduate Studies of
the University of Manitoba in partial fulfillment of the requirements
of the degree of

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ABSTRACT

Exercise and the Alleviation of Premenstrual Syndrome (PMS) Symptoms

The purpose of this time-series study was to examine the relationship between exercise and the alleviation of four self-reported PMS symptoms: depression (D), anxiety (A), carbohydrate cravings (CHO) and water retention (H₂O) (Abraham, 1980). A multiple N (N=6), single intervention (I), ABA experimental design was employed over eight months. The subjects were previously diagnosed PMS patients, sedentary and aged 30-40 years. A modified menstrual calendar featuring 4, 100 mm visual analog scales was charted daily by each subject. The intervention consisted of participation in one-hour aerobic exercise sessions, three times per week for twelve weeks. The intervention was sequentially introduced after two menstrual cycles or two lunar months. A series of seven physical fitness tests was administered at selected intervals. Physical characteristics, flexibility, grip strength and predicted maximal oxygen uptake (VO₂ max) were measured at each testing session. Strength of the exercise intervention was based primarily on results concerning increased fitness levels. Changes in food consumption patterns were not desired and monitored via Nutriprofile computer analyses of three, 3-day diet recalls. The full data did not disclose any significant differences between means of any fitness variable when submitted to the

SAS (GLM) Analysis of Variance. Six case studies were presented which discussed individual training responses with reference to dietary and body composition parameters. Modifications regarding PMS symptoms were related to the above results via graphs of the prospective ratings. The graphs were analysed by visual inspection of configuration, height and interval length of symptoms. The intervention was associated with alleviation of PMS symptoms in twenty of the charted symptoms. These results provided evidence that exercise is related to the attenuation of PMS symptoms and may be a viable therapeutic modality for this clinical disorder.

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DEDICATION

To my husband, Bill and our daughters, Kaleigh and Brennagh, I dedicate my thesis. Thank you all for your infinite patience and encouragement.

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CHAPTER 1

INTRODUCTION

PMS refers to the cyclical variations, somatic and affective, experienced during the luteal phase of the menstrual cycle. The symptoms are relieved by menses and the postmenstrual phase must be characterized by a minimum of seven consecutive asymptomatic days. PMS has emerged as a medically recognized complex condition constituting a major health concern for a large percentage of women (Cassara & Egger, 1983).

Up to 90% of females of reproductive age admit to some PMS affliction, and from 20% to 40% perceive their symptoms to be of sufficient severity to temporarily interfere with social or professional performance (Abraham, 1980). Almost all menstruating women exhibit, to some degree, an awareness of their approaching menses. For approximately half of this population, the premenstruum time is accompanied by a variety of disorders. Approximately seventy physical and psychological symptoms have been recognized, and each may range from mild to severe in intensity. Symptoms differ both intra and inter individually with respect to quantity and quality. For some sufferers, the discomforts may be manageable yet others may be affected such that normal daily functioning is impossible.

Historically, PMS was not regarded seriously by the medical profession, and as a result its etiological basis

has not yet been determined. Although there is a lack of definitive knowledge, research has suggested a number of possible causation theories (Pelosi, 1984). Generally, it appears that PMS has a physiological basis which is related to the activity of several reproductive hormones. Moreover, the control mechanisms appear to be centered above the ovaries, in the hypothalamic-pituitary axis. The traditional relegation of PMS as strictly psychogenic has been largely discounted, although disorders such as depression and anxiety are aggravated, probably due to the physiological manifestations (Rubinow, Roy-Byrne, Hoban, Gold & Post, 1984).

Most treatments of PMS are contingent on hypothesized or measured chemical imbalances and/or abnormal interactions of the sex steroids. The more popular treatments are, to a large extent, conflicting in nature and require further scientific research to substantiate their claims of effectiveness. Examples of current prescriptions to rectify hormonal and/or metabolic deficiencies are progesterone, vitamin B₆ (Pyridoxine), vitamin A, Bromocriptine, Danazol and oral contraceptives (Livingstone, Torchia & Cote, 1983). Other drug and vitamin treatments are available, yet all have been designed to treat the symptoms rather than the cause of PMS.

PMS diagnosis depends largely on the subjective, concurrent charting of symptoms, both somatic and affective. The issues of individual awareness and self-control are

prime concerns in the affective domain. Consequently, programs which encourage patients to adopt sound healthy nutritional and exercise practices have been suggested (Lauerson & Stukane, 1983; Harrison, 1984). This holistic approach in moderating the disagreeable effects of PMS has important implications for exercise as a possible ameliorating influence.

Information regarding the effects of exercise on PMS is required as the physical and mental health of menstruating women encompass a large and important segment of the population. Although the primary health concern is the individual's relief from cyclical debilitating symptoms, a multitude of contingent health issues such as marital discord, family violence and incidence of absenteeism in the work force are involved. PMS constitutes a major health problem for the patient, and creates the potential for a plethora of allied health concerns. The numerous social ramifications dictate that effective intervention and prevention methods are needed. Exercise may prove to be instrumental in the alleviation of PMS symptoms as well as the understanding of its pathophysiology and ultimately its etiology.

Statement of the Problem

The purpose of this study was to examine the effect of physical activity on the reported changes of PMS symptoms in sedentary females. The testing hypothesis postulated that during a twelve week aerobic fitness program, six sedentary

women who were confirmed moderate PMS sufferers would experience an alleviation of their symptoms associated with the exercise intervention.

Limitations

This study has been limited by the following factors:

1. The premenstrual syndrome is a complex condition which has not been defined according to etiology or treatment.
2. PMS symptoms are variable with respect to number, type, intensity, appearance and duration within and between individuals.
3. There is no known biochemical marker to indicate the presence or extent of PMS.
4. Subjective, prospective charting of menstrual cycles and recording of symptoms is presently the most reliable method of diagnosing PMS.
5. PMS symptoms have, in controlled studies, exhibited a high placebo response.
6. The manner by which exercise induces changes in the menstrual cycle is unknown.
7. The inclusion in this present study may have elicited symptomatic changes due to a 'Hawthorne effect' rather than the nature of the exercise program.

Criteria for subject selection were established to minimize the effect of confounding variables on PMS symptoms. The establishment of baseline symptom patterns

prior to sequential exercise intervention provided additional assurance that changes resulted from the intervention. Although the single subject design enabled the investigator to utilize the subjects as their own controls, systematic changes other than the exercise intervention may have affected the results. Measurement of body composition and diet recall enabled the investigator to monitor such changes. The subjects were also instructed to maintain their normal diets and activity patterns throughout the study. As a supplementary source of information, an anecdotal assessment form was administered upon completion of the 12 week exercise program.

A tangential limitation concerned the perception of individuals with regards to the severity and exacerbation or attenuation of their symptoms. Additionally, personal physical levels of effort may have been variable and inner motivation may have been a problem for some subjects. Consequently a high profile, popular yet effective program was selected as the exercise mode. The exercise intervention consisted of regular (3 times weekly) participation in aerobic fitness classes. It was desirable to utilize this program for several reasons: popular support from the population being studied; accessibility; and inherent motivational qualities.

Delimitations

In this study, the two major variables of PMS and

exercise have been restricted according to the following criteria:

1. The subjects were selected on the basis of clinical diagnosis of PMS by a physician at least one and one half years prior to the inauguration of this study.

2. The subjects fulfilled secondary criteria regarding sedentary activity levels and moderate manifestation of PMS symptoms prior to inclusion in the present study.

3. The subjects also complied with tertiary eligibility requirements relating to PMS therapy, age and body composition as presented in Chapter 3.

4. The method of exercise intervention selected was an aerobic fitness program, with minimum weekly participation requirements of three, one hour classes.

Definition of Terms

Estrogen This is one type of ovarian hormone that promotes the development and maintenance of reproductive organs and secondary sexual characteristics. Estrogen is also essential to the production of ova during the menstrual cycle.

B-Estradiol Estradiol is the major component of estrogen. It is the most potent form of estrogen, virtually all of it is produced in the ovaries, and it is frequently used in hormonal assays as an indicator of estrogen levels.

Progesterone Progesterone, another ovarian hormone, is primarily concerned with uterine and related preparations

for pregnancy. The corpus luteum produces most of the progesterone in the luteal phase.

Gonadotropins These are the hormones produced in the pituitary gland. Follicle stimulating hormone (FSH) and luteinizing hormone (LH) are two important gonadotropins which control ovarian hormone production.

Gonadotropin-releasing hormone (GnRH) This hormone produced in the hypothalamus, is released in a pulsatile fashion and regulates the activity of the gonadotropins.

Hypothalamic-Pituitary-Ovarian Axis (HPO) This term refers to the highly sensitive and integrative functions of the three major organs involved in the delicate regulation of the female reproductive cycle.

Menstrual Cycle The female reproductive cycle or menses, in its optimal state, consists of a 28 day cycle which is timed from the beginning of menstrual bleeding (Day 1). The proliferative phase, characterized by high estrogen levels completes the first half of the cycle which is often termed the follicular phase. Ovulation occurs at approximately day 14 and the following luteal or secretory phase is dominated by progesterone (see Appendix A).

Menarche Menarche refers to the onset of menses in conjunction with the pubertal progression towards mature female status. Menses proximal to menarche are characteristically anovulatory.

Menopause As used in this paper, menopause refers to the age-determined cessation of menses and signals the end of

the reproductive functions.

Primary Amenorrhea Primary amenorrhea refers to the absence of menarche when pubertal development indicates readiness for this stage.

Secondary Amenorrhea This menstrual dysfunction refers to the absence of a normal cyclic menses and is characterized by three or fewer cycles per year (Gray & Dale, 1983).

Oligomenorrhea The occurrence of a menstrual cycle on an irregular, decreased basis is termed oligomenorrhea which is characterized by less than nine but greater than four menses per year (Gray & Dale, 1983).

Dysmenorrhea Chronic uterine cramps, spasms and general discomfort of the pelvic region in association with the onset of menses has been termed dysmenorrhea.

Eumenorrhea Optimal, regular functioning of the reproductive cycle is labelled eumenorrhea; at least nine menses per year is considered normal menstrual frequency (Gray & Dale, 1983).

Premenstrual Syndrome PMS refers to the cyclical variation in physical and psychological well being experienced following ovulation and ceasing near menses.

PMS Subsets Abraham (1980) postulated that PMS was composed of four subsets which could be characterized by one major symptom. These subsets are defined below and are acceptable when considered as general guides. Most PMS patients exhibit symptoms which cross over these four subsets.

PMT-A: This group is categorized by the major symptoms of anxiety, irritability, tension and mood swings.

PMT-D: Depression, forgetfulness, easy crying, insomnia and confusion characterize this group.

PMT-H: The symptoms of water retention, edema, weight gain and mastalgia are the main concerns.

PMT-C: Headaches, fatigue, dizziness, heart pounding, increased appetite and cravings for sweets are symptoms included in this group.

Molimina: As used in this paper, molimina refers to the mild manifestations of PMS symptoms.

CHAPTER 2

REVIEW OF RELATED LITERATURE

Introduction

This chapter contains a review of literature of the following: (1) the menstrual cycle, (2) exercise considerations, (3) the premenstrual syndrome, and (4) exercise and the premenstrual syndrome.

The Menstrual Cycle

The menstrual cycle is a complex function characterized by highly variable parameters. The wide range of what is considered acceptable with reference to female reproductive status emphasizes great individual variability. In order to establish PMS within the frame of reference of menstrual dysfunctions, the following literature concerning normal menstrual cycles has been presented. Thus, this initial section addresses the broad topic of menstrual function in order to place PMS within this context and to further apply to PMS, the principal tenents and models governing this body of knowledge.

The menstrual cycle is a unique function of the female reproductive system. Gynecological structures and endocrine metabolism act synergistically to produce cyclical procreative developments in the female body. The normal menstrual cycle has been defined in terms of four major hormones: estrogen, progesterone, luteinizing hormone (LH), and follicle

stimulating hormone (FSH). Briefly, the composite of a normal menstrual cycle consists of the rhythmical production and release of these hormones due to highly integrated endocrine messengers and precursors. The normal menstrual cycle has been determined to be approximately one month in duration and consists of two distinct phases (see Appendix A). Sperryn (1983) stated that menstrual periods vary greatly with the average menses being four days in each twenty-eight day cycle. Wills, Harvard and Roylance (1979) presented a scheme of hormonal events that typify the usual menstrual cycle. A brief summary of these events has been extracted from the preceding reference. The secretion of ovarian hormones is controlled by the pituitary gland and the gonadotropins FSH and LH. The secretion rates of FSH and LH are under estrogen feedback control, while progesterone only influences LH secretion. The ovaries also secrete androgens, a fact which is important in clinical disorders. The menstrual cycle begins with the onset of menses. This interval, termed the follicular phase, is characterized by pituitary production of low tonic amounts of FSH acting in concert with LH (Wills et al., 1979). The LH stimulates follicular development, and the ovaries as well as the graafian follicle secrete estradiol. This dual production of estrogen culminates in a peak which initiates a discharge of the hypothalamic gonadotropin releasing hormone (GNRH). In turn this results in a sudden mid-cycle surge of FSH and LH (Wills et al., 1979, p. 76). Ovulation occurs

approximately twenty-four hours after this discharge and the luteal phase commences. The corpus luteum secretes principally progesterone for five to seven days (Wills et al., 1979). Hormone levels decline toward the end of the cycle in the absence of fertilization. There is a consequent diminished inhibitory effect on the hypothalamus and, consequently, an increase in FSH initiates a new cycle (Wills et al., 1979, p. 76).

The normal menstrual cycle is operable from menarche to menopause. Wills and colleagues (1979) have established that prior to puberty the HPO axis is characterized by low functional levels of FSH and LH and undetectable amounts of estradiol. Pubertal progression is accomplished via a steady increase of FSH and LH which stimulates the ovarian secretion of estrogen and the subsequent manifestations of secondary sex characteristics (Wills et al., 1979, p. 74). At the other end of the spectrum, the onset of menopause is reported to be due to a decline in ovarian activity. These same authors cited reduced negative feedback associated with hyperactivity of the hypothalamus and the elevated levels of plasma LH and FSH (Wills et al., 1979, p. 78).

In summary, the menstrual cycle is a complex function of the HPO axis. The normal cycle can be viewed as one stage of reproductive performance which changes with time and/or interventions. There are different states of menstrual function as defined by high or low, individual neuroendocrine and metabolic status. Both endogenous and

exogenous variables can alter the menstrual pattern. A multitude of permutations are possible knowing that individual factors may effect changes in isolation or in concert according to highly variable conditions and labilities.

Exercise Considerations

Physical activity is one major effector in the modification of the menstrual cycle. Changes in physiology and metabolism can occur in varying degrees according to a host of integrated exercise variables. The following section will present an overview of hormonal responses to acute exercise then address those endocrine changes associated with chronic exercise.

Acute Exercise

Endocrine Responses Terjung (1979b) stated that although physical activity tended to increase circulating levels of both estradiol and progesterone in both phases, only the increases in the mid-luteal phase were significant. He suggested that this increase directly correlated to intensity of exercise. During the mid-luteal phase, however, serum hormonal levels are so great that the additive exercise effects may not be effectual (Terjung, 1979b). Felig (1983) agreed with reports that acute exercise does not alter FSH or LH, and that temporary elevations in plasma progesterone and estradiol are proportional to the exercise intensity. He stated that exercise induced alterations were primarily a

consequence of reduced hormone clearance as opposed to hypersecretion (Felig, 1983).

When Shangold (1984) reviewed the hormone changes that occur in response to acute exercise, the following pattern emerged: an increase or decrease in gonadotropin levels; a rise in prolactin probably related to the temperature rise and normal status; and a temporary elevation of estrogen and progesterone. Other responses documented were a rise in endorphins, melatonin, and catecholamine concentrations.

An attempt to monitor hormonal responses in relation to phases of the menstrual cycle was made by Jurkowski (1982). This author studied the relationship of hormonal responses to several bicycle ergometer workloads at two specific times; mid-follicular and mid-luteal. The work intensities were 20 minutes at 30% maximum power output, 20 minutes at 60% maximum power output, and to exhaustion at 90% maximum power output. Due to the significant variability in individual hormonal profiles, this study graphed hormone responses as changes from resting values instead of citing absolute levels. The results of this research showed evidence that hormonal responses were different for the follicular and the luteal phase.

The key findings in this research appear to be concerned with possible aberrant quantities and/or ratios at specific phases of the cycle. In the mid-follicular stage, at exhaustion, estradiol, LH and FSH were elevated. Progesterone levels remained insensitive to change. Conversely, the mid-

luteal data evidenced a significant rise in progesterone levels at all exercise intensities. Estradiol levels showed a marked increase directly proportional to the work load intensities. LH responded with no consistent alterations and FSH remained stable. Jurkowski (1982) agreed that the modifications in estradiol and perhaps progesterone resulted from decreased hormone clearance. The pulsatile release of the gonadotropins prompted Jurkowski to suggest: "More frequent sampling over a considerably longer period of time . . . will be required to properly delineate the response of LH and FSH to exercise" (Jurkowski, 1982, p. 87). Thus, sex steroid responses to acute exercise are transitory but the feedback systems may be affected and accumulated interventions such as those associated with chronic exercise warrant inspection. However, Jurkowski (1982) emphasized the variability of related studies, the complexities of hormonal feedback systems, the unique pulsatile secretion of LH and FSH and other difficulties with obtaining valid hormonal measurements. Regardless, this study reported exercise induced increased concentrations of estradiol, progesterone and perhaps also LH and FSH (Jurkowski, 1982).

Chronic Exercise

Endocrine Responses Shangold (1984) presented several topics in her discussion of modulations associated with chronic training: body composition, chronic hormone alterations, fertility, diet and thyroid hormones and

estrogen metabolism. She asserted that a causal link between exercise and menstrual aberrations has been difficult to establish due to the high number of impinging variables.

With respect to chronic hormone alterations, Shangold noted that mid-luteal phase progesterone levels were lower in active female runners and that a strong correlation existed between decreased luteal phase length and increased mileage. Again the author advised caution due to the complex involvement of other factors especially emotional stress. Other hormonal changes associated with activity, noted by Shangold were: lower levels of estradiol and sex hormone binding globulin, higher ratio of estrone to estradiol, and higher concentrations of a stress-related adrenal androgen. The metabolism of estrogen was discussed in terms of the different metabolic pathways utilized by thin and obese women. Shangold concluded that discrepancies in circulating levels of estrogen were minimal and that the employment of different mechanisms to achieve equitable estrogen levels has not been proven to account for the higher prevalence of amenorrhea in thin women.

In a study of teenage swimmers, age-matched controls, and parous adult women, Bonen, Belacastro, Ling, and Simpson (1981) investigated the endocrine basis of the training induced amenorrhea. Their results are briefly highlighted below:

1. The menstrual cycle of the swimmers was shorter than that of the adult women and the controls.

2. The swimmers' luteal phase duration was significantly shorter than the other two groups.
3. The swimmers displayed significantly higher concentrations of LH in the follicular phase.
4. The characteristic FSH peak was not observed in the swimmers and cyclic fluctuations were significantly damped during both phases.
5. The FSH/LH ratio was significantly reduced throughout the cycle in the swim group.
6. During the follicular phase estradiol concentrations were prematurely maximal in the swimmers and were significantly lower on the day of the LH surge and during the luteal phase.
7. During the follicular phase progesterone concentrations were higher in the swimmers but the characteristic elevations of progesterone with the luteal peak and during the luteal phase were significantly depressed in the swim group (Bonen et al., 1981).

According to Bonen and Keizer (1984), athletic menstrual irregularity (AMI) appears to be due to changes in gonadotropin levels caused by regular training. These authors reasoned that the accumulation of many chronic repetitive training sessions probably stimulates AMI (Bonen & Keizer, 1984, p. 87). Thus, they examined the hormonal modifications which occur during acute exercise to determine which responses might contribute to AMI onset. The two major changes were thought to be the marked increases of

estradiol and progesterone with little or no evident changes of FSH and LH concentrations. It is important to note these acute responses in addition to the subsequent remarks by Bonen and Keizer; steroid response is greater with increased exercise intensity, elevated prolactin levels have been reported, metabolic clearance rates are depressed post exercise and the post exercise state may prolong residual physiological effects. These authors concluded that ". . . sex steroids are easily increased during exercise and can influence the hypothalamic pituitary hormone release" (Bonen & Keizer, 1984, p. 88). More importantly, this belief emphasizes the cumulative effect of acute activity on the feedback systems of the HPO axis which perhaps is the initial mechanism of change that is eventually manifested in chronic modulations of hormones.

Bonen (1984) also proclaimed the available research as confusing and lacking systematic criteria to define AMI. Typically, secondary amenorrhea has been utilized to signify abnormality. He stipulated that less obvious endocrine disturbances have not been detected and consequently research findings may not present a true representation of alternate menstrual states. Although the monitoring of length and frequency of the menstrual cycle identifies amenorrhea, it does not detect a milder, perhaps progressive, disturbance--the shortening of the luteal phase. Bonen (1984) suggested that future research should examine individual endocrine profiles.

Dale and Goldberg (1982) reviewed the hypothesized causative factors concerning exercise and the alteration of menses (see Appendix B). Most presumed relationships have been previously mentioned except for thermoregulatory dysfunction, diet/nutrition and the production of melatonin by the pineal gland. The importance of hormone balance is evident and other factors may have to be considered in reference to endocrinal function.

The evaluation of endocrine status is complicated by the production, conversion, degradation and excretion of sex hormones (Keizer et al., 1982; Prior, 1982a). Due to the fact that definitive, reliable hormonal measurements are difficult, there have been attempts to establish the sex-hormone-binding globulin (SHBG) as a biochemical marker. This protein binds estradiol and testosterone and has been reported at lower levels in amenorrheic runners (Shangold, 1984). Keizer and his colleagues (1982), however, cautioned against the acceptance of measurements concerning SHBG, as exercise and its increase of body temperature produced artifacts with regards to free estrogen levels. Prior (1982a) supported this by her dismissal of the theory that lowered SHBG is caused by decreased estrogen.

Wakat, Sweeney, and Rogol (1982) directed research regarding reproductive functioning in female cross-country runners. This study investigated athletic amenorrhea and proposed that this term was a misnomer with oligomenorrhea being more correct. The results determined that normal

hormonal profiles were present with normal LH and FSH responses to exogenous gonadotropin releasing hormones. The researchers concluded that alterations in the HPO complex occurred above the level of the pituitary, and that oligomenorrhea may be a response to excessive energy expenditure or limited caloric intake (Wakat et al., 1982). They stipulated that there may be three reasons for oligomenorrhea: endocrine fragility, delayed menarche, and critical body weight.

Felig (1983) addressed the topic of training related menstrual disturbances by remarking upon the seasonal variation in activity and lack of menses in some sports. He cited evidence that correlated decreased training and increased normality of menstrual function. Felig (1983) supported the tenets of hypothalamic-pituitary origination of athletic menstrual disorders as opposed to ovarian failure. The hormonal pattern of an oligomenorrheic subject was typified by reduced concentrations of FSH and LH while the endocrine profile of an anovulatory eumenorrheic subject consisted of reduced luteal phase serum progesterone levels and depressed FSH concentrations during both the luteal and follicular phases (Felig, 1983, p. 318).

Prior (1982a) suggested that exercise modifications were not confined to the ovaries but were operative at the hypothalamic-pituitary regions. She cited an earlier pilot study of four eumenorrheic female runners in which the following results were observed: significant shorter cycle

length, nearly significant shorter luteal phase and significant decreases of FSH and LH (Prior, 1982a). Moreover, the data demonstrated a subtle change in the GNRH response, quite similar to clinical presentations of hypothalamic dysfunction. In another study which chronicled menstrual cycle changes of fourteen normal females who had undertaken a program of marathon training, Prior (1982b) collected information on 48 menstrual cycles. There was a high incidence of abnormal cycles with one third being monophasic and probably anovulatory, and another third being premenstrually short and perhaps luteally deficient. Thus, exercise in the form of long training runs (approximately 10 miles) was associated with the aforementioned abnormal cycles (Prior, 1982b). One novel, crucial difference denoted by Prior was that the maintenance of normal biphasic cycles correlated with shorter training distances per training session. This observation corroborates evidence which purports to link intensity and athletic menstrual irregularity without dismissing the acute exercise modulations.

In summary, the effects of chronic exercise have been more thoroughly documented than acute exercise responses, but definitive comprehension of both hormone responses has not yet been substantiated by research. The importance of acute exercise effects should be recognized as setting precedents for chronic hormone alterations through the HPO complicated feedback system. The reported changes with

acute exercise were elevations in estradiol and progesterone with a follicular phase increase of FSH at exhaustion but no other consistent alterations in FSH or LH. The chronic training program modulated basal hormonal levels with different results. With respect to the major sex steroids and gonadotropins, the repetitive, cumulative effect of physical activity has been associated with decreased estradiol, progesterone, FSH and LH while testosterone was elevated (Wells, 1985). The area of endocrinology and exercise requires more research and technical refinement. The plethora of variables related to exercise need to be investigated further. The many possible combinations of integrated or interference in menstrual cycle modifications have to this point hindered any serious allocations of direct causality. However, general patterns of exercise induced menstrual abnormalities have been evident in the preceding literature review. Shangold (1984) has summarized athletic menstrual irregularity according to this progression of dysfunctions:

1. luteal phase deficiency (with or without prolongation of the follicular phase);
2. euestrogonic anovulation; and
3. hypoestrogenic amenorrhea (p. 73)

Changes in menstrual function occur as a result of single exercise bouts and cumulative responses are manifested as responses to chronic exercise. The progressive sequence of altered menstrual states signifies that underlying mechanisms of change must involve the entire HPO axis.

In conclusion, the female body is capable of complex and varied adaptations to exercise. The changes which are most relevant to this project are endocrinal in nature. Although laboratory techniques have been refined, the measurement of hormones is currently relegated to a secondary status due to the instability of hormones, the complexity of their actions, the diurnal and cyclical secretory phases, and great individual ranges. On the other hand, with care and further refinement, assessment of conditioning with reference to hormones and endocrine evaluations pertaining to menstrual abnormalities will become practical diagnostic procedures. The higher center, the hypothalamus, has been supported as the orchestrator of the complex hormonal interplay involved in the delicate balance of the organs and substances of the female menstrual cycle. Exercise does affect this pattern in transient and profound ways, the mechanisms of which are not completely documented nor understood.

The Premenstrual Syndrome

Introduction

For centuries, women's complaints regarding their menstrual cycles have been largely ignored or dismissed. The changing roles of women in society have reinforced the need for logical and accountable approaches to female health concerns. Menstrual disturbances, once considered undeserving of medical scrutiny, are presently receiving

their warranted attention. One major health problem which has extensive implications for physical and mental well being, is the premenstrual syndrome. According to Reid (1983) PMS is a "complex psychoneuroendocrine disorder that results in recurrent temporary disruption of the personal and professional lives of a substantial number of women throughout their reproductive years" (p. 1).

Although the current popular appellation is 'syndrome,' PMS can not be correctly labelled as such because each individual displays variability with respect to periodicity and manifestations. According to Witt (1984) it may more accurately be described as a 'condition.' Premenstrual tension, another common label, refers to only one category of possible symptoms (Witt, 1984).

PMS is "a major clinical entity which affects a large proportion of the female population" (Reid & Yen, 1981, p. 85). It has been estimated that approximately 90% of all women experience some degree of premenstrual discomfort (Cassara & Egger, 1983; Reid 1983; Witt, 1984). Of these, an estimated 40-50% display symptoms that may affect their behavior (Abraham, 1980; Reid, 1983; Witt, 1984). The degree of interference with normal intellectual, emotional and physical functioning has been well addressed by Cassara and Egger (1983). These authors report that a high level of interference is a distinguishing factor between PMS and normal menstrual discomfort or molimina. Abraham (1980) referred to the degree of temporary dysfunction in social

and/or occupational interactions as affecting 20-40% of women. A further 10% of women in their reproductive years could be diagnosed as severe PMS sufferers (Dalton, 1964; Witt, 1984). Simkin (1985) reported that it is now generally accepted that 40% of women in reproductive years have PMS. She further calculated that 3 million Canadian women were affected with approximately one quarter million being severely incapacitated (Simkin, 1985).

A primary health care concern should be the substantial number of women who experience uncharacteristic or aggravated symptoms which interfere with normal functions. By this definition those patients victimized by PMS are generally incapacitated from the moderate to severe range. Due to a 'ripple effect,' PMS claims secondary victims in the form of family, friends and co-workers (Gonzalez, 1981; Cassara & Egger, 1983). Relationships may be tested as the PMS sufferer displays inappropriate behaviour and attitudes on a cyclical basis (Gough, 1982; Lauerson & Graves, 1983; Witt, 1984). PMS has been cited as a factor in cases of marital breakdown, family discord, child abuse, alcoholism, work inefficiency, absenteeism and other social ills (Abraham, 1980; Dalton M., 1981; Dalton K., 1977). Thus, the ramifications of PMS are manifold and further investigations concerning etiology and treatment are crucial to the optimal health status of millions. A further review of literature is presented under four headings, namely: (1) definitions and diagnosis; (2) symptoms; (3) etiologies; and

(4) treatments.

Definition and Diagnosis

PMS is the clinical categorization for a multitude of symptoms displayed during the premenstruum. According to Reid (1983), PMS refers to the cyclic luteal phase recurrence of a combination of physical, psychological and behavioural changes of sufficient distress which result in interference with interpersonal relationships and/or normal activities.

Prior to the diagnosis of PMS, routine medical procedures and tests should exclude the possibility of other pathological or psychological conditions (Debrovner, 1982). The distinction between degrees of PMS impairment is also critical in the diagnosis and eventual treatment prescription. According to Abraham (1980) if mild symptoms have been discounted then there is an increased consistency of definition.

Diagnosis of PMS usually includes information regarding personal history, symptoms as observed through charting of a monthly menstrual calendar and data from a premenstrual rating scale (Abraham, 1980; Rubinow et al, 1984). There are various opinions regarding the length of the premenstruum and symptom eligibility. Pelosi (1984) stipulated that the symptoms begin with the luteal phase and terminate the day before menses. References generally concurred that the symptomatic stage begins approximately two weeks prior to and ends around the time of menstruation (Rubinow et al., 1984). From a self reporting menstrual calendar, there

should be graphic evidence of congregation of symptoms during the luteal phase. These symptoms intensify towards the end of the cycle and either taper off gradually or disappear immediately upon bleeding. Reid (1983) extended this definition and referred to four patterns of distribution of premenstrual symptoms; the first two being most frequently displayed (see Appendix C).

Several diagnostic aids have been developed to obtain information regarding somatic and affective characteristics of PMS. One of the original scales was the Moos Menstrual Distress Questionnaire (Moos, 1968). Other scales have attempted to refine and improve upon this model. Useful instruments have been developed by various authors (Steiner, Haskett & Carroll, 1980; Halbreich, Endicott & Schact, 1982). Their reliability and validity have been examined and critically analysed by Rubinow and Roy-Byrne (1984). Rubinow's group (1984) proposed a 100 mm visual analogue scale to chart PMS symptoms on a twice daily longitudinal basis. These authors employed this assessment method for depression and anxiety in a preliminary study of twenty women. This rating scheme was preferred for reasons of; simplicity, compliance, graphic translation, discrimination of PMS patterns and uniformity with associated psychological research (Rubinow et al., 1984, p. 684). Although there are limitations regarding the use of rating scales as diagnostic aids (Ruble, 1977), current literature supports the practice of concurrent subjective reporting as opposed to retrospective

observer reporting (Haskett, 1982; Rubinow et al., 1984; Keatley & Baldonado, 1984). The novel visual analogue scale applied by Rubinow and colleagues (1984) shows considerable promise as a reliable charting and assessment instrument.

The obvious endocrine foundations of PMS have led to research involving various hormonal imbalance theories and respective hormonal assays. As a result, recent research has focused on the possibility of a biochemical marker that might indicate PMS. M. Dalton (1982) conducted a compact study which yielded evidence of significantly lower serum levels of the sex hormone binding globulin in women with severe PMS as compared to normal age matched controls. The possibility of a simple blood test is supported by Gough (1982). She further advised that utilization of a hormonal test as a diagnostic procedure may be contraindicated at the present time due to practical aspects such as cost. However, hormonal blood tests such as the binding capacity of the sex hormone binding globulin may, in the future, be major diagnostic aids.

According to Dalton (1977), the woman with true PMS will exhibit the following characteristics beyond the criteria already stated; sugar cravings, intolerance of long periods between food ingestion, and atypical hormonal assay. Age of onset of PMS has been researched and discussed and it has been determined that PMS can begin with menarche and continue to menopause (Dalton, 1977). Symptoms appear to worsen with age and/or hormonal traumas such as childbirth

and tubal ligations (Abraham 1980). Simkin (1985) presented results of a retrospective chart review of 237 PMS patients from Alexandria Community Health Centre in Calgary, Alberta. She concluded from this study that the profile of a typical PMS sufferer included these characteristics; 37 years old, university or college education, married, two children, abstainer from smoking and alcohol, healthy pregnancies, post-partum depression, and negative side effects associated with oral contraceptives (Simkin, 1985). Other studies have attempted to correlate the incidence of PMS with other factors such as past menstrual history, parity, stress, high intake of refined carbohydrates, tubal ligation, lack of outdoor exercise and use of oral contraceptives (Abraham, 1980; Reid & Yen, 1981). There are controversial results, however, and charting risk symptoms through self-reporting and the use of a rating scale are the most creditable means of clinical diagnosis.

In summary, diagnosis of PMS has been characterized by poorly defined and divergent procedures (Keatley & Baldonado, 1984). Presently, patients may be diagnosed by several available methods with Halbreich's 'PAF' rating scale being highly recommended (Rubinow et al., 1984). Of particular importance is careful monitoring of the menstrual calendar which should be done by the subject for several menstrual cycles. From patterns displayed in the calendar, an individual PMS profile can be observed and accordingly used as the basis for diagnosis and subsequent treatment. The

establishment of the intermenstrual baseline fulfills several elements of diagnostic control. According to Rubinow and colleagues (1984), the baseline menstrual chart differentiates between symptom occurrence and exacerbation, illustrates degrees of fluctuation and consequently reflects the severity of symptoms, excludes random changes and permits assessment of the consistency and predictability of subsequent cycles (p. 169).

Symptoms To date, PMS has eluded definitive medical taxonomy. A multitude of factors such as society's reluctance to openly discuss this issue as well as the complex pathophysiology of PMS have impeded progress. PMS is characterized by a lack of clarity with reference to a number of parameters. In the first place, PMS is cyclical in nature, with time and duration of the condition being changeable. There are a multitude of symptoms which can be loosely categorized as physiological or affective. The most frequently noted symptoms are: irritability, depression, edema, fatigue, mastalgia, headache and abdominal cramps (Livingstone et al., 1983). Other common clinical manifestations are binging, cravings for chocolate, sugar and/or salt, emotional instability, alcoholism, clumsiness, and acne (Abraham, 1980; Reid & Yen, 1981; Pelosi, 1983). In spite of the preponderance of negative symptoms, Lever and Brush (1981) emphasized the infrequently reported positive behaviours associated with PMS. Evidence of increased creativity, sexual arousal, and accomplishment of

major tasks due to elevated energy levels have been reported by some individuals. The positive facets of PMS are limited and overshadowed by the more commonly reported aggravated, destructive impairments. Simkin (1985) suggested that the three major symptoms were anger, irritability and depression. Together with headache, lack of control, cravings and fluid retention, these account for the seven most commonly reported symptoms. The most current and extensive list of symptoms has been compiled by Rubinow's group (1984) (see Appendix D). To further complicate matters, this plethora of symptoms may be exhibited differentially with regards to quality and quantity with reference to subjects and menstrual cycles. PMS manifestations are unique to individuals and concerns regarding degrees of difference from normality are critical in evaluating research (Keatley & Baldonado, 1984; Rubinow et al., 1984).

Etiologies For over fifty years, research has been conducted in a variety of ways to explore the complexities of PMS. Investigations regarding diagnosis, development of rating scales and possible causes have also been conducted. There have been attempts to categorize symptoms and distinguish between degrees of affliction to minimize the variability that has inundated the major body of research (Haskett & Abplanalp, 1982; Steiner et al., 1980; Halbreich et al., 1982). Historically, research has examined PMS etiology through association and postulation regarding symptoms. Pelosi (1984) presented a brief but thorough

chronological analysis of the major empirical and controversial proposed causes of PMS (see Appendix E). Most of the etiological hypotheses have addressed the symptoms of water retention and/or mood disorders (Rubinow et al., 1984). Presently, there are a number of theories concerning the cause of PMS which Keatley and Baldonado (1984) have classified as hormonal imbalances, nutritional deficiencies, prolactin levels and psychological aberrations. There is still some evidence of support, however, for the psychogenic basis of PMS (Ruble, 1977; Bernsted, Luggin, & Petersson, 1984). According to Simkin (1985), the 1980 Conference on Psychosomatic Obstetrics and Gynecology issued a statement that in essence said PMS was regarded as an endocrinopathy. Simkin (1985) reiterated that PMS should be considered a defect of physiology not one of character. The majority of the literature supports the physical derivation of PMS with physiological manifestations initiating or aggravating the affective disorders. Reid and Yen (1981) supported an earlier statement by Parker (1960) which concluded that somatic factors were primary and that the psychic factors appeared secondary to the physiological, biochemical and anatomical changes resulting from hormonal influences. Harrison (1984) has recognized that symptoms may exist throughout the month but be magnified premenstrually. She has called this variation PMM and suggested that the important distinguishing factor lies within the treatments for PMM as opposed to PMS (Harrison, 1984, p. 19).

Most authorities implicated hormonal and/or metabolic abnormalities as the fundamental cause of PMS. The current list of etiologies compiled by Rubinow and colleagues (1984) confirmed this general consensus regarding the endocrinal nature of PMS (see Appendix F). Popular theories, controversial or otherwise, are; estrogen excess, progesterone deficiency. Vitamin B₆/magnesium deficiency, hyperprolactinemia, hypoglycemia, vitamin A deficiency, endogenous hormone allergies, and general menstrual aberrations (Livingstone et al., 1983; Rubinow et al., 1984). Price and Giannini (1985) discussed the popular hypotheses regarding PMS pathogenesis. They presented in tabular form PMS-related, preliminary laboratory findings:

1. Decreased beta-endorphins
2. MHPG-increased late luteal phase, decreased two days prior to menses, and elevated during menses
3. Increased estrogen
4. Decreased progesterone
5. Deficiency of pyridoxine
6. Hypoglycemia
7. Increased prolactin
8. Electrolyte disturbance secondary to fluid retention
(Price & Giannini, 1985, p. 36).

Reid and Yen (1981) conducted a thorough review of existing medical literature and consequently developed an innovative neurotransmitter hypothesis of PMS' etiology (Cassara & Egger, 1983). The endogenous opiate withdrawal theory of

Reid and Yen (1981) implicated atypical responses of peptides and endorphins in PMS. The area of research involving the "endorphins-endogenous opioid peptides" was declared by Price and Giannini as "most promising" (p. 36). Through the culmination of research, though divergent and discrete, PMS has been defined as a "multifaceted psychoneuroendocrine disorder involving a dysfunction in the hypothalamus, pituitary and ovarian axis" (Pelosi, 1984, p. 304). However, there is no single hypothesis or precise physiological origin that adequately explains PMS or is empirically substantiated.

Treatments The fact that there is no determined cause of PMS precludes the development of a single prescription. Most treatments have been developed in conjunction with proposed etiologies. Consequently, treatments have been as diverse and creative as their respective hypothesized rationales. Rubinow and colleagues (1984) have compiled a list of treatments which are correlated with their similar presentation of etiologies (see Appendix G). Some therapies or treatments in vogue are natural progesterone supplements, synthetic progestens, vitamin and mineral supplements (B₆, Pyridoxine, megadoses A, magnesium), oral contraceptives, diuretics and prostaglandins (Livingstone et al., 1983; Rubinow et al., 1984). Life-style modifications concerning stress reduction, dietary restrictions and physical activity have also been suggested as having therapeutic benefits (Reid & Yen, 1981; Lauerson & Graves, 1983; Pelosi, 1984). For the most part, the effectiveness of any one of the

preceding treatments has not been substantiated. Reid and Yen (1981) criticized most treatments because of their lack of double blind controlled investigative trials. Results have also been notoriously inconclusive and contradictory in general and more specifically between similar trials (Rubinow & Roy-Byrne, 1984).

To compound the issue, the placebo response rate in treatment studies has been high, 40%-50% (Pelosi, 1984; Rubinow & Roy-Byrne, 1984; Keatley & Baldonado, 1984). The elevated placebo response, although disconcerting to the promoters of certain treatments, supports the fundamental existence of PMS. Clare (1979) suggested that the placebo influence was not an impediment, rather it should justify and validate further research. The implications for future research have been alluded to by several authors. Rubinow and colleagues (1984) emphasized several design considerations that would ensure adequate selection criteria of subjects and avoidance of common methodological errors. They emphasized that inclusion of subjects on the basis of retrospective rating was a major factor compromising the results of previous studies (Rubinow et al., 1984). Reid and Yen (1981), in their systematic critique, underscored the need for further studies under controlled conditions. Clare (1979) also supported this stance and further suggested that more attention should be focused on the rating of premenstrual complaints rather than biochemical measurements.

In summary, there are a number of treatments associated

with their respective hypothesized etiologies. Inconclusive evidence of spurious relief of selected symptoms in random subjects has been observed. Due to the lack of conclusive, statistical support and the high placebo response in conjunction with shortcomings in research design, there is no single treatment for PMS. As a result there have been various attempts to modify life-styles with reference to decreasing stress levels, modifying dietary regimen and increasing physical activity. Any beneficial, ameliorating effects of such endeavours are largely subjective and anecdotal in nature and consequently can at this time only be allocated to a secondary therapeutic status.

Exercise and the Premenstrual Syndrome

Physical activity has been reported to be a possible alleviator of PMS dysphoria. There is a paucity of controlled experimental research concerning the effect of exercise on PMS symptomology. In a study which somewhat confused menstrual distress and PMS, Timonen and Procope (1971) compared various menstrual related disturbances and complaints in two groups; athletic and non athletic females. The resulting evidence indirectly generally supported the association between activity and fewer reported symptoms of PMS, as well as fewer menstrual related difficulties. Elite athletes were also involved in the study and generally reflected the attenuation of symptoms, although stress of competition was a variable which perhaps biased the results.

A further presentation of the literature reviewed considers the current status of exercise and PMS.

Woods, Most and Dery (1982) discovered a positive relationship between the length of menses and PMS exacerbation. Consequently, athletes who tend to experience shorter or amenorrheic cycles generally experience less PMS. This led to the conjecture that exercise may alleviate PMS via the indirect method of reducing or eliminating menstruation. Moreover, these authors reported that although the effects of strenuous exercise were not clear, physical exertion could possibly relieve symptoms in eumenorrheic women (Woods et al., 1982). Abraham (1980) suggested "prolonged outdoor exercise" would be effective in improving the anxiety and irritability syndrome of PMS but would have no effect on the water retention category. Pelosi (1984) agreed that exercise may also be beneficial, excluding the constellation of edema symptoms. He suggested that increased physical activity and exercise would promote metabolism and rebuild muscle mass, and urged a coincident reduction of calories to decrease the percentage of body fat, a factor which affects estrogen levels.

The strategies of positive stress management and increased rest can also be achieved through exercise. Lauerson and Graves (1983) promoted exercise, although they suggested the relaxation effect was primary and that an undesired hormonal imbalance could result from over-prescription. Yoga and meditation were also suggested as

possible therapies by Livingstone, Torchia and Cote (1983). Reid and Yen's (1981) theory of endogenous opiate withdrawal can be related to the effect of endorphins claimed by runners. To achieve this "runner's high," one must participate in activity of a strenuous nature, consequently physical activity may act as a substitute source of endogenous opiates. Harber and Sutton (1984) linked B-endorphin increases to menstrual cycle alterations (see Appendix H).

Price and Giannini (1985) suggested that in the clinical diagnosis of PMS, an evaluation of daily activities should be undertaken "because lack of exercise is associated with increased incidence of premenstrual tension" (Price & Giannini, 1985, p. 35).

Harrison (1984) also referred to the addictive nature of exercise. She recommended a daily regime of at least thirty minutes of exercise. In an interview transcript, Prior suggested that exercise conditioning adaptations of increased endorphin levels and decreased estrogen and catecholamine levels, may have been responsible for the relief of PMS symptoms in her current study. Caldwell (1984) reviewed Prior's report of the initial phase of a study concerning molimina (mild PMS) and moderate endurance training. Preliminary results of Prior's study reported that eight subjects experienced an overall decrease in symptoms with significant relief of mastalgia and fluid retention. The analysis of data concerning hormone levels,

not yet completed, was viewed as an important aspect of Prior's study.

In summary, exercise has been recommended as practical, successful therapy in the management of PMS symptoms. Few studies have scientifically examined the effects of exercise on PMS symptoms.

CHAPTER 3

METHODS AND PROCEDURES

Introduction

This study has attempted to fulfill methodological criteria relating to subject selection and experimental design as presented in the previous chapter. Where possible, factors which may have confounded the outcome have been controlled or monitored. Due to the cyclical and individual nature of both the female reproductive cycle and PMS, the design selection of time series analysis was deemed appropriate. The intent of this study was not to prove a cause and effect relationship between the aerobic program and PMS symptoms. This investigation was conducted to explore the association between these two variables by first establishing that the exercise intervention produced a training effect, and that documented daily ratings of the PMS symptoms demonstrated changes that could be associated with the exercise intervention. The null hypothesis stated that there would be no discernible changes in the PMS symptoms coincident with the exercise intervention of this study and the subsequent withdrawal of the intervention.

The following subsections are included in this chapter: (1) subjects; (2) experimental design; (3) data collection; and (4) data analysis.

Subjects

The subjects were selected from participants in the PMS Support Group or from patients at the Women's Health Clinic, Winnipeg, Manitoba. Criteria for inclusion in the study included clinical diagnosis of PMS, age between 30-40 years (as of January 1st, 1985), absence of ongoing physical or psychiatric disorders, and a sedentary activity pattern. Pregnant women and women taking progesterone treatments or oral contraceptives were excluded from the study. Self-report menstrual charts completed during the past year were examined to confirm the temporal specificity of the disorder prior to final inclusion in the project. Clinical diagnosis, by a medical doctor, (at least one year previous to this study) labelled the women as well-defined moderate PMS sufferers. Volunteers were requested from this group. The initial plan was to randomly select the final study participants. However, there were only 35 women among randomly selected PMS medical charts who met the above qualifications. Of this number, 12 responded to the letter requesting volunteers, with 8 embarking upon the initial phase of the study. All subjects were requested to sign an informed consent form prior to inclusion in the study (see Appendix I). Medical consent was obtained for each subject to participate in the exercise program. Of the 8 registrants in the study, only 6 completed the exercise program requisites and participated for the duration of the research. The non-compliance of 2 subjects was reportedly due to personal

problems. The remaining 6 subjects performed the required fitness tests, responded to the diet and exercise surveys, complied with the exercise program criteria, and completed daily charting for 28 weeks.

Experimental Design

Six women participated in a twenty-eight week experimental study in order to investigate the relationship of regular aerobic exercise intervention and the reported symptom changes to PMS. A single subject time-series design (ABA) was used to compare the baseline and intervention patterns for symptoms of PMS, as determined from self-report charts (Kratochwill, 1978). The type of time-series employed in this study is also known as a multiple N, single intervention design. Kratochwill (1978) referred to this experimental design as the ABA time-series wherein there was ". . . greater certainty that the intervention was responsible for change . . ." as compared to the AB design (p. 41). Another advantage of this design is that it ". . . permits an investigator to examine the pervasiveness of an intervention over groups or individual subjects" (Kratochwill, 1978, p. 69).

Further to these advantages for this particular type of time-series research, the choice of design was governed by the following factors: the individual menstrual and premenstrual profile of each PMS subject is important; the individual subject's response to the intervention is

critical; with time-series research, a small N is permitted whereas a traditional research method would demand a large, costly sample; and additionally, more traditional research methods mask or moderate the interindividual variability characteristic of PMS. Within the 'Multiple N, single I design' the examination of individual responses is fostered as well, the graphical representation of data highlights the cyclical nature of the reproductive cycle and associated PMS symptoms.

During a minimum initial baseline period of two menstrual cycles or eight weeks, the subjects completed a daily report of somatic and emotional symptoms. Following establishment of a consistent baseline pattern, exercise intervention in the form of regular aerobic fitness classes, was sequentially introduced for a twelve week period. The subjects continued to record daily symptoms of PMS throughout the exercise period and for eight additional weeks. Aerobic fitness and body composition were evaluated at weeks 0, 8, 12, 16, 20, 24 and 28 in order to monitor the effect of training and detraining (see Figure 1). Test I at week 0 was used as a familiarization process. At weeks 0, 16 and 28, a three day dietary recall was administered.

Campbell and Stanley (1966) defined time-series research:

The presence of a periodic measurement process on some group or individual and the introduction of an experimental change into this time series of measurement, the results of which are indicated by a discontinuity in the measurements recorded in the time series (p. 37).

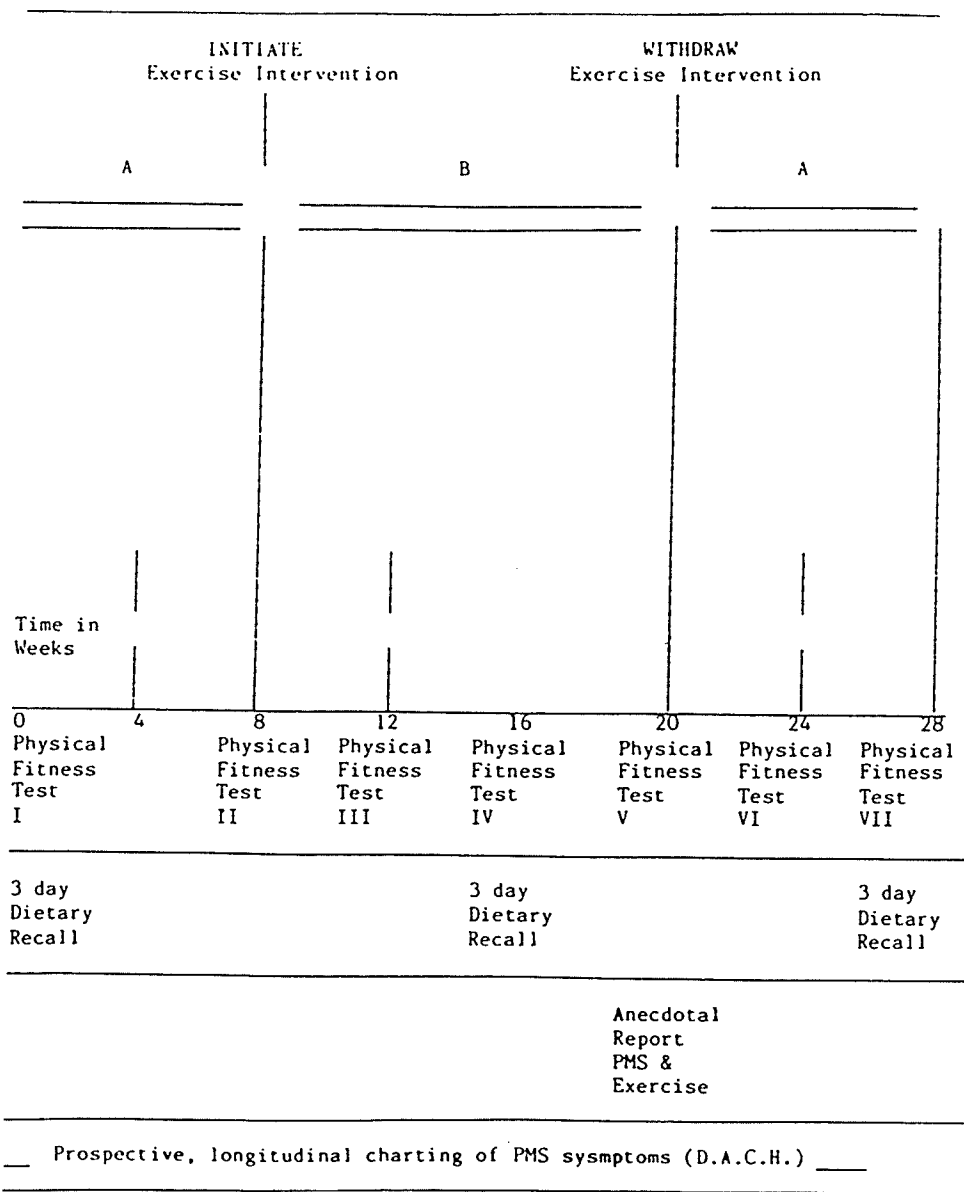


Figure 1
 Chronological Representation of
 Measurements Made During ABA Research Design

According to Kratochwill, in time-series research, it is important to define the group well and to 'match' subjects in ABA designs such as this; a 'multiple N, single intervention' (Kratochwill, 1978, p. 35). By imposing the entry criteria, several inadequacies as cited in analyses of past PMS research were hopefully avoided (Keatley & Baldanado, 1984; Rubinow et al., 1984). One of the primary tasks of this research was to attract subjects who fulfilled criteria as specified in this chapter and who represented a well-defined group of PMS subjects.

Although the replication of the ABA design on six different subjects strengthens postulations and conclusions regarding the intervention, there are several problems associated with the ABA design itself. In the first place, the experiment ends on the baseline thus the ethics of withdrawing the exercise intervention then terminating the study have been considered. This point was discussed and agreed upon by the subjects. Although time constraints did not allow a second B phase to be added, the opportunity to return to the exercise program was offered to all of the subjects. Secondly, it was questionable whether the post-exercise time period would show evidence of a return to baseline. It was considered possible that the physiological effects might have tapered off to baseline levels within the 8 weeks but the PMS symptoms might not meet pre-exercise ratings. Thirdly, to avoid the susceptibility of this research to an 'historical effect' the start of the study

was staggered or sequential according to the individual subject's menstrual phases or the completion of two lunar months. These two methods were employed to serially initiate subjects to the intervention due to the lengthy nature of one subject's first cycle and the temporal limits of the study. Additionally, the intra-individual variability with regard to cycle length and PMS symptoms has previously been clinically documented for these subjects.

An ABAB design would have provided a greater measure of reliability but time constraints disallowed this format. The reliability of the ABA design depends upon the replication over the six subjects. A contraindication regarding dieting and the monitoring of such through the Nutriprofile procedure was one feature specifically included to reinforce exercise as the treatment variable. One recognized threat was that most subjects became more familiar and comfortable with testing procedures, primarily the bicycle ergometer. Although a familiarization session occurred with fitness test I, the variable of time on the bike may not be wholly attributable to increased or decreased fitness levels. The lengthy nature of the study along with the single, continuous intervention phase may have allayed most external validity threats. Measurement procedures and test protocols were uniform throughout the study. All fitness tests were administered by the author. Standardized charting of PMS symptoms was strictly adhered to including initial declaration of symptoms and time of journal entries.

Exercise Program

Following an initial exercise test the six (6) subjects were enrolled in an aerobic fitness class for twelve weeks. The rationale for the selection of this type of exercise has been included in the review of literature. The participants were requested to attend a minimum of three and maximum of four, sixty minute classes per week. An instructor taught all classes, which consisted of stretching, muscle group exercises and aerobic exercise. Each aerobic class consisted of approximately the same general format: approximately 15 minutes of warmup exercises; 20 minutes of large cardiovascular locomotor movements; 15 minutes of conditioning floor work exercises; and 10 minutes of cool-down activities. Subjects were requested to attend 'beginner workouts' which emphasized instruction and demonstration of safe positions as well as modifications of intensity according to target heart rate principles.

The intervention consisted of a 12 week 'aerobics' exercise program wherein subjects were requested to participate not less than three times or more than four times per week. All subjects who completed the study attended less than the required number of classes and compliance appeared to wane slightly after the second month of the intervention. In the first month of the study the subjects attended a mean of 3.02 classes. Mean number of classes attended for the second and third months were 2.54 and 2.17, respectively. Although five of the six individuals

complied with the minimum number of classes initially, there was an observable reduction in individual compliance over the three months for all subjects.

There were two dropouts during the study. The first withdrawal occurred in the preliminary phase of the study with 'personal problems' cited as rationale. At the beginning of the second month of the exercise program a second subject withdrew due to chronic back problems. However, six subjects fulfilled the entry criteria and, except where indicated, completed the research requirements for the 28 weeks.

Data Collection

a) Charting of PMS Symptoms

A menstrual calendar was developed for this study from a 100 mm visual analog form in current use by Rubinow et al. (1984) (see Appendix J). Symptoms, graded 0 to 100 with 0 being absent and 100, severe, were observed and indexed daily. This data was translated to graphic form (see Appendix K). The subjects were encouraged to complete their daily journal entries each evening, prior to retiring. The claiming of symptoms, explanations of charting and practice in using the visual analog scales occurred at a preliminary meeting before the baseline charting began. To further enhance continuity, subjects were periodically reminded of their original definitions of the four symptoms and thus charting of declared symptoms was reinforced. At the bottom

of each page which represented one day's entry, specific areas requested the recording of the date and the occurrence of menses. As well, the inclusion of anecdotal information regarding diet, exercise and undue stress was deemed important. Space for written comments was made available for notation of concerns, circumstances, illness or other influences regarding the data registered on the monthly calendar.

The rationale for utilizing a menstrual calendar which incorporated four 100 mm visual analog scales was based upon the following points; the data points are more discerning of variability, the data allows for better or clearer translation of the intervention effect and the ease, reliability, efficiency and methodological reliability have been supported in the literature (Rubinow et al., 1984; Bond & Lader, 1974).

With regard to the reliability of the charting instrument, qualitative data have been utilized in psychiatric research and more specifically with PMS-related affective disorders (Moos, 1968; Halbreich et al., 1982; Haskett & Abplanalp, 1982). The method of prospective, concurrent charting via the visual analogue scale has been supported by a scientific study conducted by Rubinow and colleagues (1984). These authors conducted a preliminary application of this measure to confirm the menstrually related mood changes of depression and anxiety. Their investigation supported the employment of 100 mm visual

analogue scales in the identification and assessment of menstrually related mood disorders. Additional support for the utilization of analogue scales in the evaluation of subjective feelings came from Bond and Lader (1974).

Evidence regarding the acceptance and value of verbal data was tendered by Ericsson and Simon (1980). O'Rourke (1982) presented data which supported subjective appraisals relating to menstrual cycle disturbances and mental health. Verbal reports were not documented but prompted the creation of an anecdotal report administered at week 20 (see Appendix L). This questionnaire elicited comments and responses regarding the exercise program and personal conjectures regarding PMS symptomology. The rationale behind this anecdotal form was two-fold. Personal comments were considered an important supplement to the research conducted and such use of qualitative data has been accorded scientific status (Ericsson & Simon, 1980; O'Rourke, 1982). Secondly, the form provided the subjects with an instrument by which they could attempt to formulate their own thoughts regarding exercise and PMS and as well evaluate the exercise program itself.

b) Anecdotal Survey: PMS and Exercise (Appendix L)

This form was used to gather qualitative data to supplement the charted symptoms and to document the general attitudes and perceptions regarding the Exercise Program and PMS (see Appendix L).

c) Dietary Recall

To monitor diet, the Nutriprofile method of collecting and computing information was utilized (see Appendix M). At weeks 0, 16 and 28 the subjects were required to record dietary intake over the last three days. A nutritionist conducted the computer analysis of the dietary recall. Monitoring of dietary status was necessary due to its implications for PMS, and the information was used as supplementary data to corroborate self-reported charting of symptoms. The Nutriprofile data was collected as a control mechanism in that reduction of caloric intake, along with the exercise intervention, was not desirable. Subjects were requested to not diet and to adhere to their lifestyle regime, i.e. maintain the same eating habits throughout the study.

d) Fitness Evaluation

Each subject reported to the fitness studio prior to the exercise test for a familiarization session. For all subsequent fitness tests, subjects were instructed not to engage in any strenuous activity for twenty-four hours and to consume only a light meal two to three hours prior to the test. Participants were also requested to refrain from caffeine products and cigarettes during the three hours preceding the evaluation. In total, there were seven fitness tests, numbered herein I-VII in chronological order. Test I was the familiarization session and tests II-VII comprised the fitness results. Prior to the following physical tests,

each subject was weighed on the calibrated Seca scales.

i) Predicted Maximal Oxygen Uptake

The subjects performed a progressive, eighteen minute, three stage cycle ergometer (Monark) test. Maximal oxygen uptake ($\text{VO}_2 \text{ max}$) was predicted from the steady state heart rates at three work loads using an age corrected nomogram (Astrand & Rhyning, 1954). Heart rate was recorded, using a stethoscope and stopwatch, for 30 seconds at the second half of each minute of the test. The predicted maximal oxygen uptake has been expressed in absolute terms ($\text{l}\cdot\text{min}^{-1}$) and relative to body weight ($\text{ml}\cdot\text{kg}\cdot\text{min}^{-1}$).

ii) Body Composition

Skinfold measurements were taken at four sites (biceps, triceps, subscapular, and suprailiac) for calculation of the sum of skinfolds and percent body fat (Durnin & Wornersley, 1974). Measures at each site were taken a minimum of twice until a pair of readings were within 0.5 mm of each other. The average of a paired reading represented the value for a given site.

iii) Flexibility

The sit and reach flexibility test was administered according to standard test procedures.

iv) Strength

The hand dynamometer was utilized to measure right and left grip strength according to standardized testing protocol.

v) Compliance

Compliance to the exercise program was recorded at the exercise studio and in the menstrual journals.

Data Analysis

It was considered of primary importance to establish whether or not the exercise intervention and withdrawal were influential in establishing a training effect and detraining effect, respectively. An Analysis of Variance with Repeated Measurements was utilized to evaluate the significance of changes in the dependent fitness variables: VO_2 ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), % body fat, flexibility and total grip strength. Where a significant difference between means was obtained, the Newman-Keuls Post-Hoc Test was applied. Data relating to performance on the bicycle ergometer: maximum work load achieved; and time at this work load have been presented along with the primary fitness variables of predicted maximal oxygen uptake and percent bodyfat for each subject. The total test results have been inspected for individual exercise responses. After the significance of the training effect was established, the relationship of the exercise intervention to reported changes in PMS symptoms was analysed.

The PMS parameters, the charted symptom changes, provided a second major source of data. The information contained on the visual analogue scales has been translated into graphs and included in the next chapter. Although

there was no statistical analysis of these graphs, they were examined visually to determine differences and extent of differences in the cyclical patterns from baseline throughout the exercise intervention and back to baseline.

The issues regarding the employment of traditional statistics to time-series studies have been explored. According to Kratochwill (1978), statistical tests may be more sophisticated but the relative insensitivity of ocular analysis may be valuable. This source stated that the lack of refinement characteristic of visual inspection was redeemable in that an effect would have to be pronounced or clearly evident before proclaiming the intervention responsible for change. Thus, statistical tests of significance may detect less powerful effects but graphed data must demonstrate convincing modifications. Kratochwill (1978) suggested that visual inspection procedures probably increase the possibility of a Type II error and decrease Type I error probability.

The usual criteria for assessing change through visual analysis are changes in level and trend (slope). This study has generated data which is cyclical in nature and consequently cannot be easily submitted to statistical analyses. The reported symptoms may represent serially dependent data. Secondly, the essence of PMS research, and in particular of this investigation, is the individual. Therefore, the use of statistics which may mask individual effects is contraindicated. The initial purpose of this

study was to determine if any relationship exists between exercise and reported PMS symptoms. Consequently, visual inspection of the data was valuable in looking at individual subject's and symptom's response to the exercise intervention and withdrawal phases.

CHAPTER 4

RESULTS AND DISCUSSION

Introduction

The contents of this chapter have been presented under the following headings: fitness parameters which includes group and subject analyses; PMS parameters; and the relationship between exercise and PMS. The initial section which encompasses the evaluation of the exercise intervention has been addressed from two perspectives. The results from the fitness tests for the group were submitted to traditional statistical treatment and have been presented and explained under the first fitness subsection. The subsequent fitness subsection has examined the six subjects' individual responses to the exercise intervention. In the second major section, the results pertaining to PMS symptomology have been displayed in graphic form. Under the final heading concerning the relationship between PMS and exercise, there is an introduction followed by a case study of each subject. Further discussion with reference to this study and other research completes this chapter.

Fitness Parameters

Group Analysis

The age, weight and height of the six subjects are presented in Table 1 along with the means and standard deviations for these measures. Data from the seven fitness

Table 1
Physical Characteristics
Of The Six Volunteer Subjects

Subject No.	Age (yr)	Weight (kg)	Height (cm)
1	30	59.09	162.56
2	34	49.09	165.10
3	40	68.18	162.56
4	34	52.27	160.00
5	37	64.50	167.64
6	37	66.36	175.26
n = 6			
Mean (\bar{X})	35.33	59.92	165.52
Standard Deviations (+S.D.)	(+3.44)	(+7.84)	(+5.43)

tests, including weight, sum of skinfolds, percent body fat, flexibility, right and left grip strength, resting heart rate, predicted maximal oxygen uptake expressed in absolute terms ($l \cdot \text{min}^{-1}$) and corrected for body weight ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$), and total time on the bicycle ergometer were submitted to the statistical analysis as described in Chapter 3. This treatment of the full data disclosed no significant mean differences in any variable for the seven testing sessions. When the fitness statistics were modified by temporarily removing the variable raw data reported on Subject 3, a repeated statistical analysis of this partial data demonstrated significant differences between means as indicated in Table 2.

Thus, there is evidence to support that five subjects demonstrated a training effect due to the exercise intervention. The results show that there is a significant difference of means between the IVth and IInd fitness tests with respect to maximum volume oxygen uptake. A corresponding significant difference was found between the exercise phase test and the two post exercise tests for this same variable. Thus, this primary indicator of fitness denotes that the exercise phase fitness test IV group mean VO_2 ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) was significantly higher than the indicated pre and post exercise means.

The percentage of body fat showed a significant difference only between Test II and Test VII, thereby indicating that the group mean did not significantly vary

Table 2

Group¹ Fitness Results

Test	II	III	IV	V	VI	VII
\bar{X}	40.15 ^{*a}	42.75	47.36 ^{*abc}	43.88	41.42 ^{*b}	41.75 ^{*c}
(ml.kg. ⁻¹ min ⁻¹)	S.D. 5.49	7.54	4.12	6.72	4.31	3.72
\bar{X}	27.60 ^{*a}	28.12	29.68	29.14	29.18	30.18 ^{*a}
Body Fat (%)	S.D. 3.28	3.42	3.63	3.59	2.51	2.72
\bar{X}	32.00	34.20	34.00	34.20	34.50	33.30
Flexibility (cm)	S.D. 5.49	4.44	5.18	4.70	4.62	3.68
\bar{X}	56.20 ^{*abc}	57.40	58.80	61.60 ^{*b}	62.60 ^{*a}	61.40 ^{*c}
Grip Strength (kg)	S.D. 6.26	10.36	7.19	6.58	8.44	6.50

Key

1 Analysis excludes subject 3's fitness results.

* Significant at level = 0.05.

a, b, c Identical letters indicate the two statistically different means.

with the exercise intervention. The exercise phase means were not significantly different from pre or post values and emphasize that reported PMS symptom changes were not complicated due to exercise-related body weight and composition decreases. There was no significant difference between means for the sit and reach flexibility variable. This indicates that the training effect would appear to have been limited with regard to this measure of flexibility.

The grip strength group means were significantly different from Test II to the last three tests in respective order of significance, VI, V and VII. These significant differences indicate that grip strength improved over the exercise intervention and this parameter of fitness was maintained after peaking one month post exercise withdrawal.

Although the fitness results are varied for the group, the above evidence does support that there was a training effect during the exercise intervention and a coincident detraining response after withdrawal for VO_2 ($ml.kg^{-1}.min^{-1}$). Grip strength results also tended to support the effect of the exercise intervention.

Subject Analyses

The design of this study necessitated the examination of the six subjects from a case study perspective. The complete results of the six fitness tests are individually presented for each subject in Tables 3, 4, 5, 6, 7 and 8. The values featuring asterisks denote missing data which have been estimated by linear regression calculations. Each

Table 3
 Fitness Parameters: Subject 1

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	IG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	59.09	48	26.0	37.5	29	27	70	2.73*	46.20*	10	450	5
III	60.00	53	28.8	38.0	31	31	68	3.13	52.17	13	600	5
IV	59.01	50	28.2	39.5	33	31	66	3.13	52.90	8	600	2
V	56.82	47	27.5	41.0	35	35	70	3.05	53.59	10	600	4
VI	59.55	50	28.2	39.5	36	33	78	2.61	43.83	8	600	2
VII	62.27	52	28.5	37.5	32	33	76	2.61	41.91	9	600	3

Table 4

Fitness Parameters: Subject 2

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	IG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	47.80	37	24.6	25.0	28	22	66	1.74*	36.40*	10	450	4
III	47.72	40	25.5	33.0	26	22	76	1.74	36.46	11	150	5
IV	48.64	54	29.4	30.0	30	20	66	2.26	46.46	10	450	4
V	47.72	60	30.0	30.5	31	25	70	1.74	36.46	8	450	2
VI	49.55	50	28.2	35.0	31	21	70	1.74	35.12	7	450	1
VII	48.18	54	29.2	34.0	30	23	80	1.74	36.11	7	450	2

Table 5

Fitness Parameters: Subject 3

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	LG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	68.64	81	36.9	28.5	28	29	76	2.90	42.40	14	600	4
III	72.27	91	38.4	29.0	28	24	78	2.96	40.93	10	600	4
IV	73.18	95	39.0	29.0	29	31	76	2.96	40.45	10	600	4
V	70.45	96	39.1	30.0	32	30	72	2.44	34.63	15	750	3
VI	71.80	99	39.6	28.0	33	30	75	2.44	33.93	13	750	1
VII	70.90	104	39.7	30.0	32	30	80	2.09	29.45	12	600	6

Table 6

Fitness Parameters: Subject 4

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	LG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	51.80	50	28.2	37.0	24	26	78	2.08	40.15	11	450	6
III	51.80	51	28.2	39.0	27	28	75	2.26	43.63	9	450	5
IV	52.73	53	28.8	39.0	26	26	76	2.61	49.49	11	450	5
V	53.18	55	29.4	37.0	27	27	78	2.26	42.53	11	450	6
VI	53.18	60	30.6	37.5	27	28	76	2.35	44.19	10	450	4
VII	53.18	60	30.6	35.5	28	28	90	2.35	44.17	8	450	6

Table 7

Fitness Parameters: Subject 5

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	LG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	68.10	73	33.0	32.5	34	29	73	2.26	33.19	13	600	2
III	66.40	75	33.4	33.0	35	28	80	2.26	34.04	10	450	4
IV	66.40	90	35.8	33.5	36	29	70	2.78	41.86	15	600	3
V	65.90	79	34.3	32.5	36	27	80	2.61	39.61	10	600	4
VI	67.27	71	32.7	33.0	37	30	78	2.61	38.79	8	600	1
VII	68.34	83	34.7	31.5	38	30	70	2.78	40.74	10	600	4

Table 8

Fitness Parameters: Subject 6

Test	Wt (kg)	SS (mm)	BF (%)	Flex (cm)	RG (kg)	LG (kg)	RHR (bpm)	VO ₂ (l)	VO ₂ (ml/kg)	Time (min)	MWL (kpm)	@
II	64.09	42	26.2	28.0	32	30	72	2.87	44.80	16	600	4
III	60.45	38	24.7	28.0	35	34	75	2.87	47.47	12	750	3
IV	62.27	42	26.2	28.0	31	32	78	2.87	46.09	13	750	3
V	62.73	37	24.5	30.0	32	33	78	2.96	47.20	12	750	2
VI	65.45	42	26.2	27.5	39	31	80	2.96	45.19	13	750	1
VII	64.55	49	27.9	28.0	35	30	84	2.96	45.82	14	750	2

of the subjects exhibited variable physical, metabolic and cardiovascular responses to the exercise program. A complete fitness profile on each subject has been included in subsequent case studies. Generally speaking, all subjects except Subject 3 fulfilled to varying degrees of different variables, the expected training and detraining patterns. During the exercise intervention, $\dot{V}O_2$ ($l \cdot \text{min}^{-1}$) and $\dot{V}O_2$ ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) reached their apex for most subjects. An exercise associated weight loss was observed for only one woman (Subject 6); all other subjects either maintained or gained weight. Body composition assessments illustrated a similar pattern as above with only Subject 6 reporting an apparent training-related decrease in percent body fat while others maintained or increased in this area. A general conditioning due to the exercise program was evident for all subjects when the following responses were considered in combination: increased time to exhaustion, elevated final work load stage, and prolonged time at higher work loads. Moreover, the series of heart rate recordings indicated a training effect for most subjects. Flexibility and strength generally increased during the exercise program. Thus, from the data as presented in the Tables, the salient feature is that a training effect occurred for all subjects. The pattern of conditioning was different across subjects and variables. The strength of these changes and the reinforcement of the intervention as a treatment modality

will be discussed with reference to individual PMS symptom profiles.

Nutriprofile Information

The six subjects exhibited a Nutriprofile dietary intake pattern similar to age-matched Canadian women (Nutrition Canada, 1976). According to nutritionist Grunau (1985), these subjects' diets were low in iron, folacin, calcium and vitamin D, and percent fat of total food ingested was higher than recommended. Specific dietary information regarding caloric intake has been included in the case studies at the end of this chapter.

Generally, there were slight fluctuations in diet with reference to the intake of food, measured in kilojoules and the percent value for the three main nutrients; carbohydrates, protein and fats. There was no indication from these parameters that any subject was restricting energy intake or modifying eating patterns to reduce body weight except for Subject 1 who reported a mid-exercise intervention reduction in kilojoules ingested. This particular subject did not experience weight reductions during the exercise program. Thus, the diets of these six women were not significant factors in that there were no serious deviations. The exercise intervention was not compromised by dietary changes.

PMS Parameters

The results of the prospective, longitudinal charting

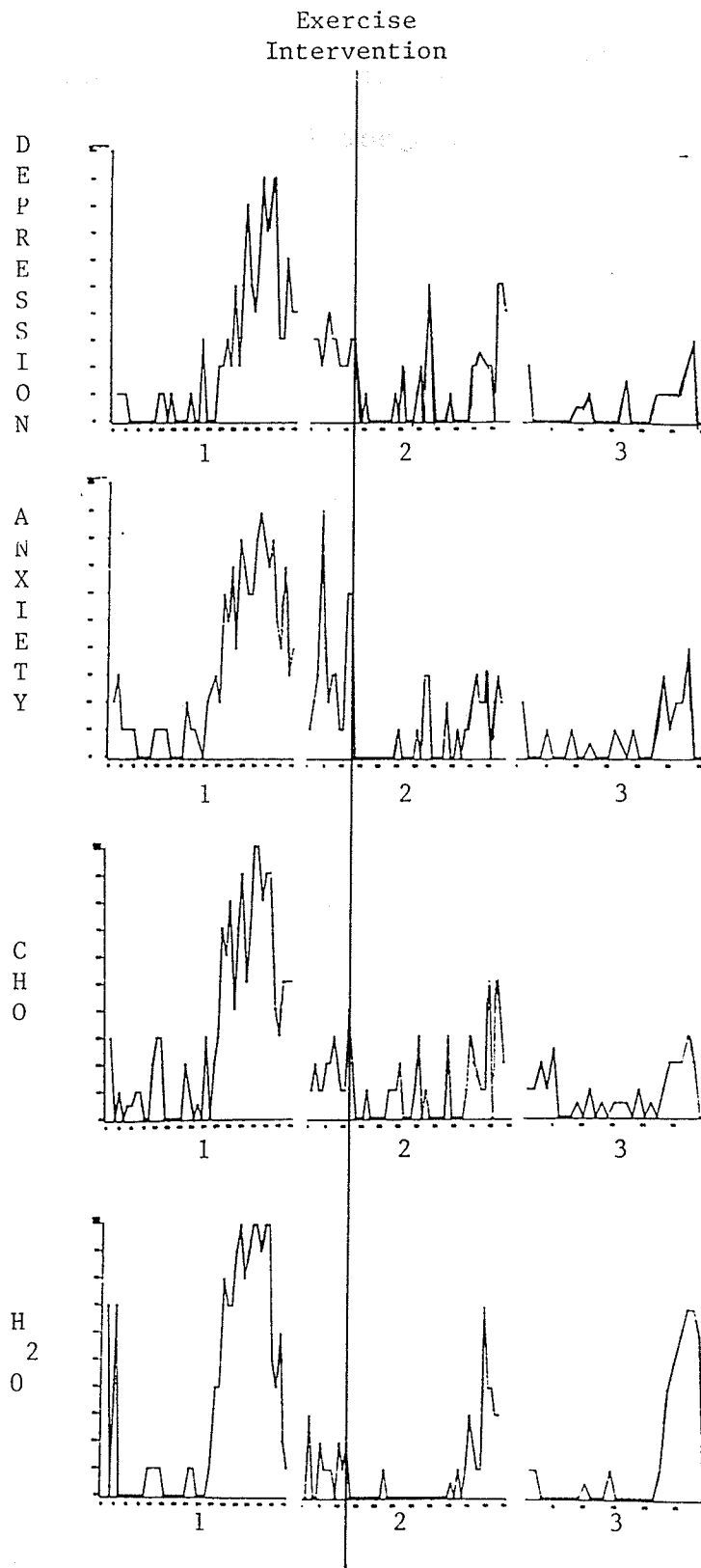
for the PMS symptoms, depression, anxiety, cravings and water retention, are presented in graphs in Appendix K. This set of graphs has been arranged so that a single page includes the four symptoms per subject per cycle, in chronological order. A complementary representation of reported PMS symptoms has been inserted in this section. The original graphs were reduced and collated according to symptom, cycle and subject and are displayed sequentially. These graphic portrayals of the longitudinal PMS self ratings are contained in Figures 2, 3, 4, 5, 6 and 7. Exercise intervention and withdrawal have been demarcated. The time axis was purposely not rendered continuous so that the variability in cycle length and consequently the number of cycles over the study's duration would be emphasized. The cycle lengths varied from 12 days to 55 days and the minimum number of cycles was 5 whereas the maximum number was 8. The normal expected values of 28 day cycles with 7 to 8 over the investigation were more commonly reported yet there was evidence of sporadic cycle length within individuals as well as between individuals.

There was huge variance with respect to the individual's vulnerability to certain of the four symptoms and the range of ascribed ratings. Although the establishment of a constant baseline is important to time-series research, the consistent menstrual pattern may evade personal definition as well as observance. The study criteria of two menstrual cycles or two lunar months appeared to be sufficient as the

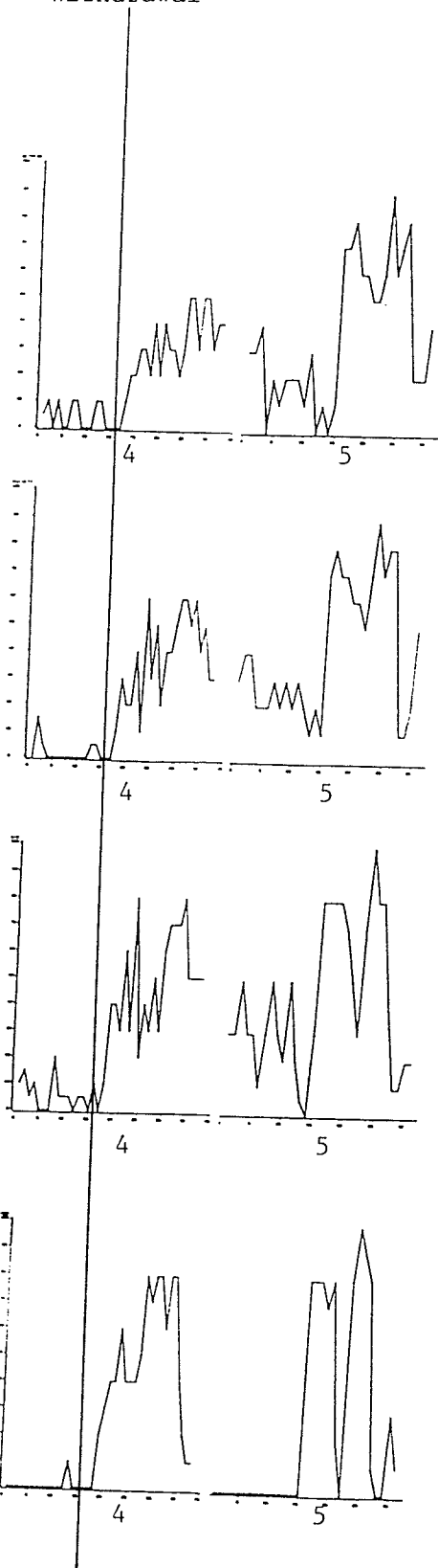
Figure 2
Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales

Subject 1

Figure 2. SUBJECT 1



Exercise
Withdrawal



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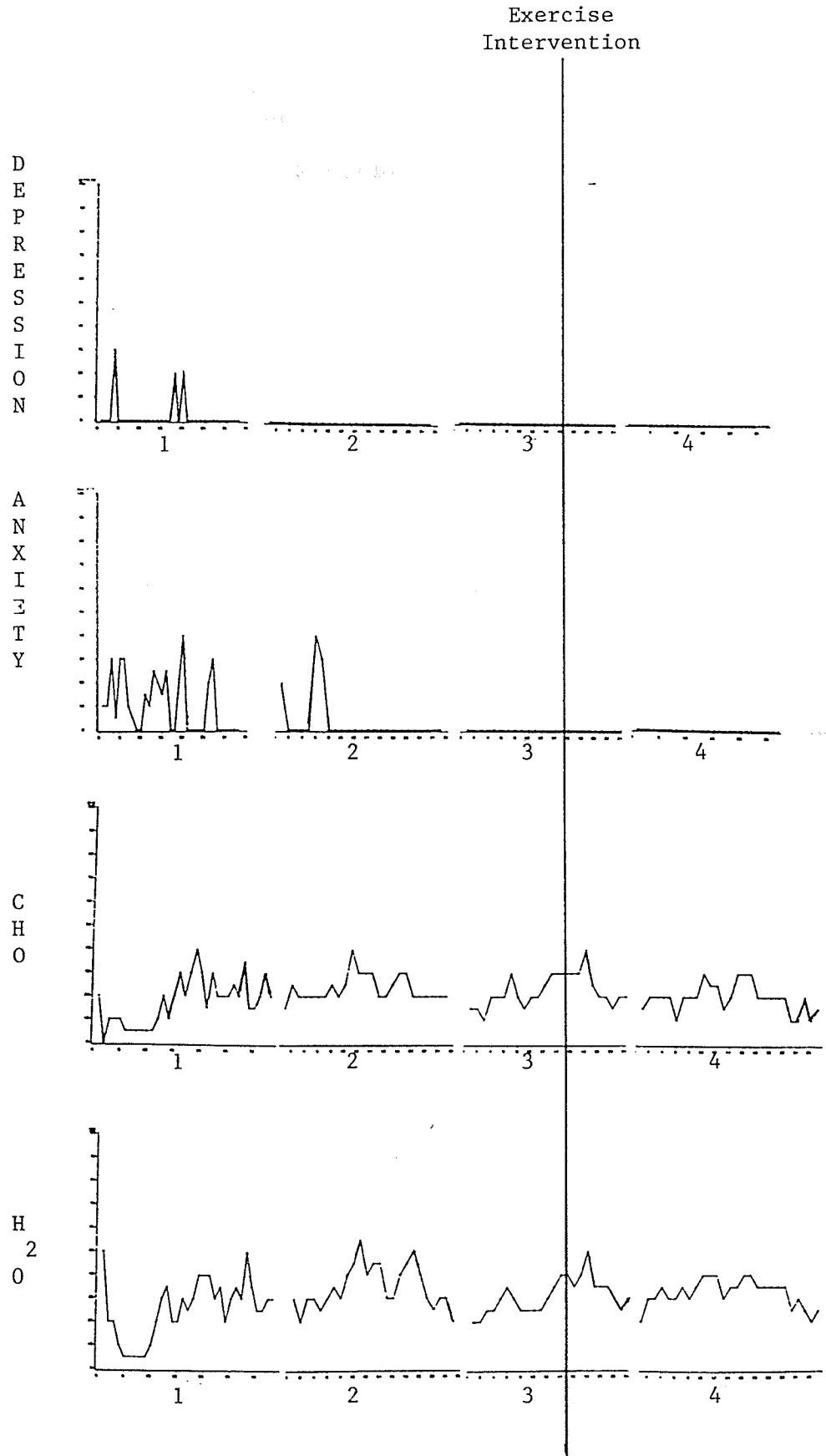
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Figure 3

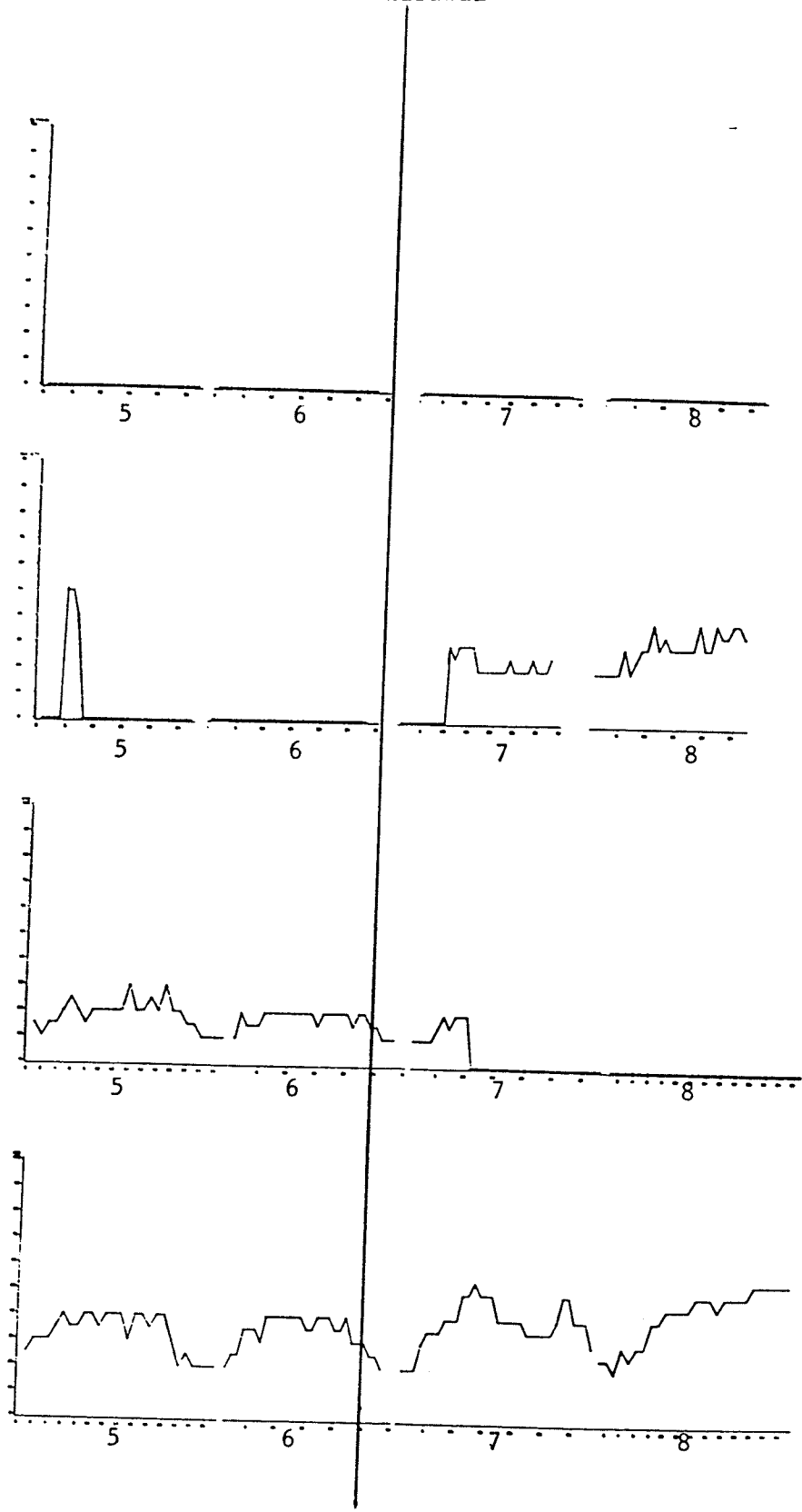
Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales

Subject 2

Figure 3. SUBJECT 2



Exercise
Withdrawal



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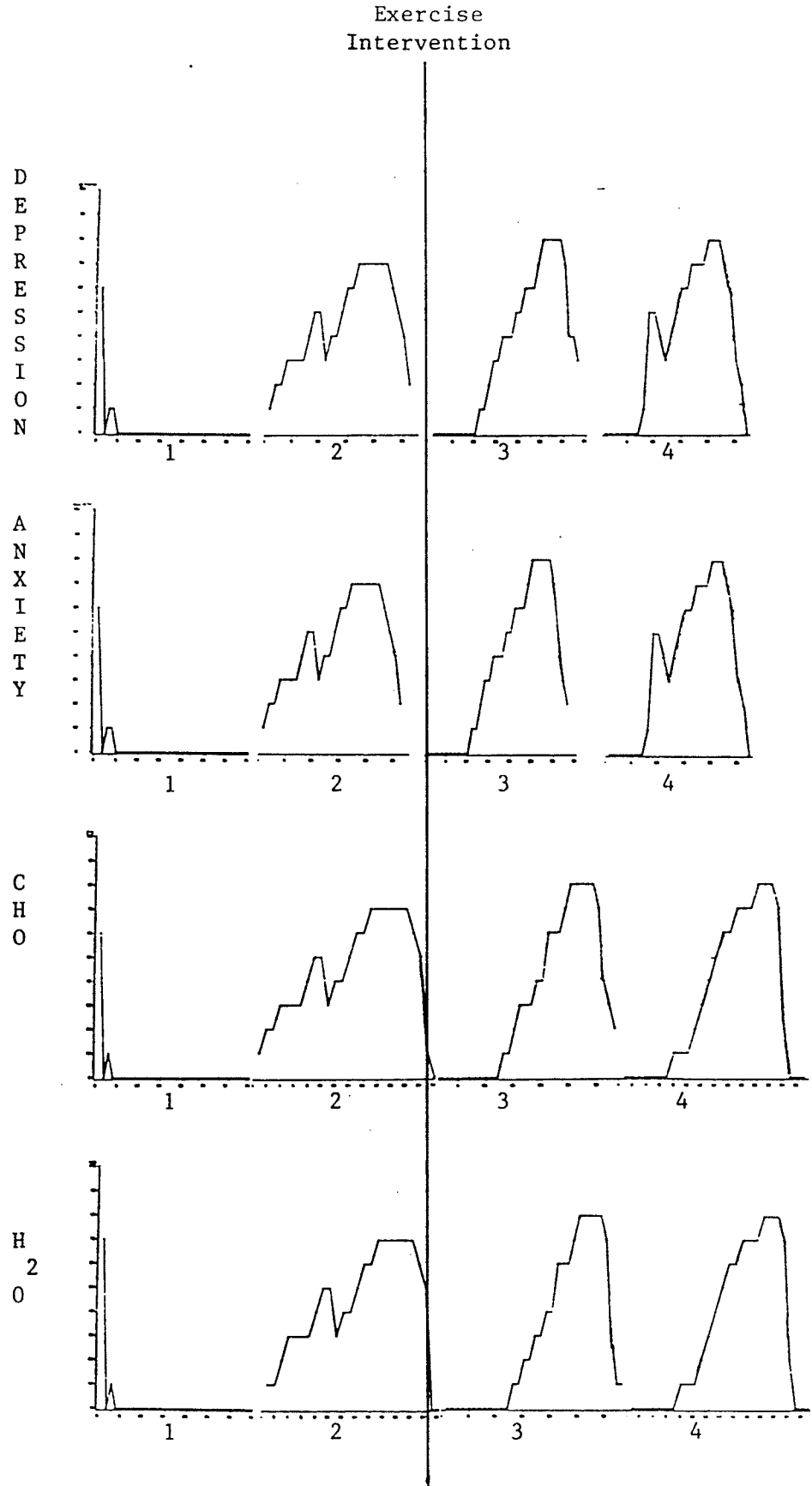
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Figure 4

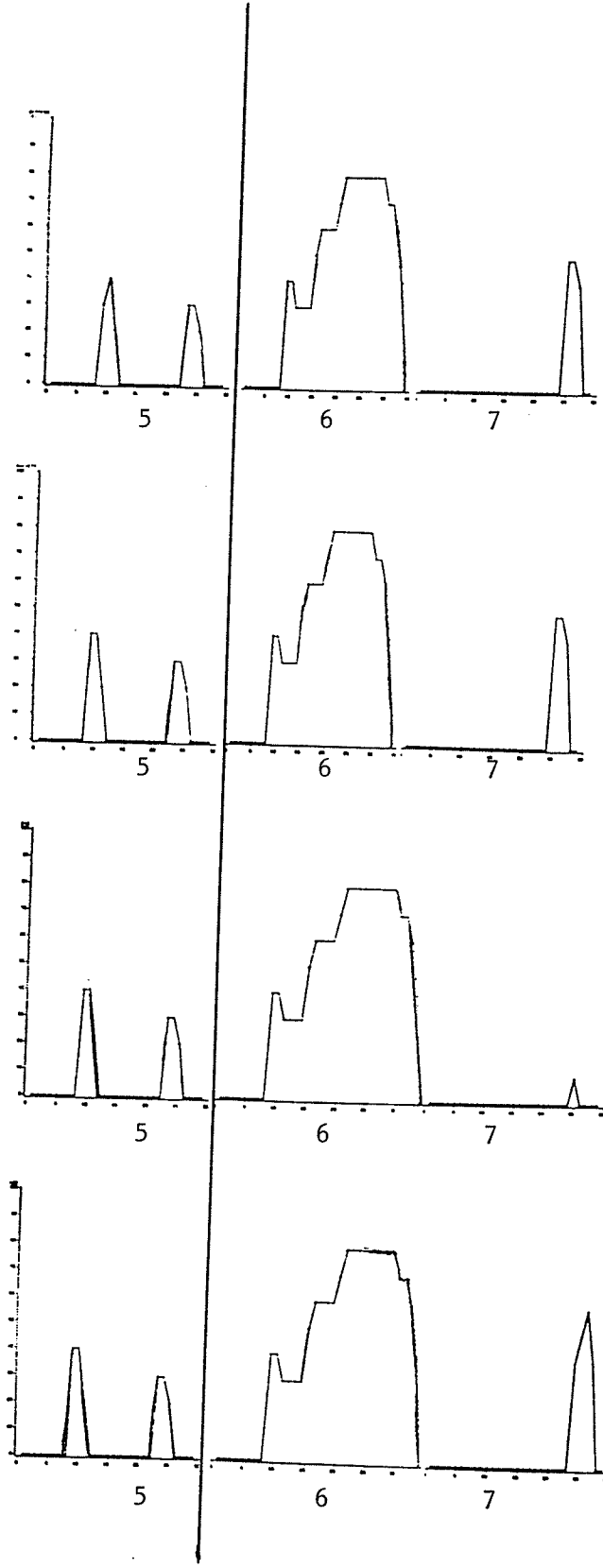
Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales

Subject 3

Figure 4. SUBJECT 3



Exercise
Withdrawal



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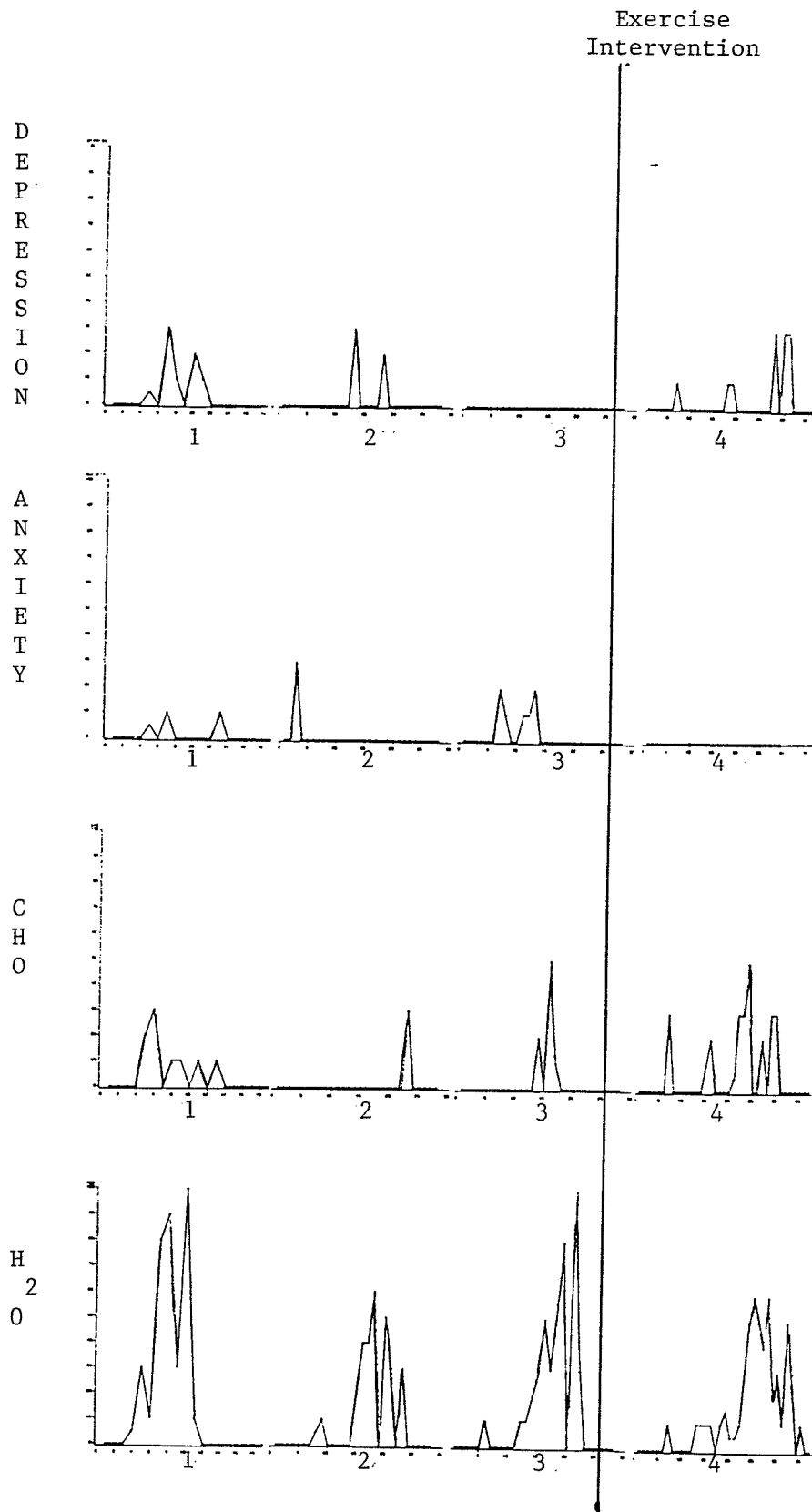
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Figure 5

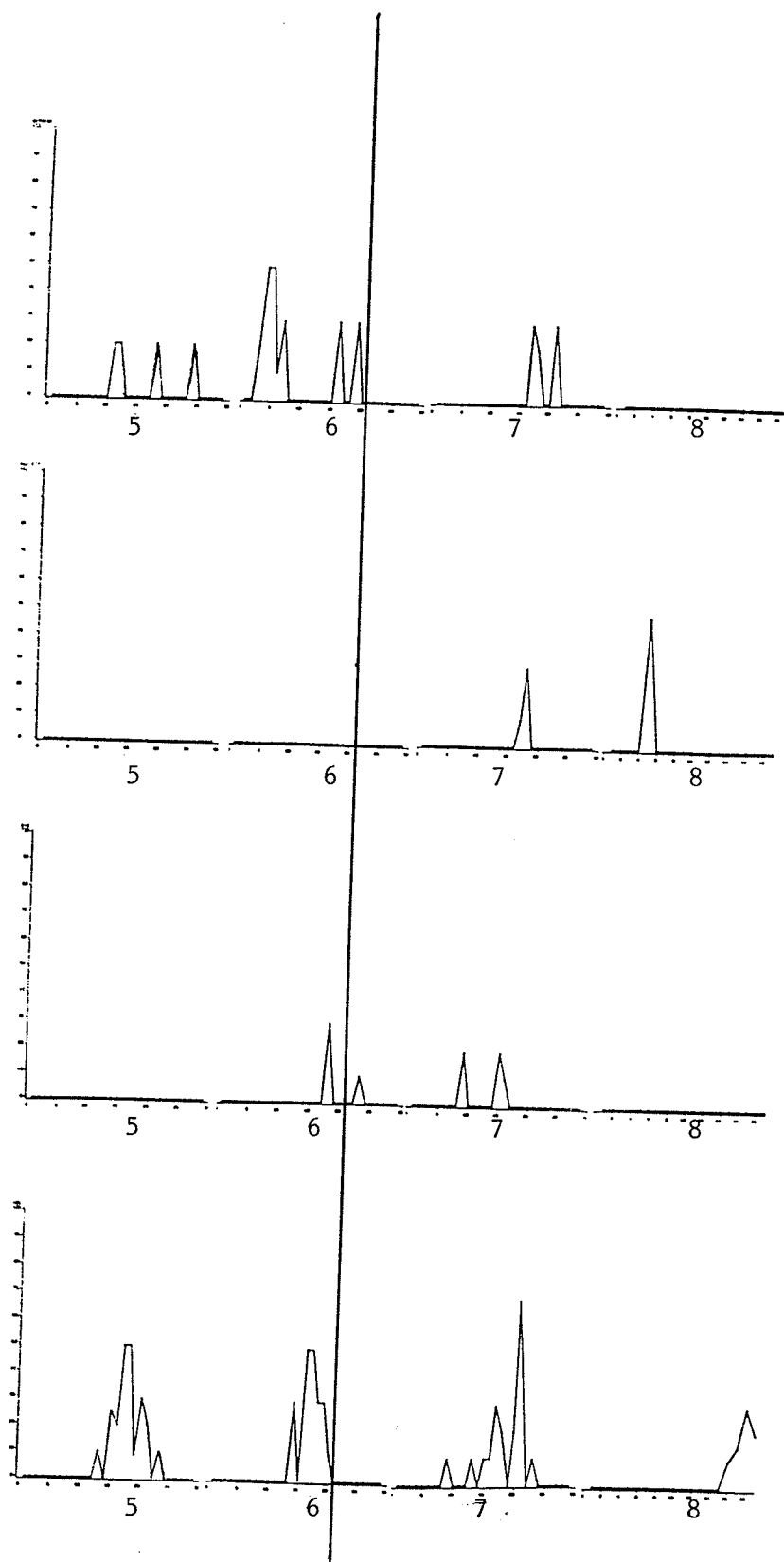
Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales

Subject 4

Figure 5. SUBJECT 4



Exercise
Withdrawal



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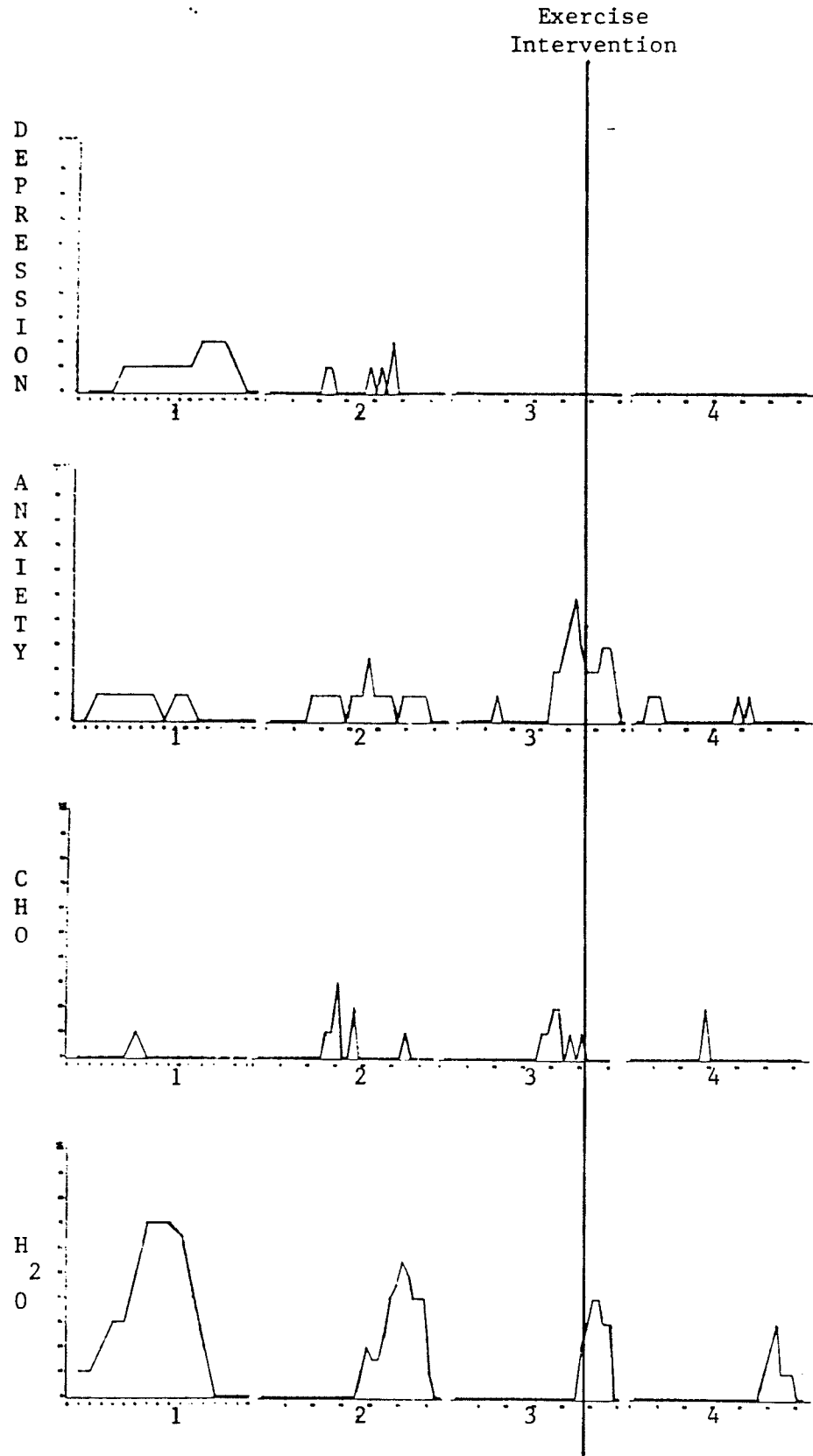
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Figure 6

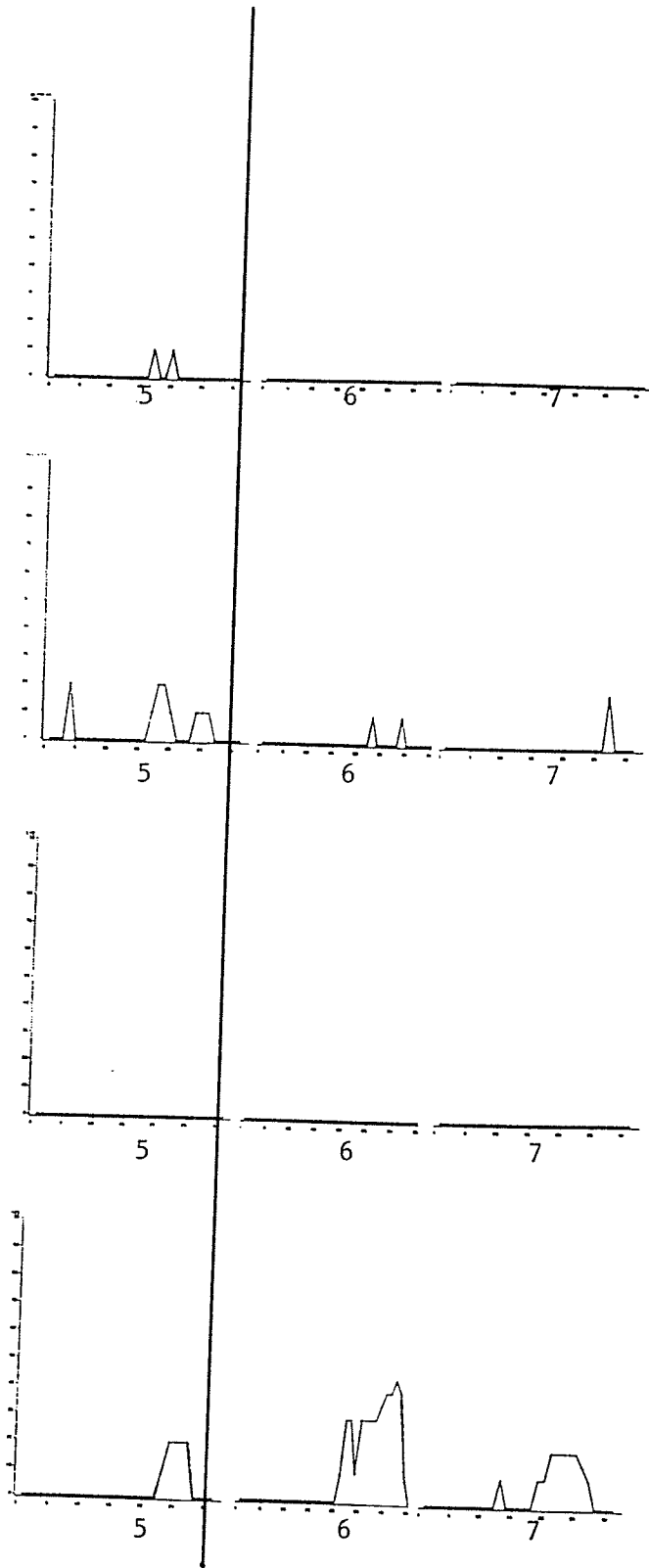
**Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales**

Subject 5

Figure 6. SUBJECT 5



Exercise
Withdrawal



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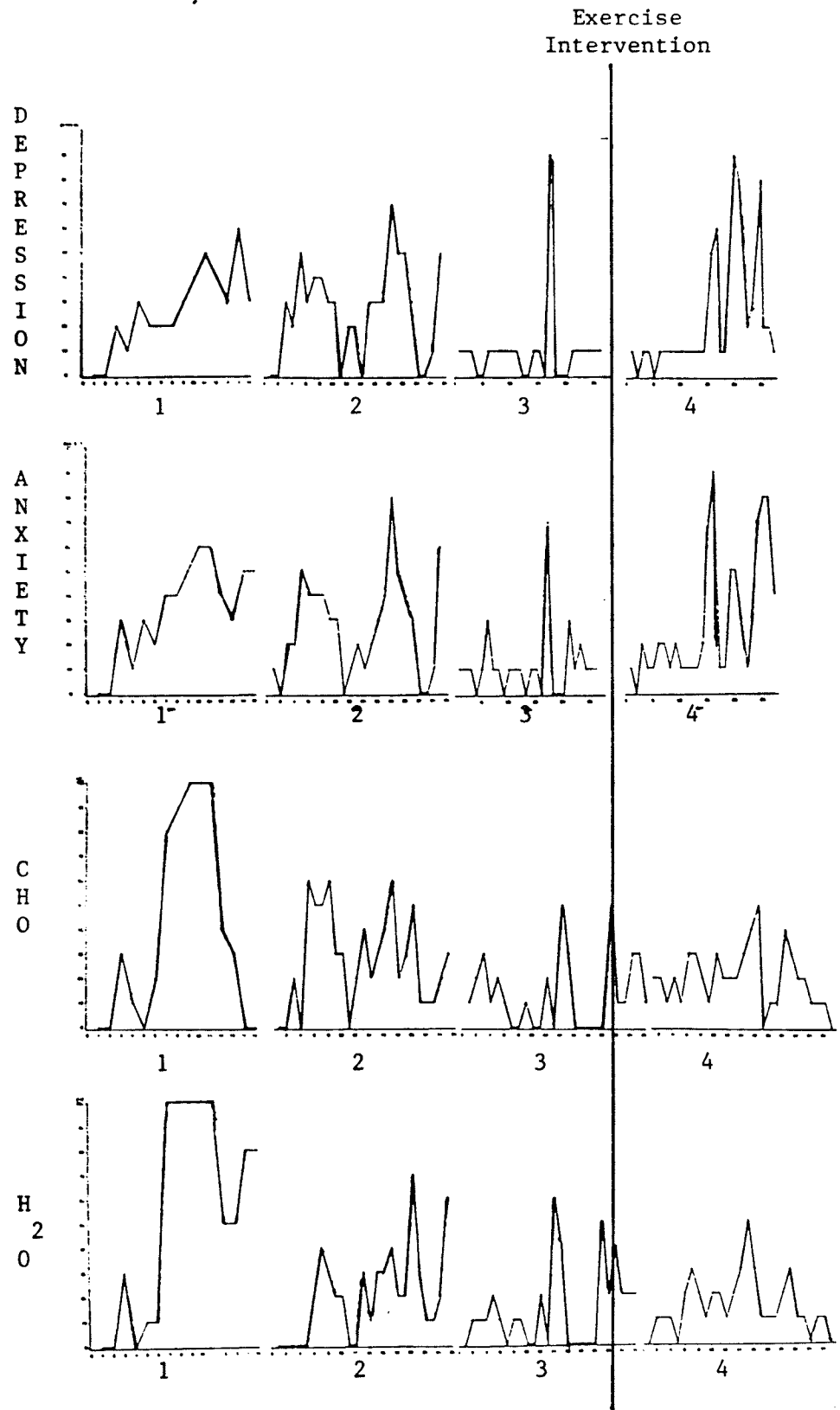
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Figure 7

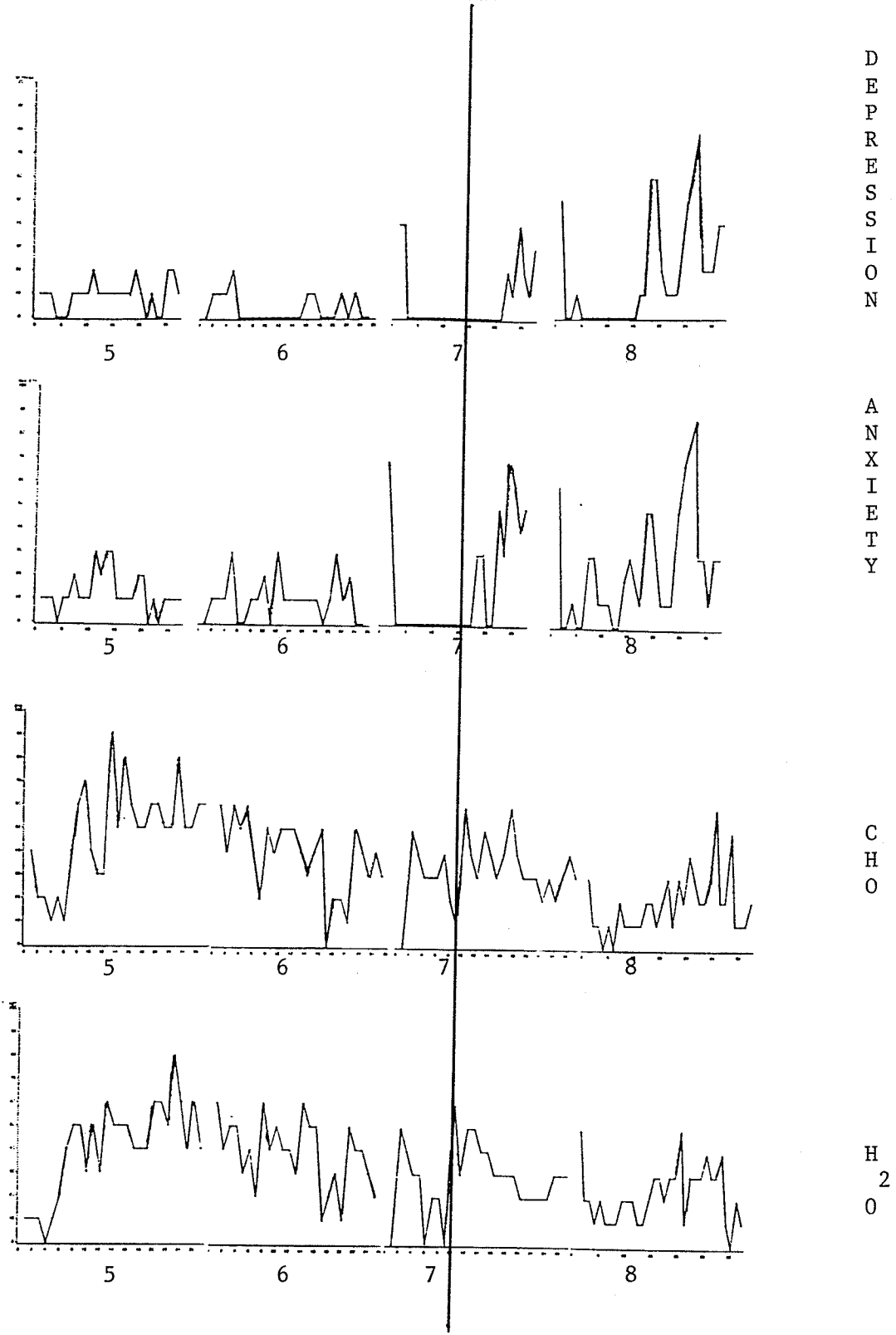
Longitudinal PMS Self Ratings
From 100 mm Visual Analogue Scales

Subject 6

Figure 7. SUBJECT 6



Exercise
Withdrawal



subjects were previously diagnosed PMS patients. Generally, there were perceived changes associated with intervention and withdrawal of exercise. A detailed visual inspection for each case study is subsequently presented but there are several noteworthy trends evident in the graphs. Exercise intervention cycles are generally characterized by an attenuated or more fragmented pattern of charting. In looking at the configuration it is evident that, in most instances, the exercise program either reduces the level of the reported symptom, alters the shape by inserting more asymptomatic intervals and/or reduces the length of symptomatic time. There appear to be different times during the exercise intervention for individual subjects to illustrate the maximum alleviation of symptoms. Several subjects reported an exercise associated increase for the carbohydrate cravings symptom. With the withdrawal of exercise, the symptoms ratings followed several patterns. Commonly, the configuration especially with reference to height neared baseline levels within two months. There were several obvious examples of the reduction of symptoms' ratings continuing through to the last fitness test. Further introspection regarding individual responses is the focus of the next section.

The Relationship between Exercise and PMS

Introduction

The focus of this multiple N, single I research has

been the six individuals' responses to the exercise and PMS parameters. The following case studies represent a concise translation of the salient changes recorded for each subject. Pertinent evidence from the fitness tests, the Nutriprofile analysis, the self-report longitudinal PMS graphs and anecdotal survey excerpts have been collected to produce individual profiles. Compliance to the exercise program has also been recognized as contingent to the individual conditioning responses (Appendix N).

These six case studies are presented and discussed in terms of the criteria listed immediately above as well as the hypothesized relationship between exercise and the attenuation of PMS symptoms.

Case Study: Subject 1

Subject 1's data, presented in Table 2, offered evidence of a training response to the exercise intervention. Both predicted maximal oxygen uptake values demonstrated increments coincident with the exercise program. Relative predicted maximum VO_2 increased 15.9% from Test II to Test V. Body weight and percent body fat parameters indicated slight reductions at Test V. These modifications cannot be wholly attributed to a training effect as the Test IV Nutriprofile results documented a changed food consumption pattern. This dietary recall computed a substantially lower amount of kilojoules ingested and revealed an obvious reduction in percent of total food intake. Consequently the control mechanism for diet and body weight registered

modifications which may have slightly confounded the exercise intervention. This subject recorded the lowest average number of classes attended per week (2.33) for the exercise program.

The self-rating PMS journal relayed the following menstrual cycle information. Menstrual cycle length was erratic with 45 days being the longest interval. The remaining cycles were longer than normal and resulted in the occurrence of only five full cycles for the study's duration. All four symptoms were equally relevant to Subject 1. Visual inspection of the longitudinal graphs (see Figure 2) showed trends which support the hypothesis of alleviated PMS symptoms related to the exercise intervention. All factors of configuration, height and length of reported symptoms displayed obvious modulations during the exercise program. A reversal of these effects was witnessed with exercise withdrawal, and the fifth cycle registered approximate baseline patterns. According to the anecdotal survey, this subject perceived participation in the exercise program to have positive, damping effects on her PMS symptoms and she expressed a desire to return to a similar program upon completion of the study.

Due to the length of the first menstrual cycle, Subject 1's symptoms are not tightly clustered in the luteal phase. For example, cycles 1, 3 and 5 were 45, 30 and 31 days, respectively. However, there are discernible differences between the exercise configurations non exercise patterns.

For all four symptoms, the exercise patterns were substantially modulated. Ratings of three symptoms during the exercise phases were of considerably reduced amplitude than pre- and post-exercise ratings. This disparity was less noticeable for the water retention symptom. Moreover, the exercise configuration illustrated that ratings tended to be consistently low for longer intervals. Similarities between the pre- and post-exercise configurations also emphasize the reduction of symptom interference in the exercise phase. These obvious differences provide further evidence to relate the exercise intervention to alleviation of PMS symptoms.

Case Study: Subject 2

The training response displayed in Table 3 for Subject 2 was moderate. There was a 27.6% increase at Test IV in VO_2 relative to body weight. Body weight and percent body fat fluctuated throughout the study but the fitness tests during the exercise intervention did not reveal a pattern of reduced values for these physical parameters. The Nutriprofile analyses revealed slight modifications in food consumption patterns with stable levels of kilojoules ingested before and during the training period. The final dietary recall noted a significantly reduced intake of food.

Records showed that this subject attended an average of 2.5 classes per week. This subject experienced seven complete menstrual cycles with the eighth cycle terminated

by the study after 27 days. Cycle lengths were within normal ranges.

The conditioning effect of the exercise intervention was related to the attenuation of the self-reported PMS symptoms (see Figure 3). Depression was not a symptom which appeared to typify Subject 2's PMS dysfunctions and was regularly perceived as dismissable. Both anxiety and water retention were reportedly reduced to varying degrees during the exercise program and returned to near baseline levels with absence of exercise. The symptom of carbohydrate cravings showed a slight damping effect during the intervention with an interesting elimination after exercise withdrawal.

The exercise cycles were reported asymptomatic for anxiety whereas it was rated moderate during the pre- and post-exercise cycles. The uncharacteristic isolated spike in cycle 2 was not matched by cycle 8's gradually heightened ratings. Carbohydrate cravings appeared to become more manageable during exercise but were completely eliminated as an aggravating factor in the post exercise ratings. The final symptom of water retention illustrated that the exercise ratings were generally depressed and consistently lower than the other two cycles of ratings. The greatest discrepancy is evident prior to menses where post exercise values received the maximal ratings from Subject 2. In summary, the ratings of the three PMS symptoms for the three selected cycles showed varying moderation of symptoms from

baseline levels to exercise levels. This subject reported positive responses on the exercise survey and generally recognized the personal benefits of exercise-related PMS changes.

Case Study: Subject 3

As demonstrated by results in Table 4, a typical training response was not observed in this subject. The primary variables of predicted maximal oxygen uptake in Tests III and IV were high but did not attain the initial test level. There was a decrease of 15.4% for VO_2 ($ml.kg.^{-1} min^{-1}$). Uncharacteristic increments in body weight and percent body fat reported during the exercise program have nullified measures of fitness gains. Fitness level may be postulated to have increased during the exercise program on the basis of increased time to exhaustion and longer performance times at higher work loads recorded for the three exercise program fitness tests. The elevated values for the variables of body weight and percent body fat could not be directly associated with changed dietary regime. Information from the Nutriprofile analysis revealed fairly constant amounts of kilojoules ingested and no variation in relative nutrient percentages except a reduction of carbohydrates and increase in fats two months post exercise. Consequently, dietary changes reportedly did not confound the exercise intervention. However, the changes in body weight and body composition reduced the strength of the exercise intervention. Compliance to the exercise program

was average with 2.5 classes attended weekly.

Menstrual cycles were consistently within normal range limits with a total of seven complete cycles over the study's duration. The longitudinal graphs displayed in Figure 4 visually demonstrated a change in length and configuration of all symptoms, progressively through the three months of intervention. Upon withdrawal of the exercise program, Subject 3 initially rebounded to exceed baseline patterns then evidenced a curious asymptomatic interval at the beginning of the final month post exercise. Another unique trend was observed in the level of symptoms with the intervention application. All four symptoms were recorded as being higher than baseline, however, the duration of symptomatic periods was decreased and the heights were considerably depressed during the final exercise month. According to the anecdotal survey, this subject perceived exercise to have some positive alleviating effects but reported highly variable monthly patterns. This individual's variability witnessed herein is supported by PMS literature. Subject 3 did not relate that exercise was worsening her symptoms but accounted for the 'bad months' as a personal PMS pattern.

Subject 3 reported a distinct amelioration of the depression symptom during the exercise phase. The ratings were substantially lower and less frequent than the non exercise phases although one isolated, preovulatory elevated rating was recorded. This heightened rating near ovulation

occurred for all four symptoms and has been accepted as one possible pattern of PMS (see Appendix C). Anxiety, carbohydrate cravings and water retention symptoms displayed results similar to those for depression. A considerable reduction of the appetite-related symptom in the luteal phase of the post exercise cycle is also noted. Subject 3 did not manifest typical fitness parameter results but according to these graphs has reported modulated PMS ratings congruent with the research design. This anomaly was addressed in the literature review and signifies the clinical considerations of exercise intervention with the perceived ratings of PMS symptoms.

Case Study: Subject 4

Table 5 contains fitness results for this subject. The VO_2 max expressed relative to body weight showed an increment of 23.3%. This, along with evident augmented capacity to accomplish more work, suggests that a definite training response was observed. Body weight and composition measurements did not markedly change during the intervention phase. According to the Nutriprofile analysis, Subject 4 registered stable patterns and amounts of food consumed with a reduction in kilojoules recorded at the termination of the study. Compliance to the exercise program was an average of 2.5 classes per week.

There were seven complete menstrual cycles and a partial eighth one of 19 days during the research with cycle length

within normal ranges. All symptoms were charted with water retention being the most obvious PMS impairment. Longitudinal graphs of PMS self-ratings are presented in Figure 5. During the exercise phase, all symptoms except depression displayed noticeable attenuation with regards to configuration, length and height of symptomatic periods. Anxiety and carbohydrate cravings were alleviated to such a degree that totally asymptomatic months were reported. The uncharacteristic exacerbation of depression during the final exercise month was synchronous with the death of Subject 4's father. The factor of depression was rated sporadically throughout the exercise cycles. When the initial high ratings were excluded, the luteal clustering of depression ratings demonstrated similar patterns for all three cycles. This finding may be rationalized as a unique individual response to initiation into the exercise program or may simply be coincidental. The analysis of anxiety yielded more typical results with the exercise cycles recording constant null ratings whereas the non exercise cycles displayed asynchronous isolated rating spikes. Carbohydrate cravings were rated as initially high and more frequent at the first exercise cycle but symptoms were almost eliminated in both successive exercise cycles.

Water retention was a more discriminative factor in Subject 4's PMS profile as indicated by the complex series of ratings illustrated in Figure 5. There was a slight peri-ovulatory rise in symptoms, and definite clusterings

were manifested in the mid-luteal phase. The exercise cycles were characterized by lower maximal ratings, with less frequent elevations than the pre-exercise configurations. This similar pattern was initially imprinted and minimized for the post exercise cycles although an isolated maximum rating was recorded toward the end of the luteal phase in cycle 7. The responses of Subject 4 generally substantiated a relationship between the exercise intervention and alleviation of symptoms.

The withdrawal of the exercise intervention elicited patterns trending to but not achieving baseline except for the anxiety symptom. According to anecdotal responses, the exercise intervention improved PMS symptoms, especially water retention, and generally resulted in a more positive management of PMS.

Case Study: Subject 5

The fitness variables as tabulated in Table 6 supported an obvious conditioning response. There was a 26.1% increase in VO_2 max ($ml.kg.^{-1}min^{-1}$). Body weight and composition were slightly higher during the mid-exercise phase. Modifications were noted in the final Nutriprofile analysis: the percentage of carbohydrates and protein increased with a concomitant decrease in fats, and the total amount of kilojoules consumed was increased. These changes did not directly affect the exercise phase and, thus, the intervention effect was recognizably the exercise program. The demonstrable training effect may be partially accounted

for by the high compliance of Subject 5 who attended an average 2.92 classes weekly.

Seven complete menstrual cycles were reported with an eighth of 12 days terminated by the research design. Cycles were normal length. Visual analysis of the graphs comprising Figure 6 was conducted. All four symptoms were charted and the exercise intervention demonstrated a progressive moderating effect overall with respect to configuration, height and symptomatic duration. Upon exercise withdrawal, the symptoms of depression and carbohydrate cravings remained unacknowledged. Water retention tended to approach baseline, but to a reduced degree. A classic relationship between PMS and exercise was characterized by Subject 5's rating of water retention symptoms. According to survey responses, this subject perceived exercise to be beneficial in alleviating her PMS symptoms.

Depression was modulated during exercise intervention in terms of height, frequency and length as compared to pre-exercise ratings for Subject 5. The post exercise cycles received constant null recordings. The anxiety ratings discerned generally moderated exercise configurations with one initial elevation coincident with the intervention. There was a similar dampened pattern of post exercise ratings with an immediate premenstrual heightened rating. As illustrated in the longitudinal graphs, the cravings symptom was definitely alleviated for exercise and post exercise cycles. The water retention tracings demonstrated that the

exercise cycles were substantially less symptomatic in terms of height, frequency and duration than either of the non exercise phases. The post exercise cycles again showed evidence of possible residual training effects as the configuration did not nearly achieve pre-exercise levels or form. Thus, Subject 5 displayed patterns which reinforce the association of minimized PMS dysphoria with the exercise program.

Case Study: Subject 6

Fitness results for this subject are presented in Table 7. The exercise intervention appears to have elicited a positive, incremental training response marked by a 5.4% in VO_2 ($ml.kg.^{-1}min^{-1}$). Data on the primary variables showed significant increases as well as an accommodation to higher work loads. However, body weight and composition reflected significant decreases over the training phases. The Nutriprofile analysis denoted increased kilojoule intake in the mid-exercise phase with a corresponding reduction in percentage of carbohydrates consumed. Thus, this subject's dietary regime was altered but the increased energy intake indicated that body weight reductions were not primarily influenced through a purposeful weight-reduction diet. Thus, the exercise intervention may have accounted for the weight losses and corresponding reduced values for body composition. The effect of the exercise program may have been reinforced for this subject due to her attendance

record averaging 2.67 classes per week and more notably four classes per week in the first month of training where weight loss was most marked. Illness reportedly affected regular attendance in the last month of exercise.

Subject 6 experienced eight complete menstrual cycles and a ninth one of 12 days, when charting concluded with the study's termination. Menstrual cycle length was normal. Visual inspection of the PMS longitudinal graphs, displayed in Figure 7, indicate a discernible response to exercise intervention for all four symptoms. Depression and anxiety ratings were progressively reduced during the exercise phase except for two isolated, synchronized spikes at the beginning of the seventh menstrual cycle. Occupational-related stress was cited as a factor in the menstrual calendar. Both depression and anxiety configurations tended to baseline patterns upon exercise withdrawal. The related symptoms of carbohydrate cravings and water retention showed initial attenuation with exercise intervention but exceeded baseline values with reference to length during the remaining two months of exercise. The configurations were sporadic and, although they did not supercede the severe ratings of baseline, they demonstrated reduced asymptomatic intervals. Upon exercise withdrawal these two symptoms were markedly reduced, receiving less than baseline ratings. Thus, the training program appears to have variable effects on this subject's PMS symptoms. Although the anecdotal responses were favorable with regard to exercise benefits, and this

subject evidenced an exercise associated weight loss, the ratings of these last two symptoms do not reflect a related amelioration except in the first month of the exercise program. The modifications in diet, perhaps related to activity-induced appetite increases, may account for this contradiction.

As depicted in Figure 7, depression was rated by Subject 6 as progressively reduced in height, frequency and duration of symptomatic interference during the exercise cycles. The pre-exercise configurations were rated as most symptomatic although the prolonged post exercise luteal phase of cycle 8 reported extreme values. The anxiety factor yielded conclusions similar to those concerning depression. There were no noticeable modulations of carbohydrate cravings in the exercise phase, whereas the post exercise cycle presented evidence of slight alleviation. Similarly, water retention interference was reported to be slightly exacerbated throughout the exercise cycle with the most noticeable improvements in post exercise cycle 8. These last two symptoms displayed variable ratings throughout all three selected cycles and the cluster of ratings was not obvious. This observation may be due to these two symptoms being aggravated by exercise as well as the evidence of premenstrual magnification of all four symptoms (Harrison, 1984). Moreover, the extended length of menstrual cycle 8 added to this cluster absence with prolonged luteal ratings. Subject 6 displayed variable ratings of PMS symptoms

associated with the exercise intervention. Depression and anxiety were demonstrably alleviated but the reverse was true for the remaining two symptoms.

Discussion

Exercise Considerations

The exercise intervention and fitness test sessions yielded some concerns beyond what has been previously discussed. The six subjects were not physically active women and their fitness levels at the preliminary test session confirmed this fact. There was only one subject who was comfortable with the bicycle apparatus and test. Others displayed anxiety more so towards the management of cardiovascular demands rather than the muscular discomforts of the exertion.

Although target heart rates according to the Karvonen method were the basis for intensity during the beginner's class, the monitoring of such during classes was not uniformly observed. Consequently, the effort expended during classes depended largely upon the individual's motivation and would directly affect the total training response. The compliance to the exercise program documented a tapering off trend, especially in the final month. This trend was supported by the fitness results which illustrated that Test IV was generally where most fitness peaks were achieved. A maintained or slightly reduced fitness level was characterized in Test V results. Thus, a 10 week exercise program may be

more efficiently employed especially where an ABAB design is considered.

The exercise modality of aerobics, originally viewed as motivating and attractive, fulfilled the criteria for inducing training responses. However, the questionable area of intensity and duration during classes prompted the suggestion that other exercise modalities may be more effectively employed and monitored. A walk-jog program might result in more specific quantifiable measures. The impact of the socialization factors present in the aerobic workouts should not be overlooked. Eickhoff et al. (1983) pointed out the positive implications for self-esteem through an aerobic dance program. The exercise intervention in this study, coupled with the control for diet, resulted in varying degrees of training responses.

Menstrual Cycle Variance

This study has raised concerns similar to those found by other investigators. The duration and frequency of menstrual cycles was instrumental in the sequential application of intervention, necessitating modification of the experimental design to adapt to individual menstrual cycle variances. The variations observed in this study were not considered abnormal as Sperryn (1983) proposed parameters to define normality but admitted that great differences occur and are accepted as normal. Examples of inter-individual and intra-individual variability were observed within this study.

The differences in menstrual cycles as documented herein may be due to varying components comprising endocrine profiles: levels of the sex steroids, gonadotropins and their precursors. The cyclical, pulsatile secretion of these substances and their complex, highly sensitive, often oppositional feedback systems have been held responsible for variances of menstrual cycles and reproductive states (Watts & Keffer, 1982). Hormonal assays have been utilized in a number of similar studies and although they have been criticized by various sources, they provide a method of characterizing and assessing endocrine status (Keizer et al., 1982; Shangold, 1984; Jurkowski, 1982). As progressive research has developed more valid techniques of radioimmunoassays, the implementation of such would be a valuable investigative feature.

Menstrual Disorders

The results of this study have alluded to the different endocrine profiles typifying various menstrual disorders (Sperryn, 1983; Loucks, 1981). In accordance with Loucks' models of altered menstrual states, these six PMS subjects manifested alternate states of reproductive function. Zhang and colleagues (1984) associated obesity with specific deviant endocrine profiles and Coney (1984) found similar results in the related disorder of polycystic ovarian disease. At the other end of the reproductive spectrum, modifications in reproductive hormones characterized research

concerning athletic menstrual irregularities (Bonen et al., 1981; Jurkowski, 1982). Consequently, both parameters of obesity and exercise have been implicated as events which cause transitions from reproductive states (Shangold, 1984; Wells, 1985).

Physiological Response to Exercise

The general physiological response of females to exercise was attested to by Fox and Mathews (1981). Evidence to support the implementation of an aerobic program was presented by Eickhoff and associates (1983). The intervention employed here generally yielded expected normal results. Further documentation regarding the effects of acute and chronic exercise support the hypothesis that exercise is implicated in modifications of menstrual cycle functions (Jurkowski, 1982; Shangold, 1984). As previously noted by Gray and Dale (1983), the important mediating factor may be the percentage of body fat as affected by training which in turn affects the menstrual cycle. In this study, changes in body composition due to changed dietary regime were not desired. There was no substantial evidence to indicate dietary based weight losses during the exercise phases. Additionally, all but one subject either maintained or slightly increased percent body fat values during the exercise program. Subject 6 showed a 9% decrease in body fat from Test II to Test V.

Effect of Training on Endocrine Function

Other sources have linked training intensity and duration to changed endocrine functions and consequent athletic menstrual disorders (Bonen et al., 1981; Wakat et al., 1982). Prior (1982a) agreed that training modifies operations at the HPO regions with subsequent shorter cycle length, shorter luteal phases and significant decreases in FSH and LH. Evidence has also demonstrated that running and swimming programs affect the complex hormonal interplay in a gradual, progressive manner (Prior, 1982b; Bonen, 1984). Specific changes in the biphasic nature of the menstrual cycle were not the focus of this study. However, the support for exercise as an agent of change as well as the proposed pathways for change, was a fundamental issue. The implications of the relationship between exercise and endocrine function, and its association with hypothesized alleviation of PMS symptoms, is critical.

Symptoms of PMS

Reid (1983) defined PMS in terms of neuroendocrine-based cyclic fluctuations of somatic and affective symptoms. PMS variability with respect to periodicity and manifestations of symptoms as suggested in Appendix C was evident in the six subjects in the current study. The clustering of symptoms and the resultant asymptomatic interval mentioned by numerous sources was generally illustrated. Harrison (1984) recommended that a magnification during the luteal phase of chronic symptoms

should also qualify one as a PMS sufferer. The manner in which symptoms were manifested did not align with Abraham's (1980) delineation of subsets as numerous combinations were observed.

The four charted symptoms; depression, anxiety, carbohydrate cravings and water retention were documented elsewhere as being of high incidence (Simkin, 1985). All but one subject, who reported depression as not an important factor, charted all four symptoms. Consequently, there were various patterns of crossovers which refuted Abraham's categorization according to symptom constellation. That is, no subject could be labelled as adhering to any one of Abraham's subtypes of PMS.

Simkin's (1985) characterization of the typical PMS sufferer as: 37 years old, college or university educated, married and with two children, and a homemaker; was also not applicable to all six subjects. Only three subjects fulfilled her portrayal although the two subjects who withdrew from the study also met her PMS stereotype.

Previous studies have generally agreed that concurrent charting of symptoms is the best method for diagnosing and evaluating PMS (Keatley & Baldanado, 1984; Pelosi, 1984). The 100 mm visual analog scale rating instrument supported by Rubinow and colleagues (1984) yielded the PMS symptom results in this investigation. The six subjects displayed unique PMS manifestations, and different qualitative and

quantitative ratings were exhibited for subjects, and over menstrual cycles.

Exercise and PMS

Support for the physiological basis of PMS as opposed to a psychogenetic origin is evident in the literature. Simkin (1983) referred to the pronouncement of PMS as an endocrinopathy. That PMS has biological derivations with physiological manifestations, somatic and affective, was supported by numerous references. Although there has been no determined cause of PMS, and treatments have not yielded conclusive or corroborative results, prescriptions of dietary regimes and exercise have yielded positive results (Harrison, 1984; Pelosi, 1984).

Timonen and Procope (1971) linked the possible alleviation of PMS dysphoria to physical activity. A positive correlation between length of menses and PMS exacerbation was substantiated by Woods and associates (1982). Additional support regarding the shortening of the luteal phase through exercise-induced changes have considerable implications for PMS management (Bonen et al., 1981; Jurkowski, 1982).

Authorities on PMS have registered conjectures regarding the therapeutic benefits of physical activity. Pelosi's (1984) recommendations were based upon an increase of lean body tissue with a subsequent decrease in body fat, resulting in decreased estrogen levels. This was not a factor in the present investigation, however, as body composition did not change except for Subject 6.

A number of reasons may account for the lack of research regarding exercise therapy for PMS. In the first place, the subjective, qualitative, and variable nature of PMS have confounded scientific documentation. Keatley and Baldano (1984), and Pelosi (1984) have attested to the lack of reliable, quantifiable data in controlled, reproducible studies. Secondly, contemporary societal and medical influences have had contradictory and counterproductive effects. The development of feminism has paradoxically fostered a demand for medicalization of PMS while simultaneously denying biologically based behaviour. The third factor of radicalism is related to the above point. The spectacular aspects of PMS with regards to legal pleas of 'diminished responsibility' in conjunction with media sensationalization of the 'raging hormone' theory have detracted from serious scientific research. Finally, the matriarchs of the PMS movement, Dalton in Great Britain and Cassara in North America, have not supported exercise as a form of PMS prevention or prescription. Cassara and Egger (1983) have suggested that the dietary regime outlined for PMS sufferers may be more difficult to follow with exercise intervention. Dalton (1977) admitted that exercise is conducive to general health and a positive attitude to life, yet pronounced exercise as irrelevant to PMS.

PMS has been established as having an hormonal basis which supercedes both the ovaries and pituitaries regulation. Exercise may be aligned with PMS on an endocrine scheme.

Although Keizer and colleagues (1982) have presented a thorough overview regarding hormonal measurements, this area presents a number of intriguing possibilities with regard to diagnosis and prescription for PMS. Additionally, the methods of endocrine influence through exercise have been studied with specific regard to the menstrual cycle. As previously mentioned, Dale and Goldberg (1982) have presented an extensive list with reference to altered menstrual function and exercise (see Appendix B). There may be future indications from this area of study which will assist in further studies of PMS and exercise.

Contemporary studies have alluded to a wide range of topics, including abnormal essential fatty acid levels, abnormal patterns of pulsatile luteinizing hormone and a radical pharmacological treatment which induced amenorrhea to provide relief from PMS. Although the psychogenetic origin has been for the most part disapproved, a few studies have suggested negative attitudes towards femininity as an instigator of PMS. Additionally, reeducation of women with regards to their perception of self-control and menses has been a recommendation of these reactive theories.

The majority of sources support the hypothesis of hypothalamic-governance and neurotransmitter activity in the global construct of PMS. Of all the topical literature, the theory of endogenous opiate withdrawal (Reid & Yen, 1981) has generated great, current interest and yielded a formidable related body of research. Reid and Yen (1981)

proposed that the luteal phase may feature abnormal or sensitive responses to hormones which would initiate a cascade of neuroendocrine changes manifested in terms of PMS. The idea of linking PMS and the theory hypothesized by Reid and Yen (1981) has elicited several commendations. The theory involves the psychoneuroendocrine permutations vital to PMS. Evidence supports the wide range of time and subject variability in sex steroid levels and clinical manifestations of PMS. Both of these factors are fundamental to this proposed etiology as well as the endorphin induced mood changes. Exogenous emotional influences can also be integrated with Reid and Yen's (1981) endorphin endocrinological approach. Finally, this perspective has direct implications to exercise and its role in alleviating PMS symptoms.

Jewelewicz (1984) hypothesized that "the opioids may be the connecting link between environmental and psychologic factors and physiologic responses" (p. 685). Shangold (1984) reported that the increased rise in plasma endorphins was facilitated by training and was relatively greater in men. Furthermore, Harber and Sutton (1984) have suggested that opioid substances may regulate secretion of gonadotropins during exercise through a feedback system involving the ovarian steroids. In a study of strenuously exercising swimmers, Russell, Mitchell, Musey and Collins (1984) theorized that increased levels of B-endorphins and catechol estrogen explained the incidences of oligomenorrhea. Harber

and Sutton's (1984) review reinforced the pervading effect of exercise induced B-endorphins. The following exercise effects were cited; hyposecretion of estrogen, increased prolactin and growth hormone, decreased serum LH and perhaps FSH. Consequently, B-endorphins could initiate the shortening of the luteal phase and progress to amenorrhea.

Endogenous opiates have also been implicated in mood alterations (Harber & Sutton, 1984). Allen (1983) refers to the "steady state" cycle of calm then abstinence with regards to B-endorphins. This calm attitude has been translated as the runner's high which in several nonrunning days becomes similar to an addict's withdrawal trauma and subsequent 'fix'. Allen (1983) has presented a thorough, compelling case for the consideration of activity-generated endorphins. He has attempted to apply the "model of thriving" to these endogenous opioids and has attended to both the advantages and disadvantages of these substances as well as a critical analysis of his model. Endorphins appear to be diversely functional and equally important to both PMS and exercise. Allen (1983) defined B-endorphins as the central biasing mechanisms that help us to thrive. In the area of PMS, endorphinergic activity may provide the missing link between physical and affective disorders. The exogenous nature of B-endorphins also reinforces the issue of prevention through exercise. Thus, physical exercise may be directly involved by aiding the balancing of functions, so that the individual can thrive. PMS may be better understood as research reveals

more about B-endorphin activity. Future ramifications for exercise should be determined concurrently with this and other topical criterion-based research.

Although the proposed forms and reasons for exercise therapy may appear to be contradictory, the diverse nature of PMS suggests that some symptoms may respond to various activity modalities. The efficacy of a PMS treatment is dependent upon subjective evaluation, therefore optional forms of activity would allow for preferential selection, increased motivation, and extended participation. The potential for exercise as an attenuator of PMS symptoms is high. Physical activity also promotes, indirectly, a compliance with other suggested life-style approaches of controlling PMS. In addition, the individual hormonal and metabolic influences of exercise should be investigated with respect to related hypothesized PMS etiologies. Exercise may also affect other secondary symptoms displayed with PMS, and control these through occupational behaviour. For example, smoking is contraindicated and regular physical exercise may promote its cessation. Finally, exercise may educate the PMS patient regarding the physical, mental and emotional aspects of her own body. Cassara and Egger (1983) emphasized the importance of women understanding PMS in terms of their own bodies so that ultimately this knowledge would determine effective treatment. The support for exercise as an adaptive and perhaps inhibitive factor in the treatment of PMS is a relatively unexplored area. The

possible implications of regular physical activity for PMS are manifold; the multidisciplinary symptoms of PMS may be holistically mediated through exercise.

Exercise, as a stress reliever, has been suggested by several sources as a possible factor in the alleviation of affective PMS symptoms (Lauerson & Graves, 1983; Livingstone et al., 1983). Anecdotal responses of the subjects in this study indicated that the exercise program positively affected such elements as sleeping habits, energy level, decision making and eating patterns, all of which may be associated with stress reduction.

Price and Giannini (1985) linked a lack of exercise with increased incidence of premenstrual tension. These authors suggested that clinical diagnosis of PMS should include a measurement of physical activity (p. 35). Prior's study as reported by Caldwell (1984) concluded that moderate endurance training alleviated molumina, including water retention discomforts. On the other hand, Pelosi (1984) suggested that this symptom may be aggravated by physical activity. The case study analyses reveal an inconsistent response of water retention to exercise, with several subjects recording aggravated responses during the program. Exacerbation of carbohydrate cravings may also accompany and possibly explain this response. Contradictory responses were alluded to in the literature review and may be accounted for by personal variability of PMS.

That exercise may prove to be a viable therapy for PMS has been suggested indirectly and directly in recent literature. Current hormonal and pharmacological treatments have been found to be lacking in scientific rationale and implicated in the production of numerous side effects. Pelosi (1984) mentioned the high placebo response of PMS to any treatment form. Clare (1979) responded by pointing out that this validated the acceptance of PMS as a medical concern. He further suggested that self-ratings were more important than biochemical analyses (Clare, 1979). Conversely, Dalton, M. (1981) and Gough (1982) supported the use of hormonal assays in evaluating PMS status.

Although the biphasic properties of the menstrual cycle were not evaluated in this study, there are important implications for PMS (Prior, 1982b). The manifestations of PMS have been linked to the luteal phase. Consequently, a shortening of the luteal phase would provide a reduced interval for symptomatic interference. Although this current study did not investigate and compare changes of menstrual cycle length, this would be worthy of inclusion in a future study.

CHAPTER 5

SUMMARY AND CONCLUSIONS

The purpose of this investigation was to examine the relationship between exercise and the alleviation of self-reported PMS symptoms categorized by depression, anxiety, carbohydrate cravings and water retention. The research featured a multiple N (N=6), single I experimental design imposed on an ABA time-series format. The review of literature pointed out the global implications regarding exercise and the reproductive cycle. The research concerning PMS as a menstrual dysfunction and the complexities besetting scientific investigation of this topic were presented. The utilization of physical activity as a therapeutic modality in PMS management was also surveyed. To relate the two major topics of exercise and PMS, six case studies were presented. These featured analyses of training responses, changes in body weight and body composition, Nutriprofile information, exercise compliance, frequency and length of menstrual cycles and graphic representations of self-rated PMS symptoms. Supportive anecdotal evidence originated from the menstrual journal and the survey forms.

The general theme apparent in all six case studies is that the exercise intervention was temporally related to the changes demonstrated in the self-ratings of PMS symptoms. There were incidences previously discussed where a reverse effect or exacerbation of the symptom could be related to

exercise intervention. However, of the remaining charted symptoms, exercise was related to varying degrees of moderating tendencies or as in the case of Subject 2, who did not perceive depression to be a personal PMS symptom, there was no demonstrable change. The attenuation of symptoms varied greatly within and between subjects. Moreover, the modulations were generally progressive in that the greatest asymptomatic patterns were demonstrated as the exercise intervention continued. Trends toward baseline patterns were for the most part displayed subsequent to exercise withdrawal, although in several cases the training response appears to have had a residual, dampening effect on the ratings of symptoms.

The exercise intervention was for the most part not complicated by changes in diet or physical modifications in body weight or body composition. The timing of the diet recalls may account for some variability in the Nutriprofile analyses. The evidence relating the exercise intervention to positive management of PMS symptoms was supported by all subjects through verbal and written anecdotes.

The following conclusions appear justified on the basis of the previously mentioned delimitations:

1. The exercise intervention, in the form of an aerobic workout program for 12 weeks, elicited normal but variable training responses for the six subjects.
2. The body weight and body composition measures were not reduced significantly via the exercise program. Most

- subjects displayed fluctuations and recorded comparable or higher exercise to non exercise values.
3. The Nutriprofile analyses denoted no dietary changes that could be directly related to exercise phase weight reductions.
 4. The exercise intervention was related through fitness and PMS parameters to demonstrate alleviation of self-reported PMS symptoms except as noted below.
 5. The two symptoms of carbohydrate cravings and water retention were in several instances exacerbated during the exercise intervention. These symptoms appeared to be related to each other, to an increase in dietary carbohydrates, and to total ingested kilojoules.
 6. Corroborating written responses and verbal comments attested to the positive influence of exercise on PMS symptoms.

Recommendations

The following recommendations have been made on the basis of the current study and may be of benefit to further research in this area:

1. A larger sample size would reinforce the time-series design of multiple N, single intervention and permit the employment of inferential statistics to support the visual analyses. A larger sample would enable the application of traditional quantitative statistical analysis such as trend and autocorrelation.

2. An ABAB design would further reinforce the intervention effect.
3. A control group which monitored PMS symptoms but did not participate in the exercise program might provide further support for the hypothesis.
4. The utilization of a different exercise modality would perhaps yield further information concerning PMS symptom responsiveness.
5. Quantitative data regarding menstrual length and frequency might provide interesting information regarding the intervention effects and PMS responses.

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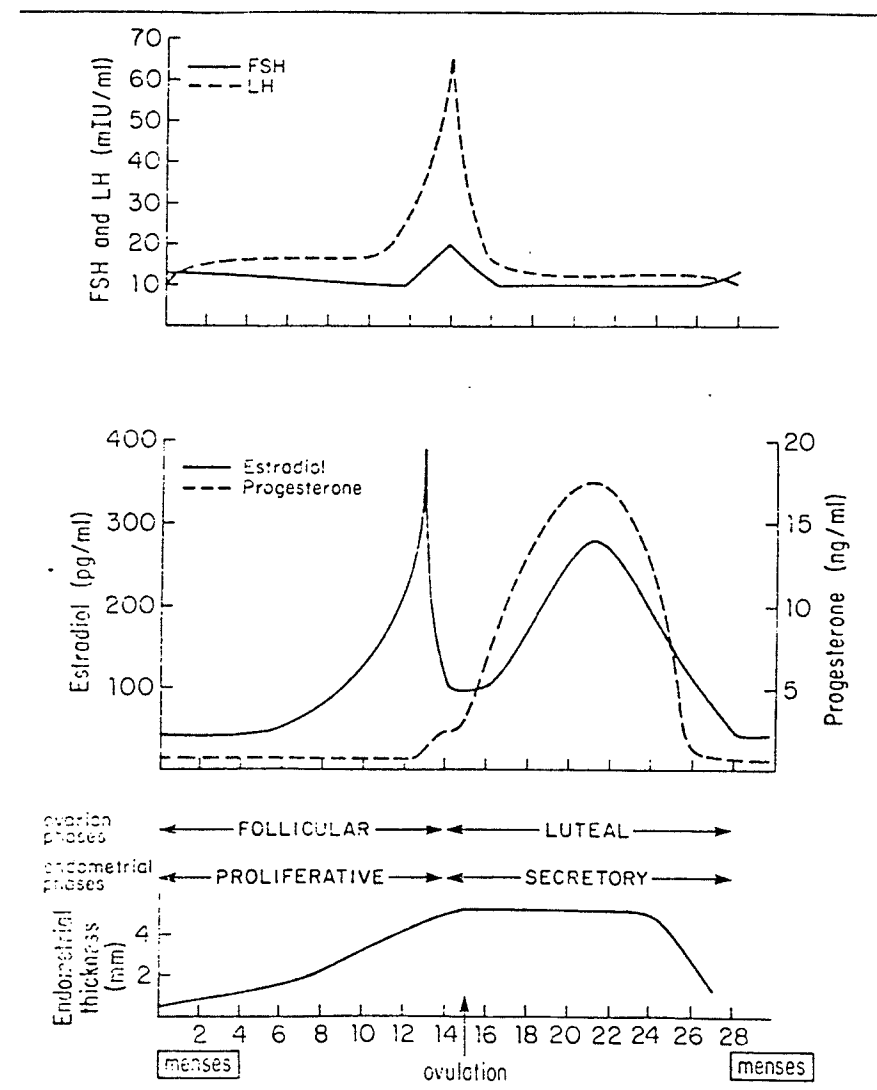
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APPENDICES

Appendix A

Appendix A

Hormonal events of the menstrual cycle. Graphs show blood concentrations of FSH, LH, estradiol, and progesterone, and endometrial thickness. Phases of the ovarian and endometrial cycles are shown. Ovulation and menstruation are indicated.



(Shangold, 1984)

Appendix B

Appendix B

Hypotheses Regarding the Presumed Relationships
Between Exercise and Alteration of Menstrual Function

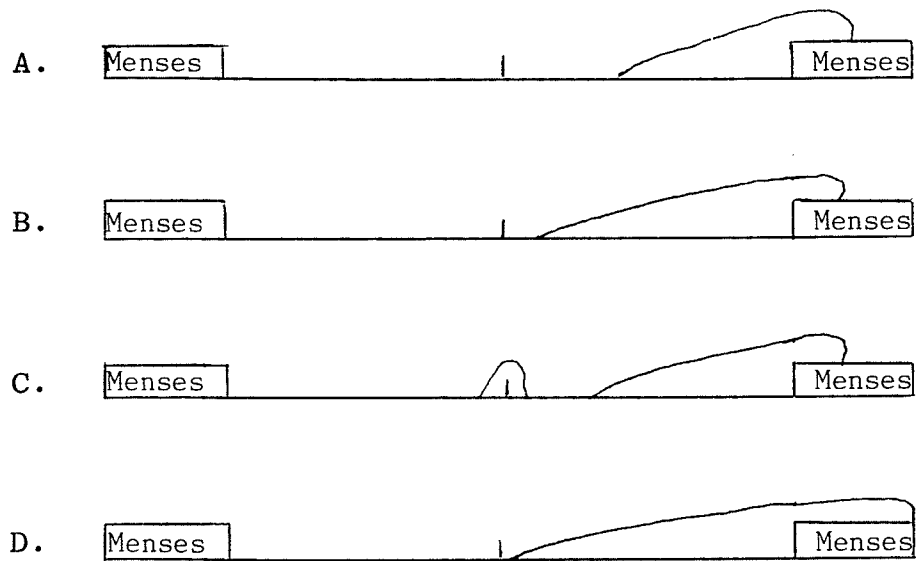
- 1) Stress of running
- 2) Mileage per week
- 3) Intensity of training
- 4) Weight loss
- 5) Body fat loss
- 6) Hormone imbalance
- 7) Suppression of reproductive endocrine axis
- 8) Reduction of steroid hormone production
- 9) Reduction of gonadotropin stimulation
- 10) Short luteal phase
- 11) Hypothalamic dysfunction
- 12) Thermoregulatory dysfunction
- 13) Diet/Nutrition changes
- 14) Other
- 15) May not be running associated

(Dale & Goldberg, 1982)

Appendix C

Appendix C

Schematic diagram showing variability in the onset and duration of premenstrual symptoms. Most patients experience pattern A or B.

PATTERNS OF PREMENSTRUAL SYNDROME

(Reid, 1983)

Appendix D

Appendix D

Common Symptoms of Premenstrual Syndrome

Affective

- Sadness
- Anxiety
- Anger
- Irritability
- Labile mood

Cognitive

- Decreased concentration
- Indecision
- Paranoia
- "Rejection sensitive"
- Suicidal ideation

Pain

- Headache
- Breast tenderness
- Joint and muscle pain

Neurovegetative

- Insomnia
- Hypersomnia
- Anorexia
- Craving for certain foods
- Fatigue
- Lethargy
- Agitation
- Libido change

Autonomic

- Nausea
- Diarrhea
- Palpitations
- Sweating

CNS

- Clumsiness
- Seizures
- Dizziness
- Vertigo
- Paresthesia
- Tremors

Fluid/Electrolyte

- Bloating
- Weight gain
- Oliguria
- Edema

Dermatological

Acne

Greasy hair

Dry hair

Behavioural

Decreased motivation

Poor impulse control

Decreased efficiency

Social isolation

(Rubinow et al., 1984)

PMS: Overview from a
methodological perspective.
American Journal of
Psychiatry, 141, 163-172.

Appendix E

Appendix E

Etiology of Premenstrual Syndrome

<u>Author</u>	<u>Year</u>	<u>Theory</u>
Frank	1931	Decreased renal clearance.
Sweeney	1934	Disturbance of the sympathetic nervous system.
Israel	1938	Defective luteinization resulting from progestin deficiency or relative hyperstrogenemia.
Thorn, et al.	1938	Decreased urinary output, with retention of sodium and chloride.
Greenhill and Freed	1940	Estrogen-induced salt and water retention.
Biskind	1943	Estrogen excess due to decreased liver clearance. Vitamin B recommended.
Billig and Spaulding	1947	Hyperinsulinism.
Stieglitz and Kimble	1949	Endometrial toxin which could be treated with ammonium nitrate.
Morton	1950	Increased carbohydrate tolerance and abnormal glucose tolerance curve. Recommended vitamin B complex, high-protein, low-salt, low-sugar diet.
Bickers and Woods	1951	Vasopressin-induced water intoxication.
Greene and Dalton	1953	Introduced for the first time the concept of a syndrome. Progestin therapy.
Rees	1953	Found psychotherapy of limited value and recommended individual therapy.

- | | | |
|-------------------|------|--|
| Mukherjee | 1954 | Found flat glucose tolerance curves during the luteal phase. |
| Jones and Gordon | 1969 | They found no allergic component to sex hormones in PMS. |
| Farah and Shbaklu | 1971 | They found no allergic component to sex hormones in PMS. |
| Reid and Yen | 1981 | Multifaceted psychoneuroendocrine disorder involving dysfunction in the hypothalamus, pituitary, and ovarian axis. PMS is due to an atypical release of/or a sensitivity to the neurointermediate lobe peptide alpha melanocyte-stimulating hormone and beta-endorphins during the luteal phase. |

(Pelosi M.A., 1984)

Pre-menstrual syndrome:
Fact or fantasy. The
Journal of the Medical
Society of New Jersey, 81,
303-308.

Appendix F

Appendix F

Proposed Etiologies for Premenstrual Syndromes

Ovarian hormonal

Estrogen
Progesterone

Fluid and electrolyte hormonal

Prolactin
Aldosterone
Renin/angiotensin
Vasopressin

Other hormonal

Endorphins/enkephalins
Melanocyte stimulating hormone
Glucocorticoid
Androgen
Insulin
Melatonin

Neurotransmitter

Monoamines (5-hydroxytryptamine, norepinephrine,
dopamine)
Acetylcholine

Other

Vitamin B₆/magnesium
Psychological basis
Prostaglandins

(Rubinow & Roy-Byrne, 1984)

PMS: Overview from a
methodological perspective.
American Journal of
Psychiatry, 141, 163-172.

Appendix G

Appendix G

Proposed Treatments for Premenstrual Syndromes

Hormonal

- Progesterone
- Progestins/oral contraceptives
- Antihormonal, danazol
- Androgens

Psychotropics

- Lithium
- Monamine oxidase inhibitors
- Sedative-hypnotics

Other

- Bromocriptine
- Pyridoxine
- Dietary restriction
- Diuretics
- Prostaglandin precursors/inhibitors

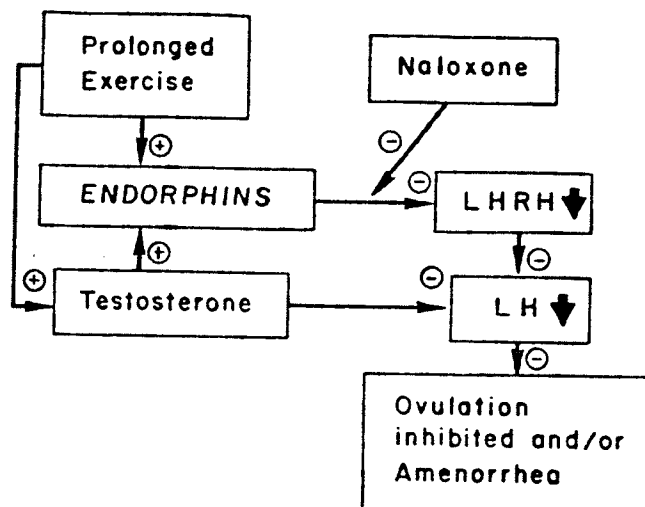
(Rubinow & Roy-Byrne, 1984)

PMS: Overview from a
methodological perspective.
American Journal of
Psychiatry, 141, 163-172.

Appendix H

Appendix H

Endorphins Effect on Ovarian Function



Model of endorphin inhibition (--) of Luteinizing Hormone Releasing hormone (LHRH) which by its absence fails to stimulate the necessary Luteinizing Hormone (LH) for ovulation. Testosterone both stimulates (+) endorphins and inhibits (--) LH directly. Naloxone blocks (--) the inhibitory affect of Endorphin on LHRH with a subsequent rise of LHRH (not shown here).

(Allen, 1983)

Appendix I

Appendix I

INFORMED CONSENT1. Explanation of the Fitness Tests

You will perform a twelve minute, submaximal exercise test on the bicycle ergometer. We may stop the test at any time because of fatigue or you may stop when you wish to because of personal feelings of fatigue or discomfort. Your heart rate will be monitored throughout the test.

Your percent body fat will be determined by weighing you underwater. This is a safe procedure, and you will be supervised by an exercise technician at all times. You will also perform several tests of flexibility including sit and reach, trunk extension and shoulder extension.

2. Explanation of Exercise Program

You will be asked to participate in an aerobic fitness program for 12 weeks. The classes are 1 hour in length and you will be expected to participate on a regular basis (minimum 3 times per week and maximum 4 times per week). These classes will be respective of your fitness level and you will be asked to monitor your workouts through observance of target heart rates.

3. Explanation of Dietary Recall

You will be asked to recall, under the guidance of a nutritionist, your dietary intake over a three day

period. This will be used to evaluate your nutritional status.

4. Explanation of Monthly Menstrual Calendar

You will be asked for the 28 weeks, to chart your monthly menstrual calendar on a form similar to the Women's Health Clinic Form. There are 6 selected symptoms as well as the 2-5 symptoms you may write in. The registering of information is according to a 1-3 scale for symptoms, a check for other data and number of minutes and type of physical activity.

5. Risks and Discomforts

There is a very minimal possibility that certain changes could occur during the bicycle test. These include abnormal blood pressure, fainting, disorders of heart beat, and in rare instances heart attack. Every effort will be made to minimize these through a preliminary examination and by observation during the testing. The exercise technicians are trained to handle any unusual situation which may arise.

6. Benefits to be Expected

The information being collected is part of an experimental study to examine the effect of physical activity on the Premenstrual Syndrome. In addition to providing needed information for the study you will receive the complete results of your fitness test (percent body fat, flexibility, cardiovascular capacity), nutritional evaluation, and the complete

results of the premenstrual assessment form.

7. Inquiries

Any question about the procedures to be used in the study are encouraged. If you have any doubts or questions, please ask us for further explanations.

8. Freedom of Consent

Your permission to take part in this study is voluntary. You are free to deny consent if you so desire.

I have read this form and I understand the test procedures that I will perform. I consent to participate in this test. I will inform the study coordinators of any extenuating circumstances that may bias the nature of my results (eg. binging, oral contraceptives, pregnancy, non participation in aerobic program).

Signature of participant

Date

Signature of witness

Appendix J

Appendix J

Menstrual Calendar (100 mm Visual Analogue)

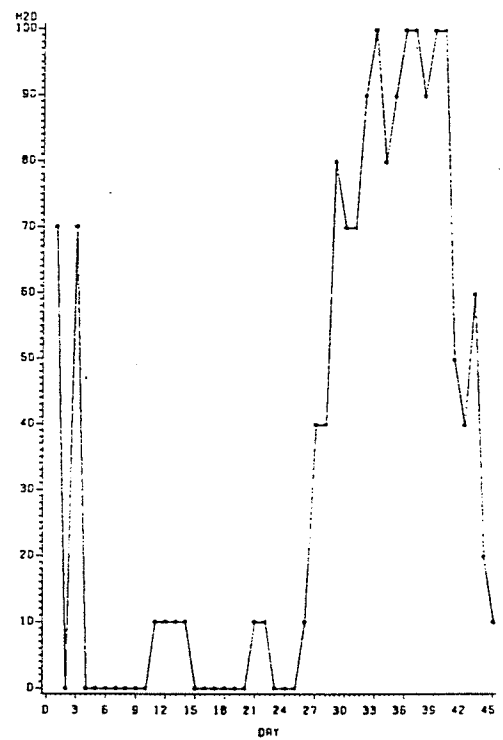
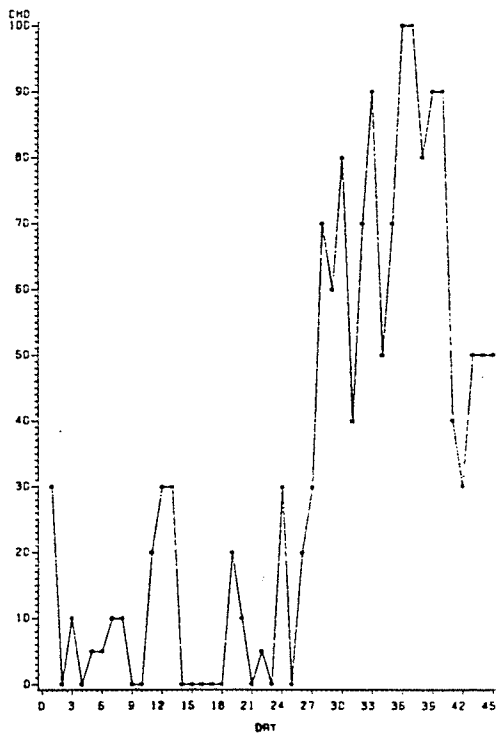
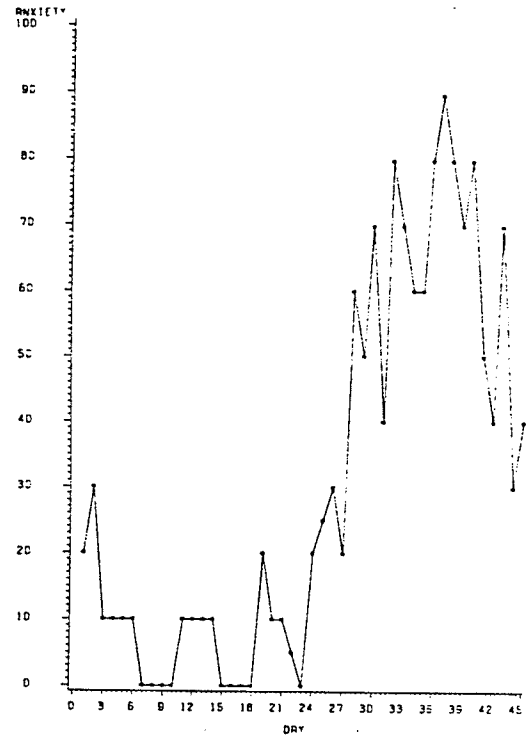
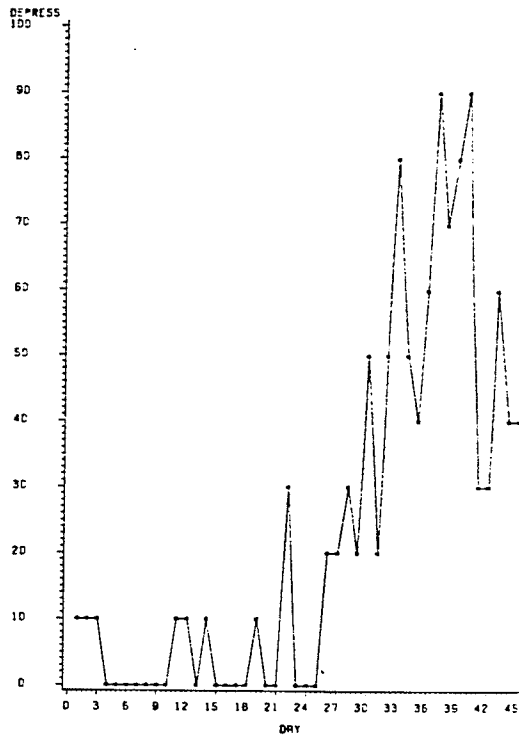
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60	60	60	60
50	50	50	50
40	40	40	40
30	30	30	30
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10	10	10	10
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A	B	C	D

Date: _____ Time: _____ Menses: Yes ___

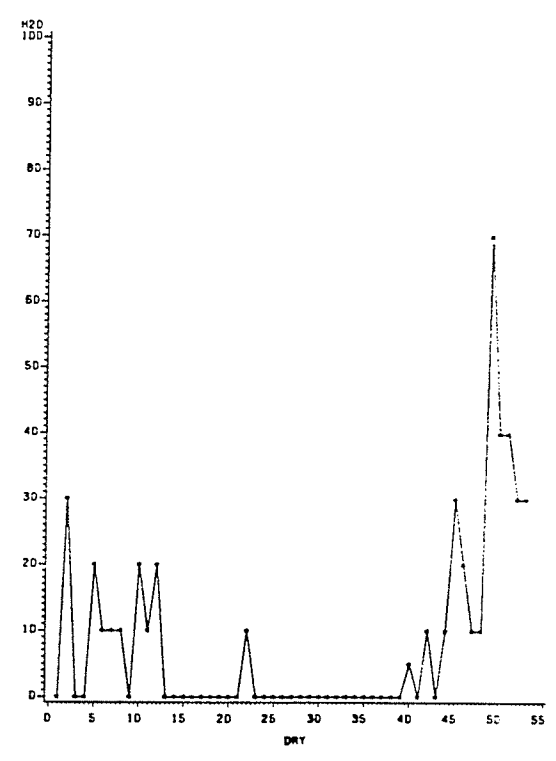
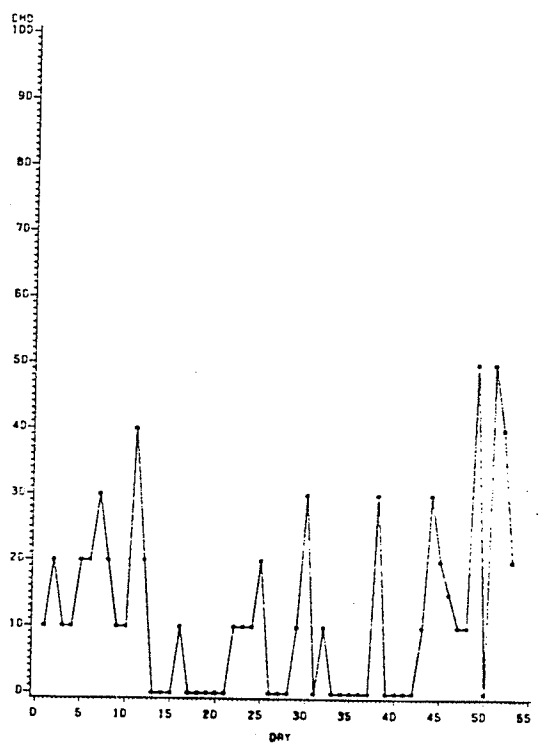
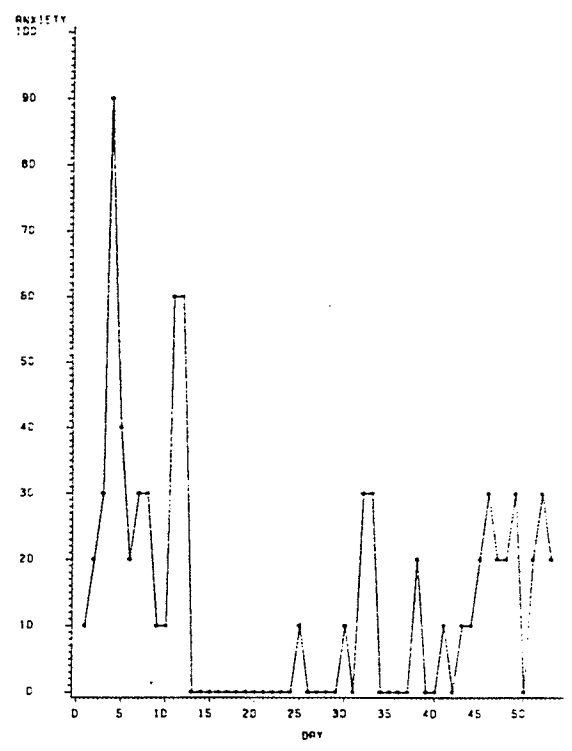
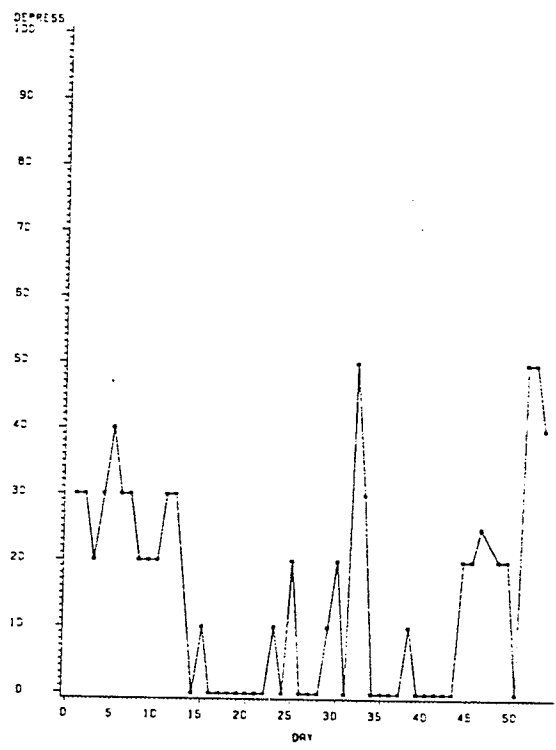
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Appendix K

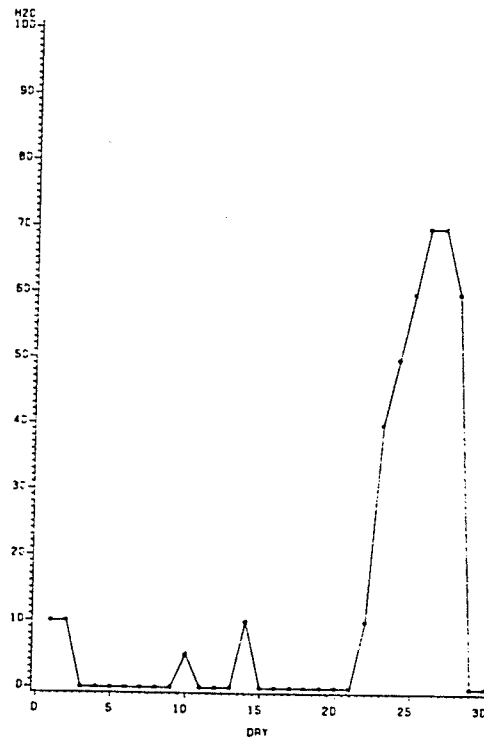
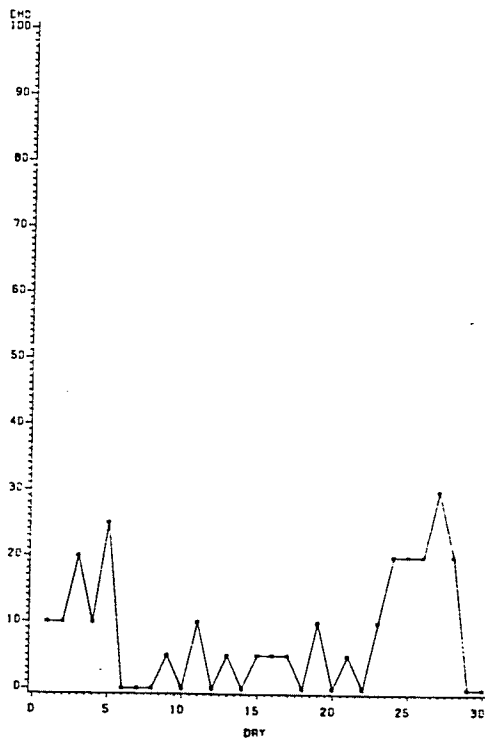
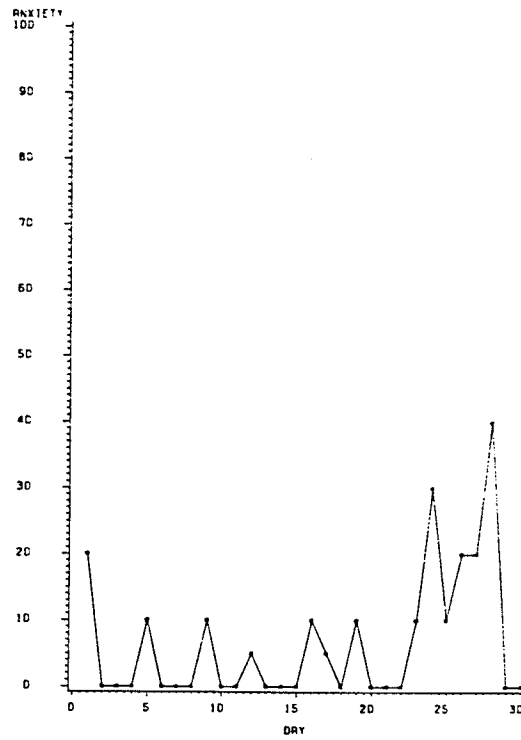
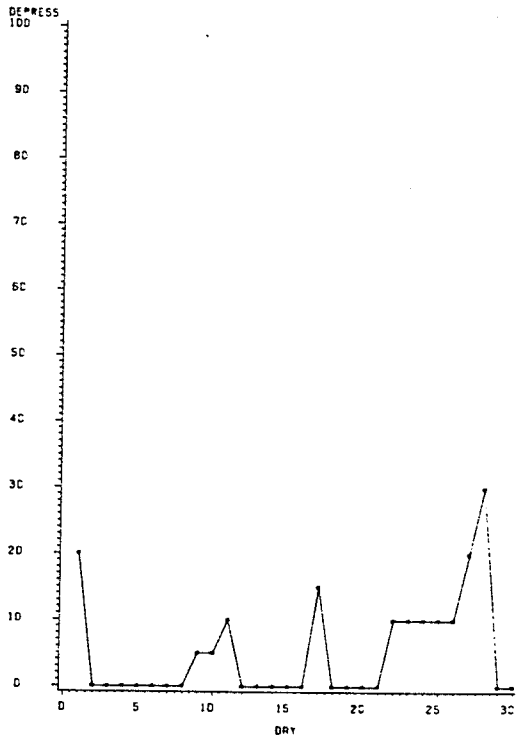
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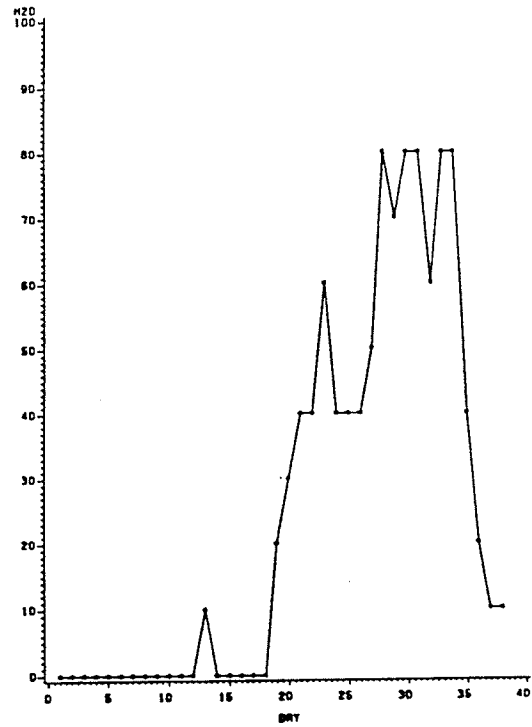
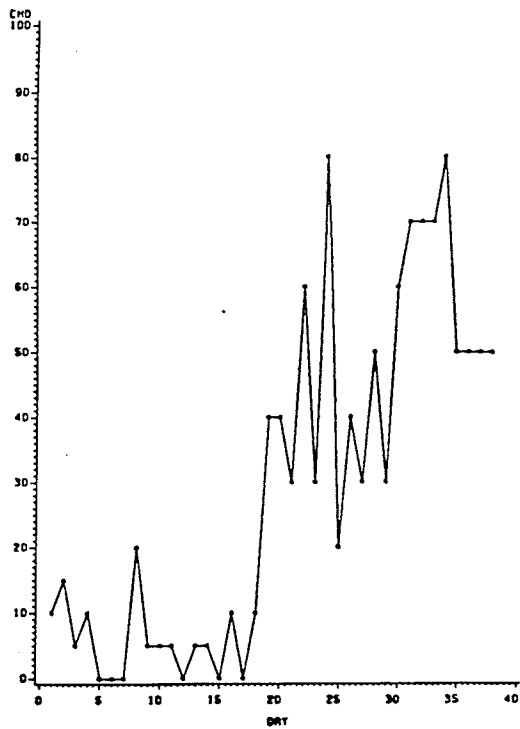
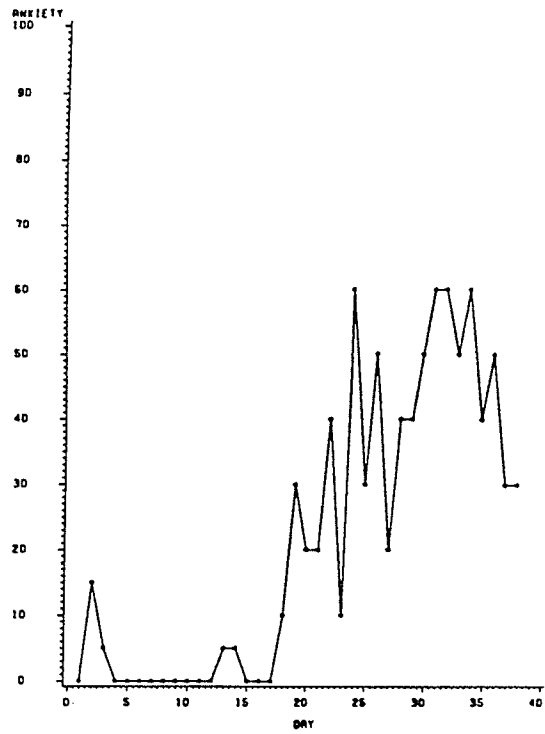
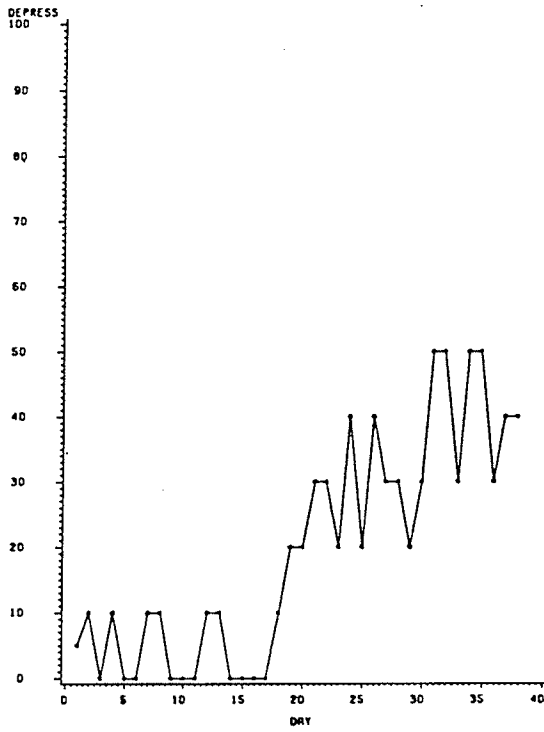
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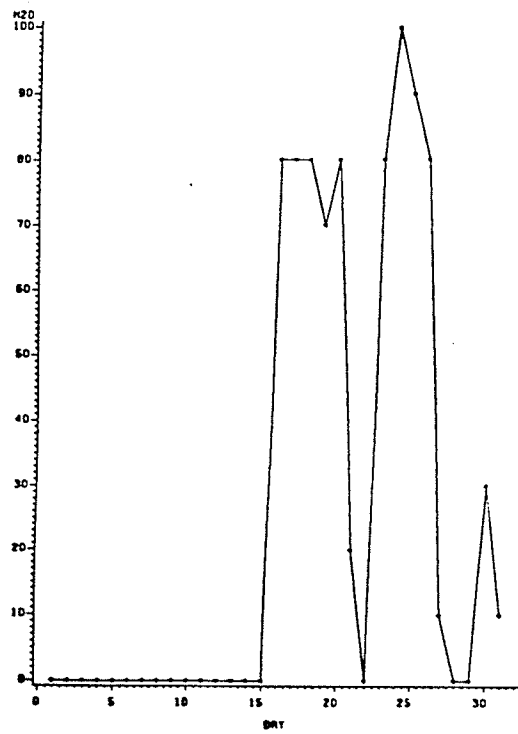
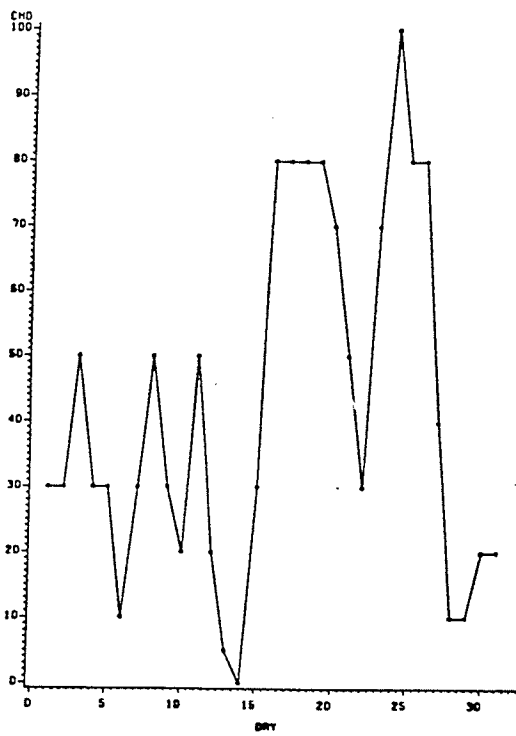
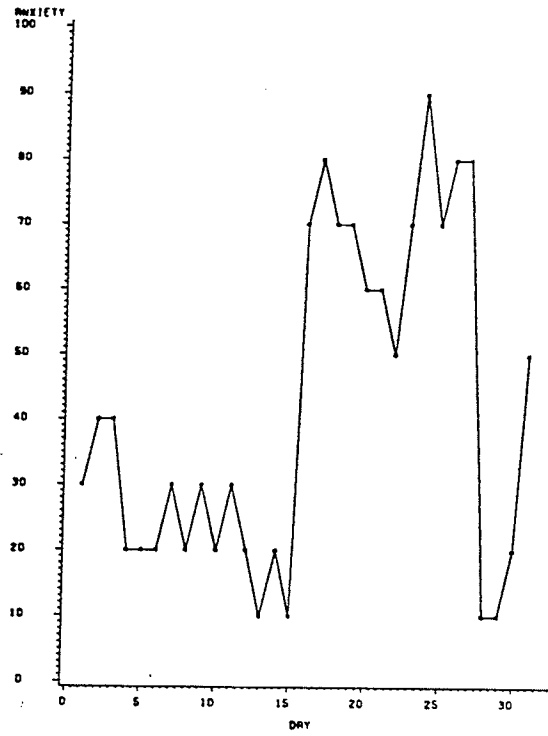
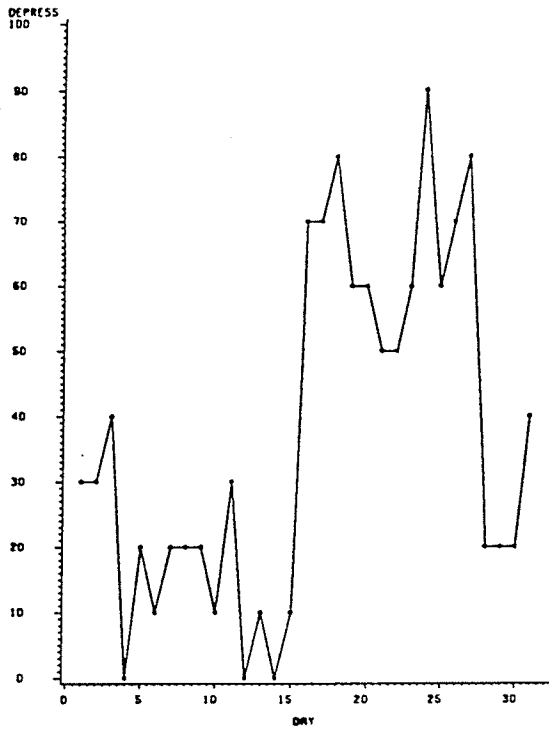


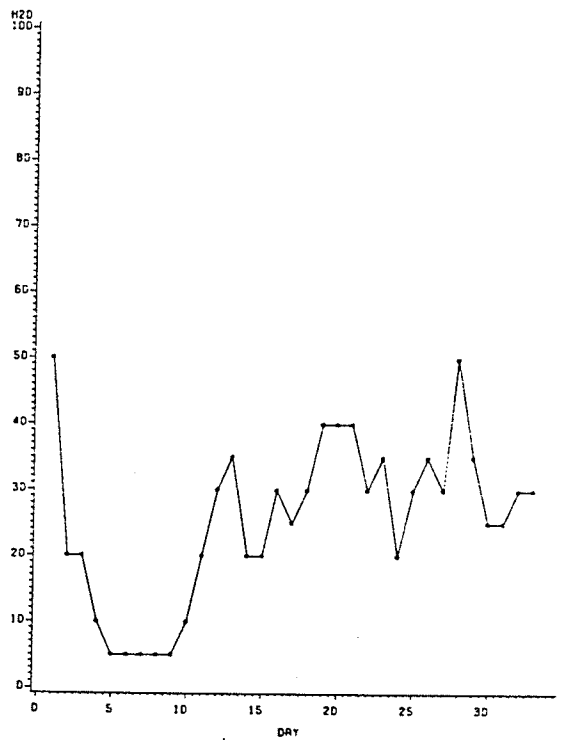
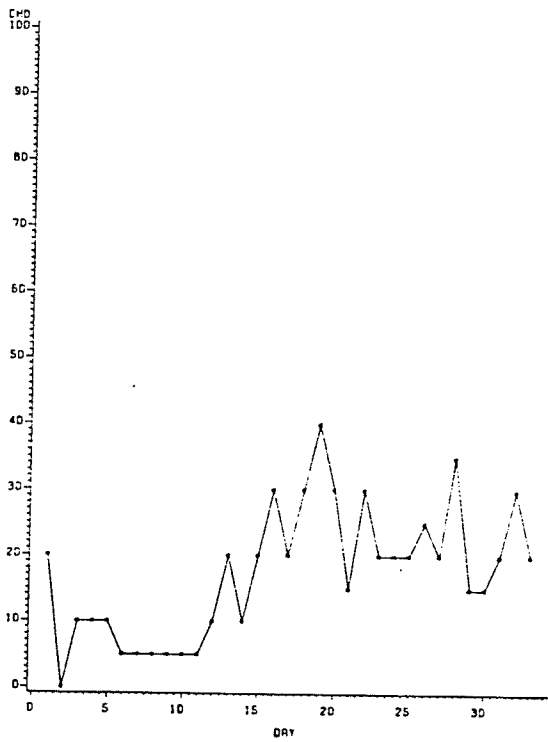
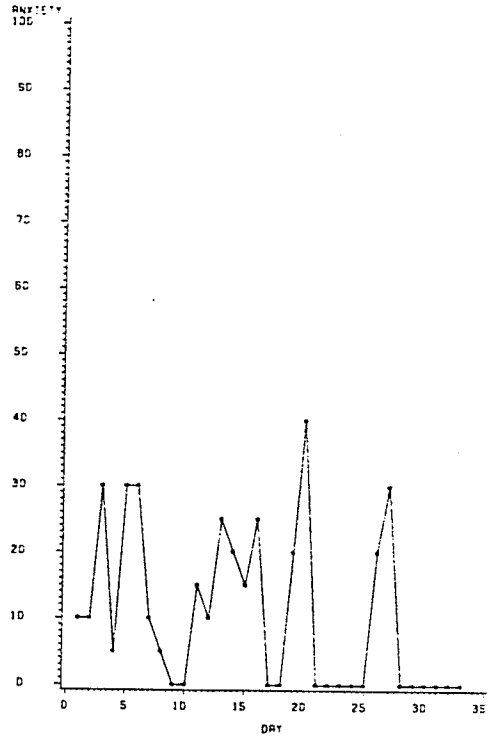
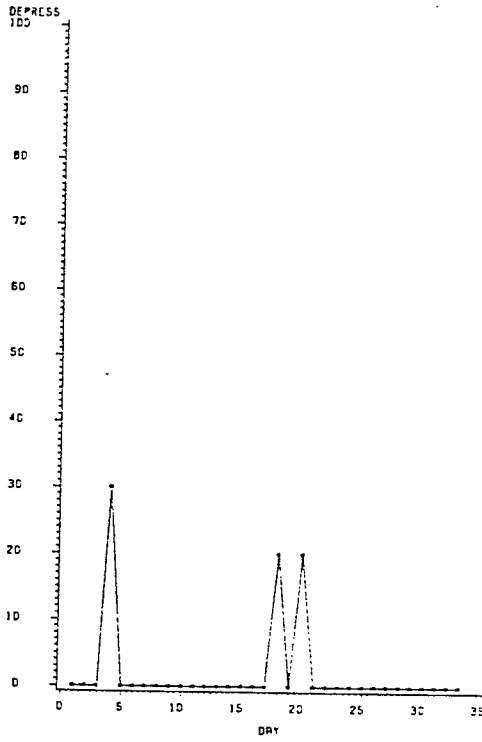
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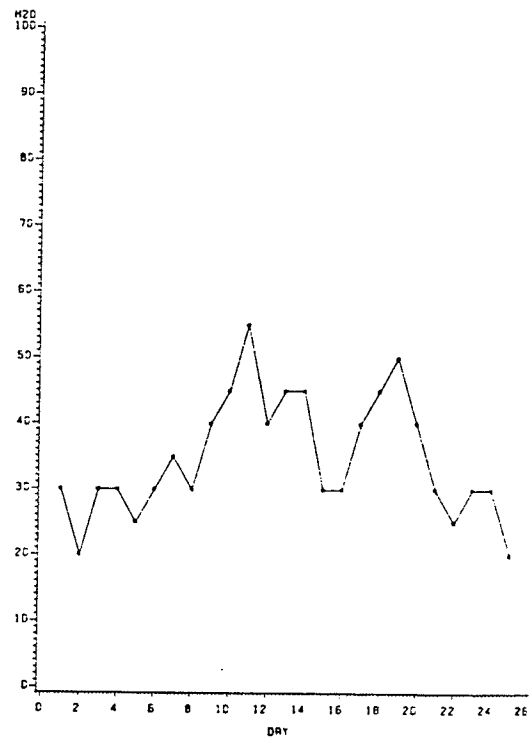
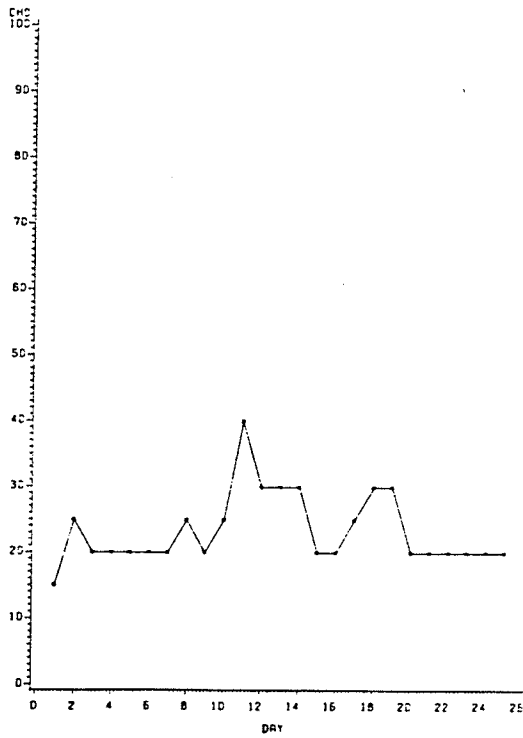
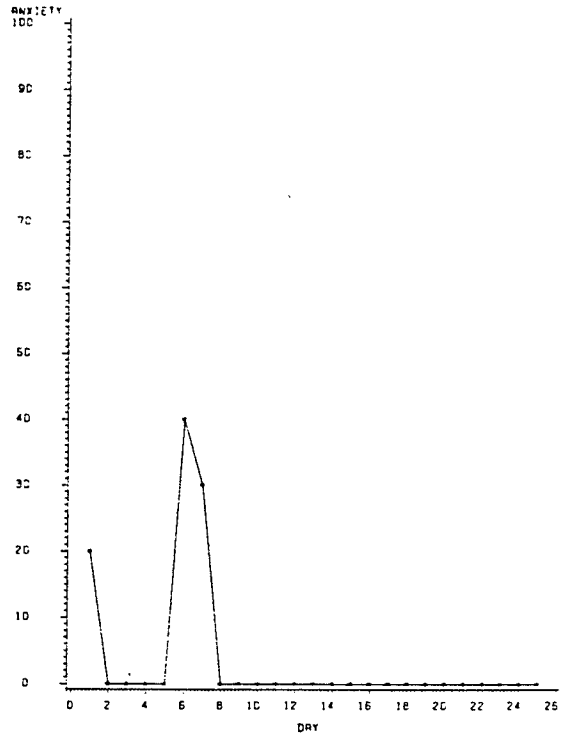
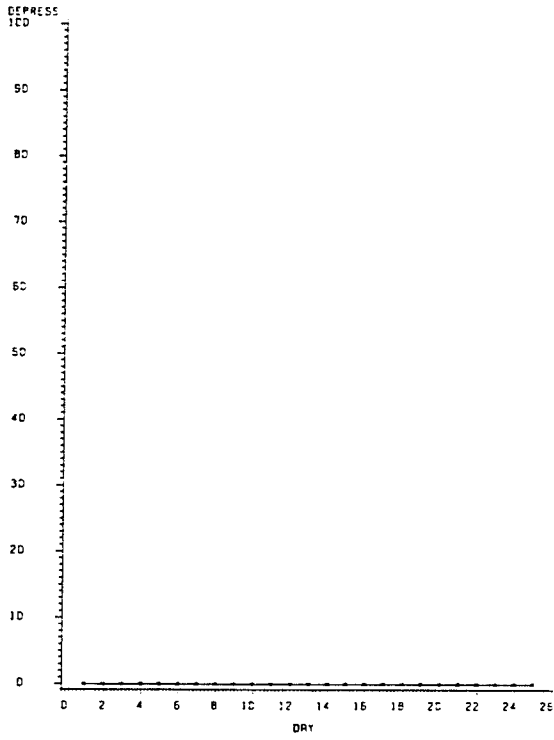


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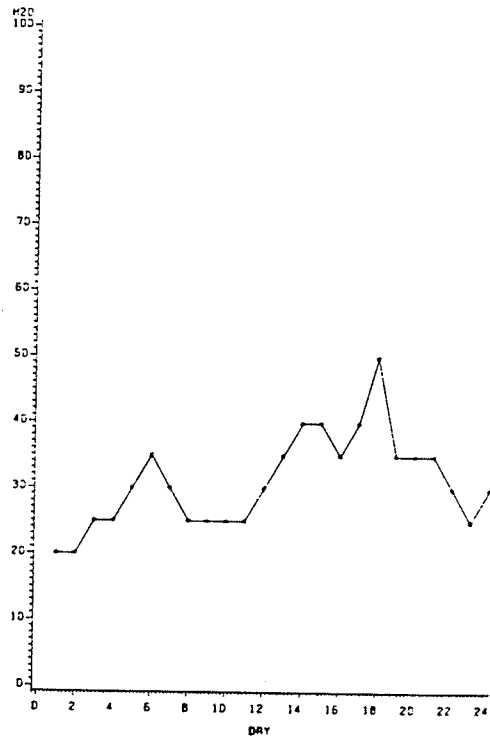
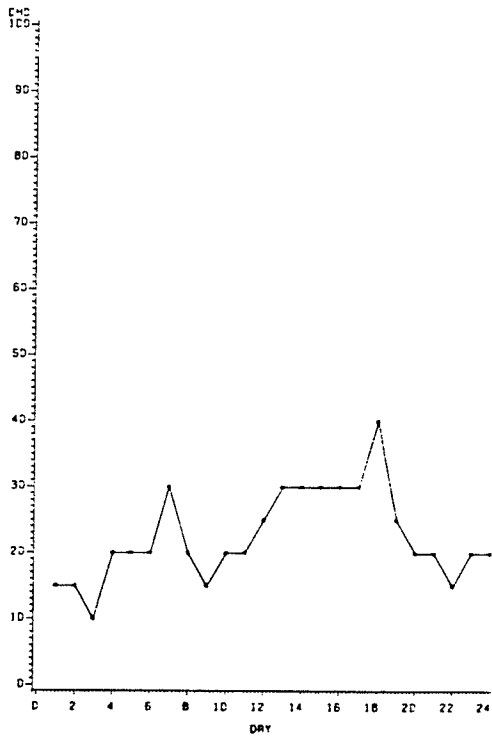
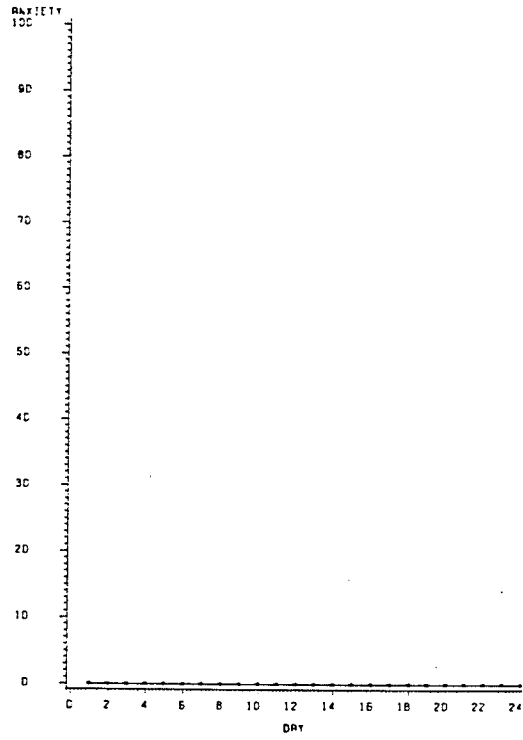
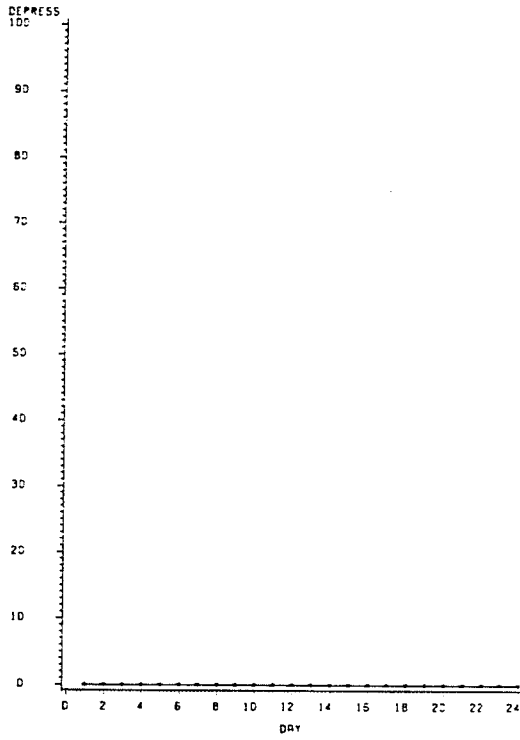




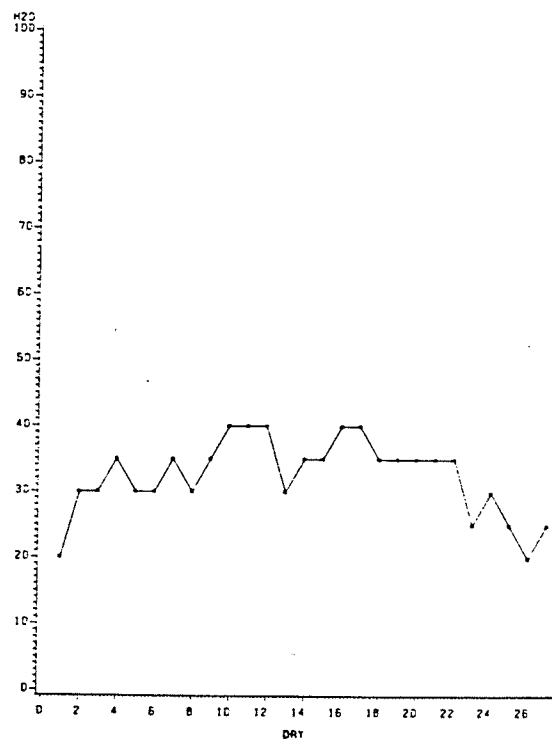
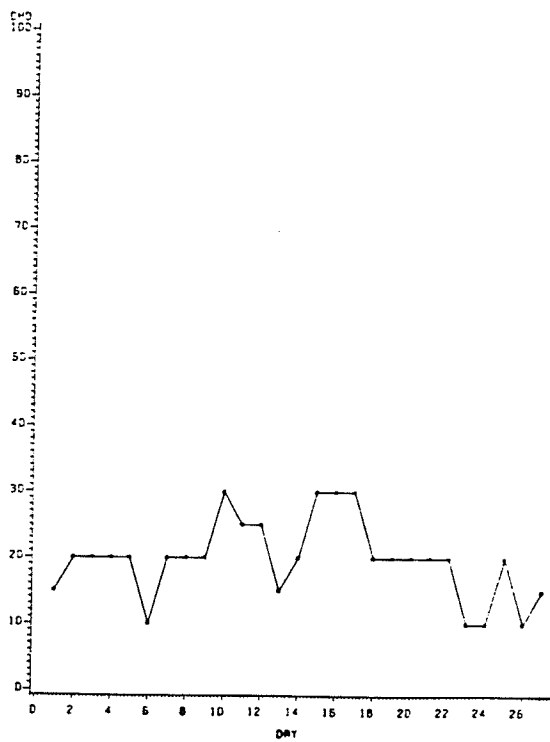
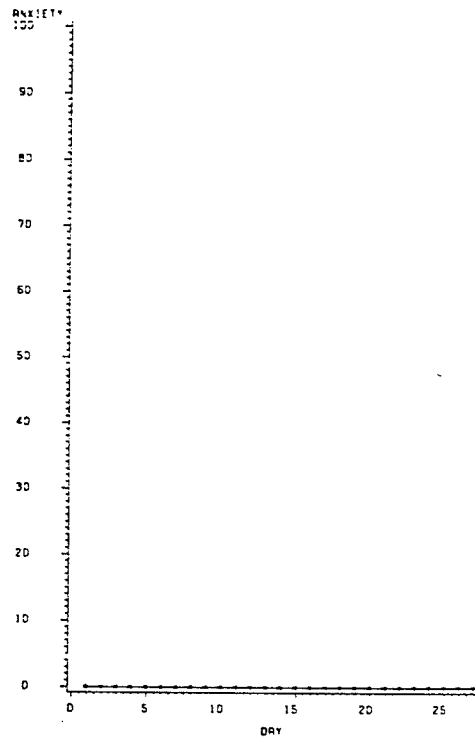
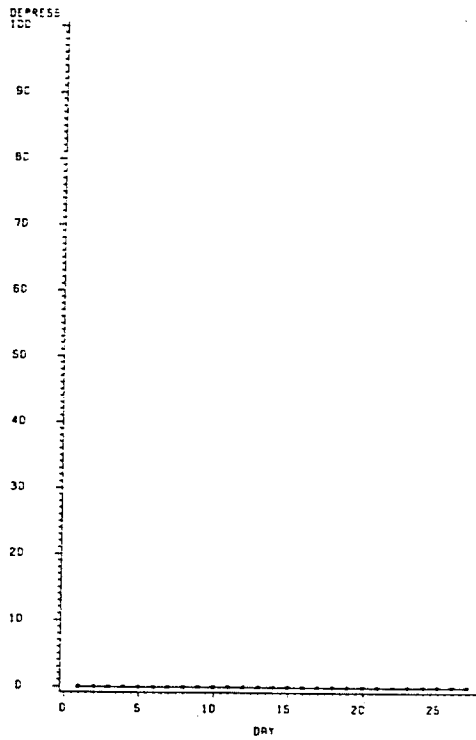




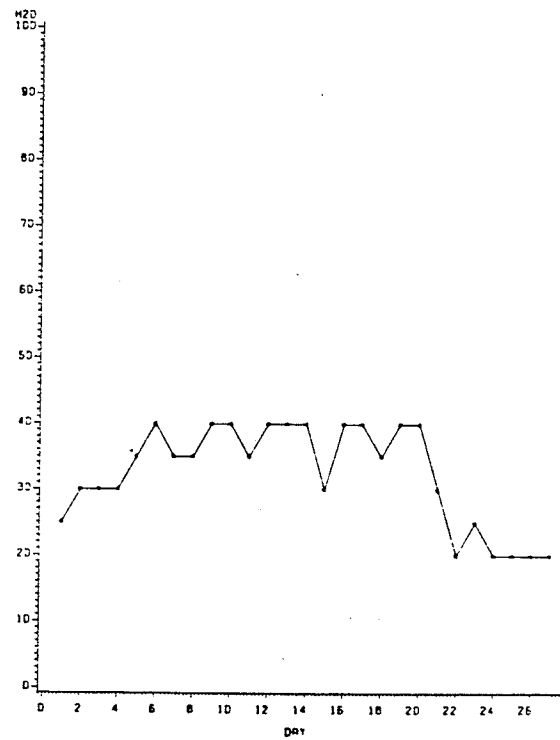
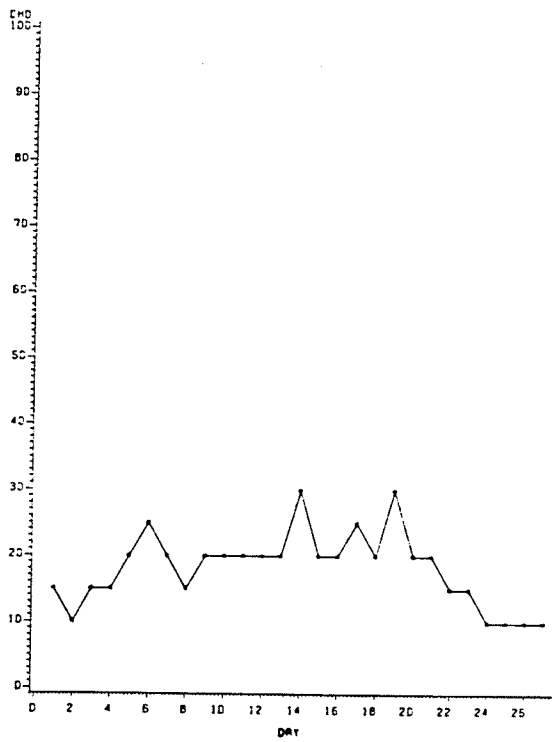
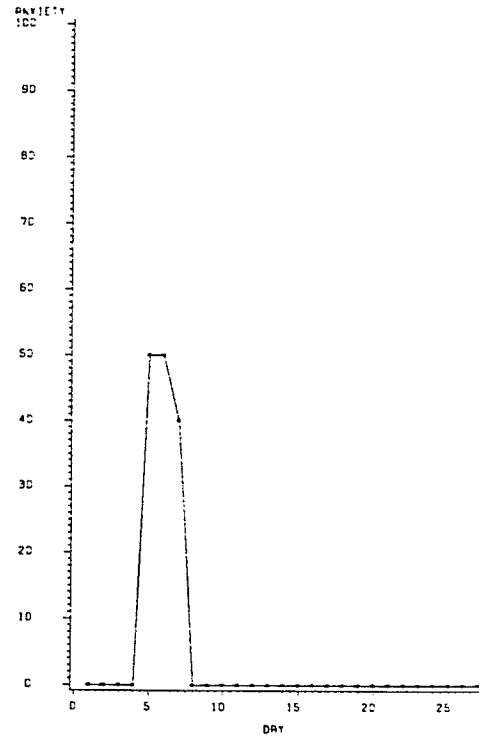
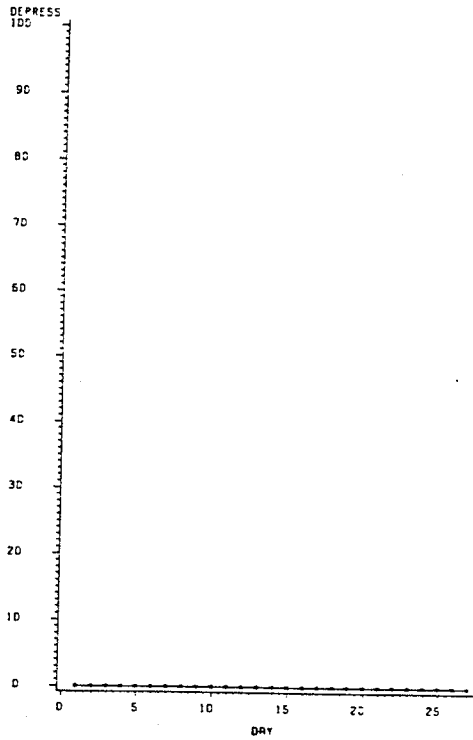
SUBJECT 2 : CYCLE 3 PMS SELF-RATINGS



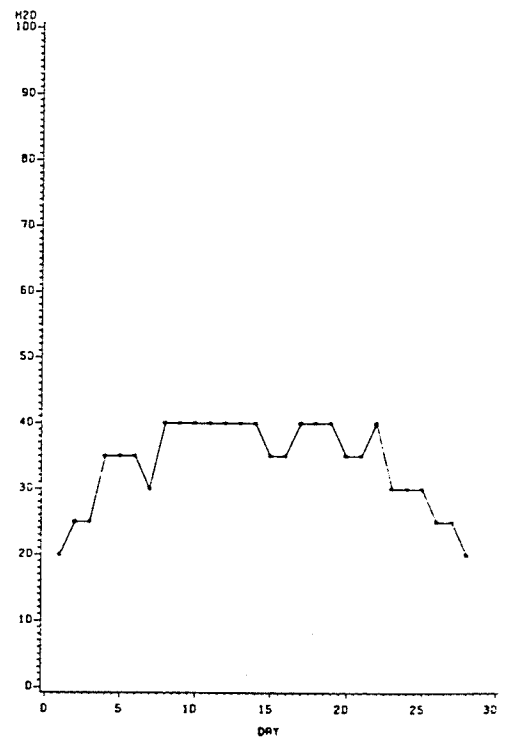
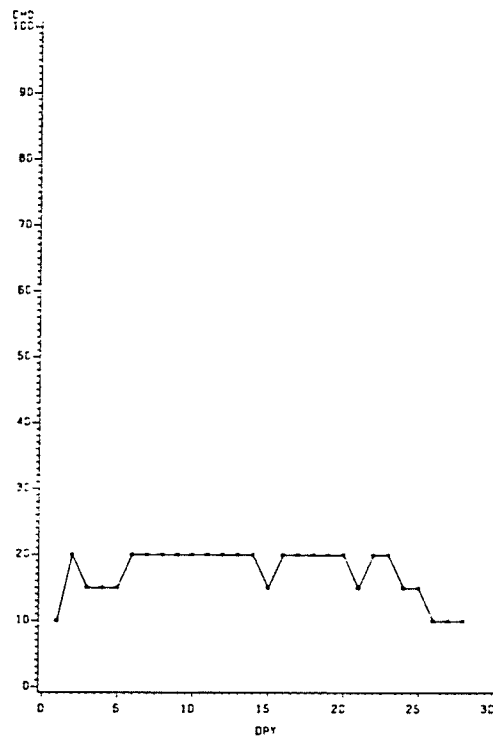
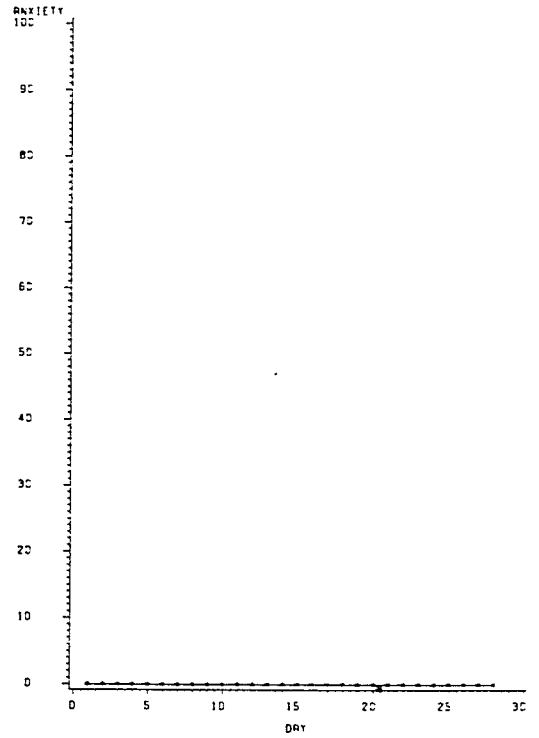
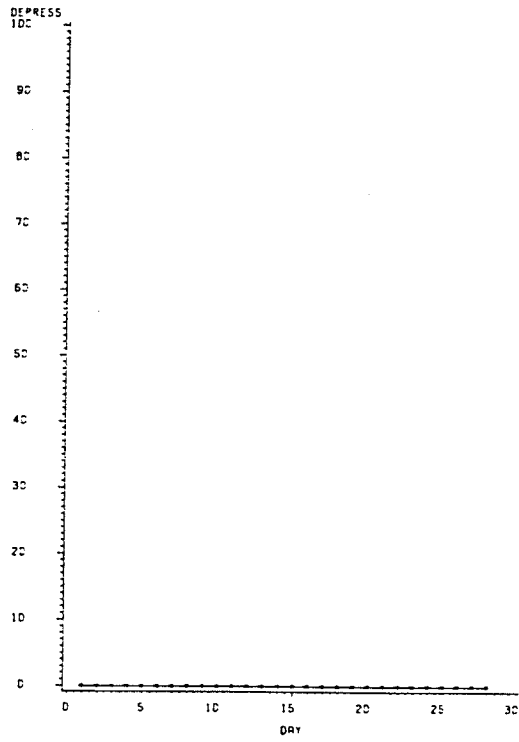
SUBJECT 2 : CYCLE 4 PMS SELF-RATINGS



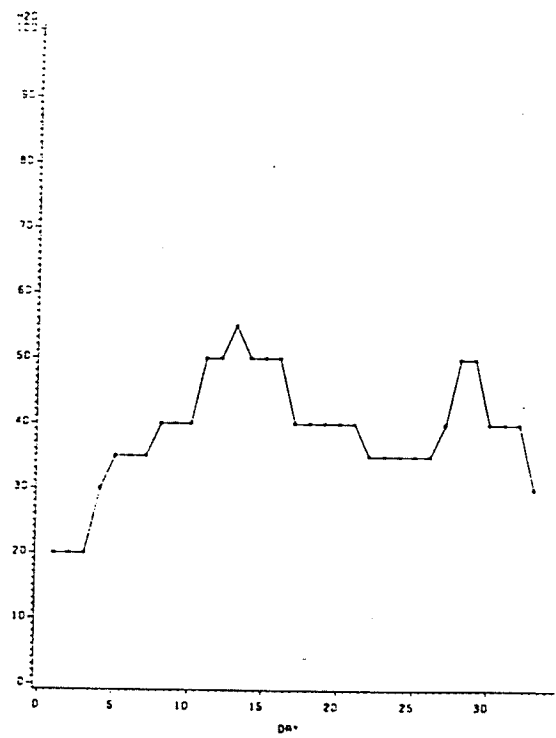
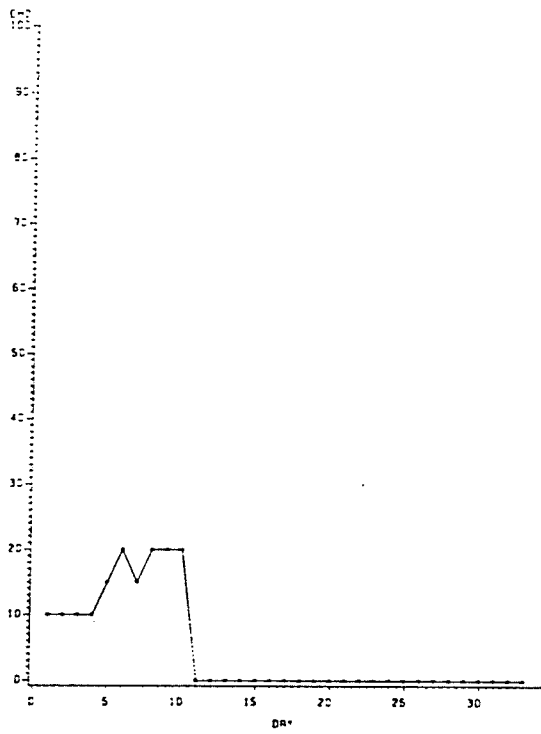
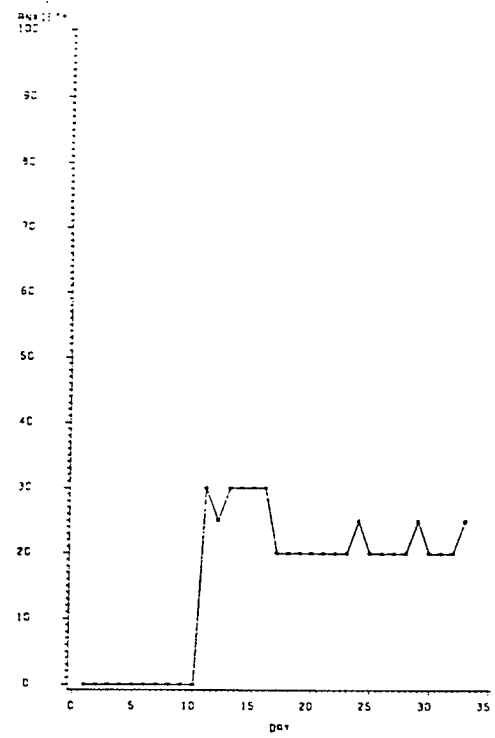
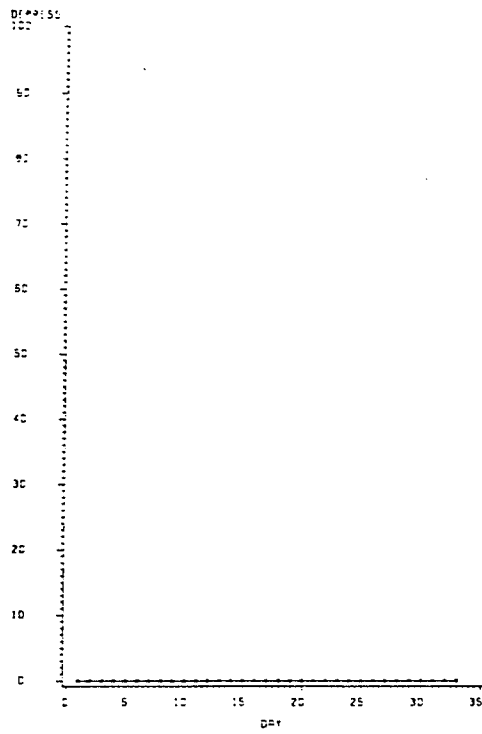
SUBJECT 2 : CYCLE 5 PMS SELF-RATINGS



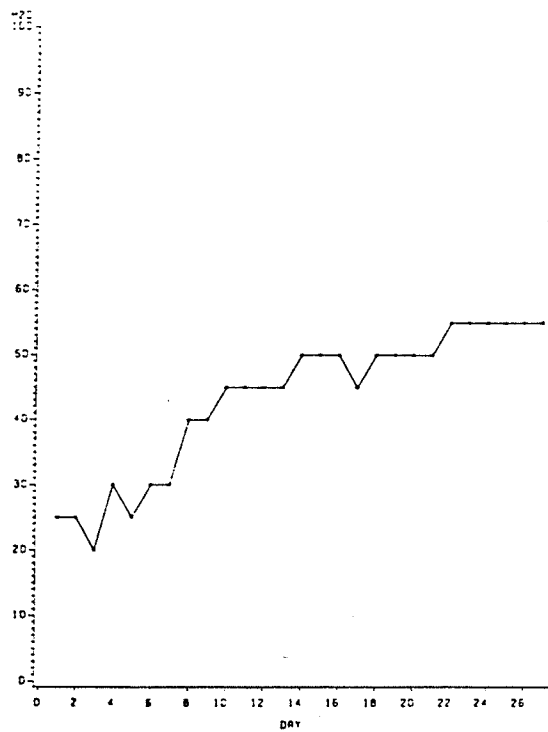
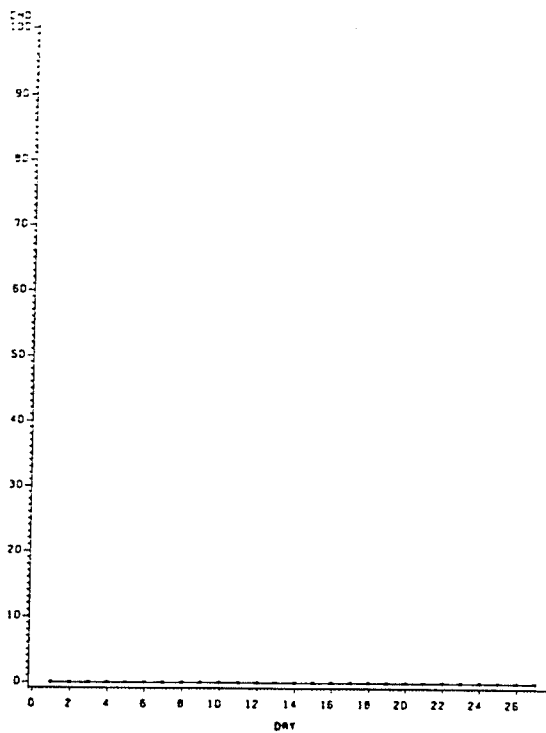
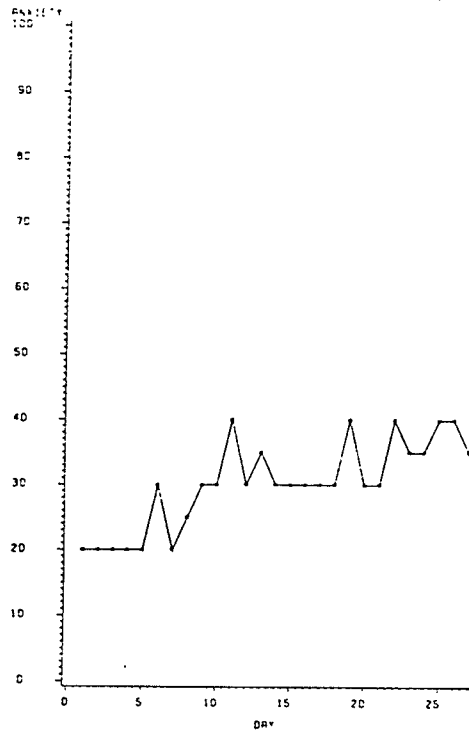
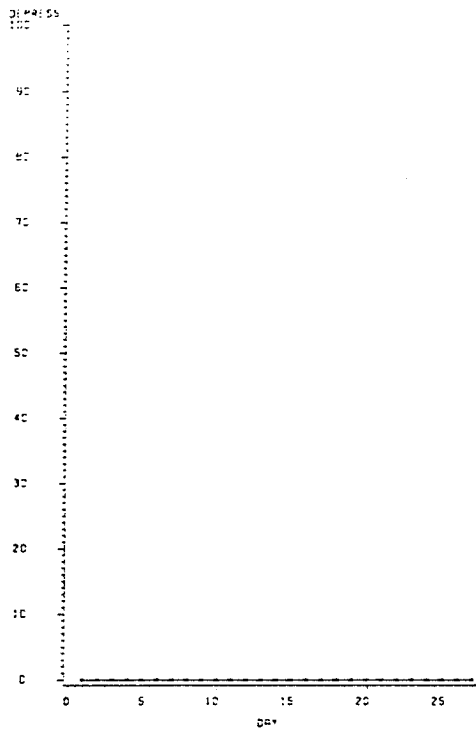
SUBJECT 2 : CYCLE 6 PMS SELF-RATINGS



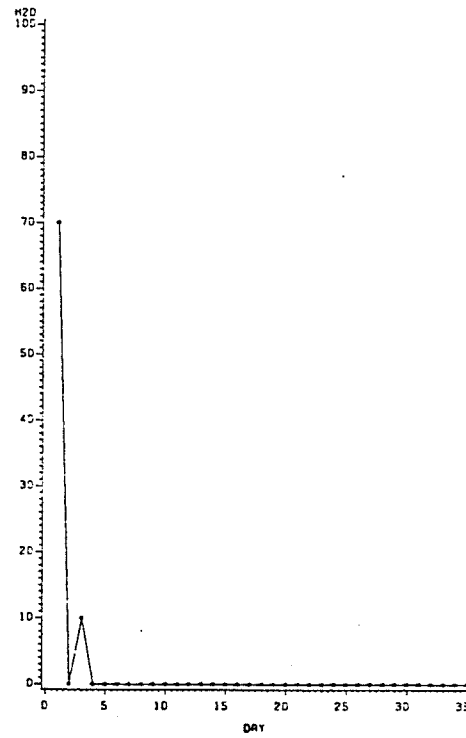
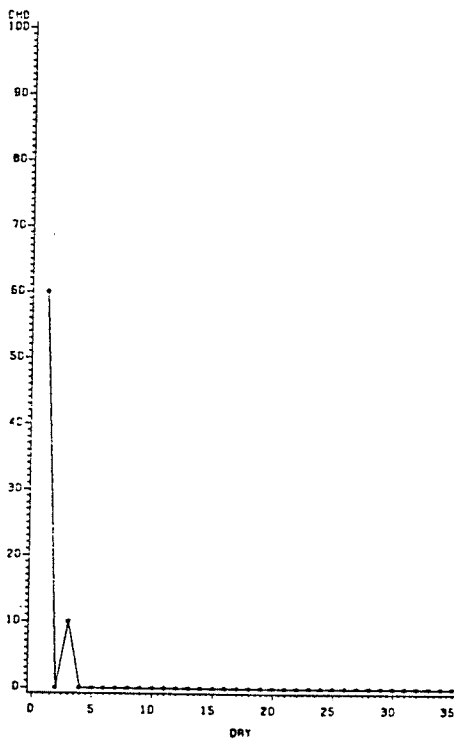
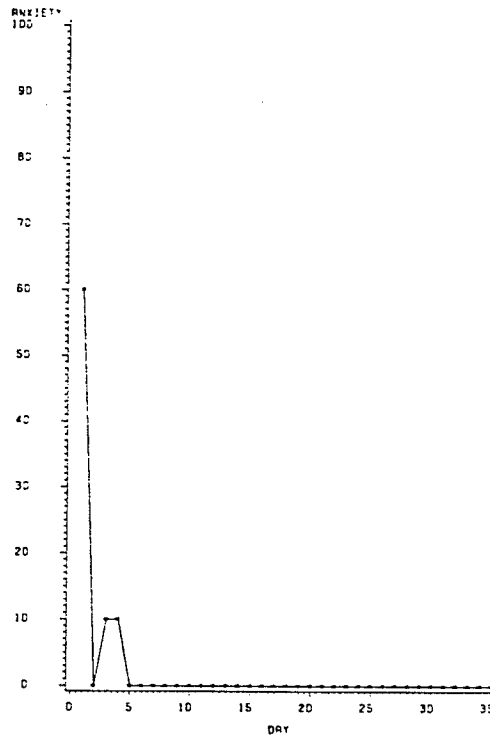
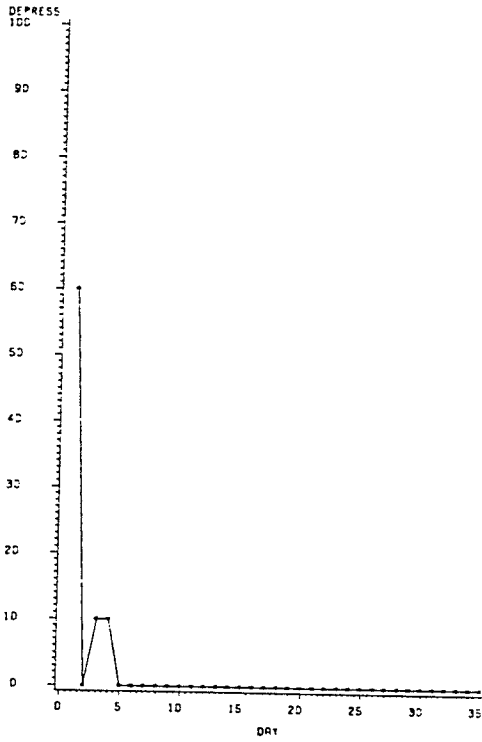
SUBJECT 2 : CYCLE 7 PMS SELF-RATINGS



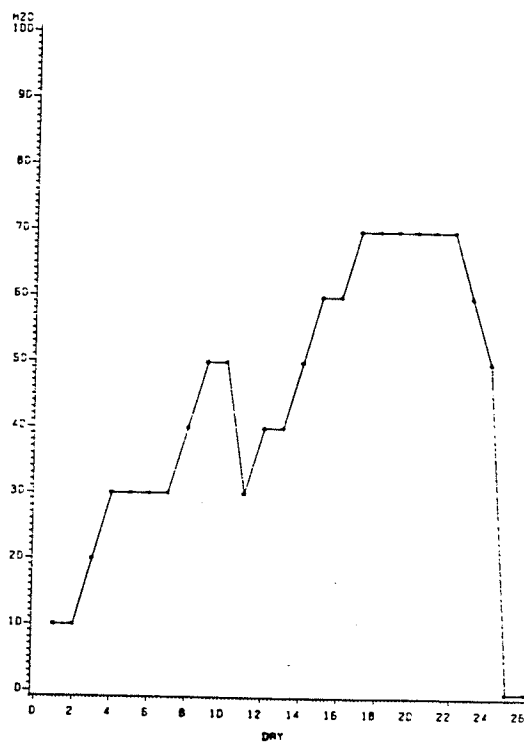
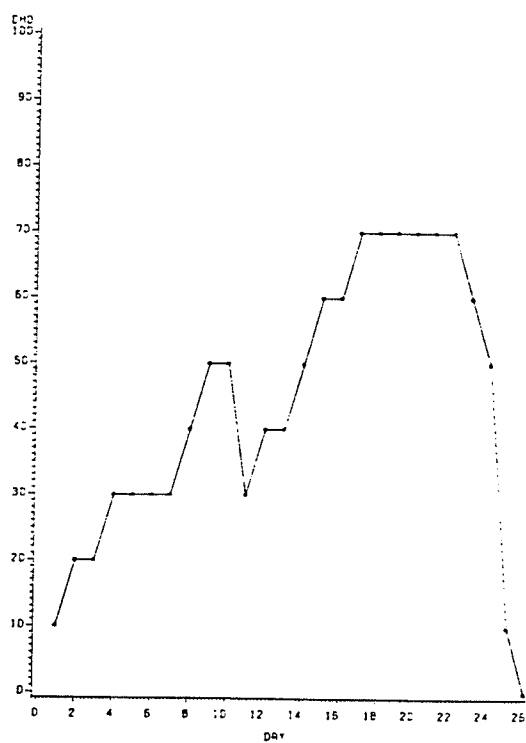
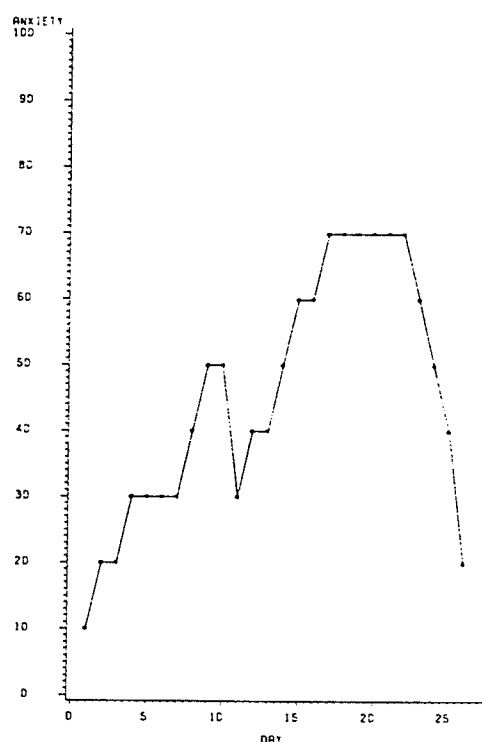
SUBJECT 2 : CYCLE 8 PMS SELF-RATINGS



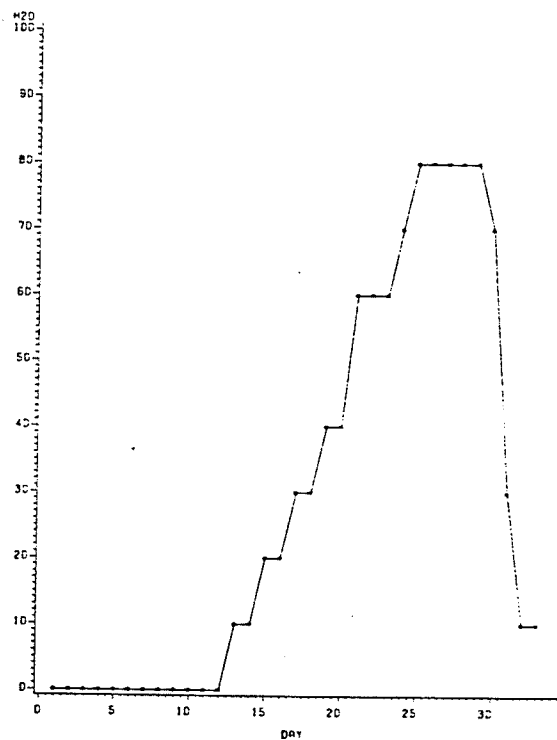
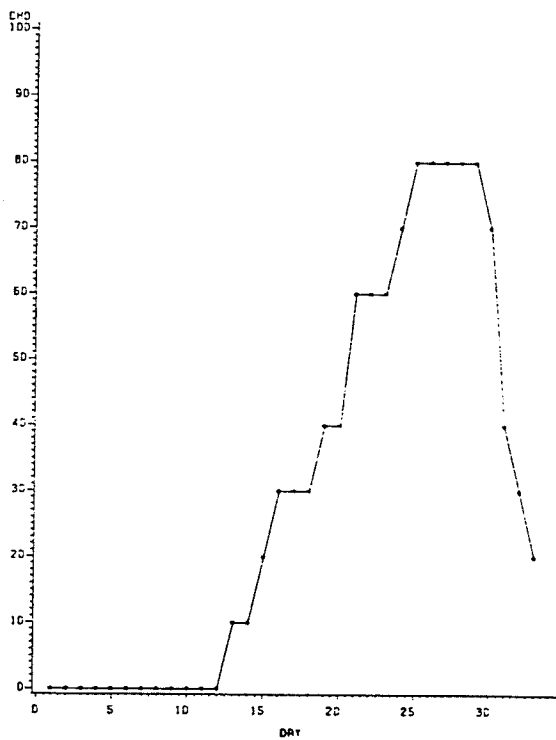
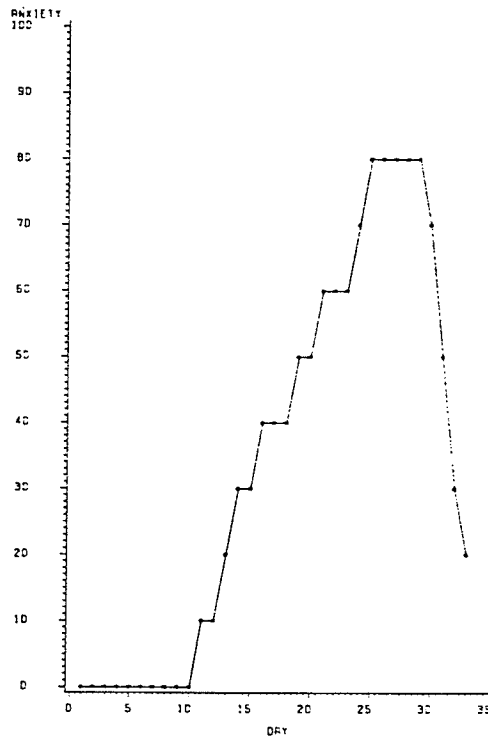
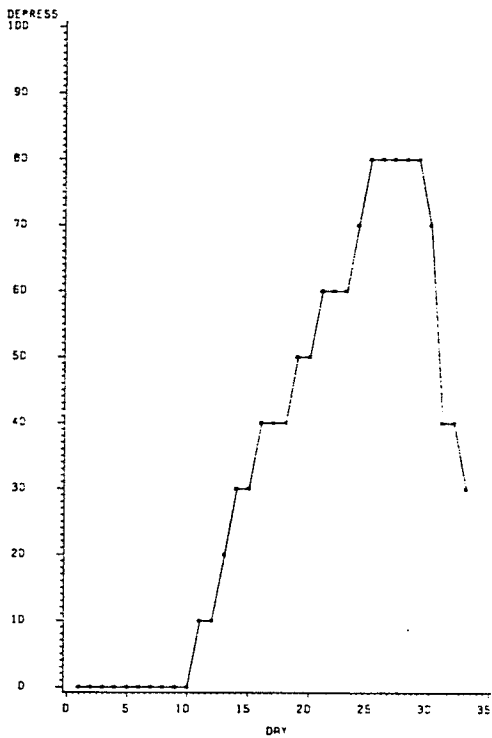
SUBJECT 3 : CYCLE 1 PMS SELF-RATINGS



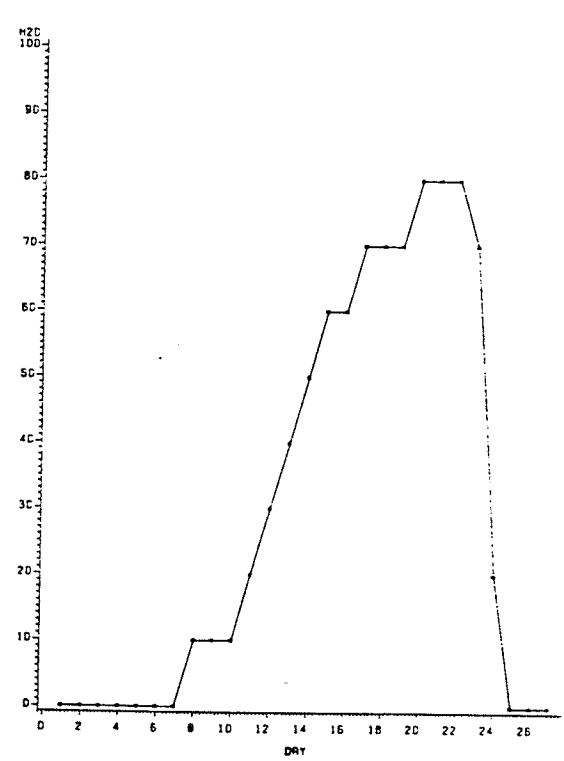
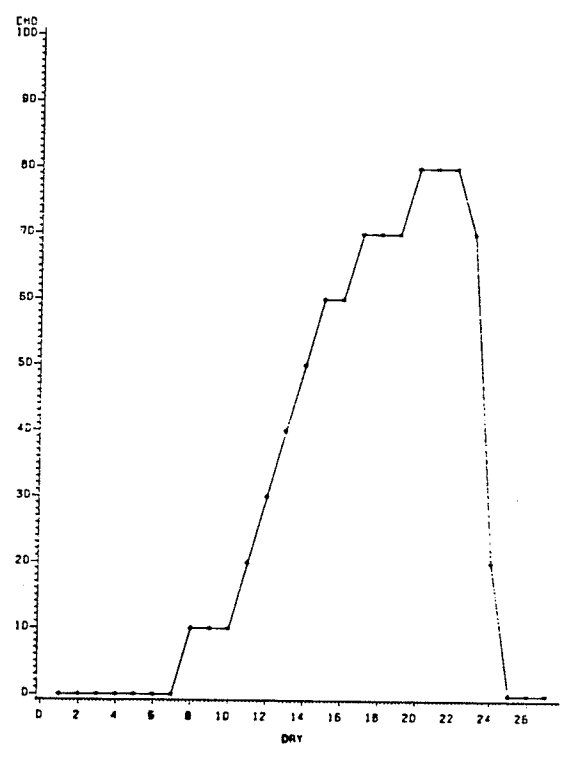
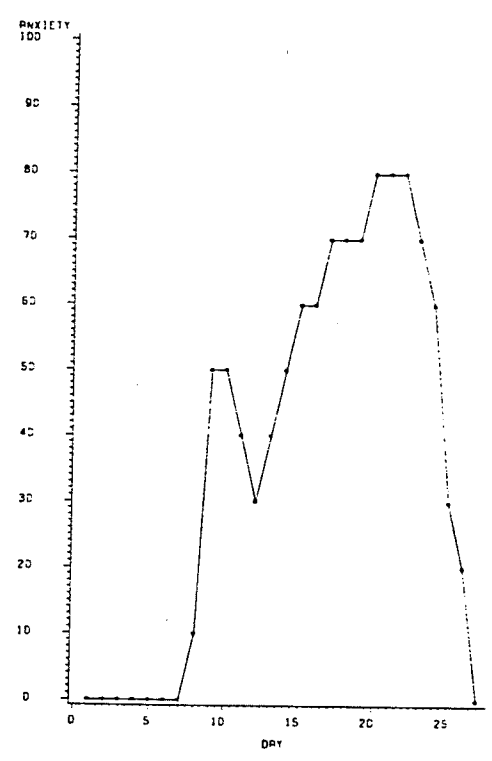
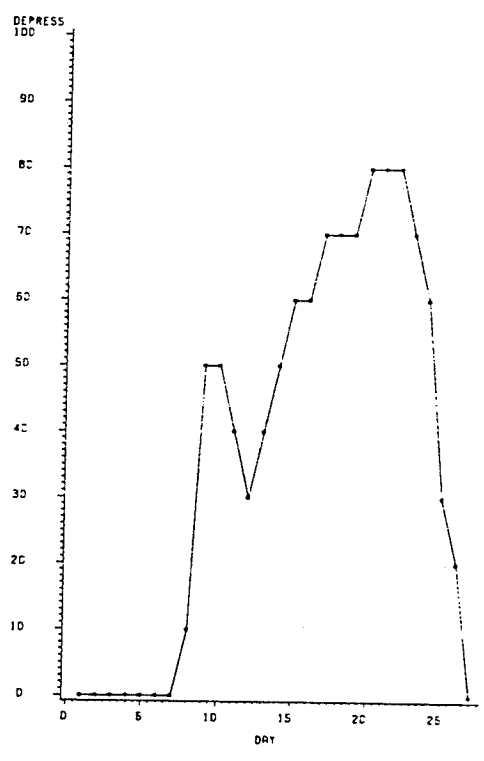
SUBJECT 3 : CYCLE 2 PMS SELF-RATINGS



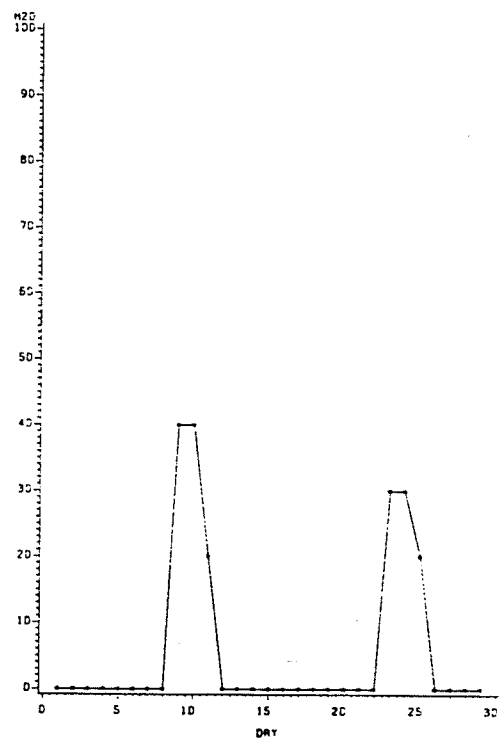
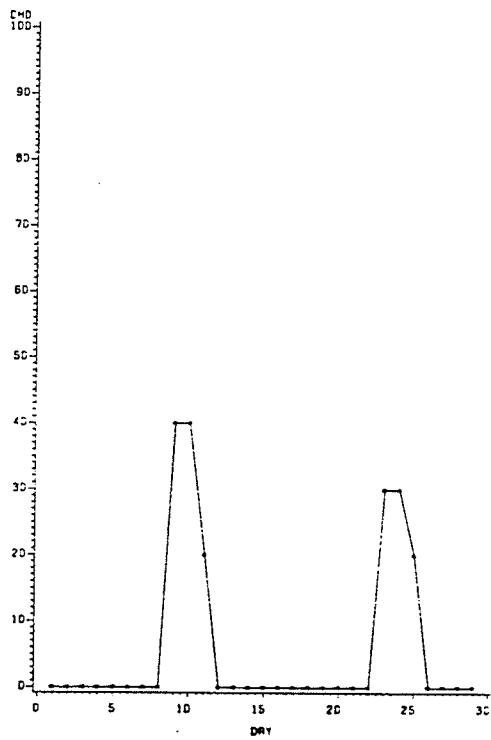
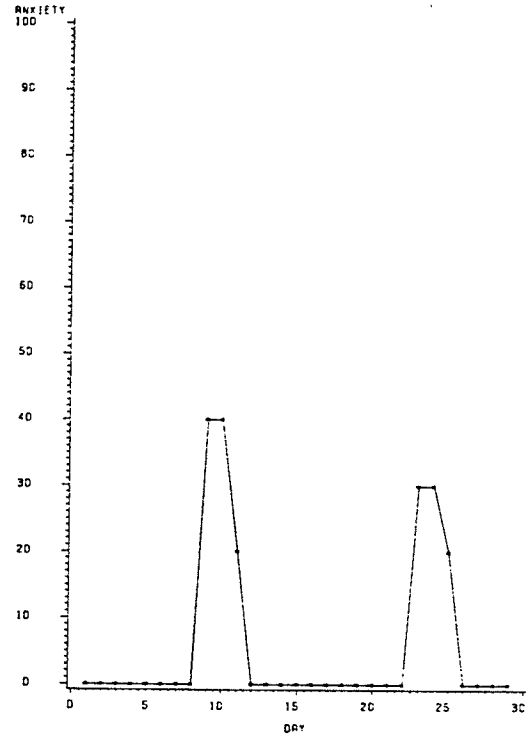
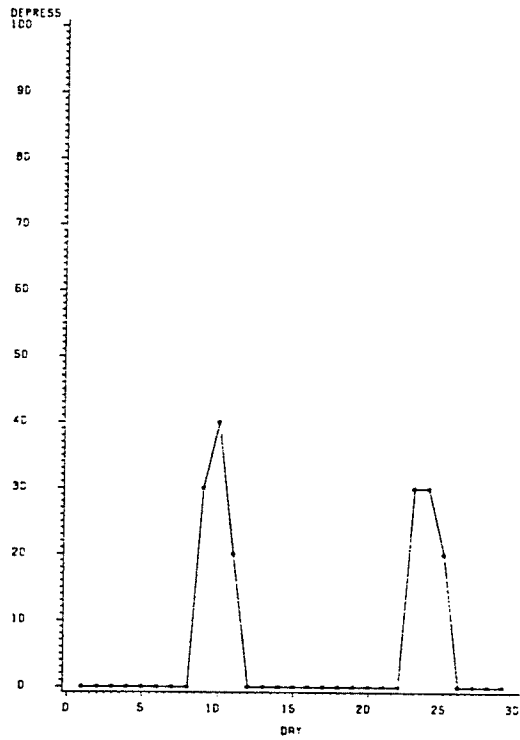
SUBJECT 3 : CYCLE 3 PMS SELF-RATINGS



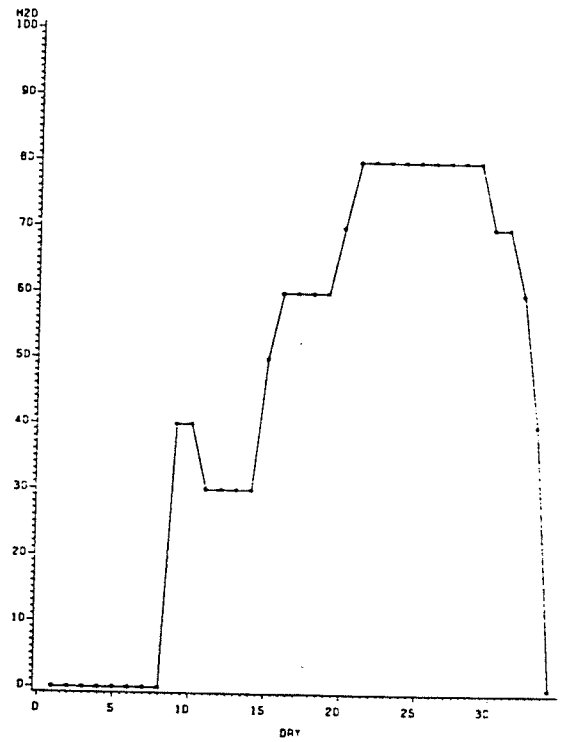
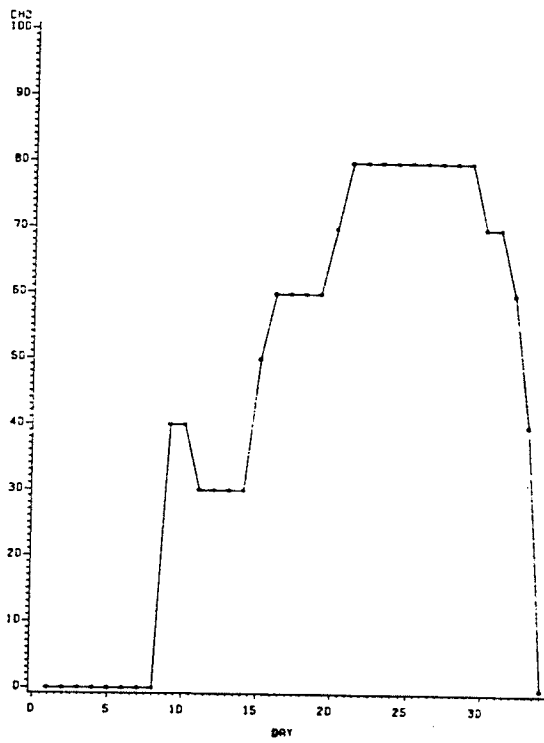
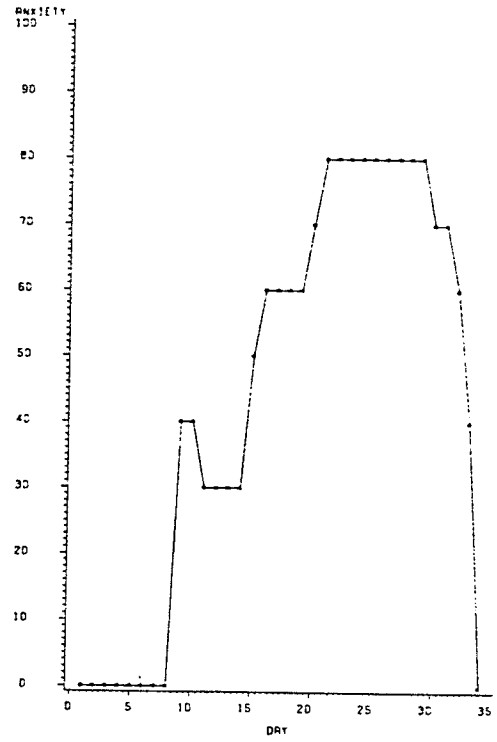
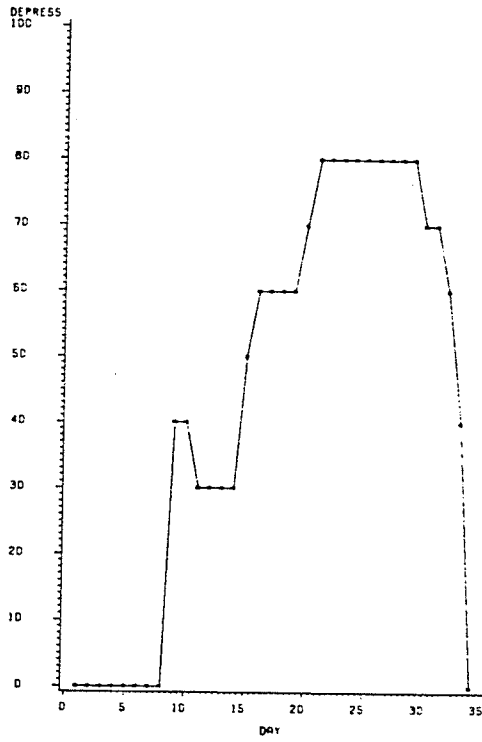
SUBJECT 3 : CYCLE 4 PMS SELF-RATINGS



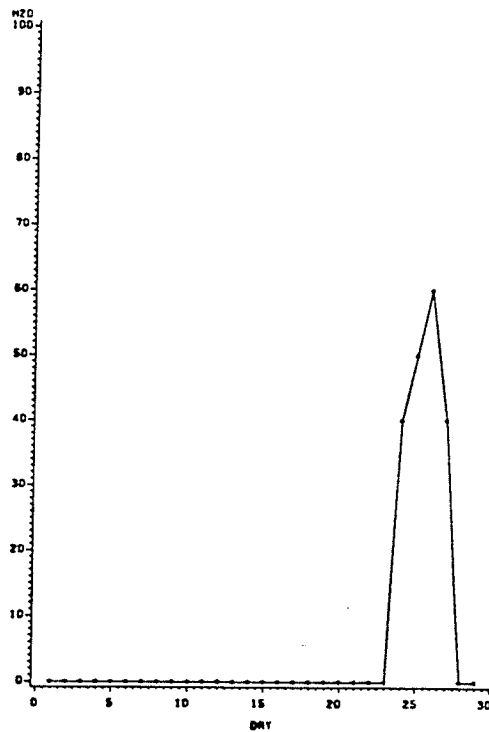
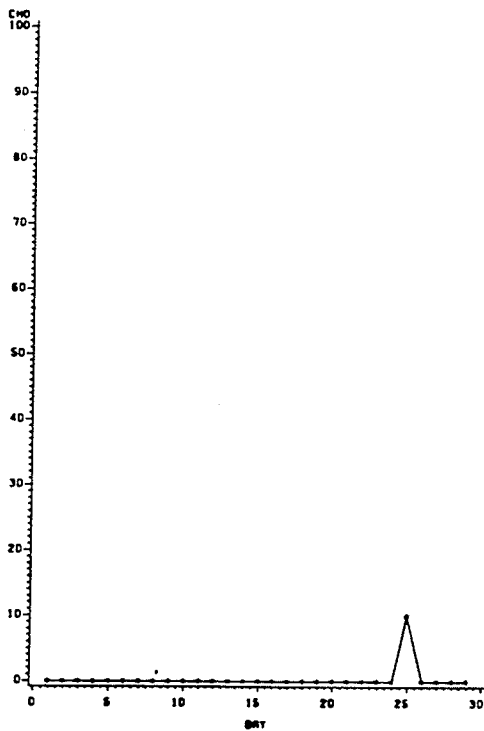
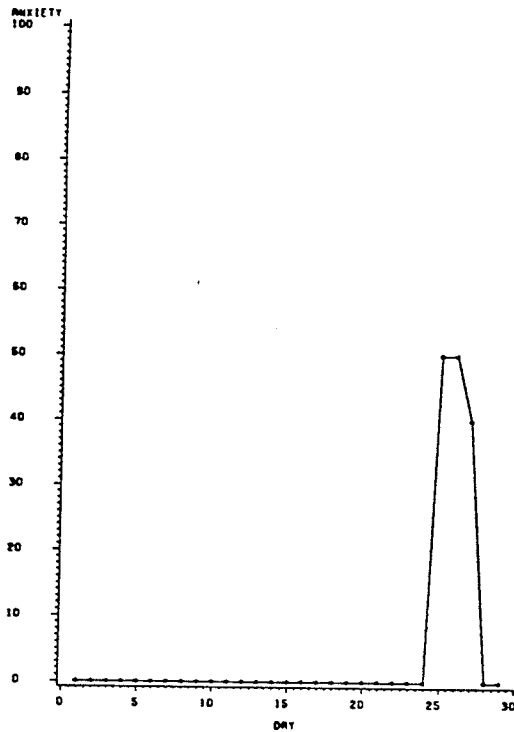
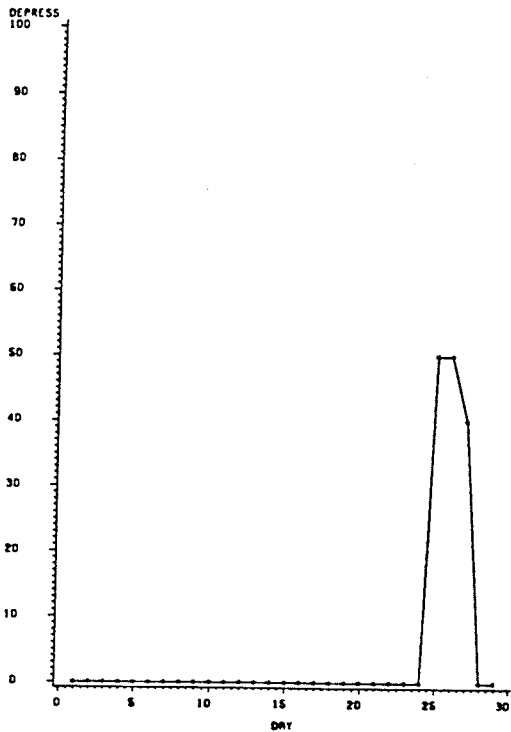
SUBJECT 3 : CYCLE 5 PMS SELF-RATINGS



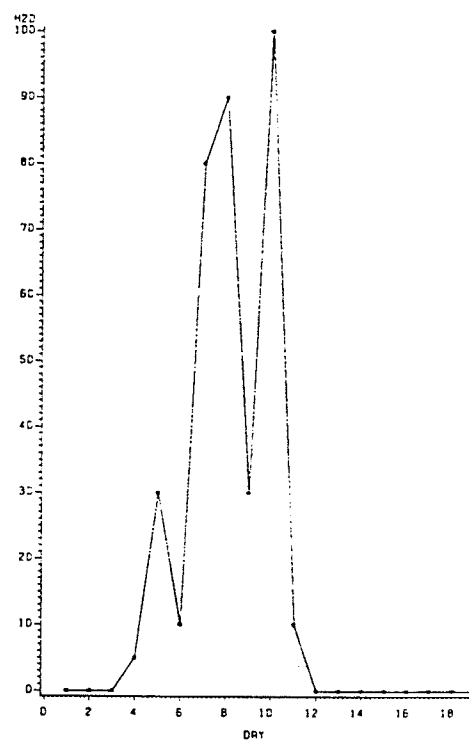
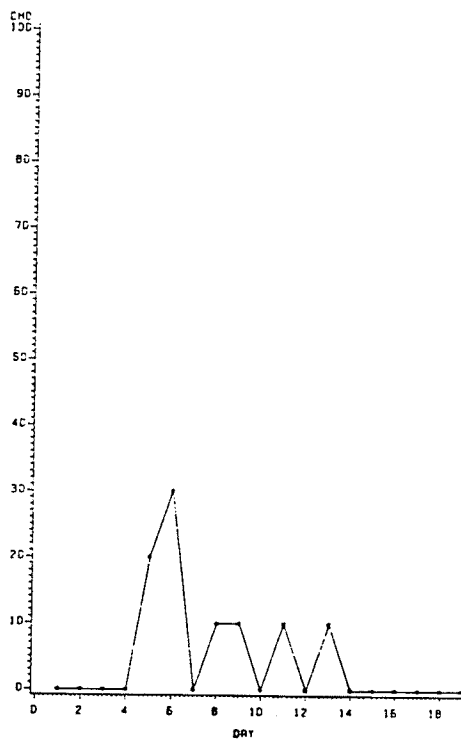
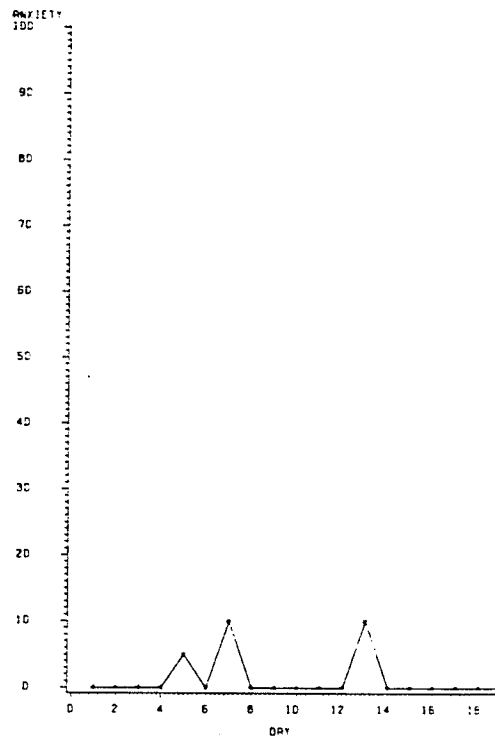
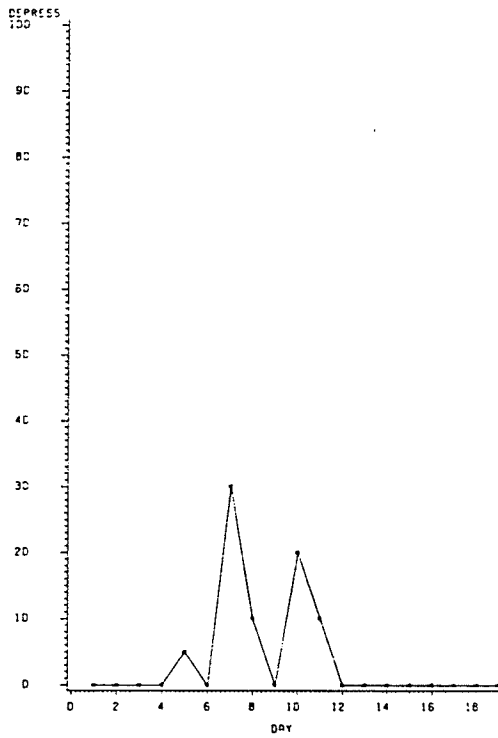
SUBJECT 3 : CYCLE 6 PMS SELF-RATINGS



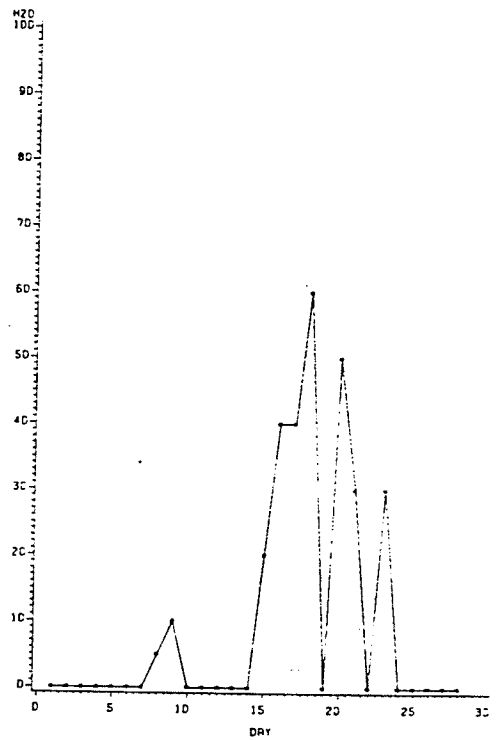
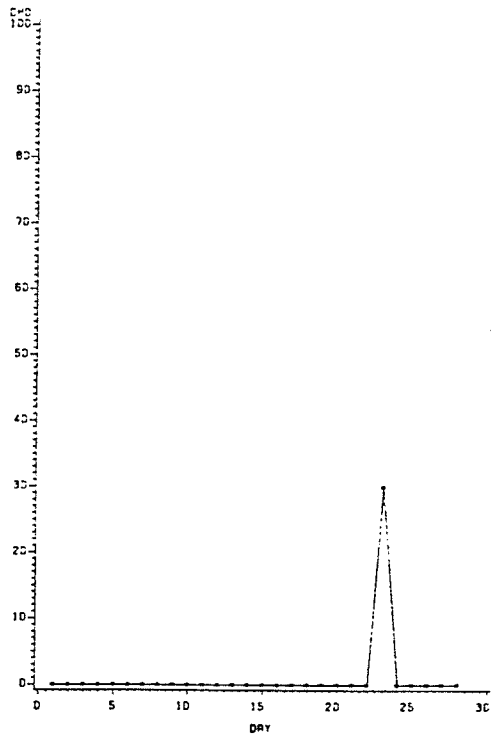
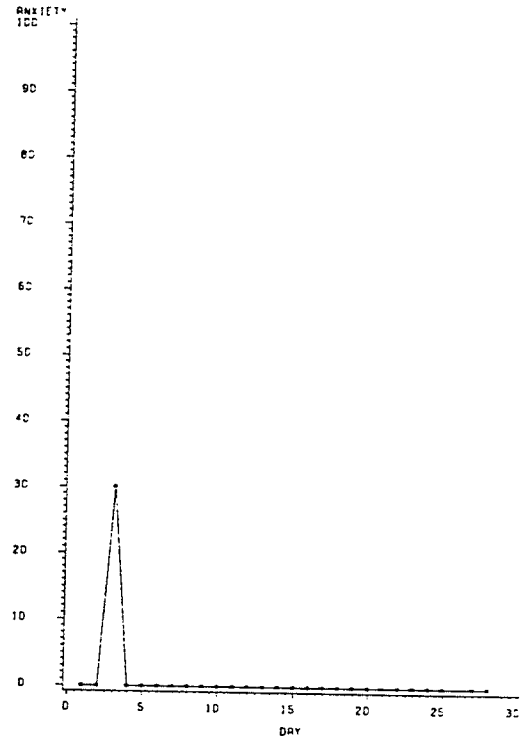
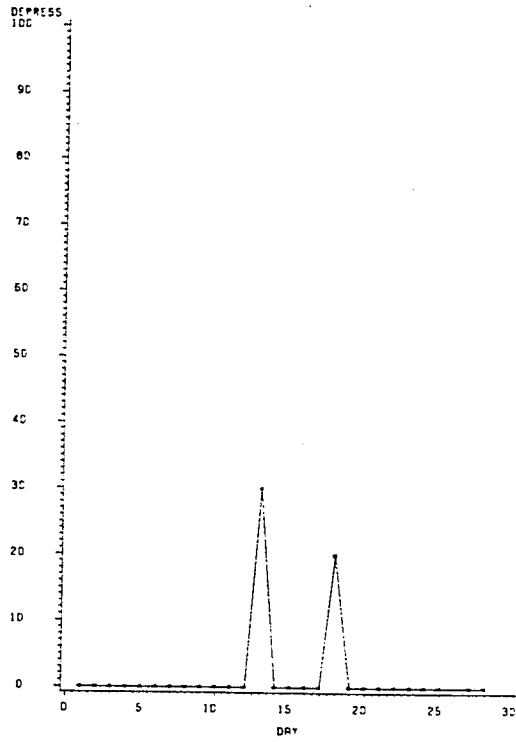
SUBJECT 3 : CYCLE 7 PMS SELF-RATINGS



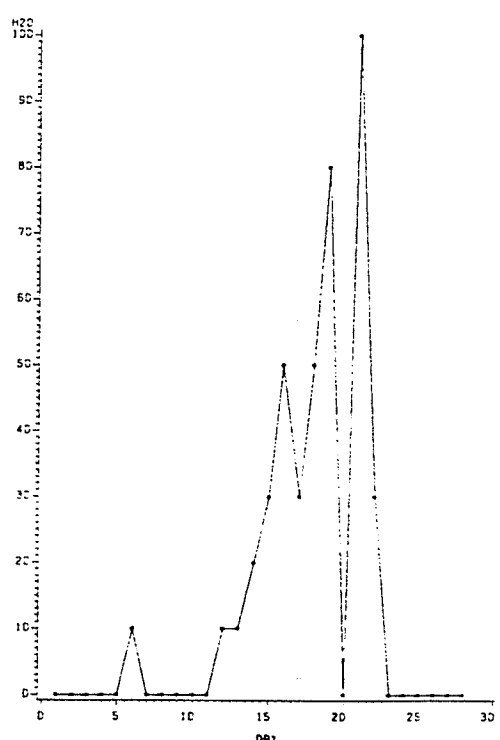
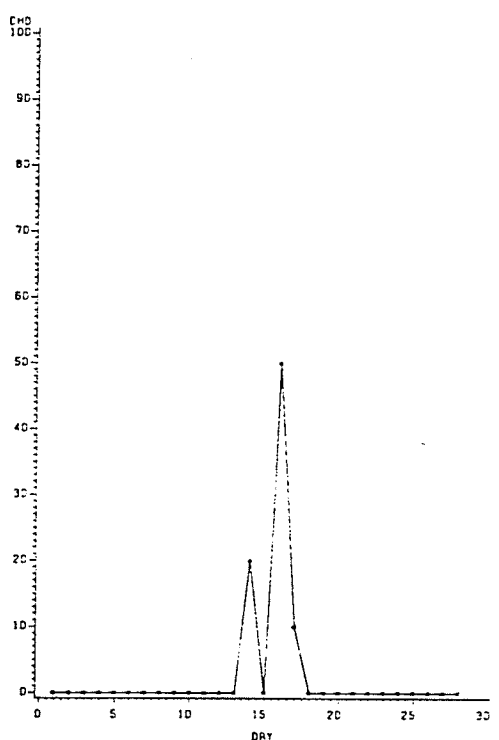
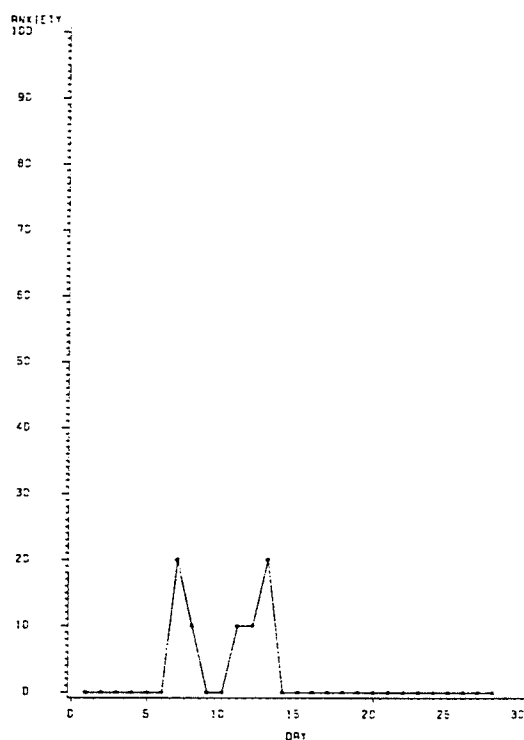
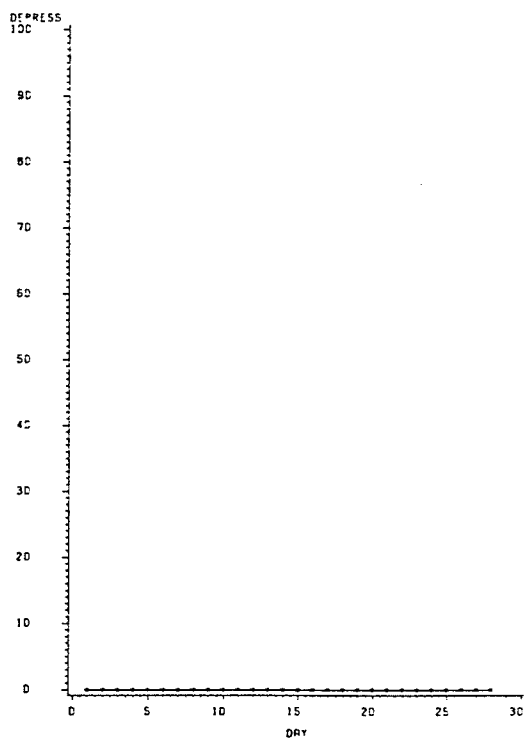
SUBJECT 4 : CYCLE 1 PMS SELF-RATINGS



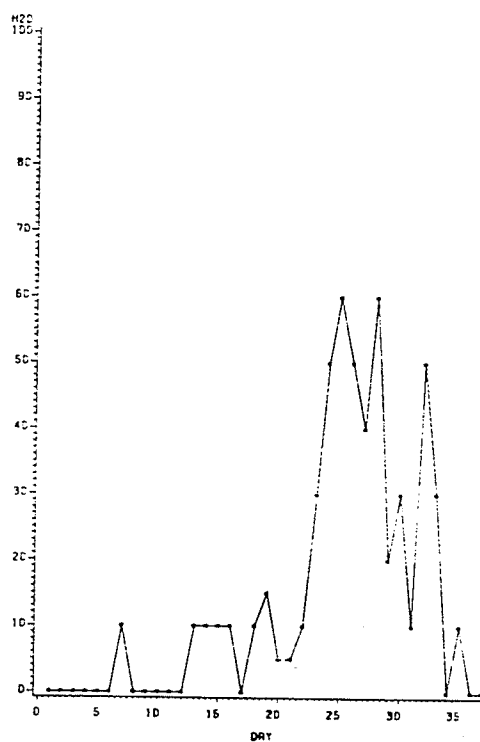
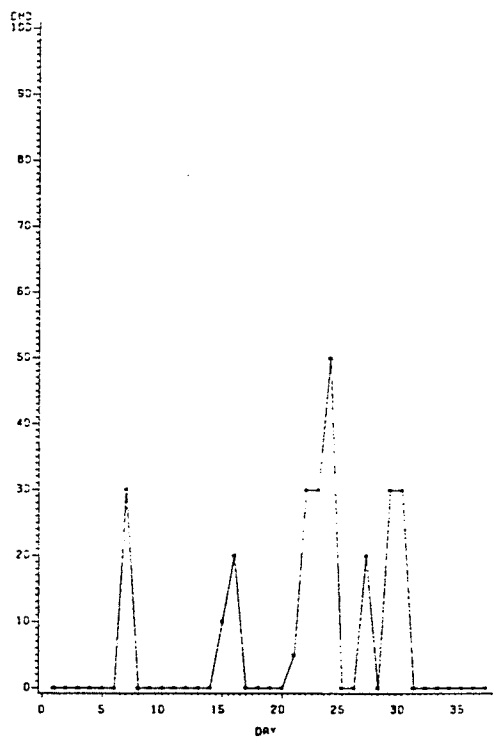
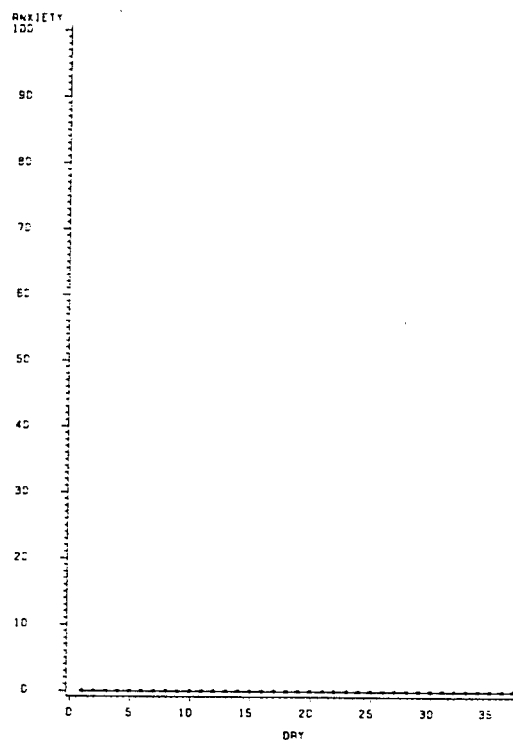
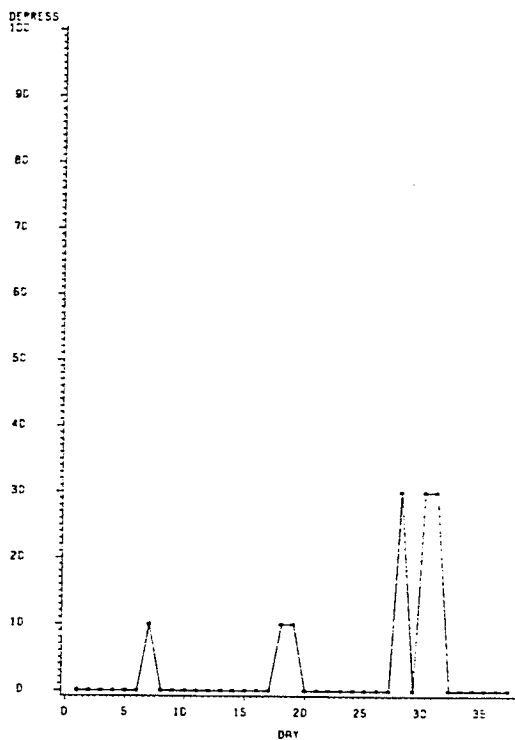
SUBJECT 4 : CYCLE 2 PMS SELF-RATINGS



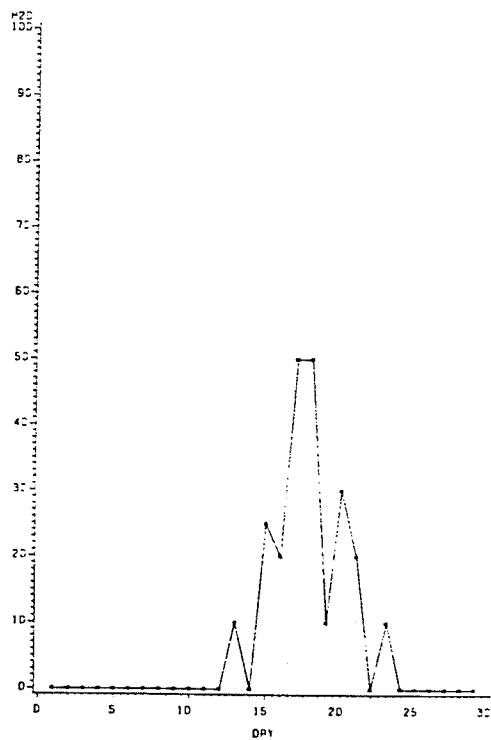
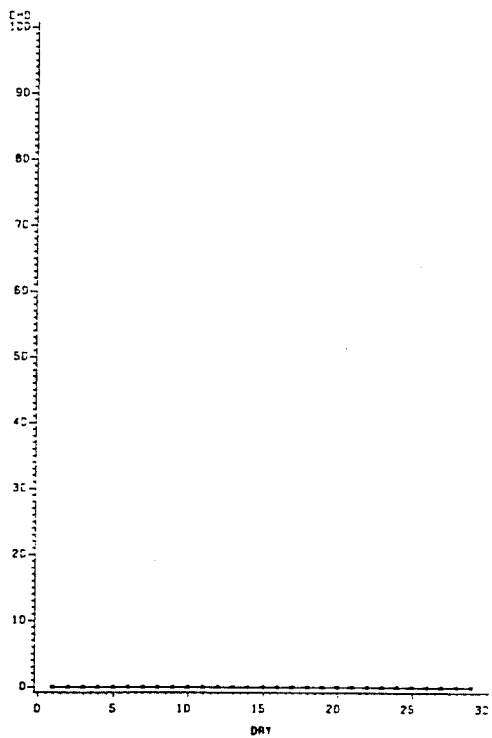
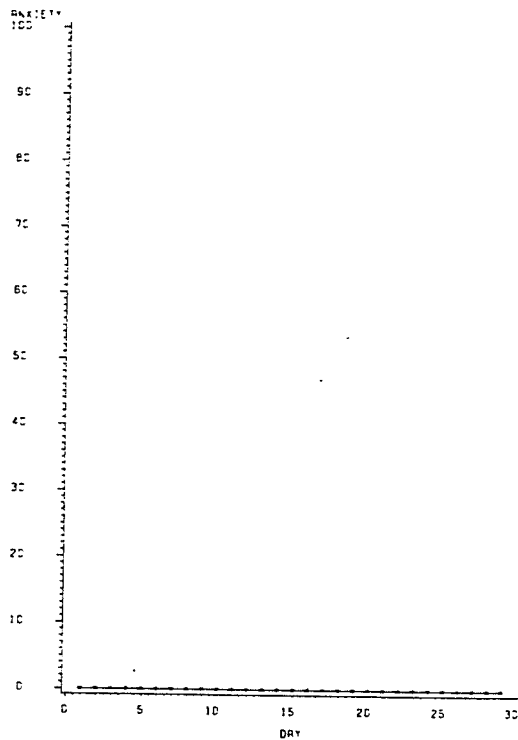
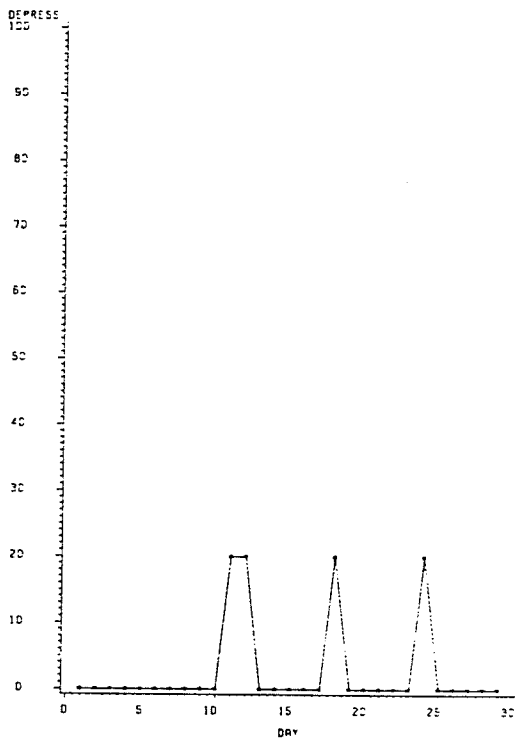
SUBJECT 4 : CYCLE 3 PMS SELF-RATINGS



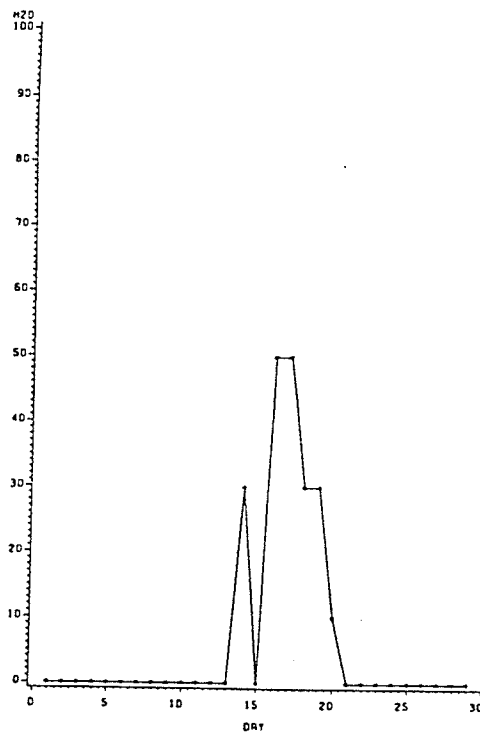
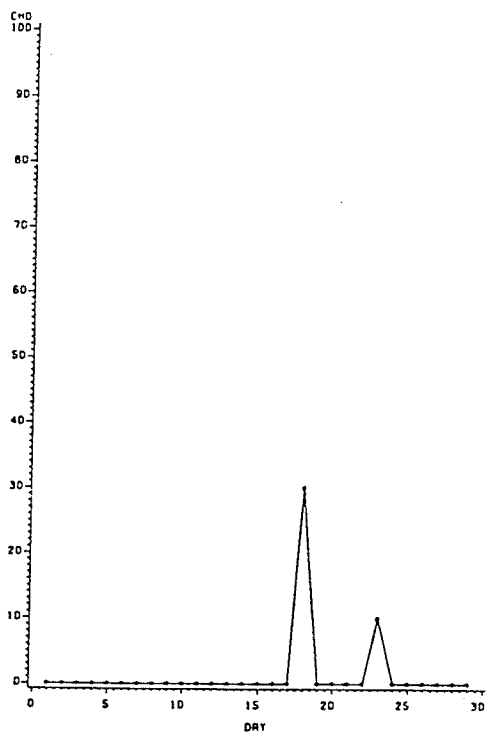
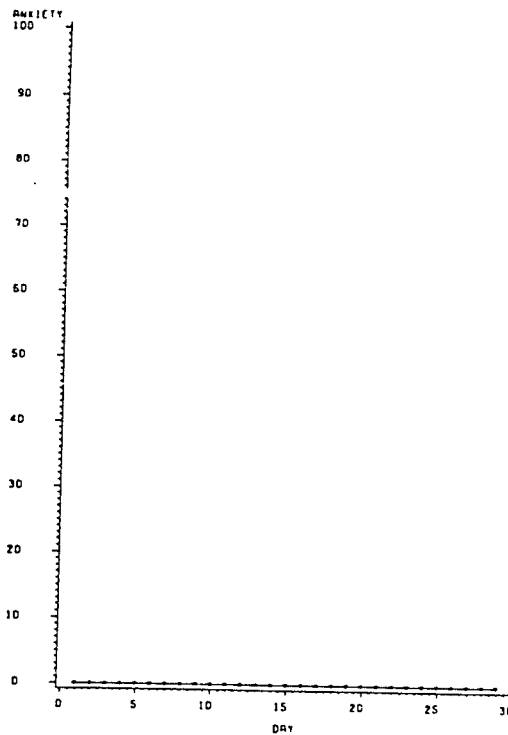
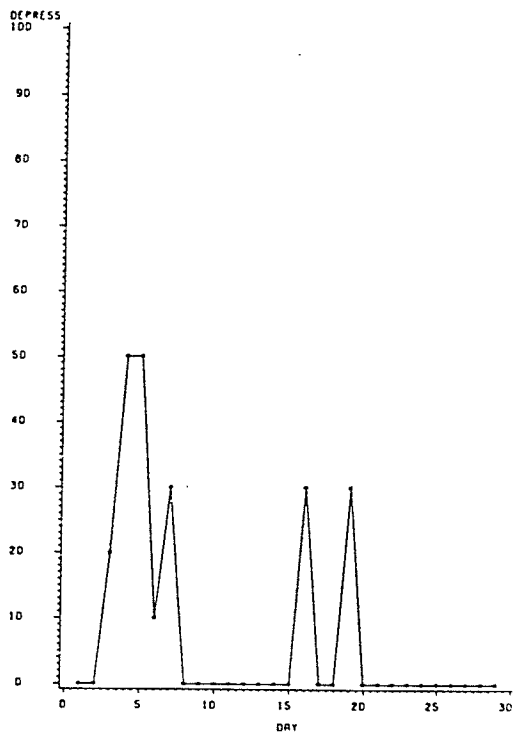
SUBJECT 4 : CYCLE 4 PMS SELF-RATINGS



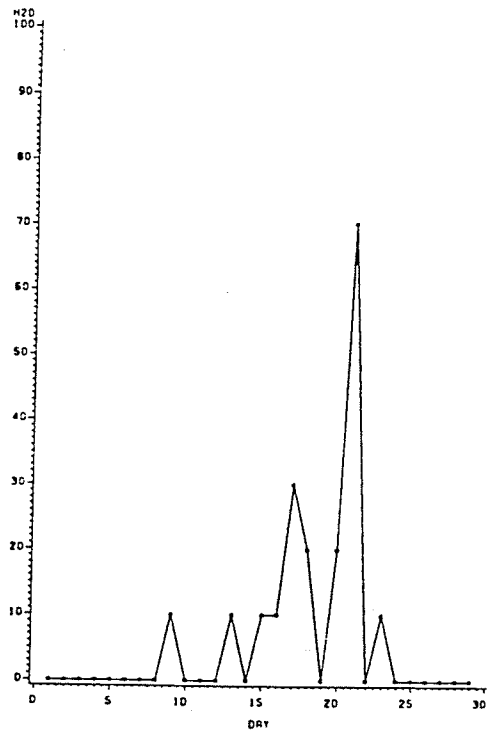
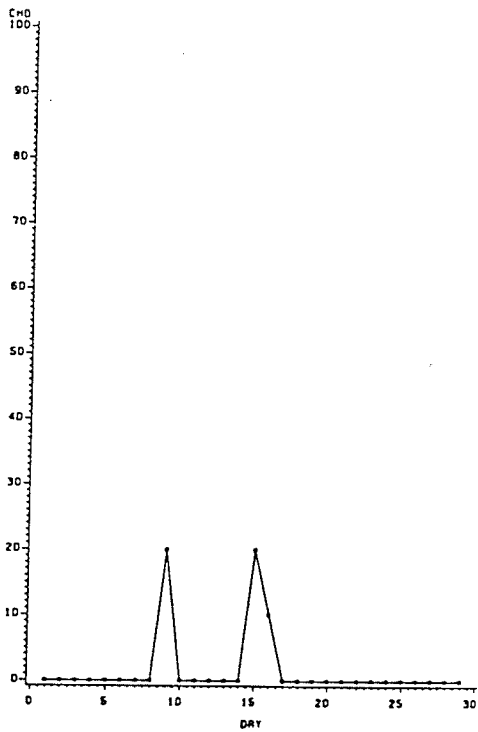
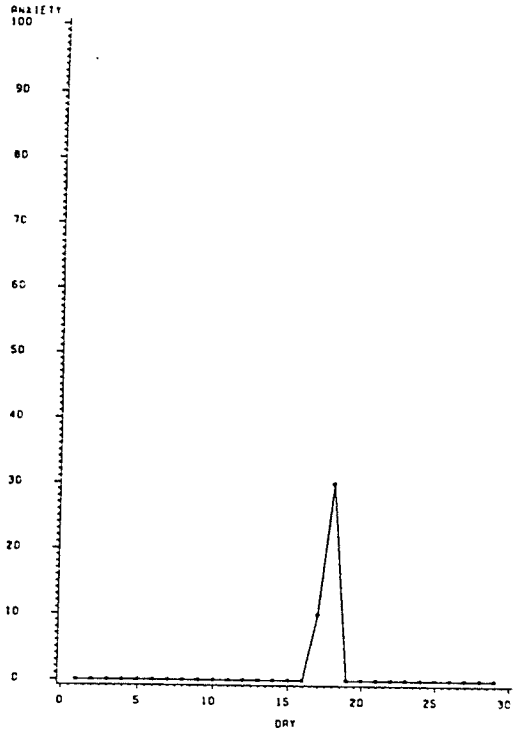
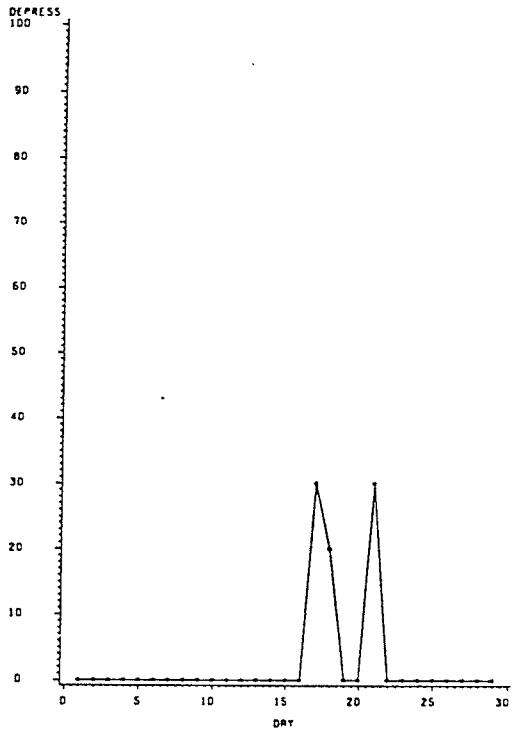
SUBJECT 4 : CYCLE 5 PMS SELF-RATINGS



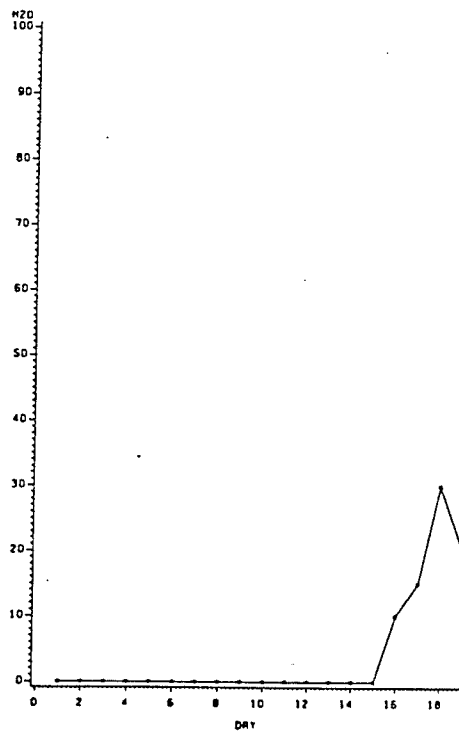
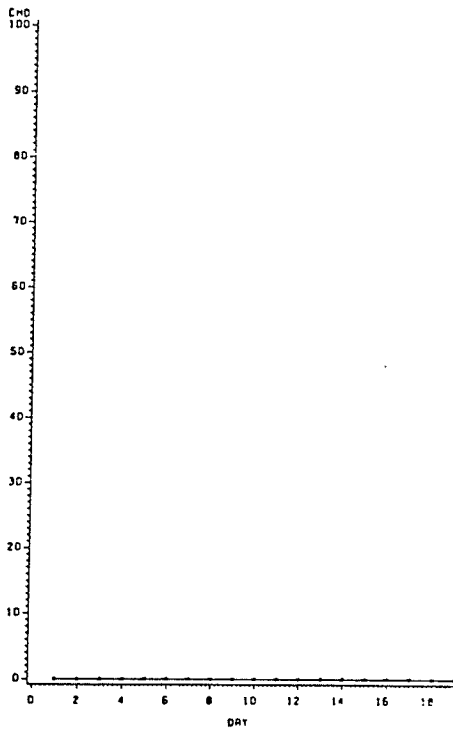
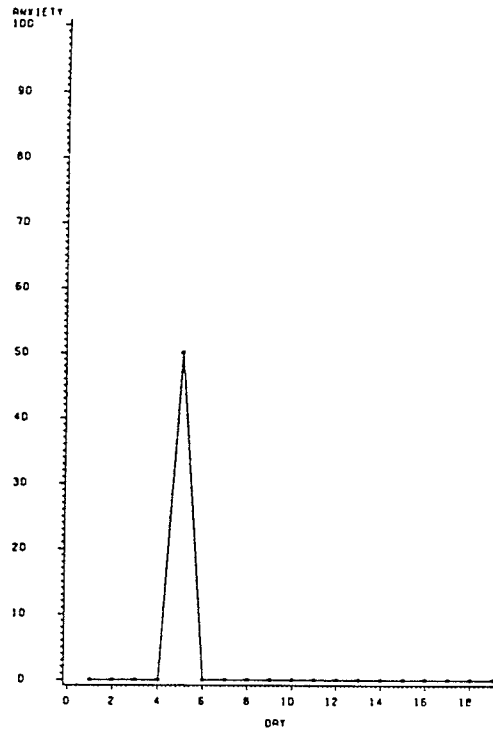
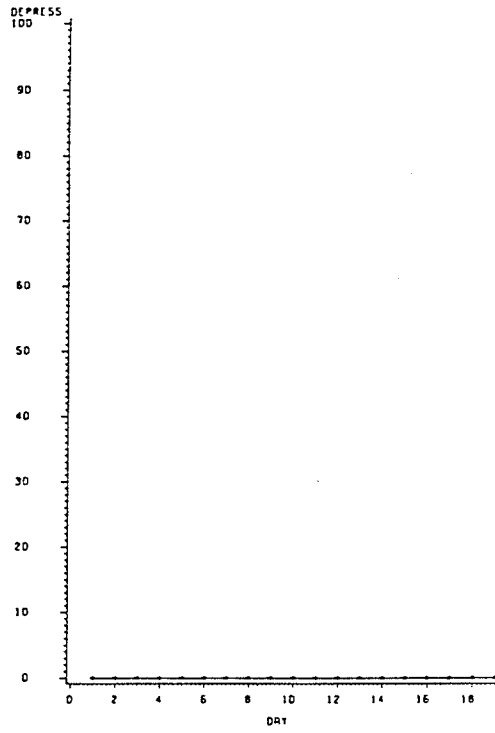
SUBJECT 4 : CYCLE 6 PMS SELF-RATINGS



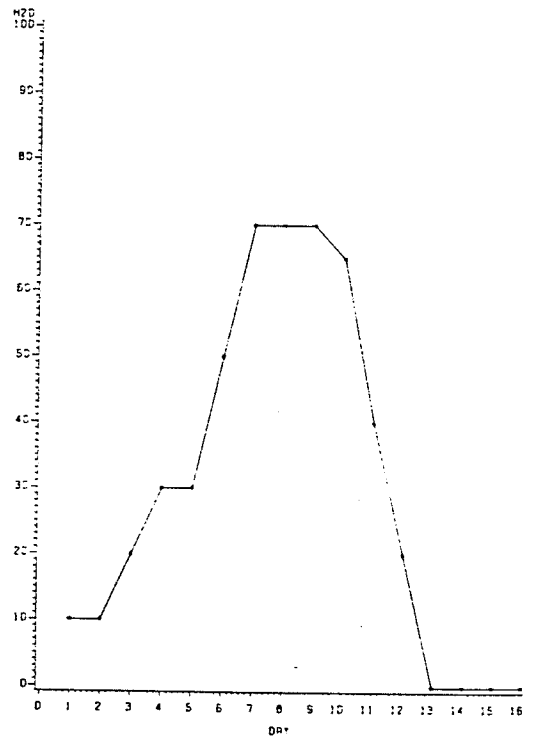
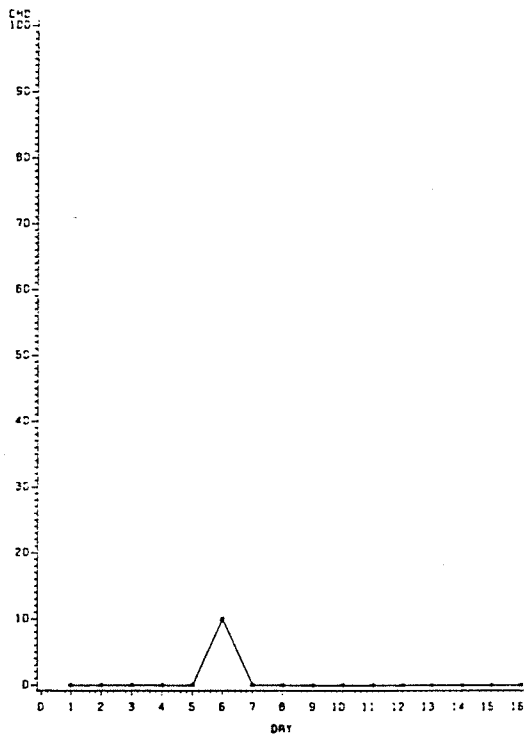
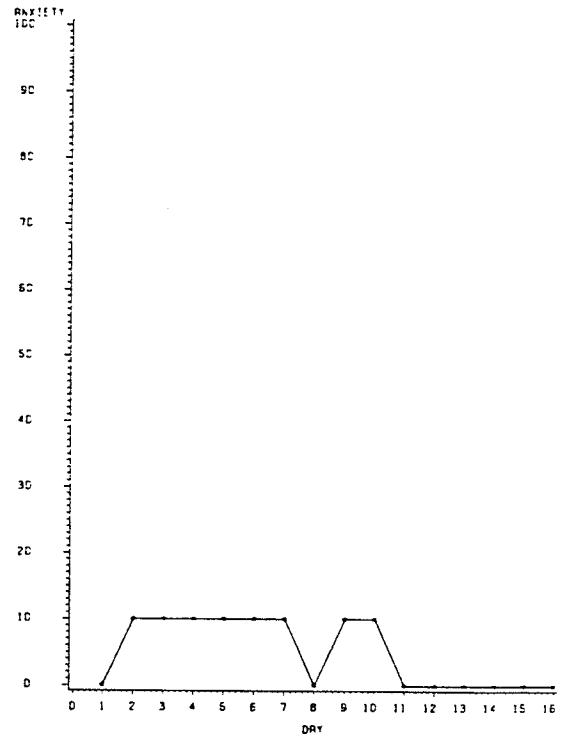
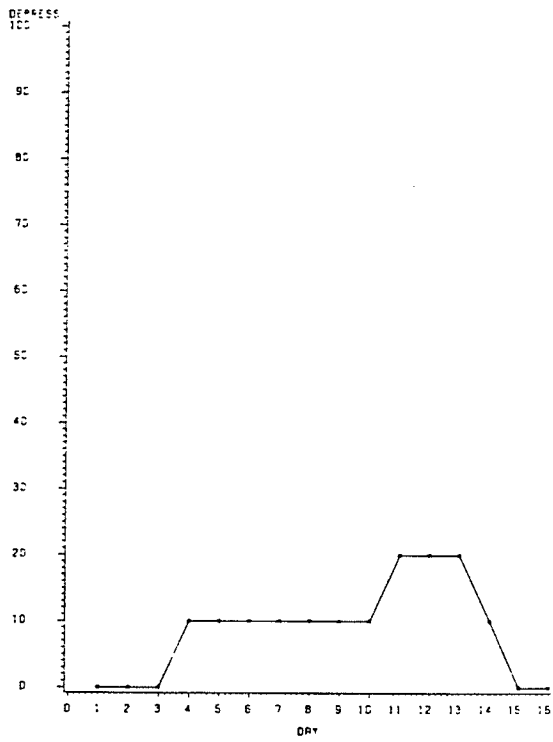
SUBJECT 4 : CYCLE 7 PMS SELF-RATINGS



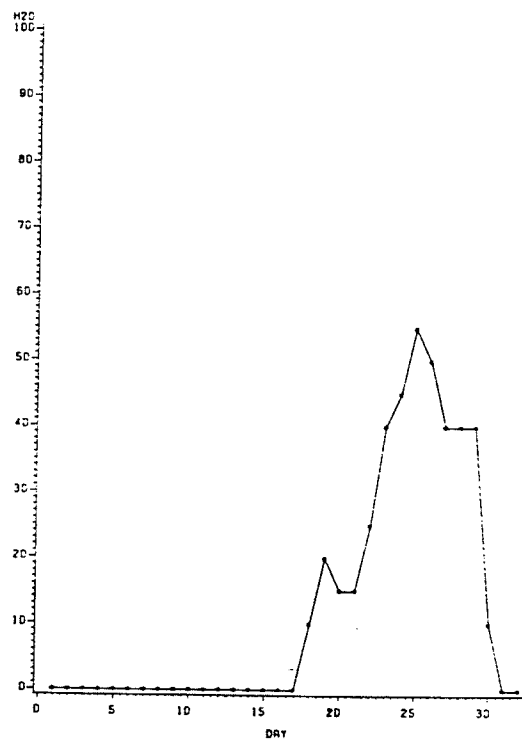
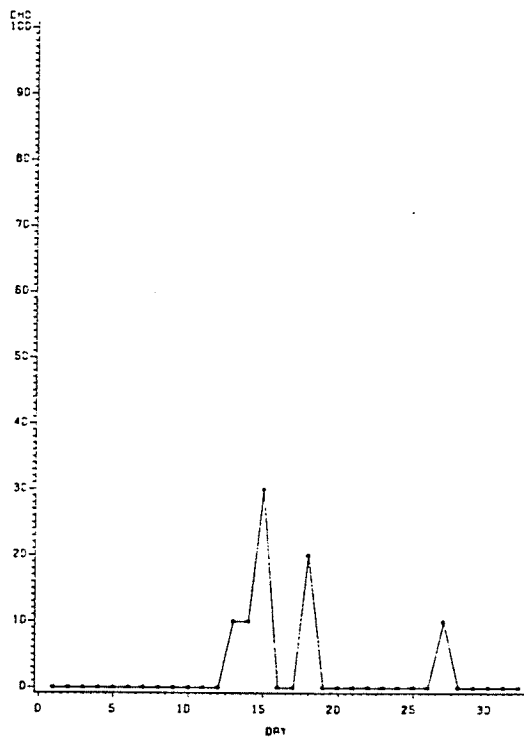
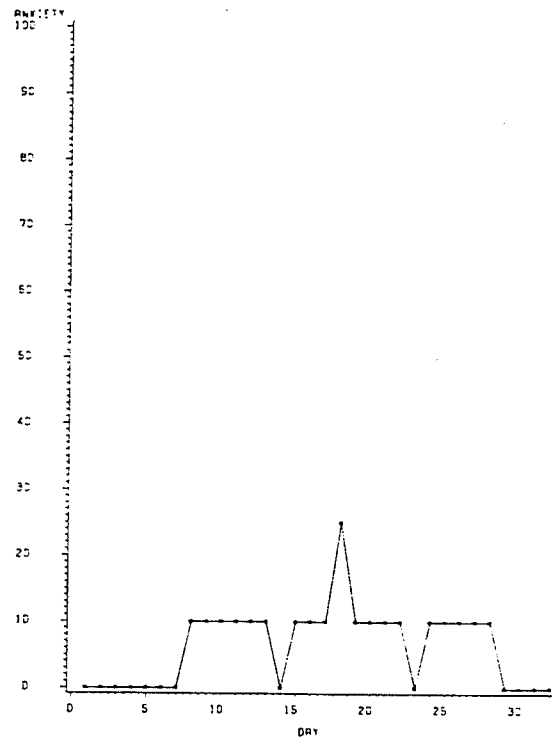
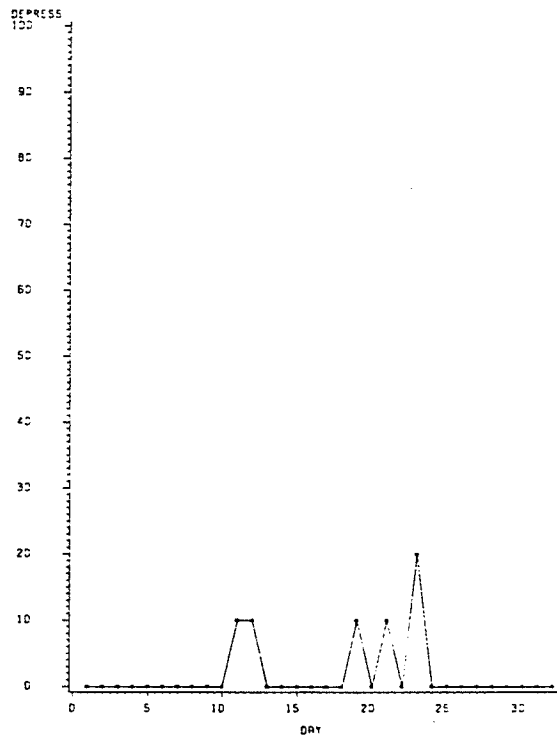
SUBJECT 4 : CYCLE 8 PMS SELF-RATINGS



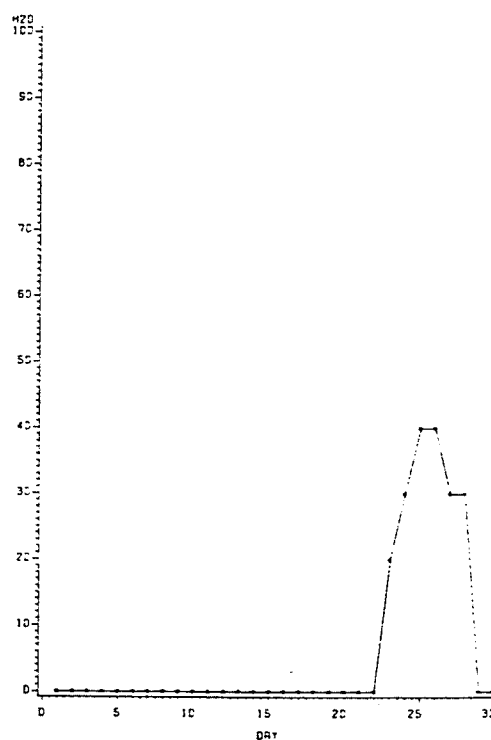
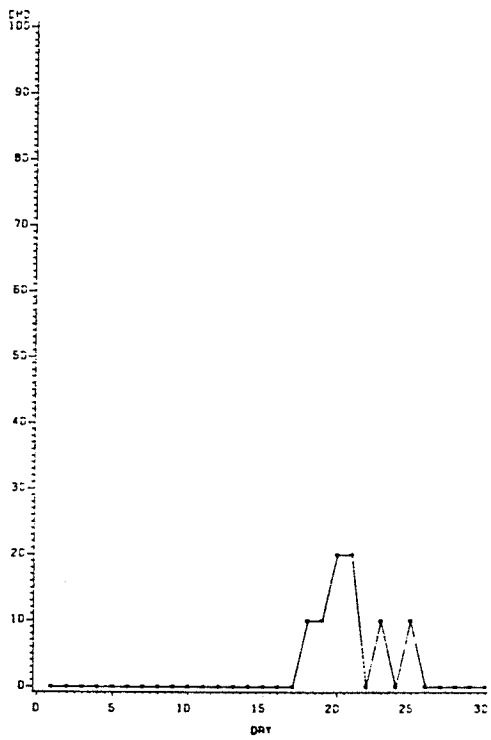
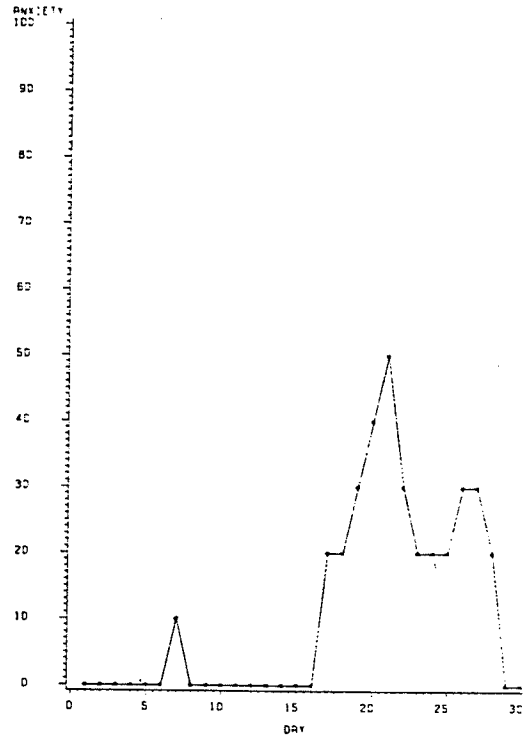
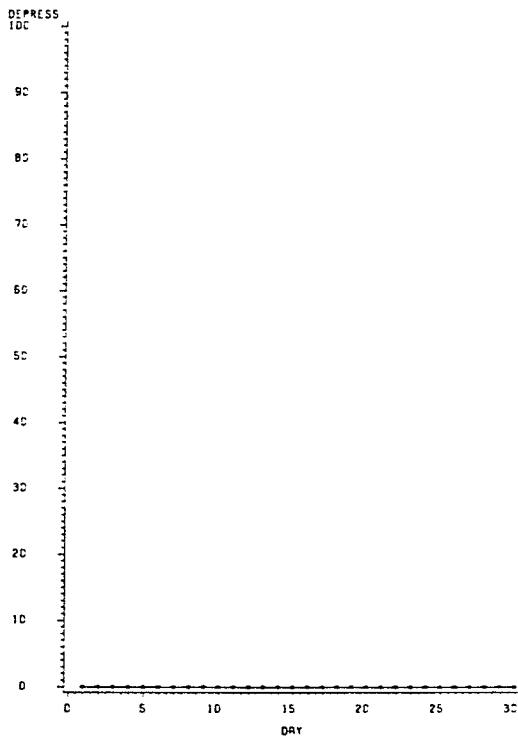
SUBJECT 5 : CYCLE 1 PMS SELF-RATINGS



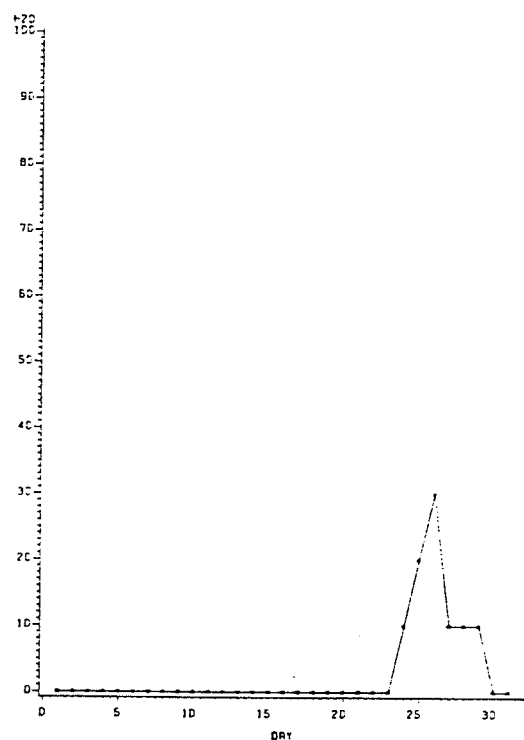
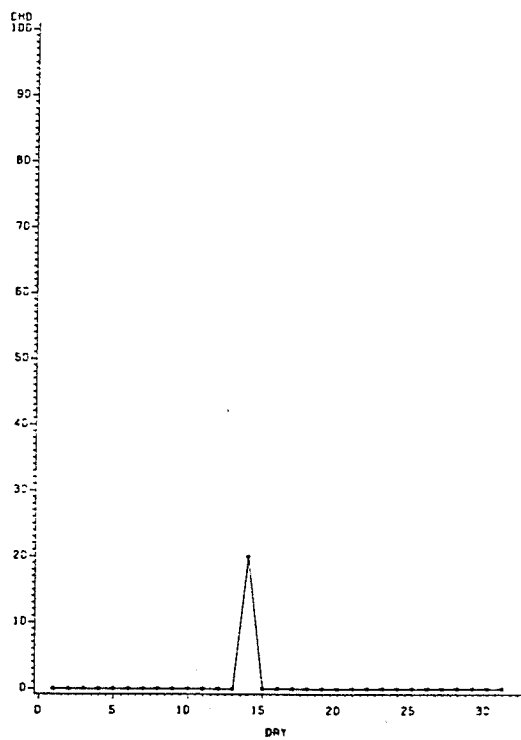
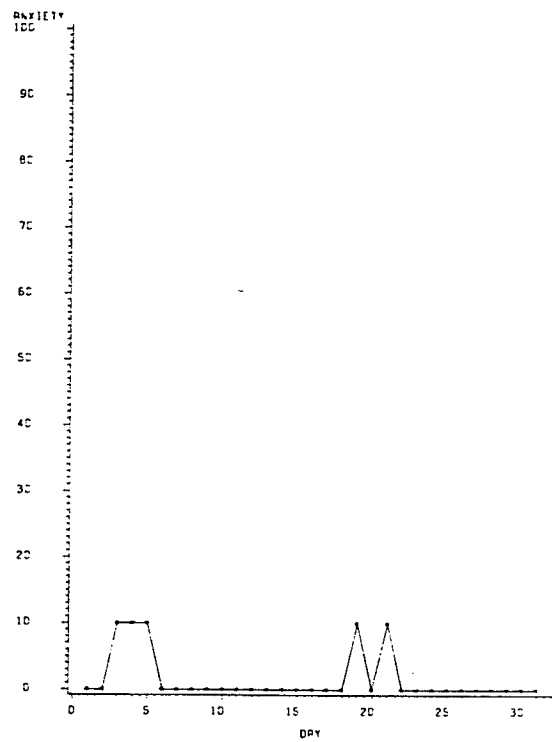
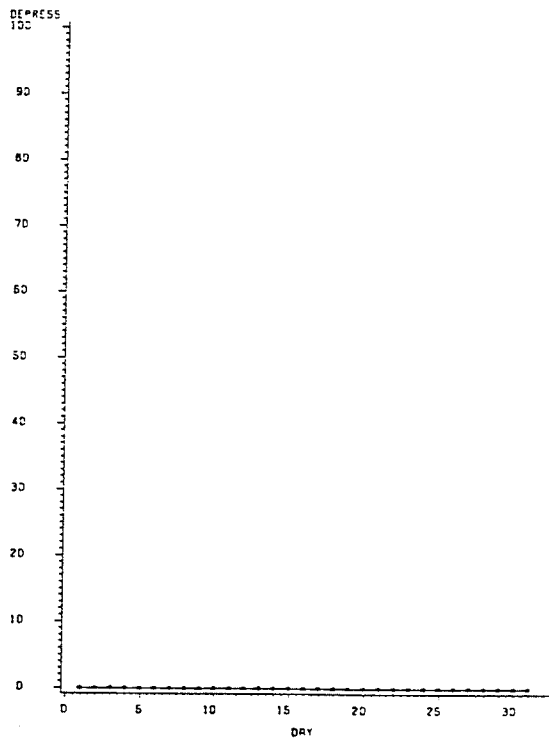
SUBJECT 5 : CYCLE 2 PMS SELF-RATINGS



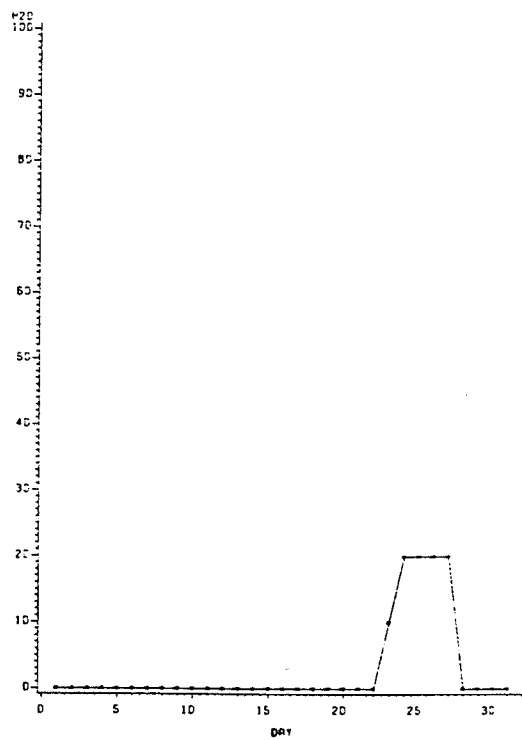
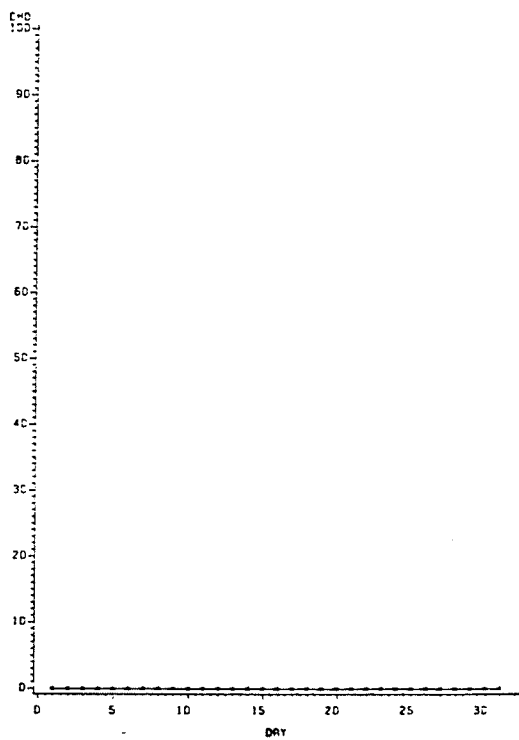
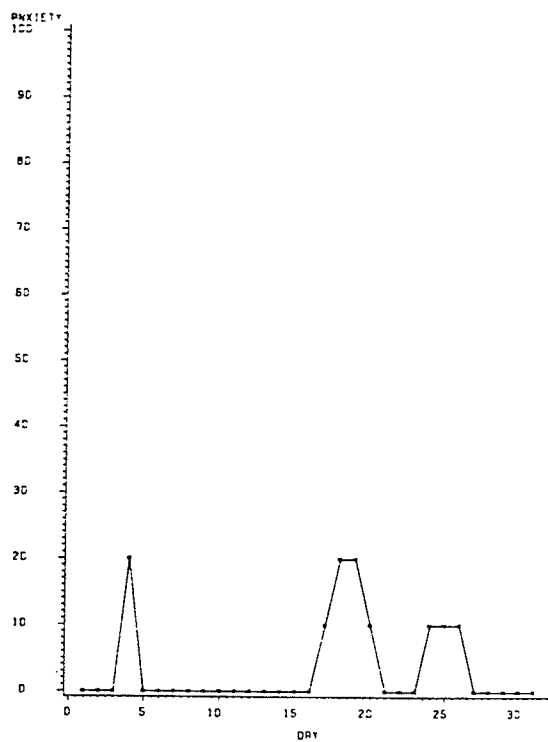
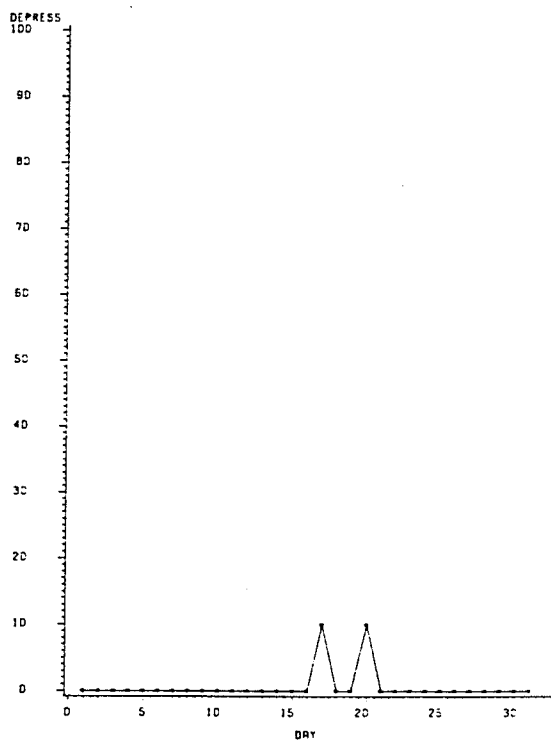
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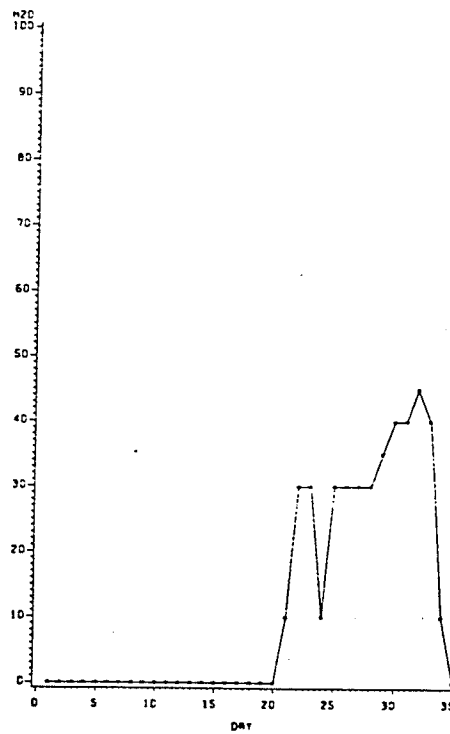
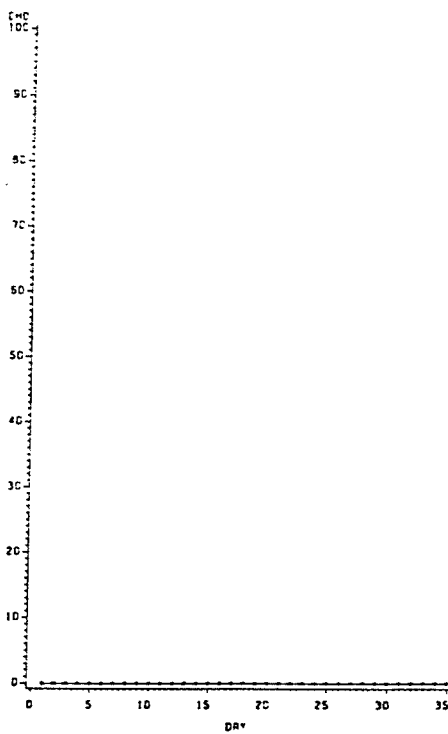
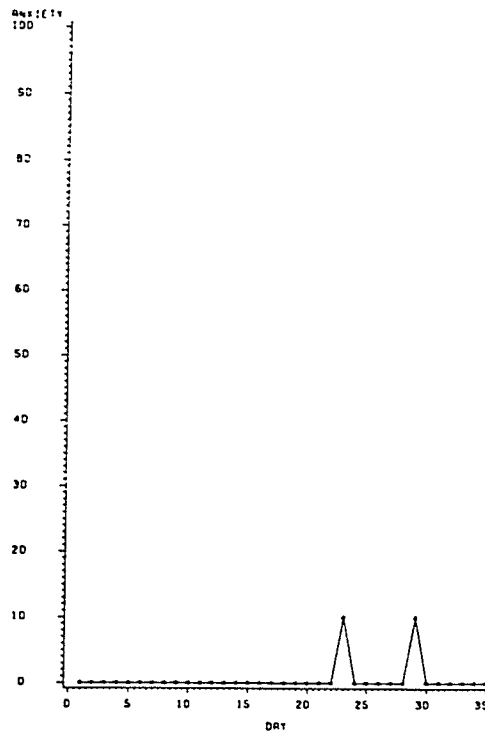
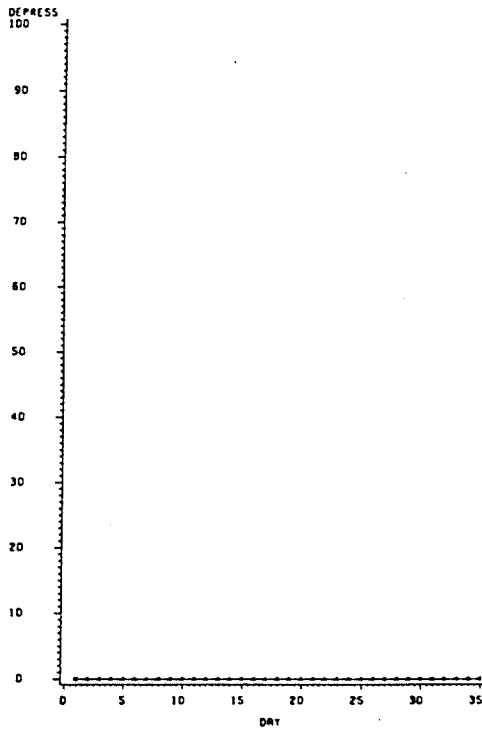
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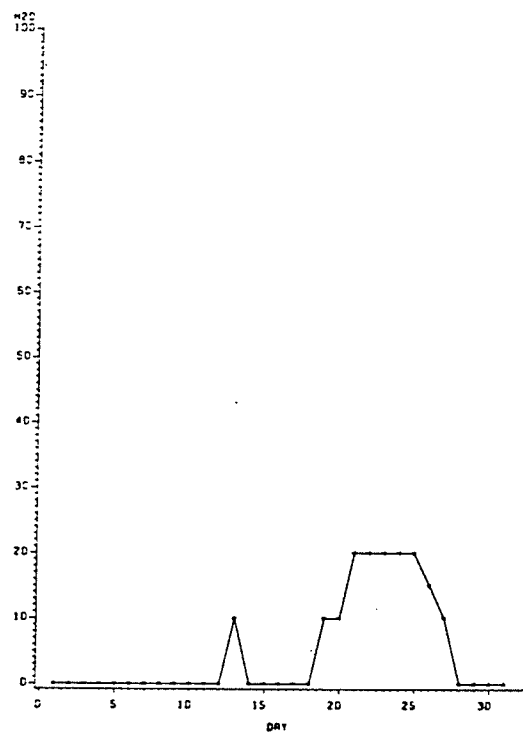
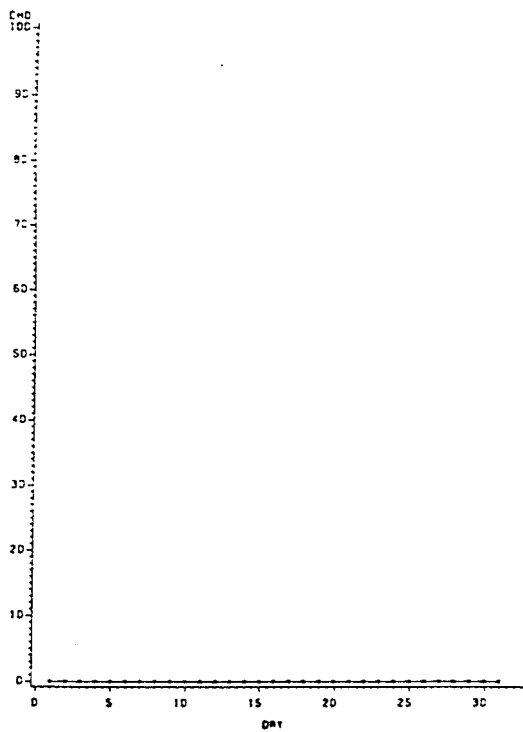
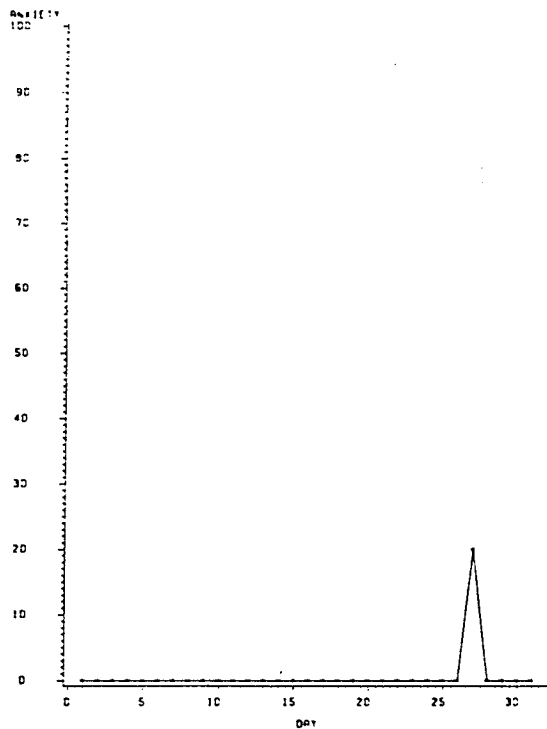
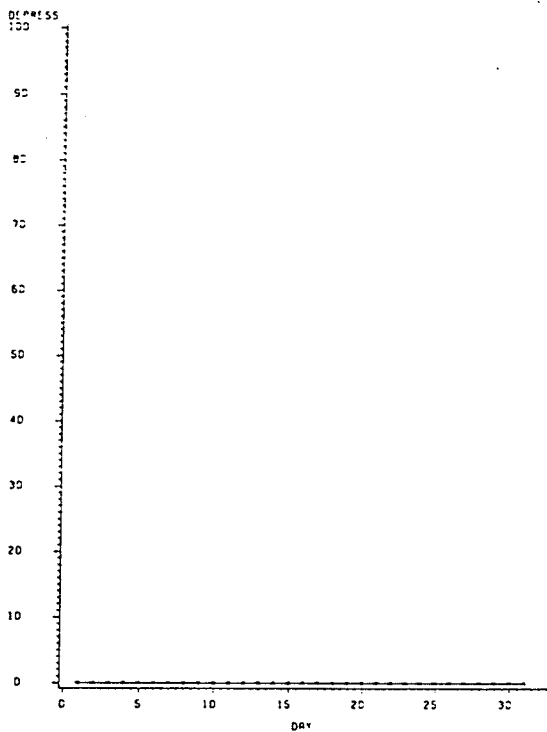
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SUBJECT 5 : CYCLE 6 PMS SELF-RATINGS

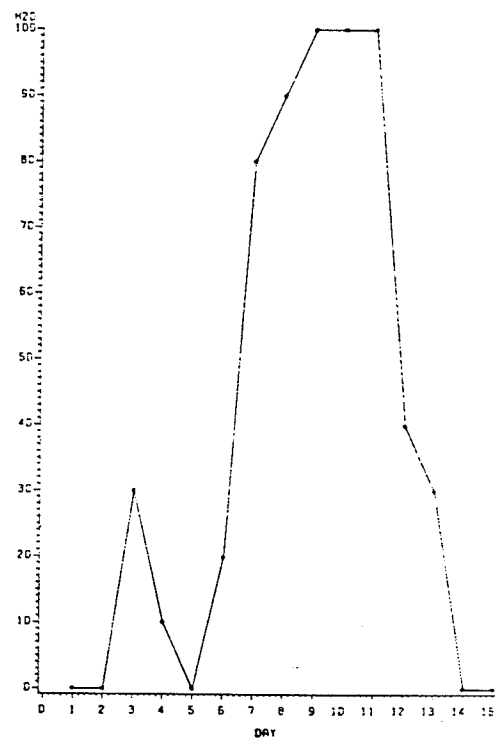
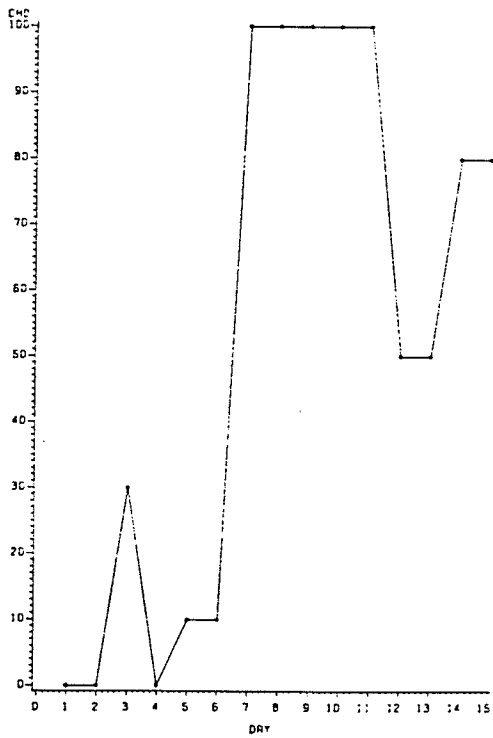
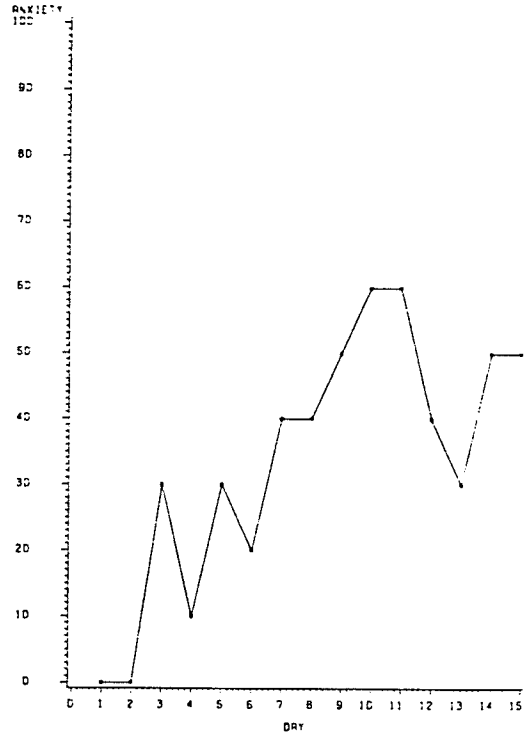
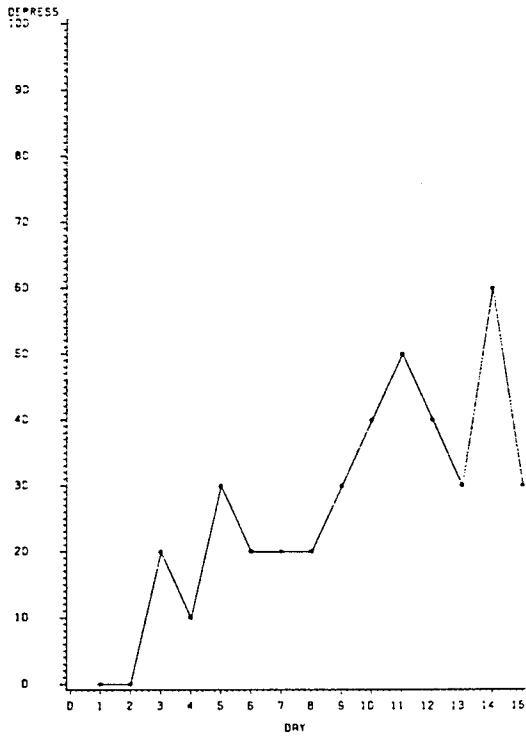


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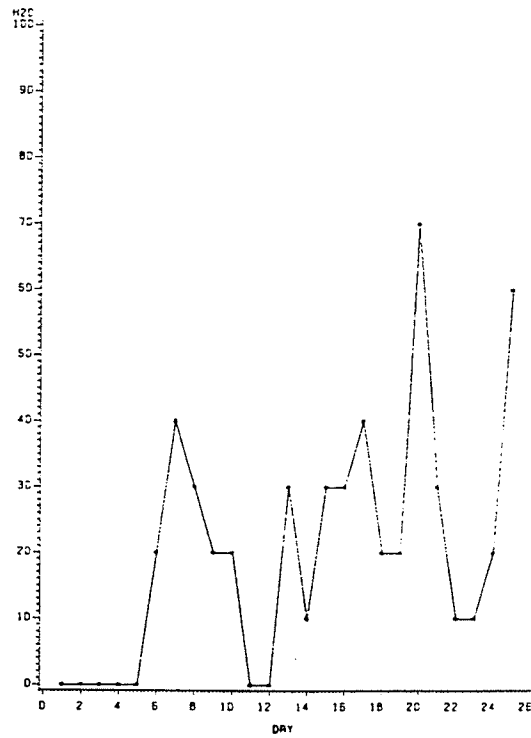
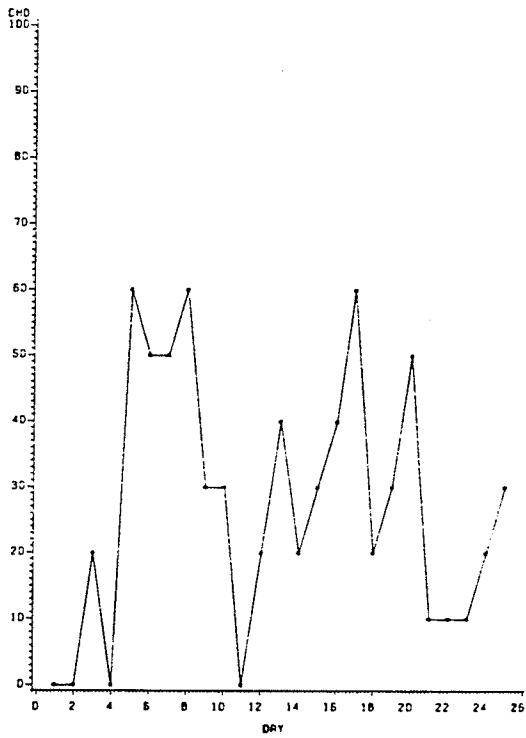
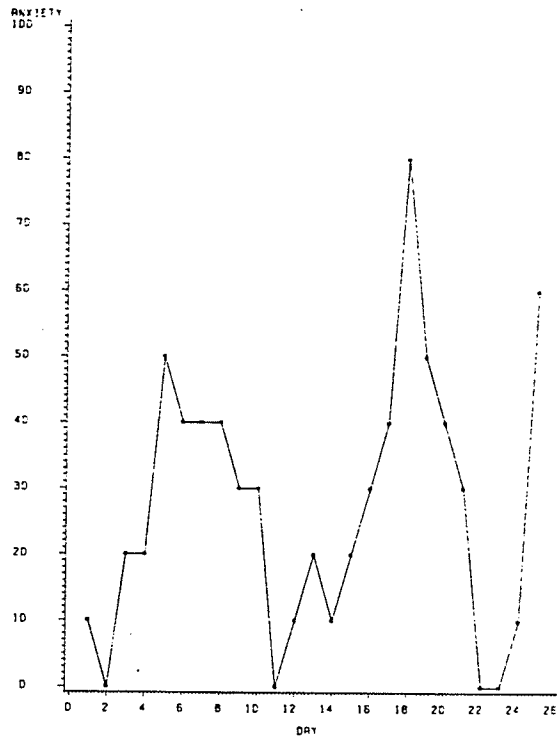
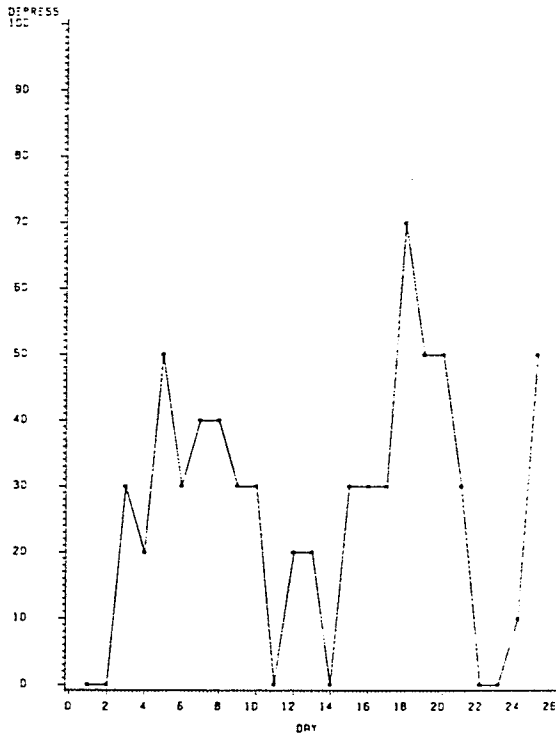


SUBJECT 6 : CYCLE 1

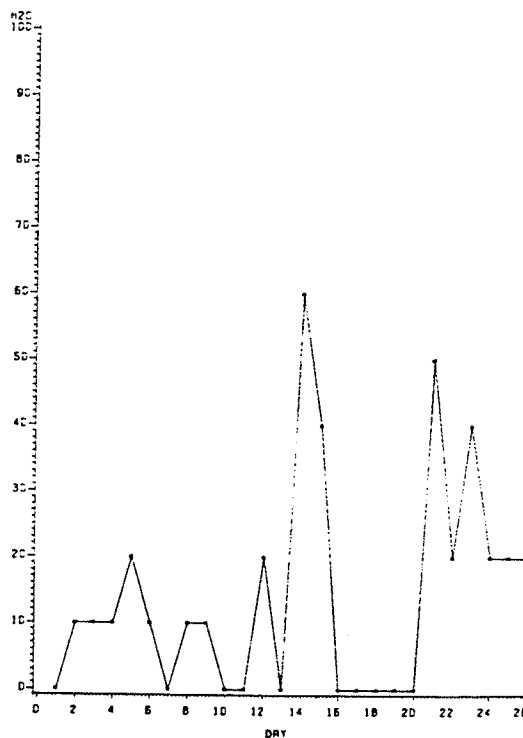
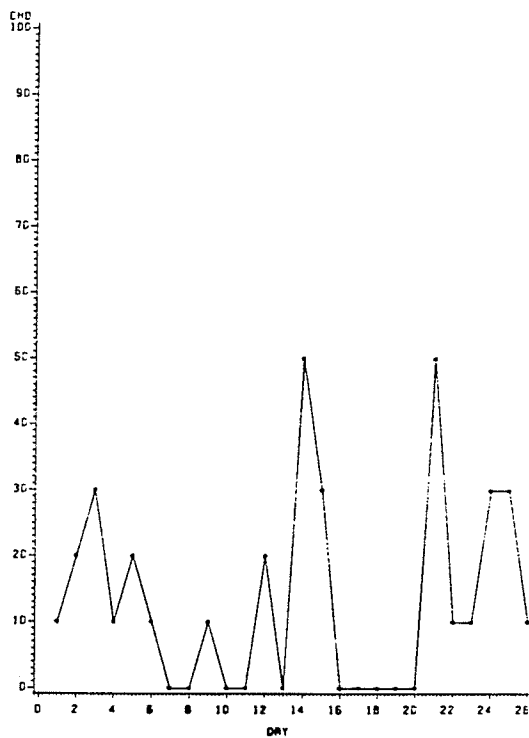
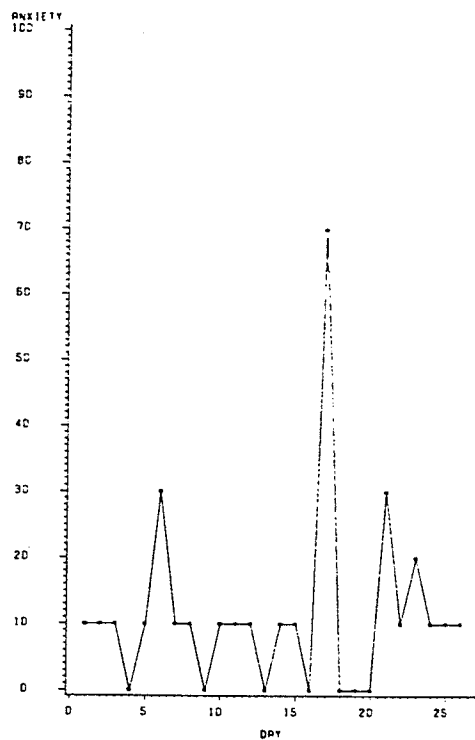
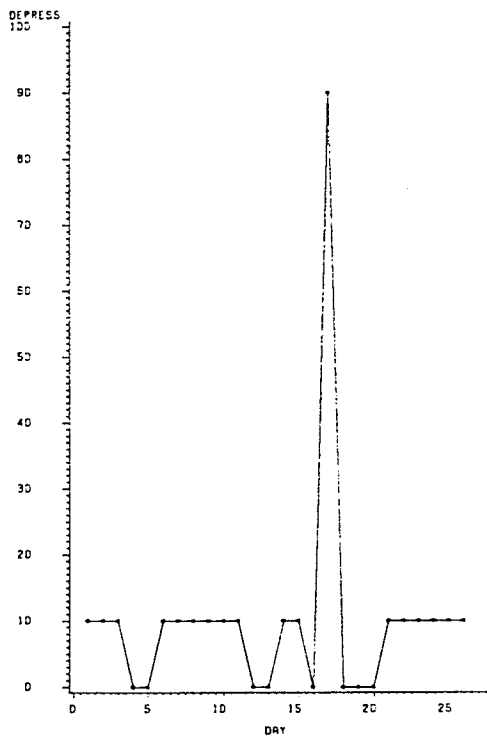
PMS SELF-RATINGS



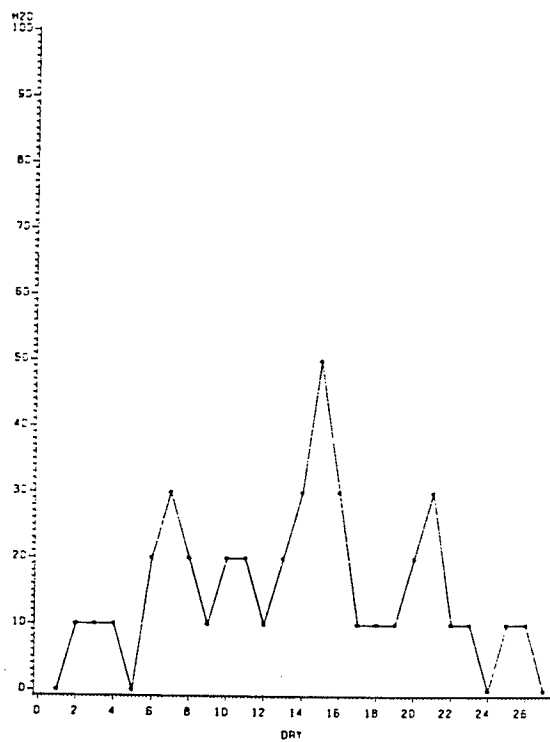
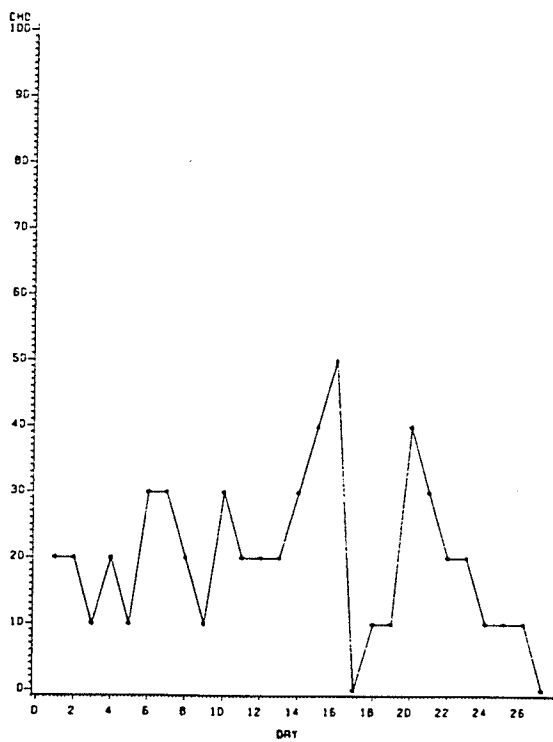
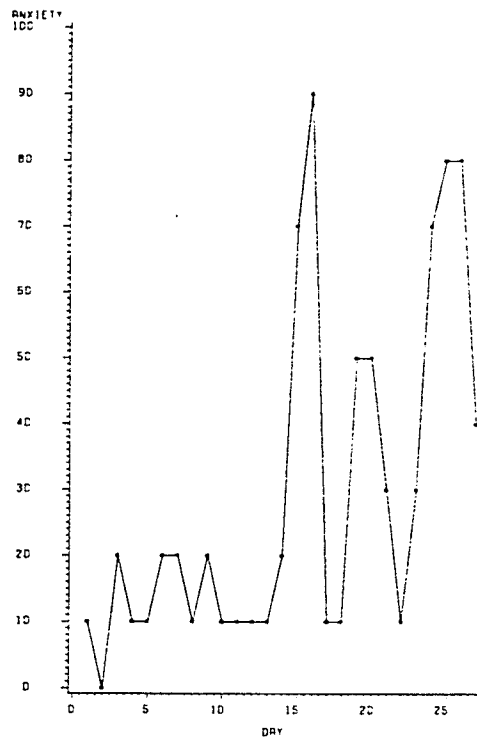
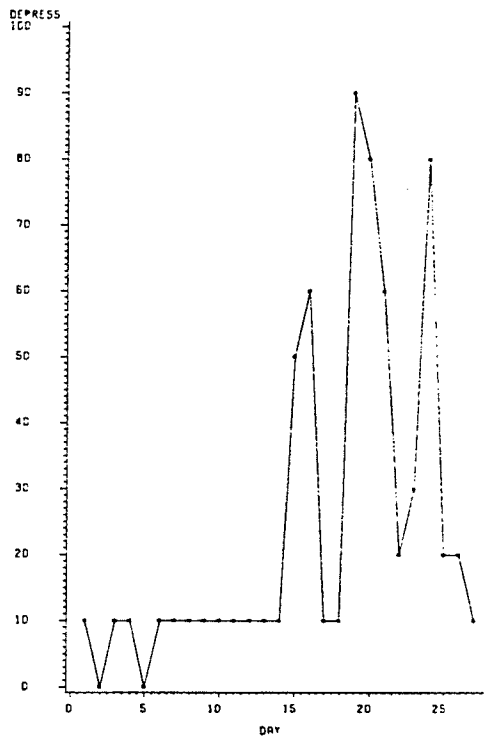
SUBJECT 6 : CYCLE 2 PMS SELF-RATINGS



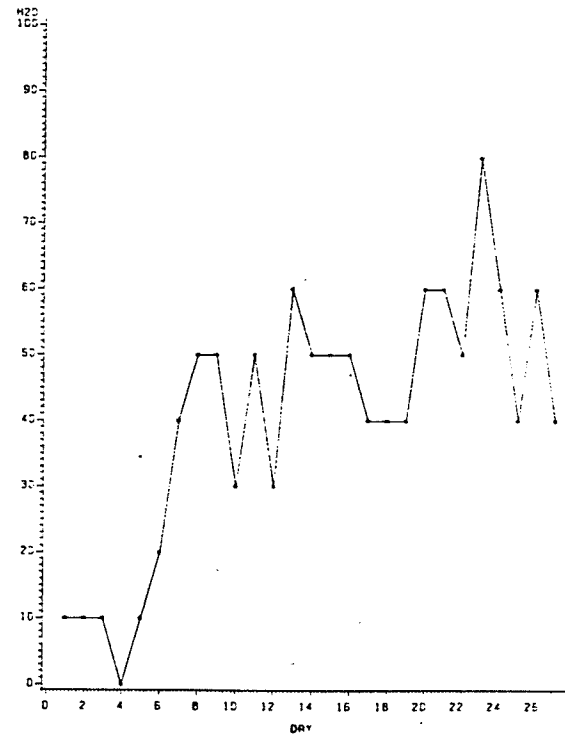
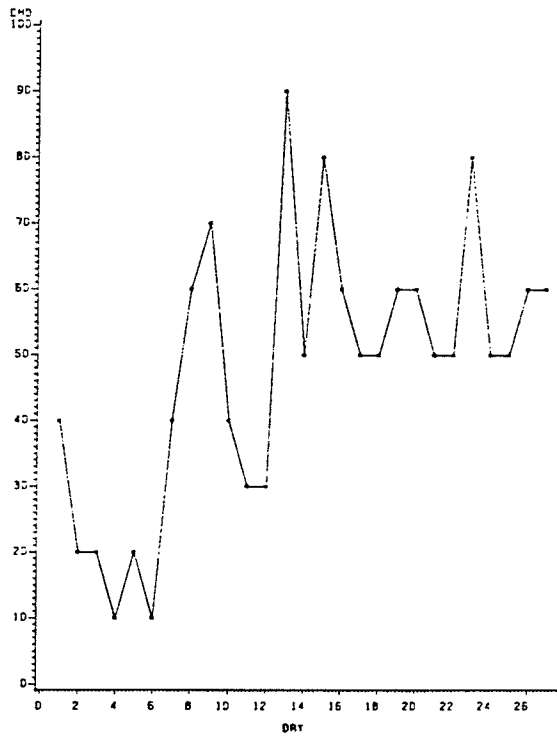
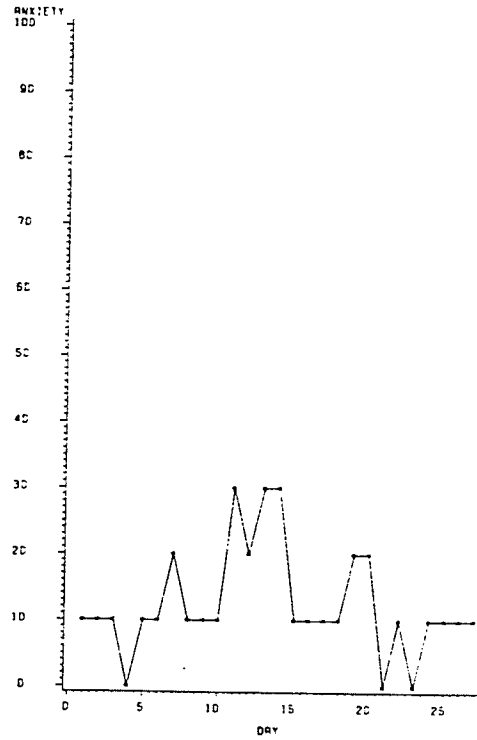
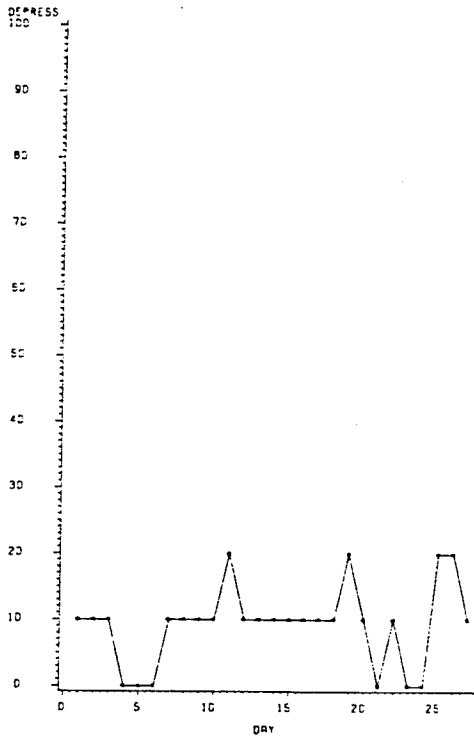
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SUBJECT 6 : CYCLE 4 PMS SELF-RATINGS

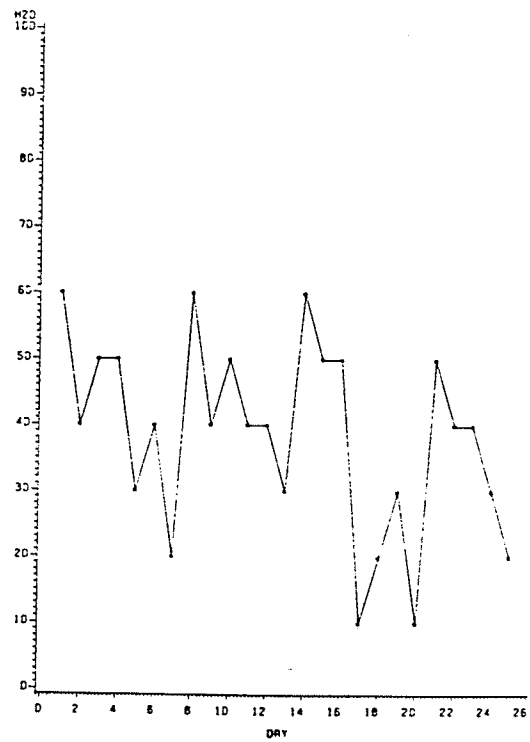
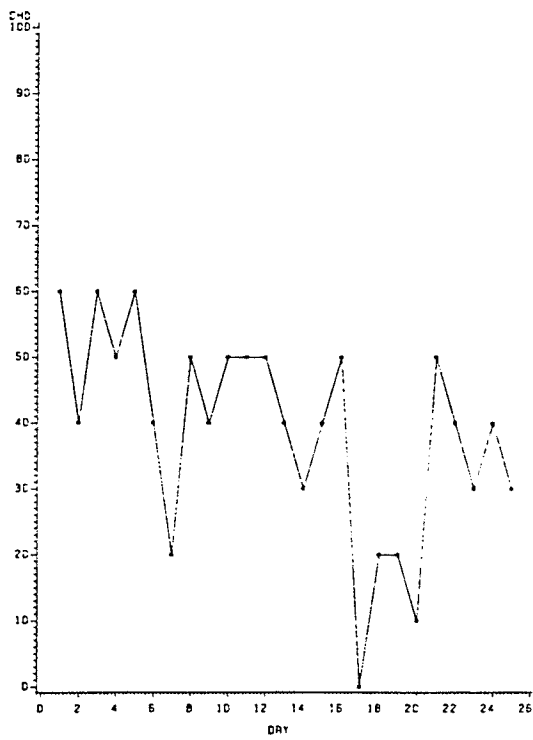
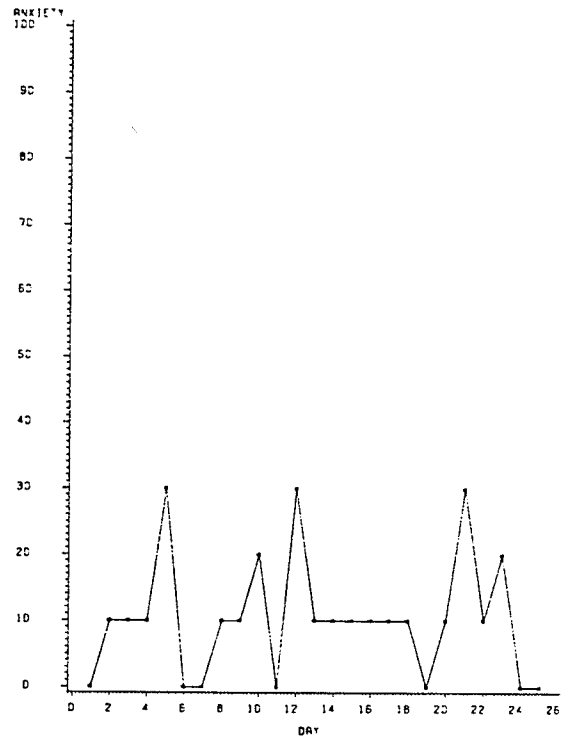
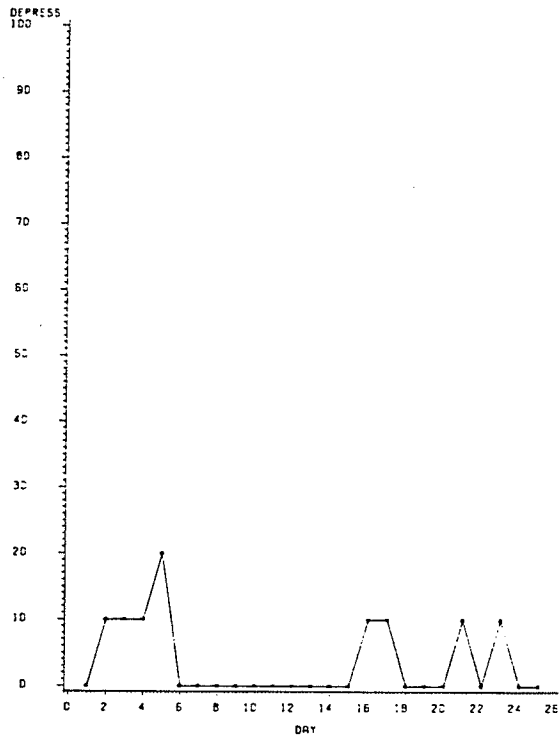


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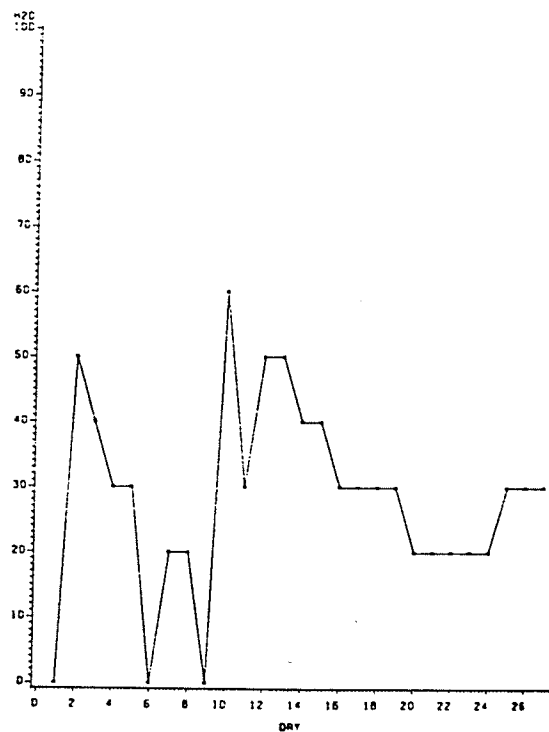
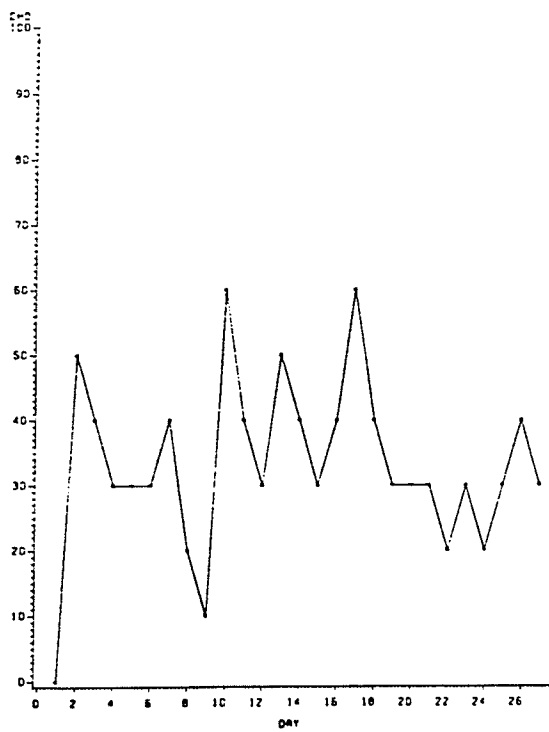
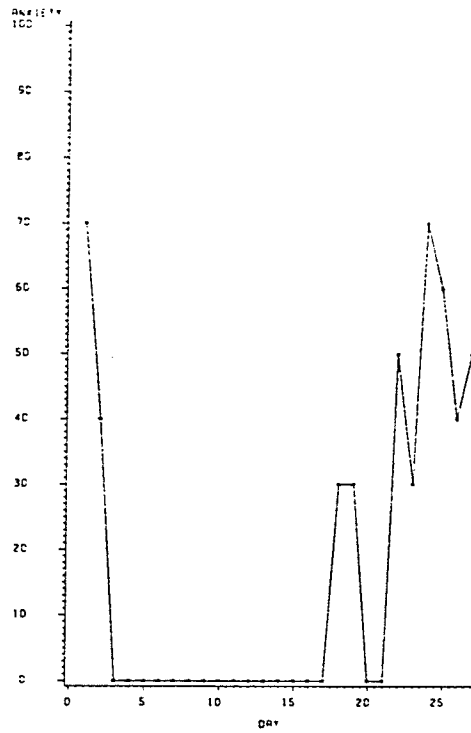
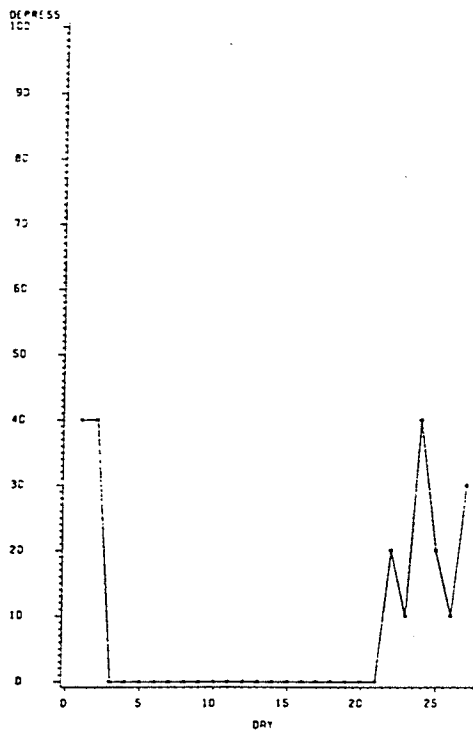


SUBJECT 6 : CYCLE 6

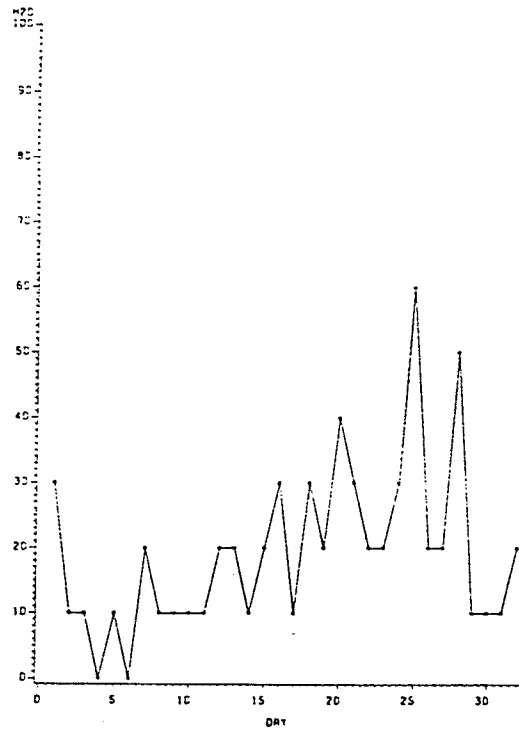
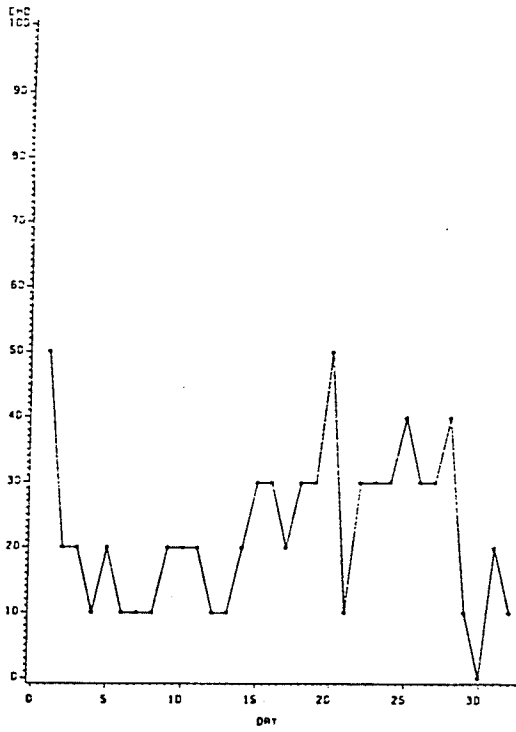
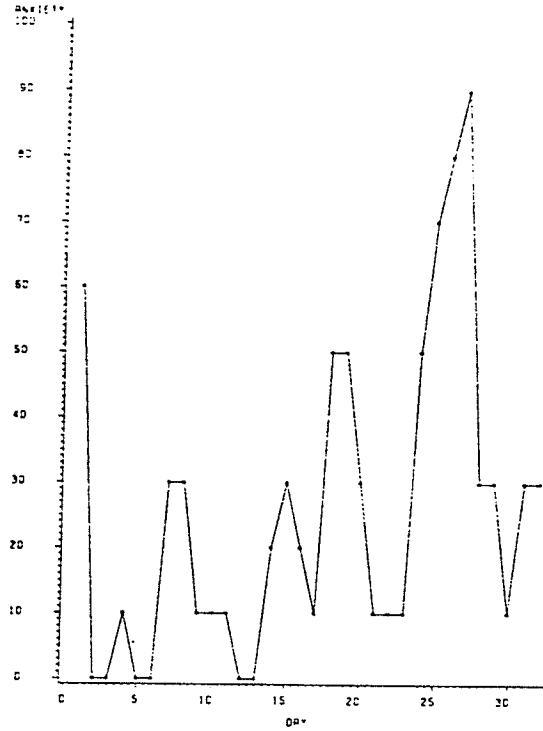
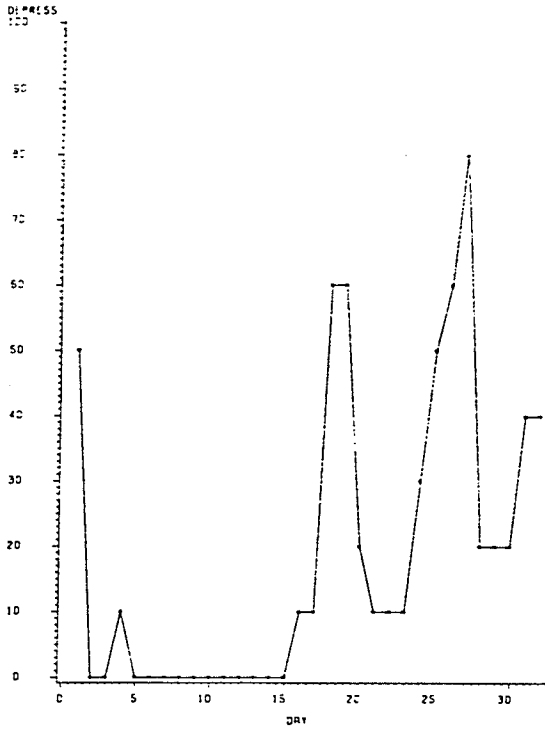
PMS SELF-RATINGS



SUBJECT 6 : CYCLE 7 PMS SELF-RATINGS



SUBJECT 6 : CYCLE 8 PMS SELF-RATINGS



Appendix L

Subject 1

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? Sometimes I was but about 1/2 or more not very regular. but even though I didn't come as often as I should have, I did walk and run more at home than usual or before.
 2. Did you notice any relationship between your general disposition and:
 - month 1 of exercise Seemed to have less symptoms but tired.
 - month 2 of exercise Felt terrific.
 - month 3 of exercise Felt good, but could have felt terrific if I had exercised more! But still felt on top of things.
 3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression: Seemed not as bad, though I felt bad, I didn't exercise more; if I was depressed exercise really seemed to make a difference.
 - (ii) A-anxiety: Improved lots, and felt I could deal with it better.
 - (iii) C-cravings: Were still there but alot subdued. Sugar has been low now, but salt is still a problem.
 - (iv) H-water retention: Still was high but exercising helped somewhat (hard to exercise with breast pain!)
 - (v) Overall trends: Felt less tired, hair & complexion improved. Felt more tolerable & patient. Noises & lights no problems at all as compared to before. Headaches lessened. World didn't look so bleak, longer periods of feeling good.
 4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight Down a little
 - (ii) muscle tone Slight difference
 - (iii) general fitness level Felt a lot better, bicycle still feels tough
 - (iv) fit of clothing Clothes felt looser
 - (v) complexion A lot better
 - (vi) energy level Up 90%
 - (vii) decision making Yes, Yes, Yes! Really made a lot of decisions incapable of before!
 - (viii) sleeping habits Didn't need as much sleep as before
 - (ix) appetite Less
 - (x) caloric intake Some days when exercising intake less--not exercising, intake more.
 - (xi) type of foods ingested More health food--fruits & vegetables
 - (xii) eating patterns More small meals than big ones
-
5. Please use the back for any additional comments regarding the study format and/or your impressions.

Subject 2

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? No--due to conflicts with work commitments and/or holidays out of town.
 2. Did you notice any relationship between your general disposition and:
month 1 of exercise Felt better during cycle.
: month 2 of exercise Felt o.k. but didn't exercise much.
: month 3 of exercise Hardly exercised--noted more water retention.
 3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression: Often mid cycle.
 - (ii) A-anxiety: Insomnia felt a lot during summer months.
 - (iii) C-cravings: Just pre menstruation--usually for chocolate--usually I didn't give in!
 - (iv) H-water retention: A real problem for me--this last month of no exercise I've gone to a larger bra size.
 - (v) Overall trends: I really see a difference in my body's symptoms with exercise but found Bodyworks Program too demanding in time and energy. The workouts were too strenuous.
 4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight A bit heavier (1 lb.) without exercise
 - (ii) muscle tone Better with exercise
 - (iii) general fitness level Better when exercising
 - (iv) fit of clothing Less swollen in tummy when exercising
 - (v) complexion No change
 - (vi) energy level Probably better with exercise
 - (vii) decision making No difference
 - (viii) sleeping habits Had a lot of insomnia when I stopped exercising
 - (ix) appetite No difference although a recent noticeable increase
 - (x) caloric intake No difference
 - (xi) type of foods ingested Problems in last cycle, not sure why
 - (xii) eating patterns No difference
-
5. Please use the back for any additional comments regarding the study format and/or your impressions.

Subject 3

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? Yes.
2. Did you notice any relationship between your general disposition and:
month 1 of exercise
: month 2 of exercise
: month 3 of exercise Improved.
3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression: Improved 3rd mo. Shorter duration and less severe.
 - (ii) A-anxiety: Less 3rd mo.
 - (iii) C-cravings: Less 3rd mo.
 - (iv) H-water retention: Less 3rd mo.
 - (v) Overall trends: The first two periods during the exercise period were severe in all PMS symptoms but all less severe and shorter duration.
4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight Increased
 - (ii) muscle tone Improved
 - (iii) general fitness level Improved
 - (iv) fit of clothing Improved
 - (v) complexion Same
 - (vi) energy level Same
 - (vii) decision making Same
 - (viii) sleeping habits Same
 - (ix) appetite Increased
 - (x) caloric intake Increased
 - (xi) type of foods ingested Carbohydrates
 - (xii) eating patterns Similar

-
5. Please use the back for any additional comments regarding the study format and/or your impressions.

Time of month for testing affected performance I feel.

Subject 4

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? For the first 2 months, yes.
2. Did you notice any relationship between your general disposition and:
month 1 of exercise A little.
: month 2 of exercise A little.
: month 3 of exercise Did not exercise. (as regularly)
3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression:
 - (ii) A-anxiety:
 - (iii) C-cravings:
 - (iv) H-water retention: Bloating was equally as bad but did not last for as long a period of time.
 - (v) Overall trends: My problem has always been a physical one--with a lot of pain. The pain used to last up to 14 days. I found during the 2 months that the maximum was only 7 days.
4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight Yes. In the beginning I lost a couple of lbs.
 - (ii) muscle tone A little
 - (iii) general fitness level Yes
 - (iv) fit of clothing Yes. I was slimmer around waist.
 - (v) complexion No difference
 - (vi) energy level Yes. Much higher energy level.
 - (vii) decision making Not affected
 - (viii) sleeping habits No change
 - (ix) appetite Increased appetite
 - (x) caloric intake No change
 - (xi) type of foods ingested No change
 - (xii) eating patterns No change

5. Please use the back for any additional comments regarding the study format and/or your impressions.

Subject 5

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? Yes--with the exception of 2 weeks of vacation.
2. Did you notice any relationship between your general disposition and:
 - month 1 of exercise + + +
 - : month 2 of exercise + +
 - : month 3 of exercise + +
3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression: Only infrequent moments of lethargy.
 - (ii) A-anxiety: Much improved. Felt periods of irritability were less frequent and shorter.
 - (iii) C-cravings: Less. Only infrequent salt cravings.
 - (iv) H-water retention: Some improvement. Same degree but for shorter periods.
 - (v) Overall trends: Generally, I felt better able to cope with symptoms as they appeared. I felt more relaxed.
4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight Same
 - (ii) muscle tone Improved
 - (iii) general fitness level Improved
 - (iv) fit of clothing Improved
 - (v) complexion Same
 - (vi) energy level Improved
 - (vii) decision making Much improved. Cope better with pressure.
 - (viii) sleeping habits Improved
 - (ix) appetite Same
 - (x) caloric intake Same
 - (xi) type of foods ingested Same
 - (xii) eating patterns Same

-
5. Please use the back for any additional comments regarding the study format and/or your impressions.

Because of the type of position I have, I found three times a week somewhat difficult, depending on my schedule. However, time constraints aside, exercise really seemed to help. The psychological benefit of overall well-being helps!

Subject 6

Anecdotal Report: P.M.S. and Exercise

1. Do you feel that you were a regular adherent to the exercise program? No. 2x, week sick 3 weeks.
2. Did you notice any relationship between your general disposition and:
 - month 1 of exercise Yes
 - : month 2 of exercise Yes
 - : month 3 of exercise Missed 3 weeks (of regular attendance)
3. During the exercise program what were your general impressions regarding your P.M.S. symptoms for:
 - (i) D-depression: Not severe--felt very good about myself.
 - (ii) A-anxiety: Less--no outbursts.
 - (iii) C-cravings: Almost nil--but returned when I was sick for 3 weeks.
 - (iv) H-water retention: Gone.
 - (v) Overall trends: The anger lessened. I'm more tolerant.
4. Did you notice any difference between your normal status as opposed to "exercise" status with regards to:
 - (i) body weight Lost 10-15 lbs. gained
 - (ii) muscle tone Better, especially thighs, stomach & buttocks
 - (iii) general fitness level Better
 - (iv) fit of clothing Better
 - (v) complexion Better
 - (vi) energy level Better
 - (vii) decision making Better
 - (viii) sleeping habits Better
 - (ix) appetite Better types of food, but less overeating
 - (x) caloric intake Less
 - (xi) type of foods ingested More nutritious
 - (xii) eating patterns More regular

-
5. Please use the back for any additional comments regarding the study format and/or your impressions.

I no longer require the Vitamin regime that I was on when I started this program and the symptoms but decreased to a normal level.

Appendix M

DATE: 85/10/25

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA
=====

NAME: SUBJECT 1 1st Recall

SEX: Female AGE: 29 years FRAME SIZE: Medium

WEIGHT: 59.1 kg (130 lbs.) HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL
=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	125.0	45.1%	at least 50%
Protein	44.6	16.1%	10-15%
Total Fat	52.3	42.5%	30-35%
Essential Fatty Acid	12.4	10.0%	3%
Saturated Fatty Acid	15.2	12.4%	
Polyunsaturated F.A.	12.2	9.9%	
Monounsaturated F.A.	20.5	16.6%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN
=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,638.0 kj	
Protein	43.6 g	44.7 g	102 %
Vitamin A	800.0 RE	211.7 RE	26 %
Vitamin C	45.0 mg	109.9 mg	244 %
Vitamin D	2.5 ug	1.9 ug	76 %
Thiamin	0.8 mg	0.8 mg	105 %
Riboflavin	1.0 mg	0.8 mg	81 %
Niacin	14.4 NE	23.1 NE	160 %
Vitamin B6	0.7 mg	1.2 mg	173 %
Folacin	165.0 ug	164.4 ug	100 %
Vitamin B12	2.0 ug	0.8 ug	42 %
Pantothenic Acid	2.5 mg	3.4 mg	132 %
Calcium	700.0 mg	392.4 mg	56 %
Phosphorus	700.0 mg	731.0 mg	104 %
Iron	14.0 mg	7.2 mg	51 %
Potassium	44.2 mmol	48.1 mmol	109 %
Sodium	23.6 mmol	79.7 mmol	338 %
Fiber, Crude		3.9 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA
=====

NAME: SUBJECT 1 2nd Recall

SEX: Female AGE: 29 years FRAME SIZE: Medium

WEIGHT: 59.1 kg (130 lbs.) HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/09/01 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL
=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	91.9	55.6%	at least 50%
Protein	29.1	17.6%	10-15%
Total Fat	21.4	29.2%	30-35%
Essential Fatty Acid	1.4	2.0%	3%
Saturated Fatty Acid	9.5	13.0%	
Polyunsaturated F.A.	1.1	1.6%	
Monounsaturated F.A.	8.5	11.6%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN
=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		2,763.0 kj	
Protein	43.6 g	29.1 g	67 %
Vitamin A	800.0 RE	1,169.4 RE	146 %
Vitamin C	45.0 mg	87.6 mg	195 %
Vitamin D	2.5 ug	0.1 ug	3 %
Thiamin	0.8 mg	0.5 mg	60 %
Riboflavin	1.0 mg	0.6 mg	64 %
Niacin	14.4 NE	10.2 NE	71 %
Vitamin B6	0.4 mg	0.6 mg	133 %
Folacin	165.0 ug	122.1 ug	74 %
Vitamin B12	2.0 ug	0.4 ug	22 %
Pantothenic Acid	1.5 mg	2.0 mg	131 %
Calcium	700.0 mg	298.2 mg	43 %
Phosphorus	700.0 mg	522.6 mg	75 %
Iron	14.0 mg	6.5 mg	46 %
Potassium	44.2 mmol	35.8 mmol	81 %
Sodium	23.6 mmol	61.9 mmol	262 %
Fiber, Crude		3.6 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 1 3rd Recall

SEX: Female AGE: 30 years FRAME SIZE: Medium

WEIGHT: 62.1 kg (137 lbs.) HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	201.1	68.0%	at least 50%
Protein	45.0	15.2%	10-15%
Total Fat	28.8	21.9%	30-35%
Essential Fatty Acid	4.3	3.3%	3%
Saturated Fatty Acid	10.5	8.0%	
Polyunsaturated F.A.	4.3	3.3%	
Monounsaturated F.A.	10.3	7.8%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,948.0 kj	
Protein	46.0 g	45.1 g	98 %
Vitamin A	800.0 RE	804.8 RE	101 %
Vitamin C	45.0 mg	202.2 mg	449 %
Vitamin D	2.5 ug	0.0 ug	0 %
Thiamin	0.8 mg	1.0 mg	123 %
Riboflavin	1.0 mg	0.9 mg	93 %
Niacin	14.4 NE	19.6 NE	136 %
Vitamin B6	0.7 mg	2.5 mg	368 %
Folacin	165.0 ug	200.8 ug	122 %
Vitamin B12	2.0 ug	1.1 ug	57 %
Pantothenic Acid	2.7 mg	3.1 mg	115 %
Calcium	700.0 mg	316.4 mg	45 %
Phosphorus	700.0 mg	618.5 mg	88 %
Iron	14.0 mg	8.4 mg	60 %
Potassium	46.6 mmol	79.5 mmol	171 %
Sodium	24.9 mmol	26.5 mmol	107 %
Fiber, Crude		7.3 g	

DATE: 85/10/25

*** NUTRIPROFILE ***

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 2 1st Recall

SEX: Female AGE: 33 years FRAME SIZE: Small

WEIGHT: 49.9 kg (110 lbs.) HEIGHT: 165 cm (5 ft, 5 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
	-----	-----	-----
Carbohydrate	151.8	50.9%	at least 50%
Protein	41.6	13.9%	10-15%
Total Fat	41.8	31.6%	30-35%
Essential Fatty Acid	10.5	7.9%	3%
Saturated Fatty Acid	13.6	10.3%	
Polyunsaturated F.A.	10.3	7.8%	
Monounsaturated F.A.	15.1	11.4%	
Alcohol	10.9	6.4%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,982.0 kj	
Protein	36.9 g	41.6 g	113 %
Vitamin A	800.0 RE	495.8 RE	62 %
Vitamin C	45.0 mg	75.5 mg	168 %
Vitamin D	2.5 ug	0.9 ug	35 %
Thiamin	0.8 mg	0.8 mg	103 %
Riboflavin	1.0 mg	0.8 mg	81 %
Niacin	14.4 NE	19.1 NE	133 %
Vitamin B6	0.6 mg	1.0 mg	154 %
Folacin	165.0 ug	129.9 ug	79 %
Vitamin B12	2.0 ug	1.2 ug	62 %
Pantothenic Acid	2.7 mg	2.7 mg	98 %
Calcium	700.0 mg	411.7 mg	59 %
Phosphorus	700.0 mg	735.1 mg	105 %
Iron	14.0 mg	9.2 mg	66 %
Potassium	37.4 mmol	48.2 mmol	129 %
Sodium	20.0 mmol	59.3 mmol	297 %
Fiber, Crude		3.6 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

NAME: SUBJECT 2 2nd Recall

SEX: Female AGE: 33 years FRAME SIZE: Small

WEIGHT: 48.6 kg (107 lbs.) HEIGHT: 165 cm (5 ft, 5 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/09/03 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	116.0	46.3%	at least 50%
Protein	47.2	18.8%	10-15%
Total Fat	39.3	35.3%	30-35%
Essential Fatty Acid	7.4	6.6%	3%
Saturated Fatty Acid	13.1	11.7%	
Polyunsaturated F.A.	7.0	6.3%	
Monounsaturated F.A.	16.1	14.5%	
Alcohol	0.9	0.6%	

NUTRIENT BREAKDOWN

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,191.0 kj	
Protein	35.9 g	47.2 g	132 %
Vitamin A	800.0 RE	578.1 RE	72 %
Vitamin C	45.0 mg	95.7 mg	213 %
Vitamin D	2.5 ug	0.5 ug	22 %
Thiamin	0.8 mg	0.7 mg	90 %
Riboflavin	1.0 mg	0.8 mg	83 %
Niacin	14.4 NE	17.2 NE	119 %
Vitamin B6	0.7 mg	0.8 mg	118 %
Folacin	165.0 ug	129.6 ug	79 %
Vitamin B12	2.0 ug	1.9 ug	95 %
Pantothenic Acid	2.3 mg	2.7 mg	115 %
Calcium	700.0 mg	325.1 mg	46 %
Phosphorus	700.0 mg	582.6 mg	83 %
Iron	14.0 mg	11.0 mg	79 %
Potassium	36.4 mmol	37.7 mmol	104 %
Sodium	19.4 mmol	65.3 mmol	336 %
Fiber, Crude		2.8 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 2 3rd Recall

SEX: Female AGE: 34 years FRAME SIZE: Small

WEIGHT: 48.1 kg (106 lbs.) HEIGHT: 165 cm (5 ft, 5 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	76.2	42.9%	at least 50%
Protein	33.8	19.1%	10-15%
Total Fat	31.2	39.7%	30-35%
Essential Fatty Acid	2.7	3.5%	3%
Saturated Fatty Acid	15.8	20.0%	
Polyunsaturated F.A.	2.7	3.4%	
Monounsaturated F.A.	10.3	13.1%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		2,967.0 kj	
Protein	35.6 g	33.9 g	95 %
Vitamin A	800.0 RE	258.7 RE	32 %
Vitamin C	45.0 mg	21.3 mg	47 %
Vitamin D	2.5 ug	1.7 ug	69 %
Thiamin	0.8 mg	0.6 mg	78 %
Riboflavin	1.0 mg	0.7 mg	65 %
Niacin	14.4 NE	12.1 NE	84 %
Vitamin B6	0.5 mg	0.5 mg	107 %
Folacin	165.0 ug	65.0 ug	39 %
Vitamin B12	2.0 ug	1.9 ug	94 %
Pantothenic Acid	1.6 mg	2.1 mg	126 %
Calcium	700.0 mg	280.3 mg	40 %
Phosphorus	700.0 mg	511.0 mg	73 %
Iron	14.0 mg	8.2 mg	58 %
Potassium	36.1 mmol	23.4 mmol	65 %
Sodium	19.2 mmol	32.0 mmol	167 %
Fiber, Crude		1.8 g	

DATE: 85/10/25

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

NAME: SUBJECT 3 1st Recall

SEX: Female AGE: 40 years FRAME SIZE: Medium

WEIGHT: 68.0 kg (150 lbs.) HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	129.3	45.1%	at least 50%
Protein	62.3	21.7%	10-15%
Total Fat	36.2	28.4%	30-35%
Essential Fatty Acid	7.7	6.0%	3%
Saturated Fatty Acid	10.1	7.9%	
Polyunsaturated F.A.	7.5	5.9%	
Monounsaturated F.A.	15.3	12.0%	
Alcohol	7.2	4.4%	

NUTRIENT BREAKDOWN

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,793.0 kj	
Protein	50.3 g	62.4 g	124 %
Vitamin A	800.0 RE	241.6 RE	30 %
Vitamin C	45.0 mg	61.6 mg	137 %
Vitamin D	2.5 ug	4.6 ug	184 %
Thiamin	0.8 mg	0.8 mg	105 %
Riboflavin	1.0 mg	1.1 mg	106 %
Niacin	14.4 NE	25.8 NE	179 %
Vitamin B6	0.9 mg	0.9 mg	98 %
Folacin	165.0 ug	92.8 ug	56 %
Vitamin B12	2.0 ug	3.3 ug	167 %
Pantothenic Acid	2.6 mg	2.8 mg	106 %
Calcium	700.0 mg	544.9 mg	78 %
Phosphorus	700.0 mg	832.3 mg	119 %
Iron	14.0 mg	8.9 mg	64 %
Potassium	51.0 mmol	38.2 mmol	75 %
Sodium	27.2 mmol	78.8 mmol	289 %
Fiber, Crude		1.2 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 3 2rd Recall

SEX: Female

AGE: 40 years

FRAME SIZE: Medium

WEIGHT: 72.6 kg (160 lbs.)

HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/08/31 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	124.4	46.5%	at least 50%
Protein	65.3	24.4%	10-15%
Total Fat	32.3	27.2%	30-35%
Essential Fatty Acid	4.9	4.1%	3%
Saturated Fatty Acid	11.8	9.9%	
Polyunsaturated F.A.	4.9	4.1%	
Monounsaturated F.A.	12.1	10.2%	
Alcohol	3.6	2.3%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,471.0 kj	
Protein	53.7 g	65.3 g	122 %
Vitamin A	800.0 RE	656.2 RE	82 %
Vitamin C	45.0 mg	72.6 mg	161 %
Vitamin D	2.5 ug	2.8 ug	111 %
Thiamin	0.8 mg	1.0 mg	123 %
Riboflavin	1.0 mg	1.2 mg	118 %
Niacin	14.4 NE	19.5 NE	136 %
Vitamin B6	1.0 mg	1.2 mg	126 %
Folacin	165.0 ug	117.5 ug	71 %
Vitamin B12	2.0 ug	3.0 ug	151 %
Pantothenic Acid	2.5 mg	4.0 mg	161 %
Calcium	700.0 mg	522.4 mg	75 %
Phosphorus	700.0 mg	879.3 mg	126 %
Iron	14.0 mg	7.7 mg	55 %
Potassium	54.4 mmol	50.6 mmol	93 %
Sodium	29.0 mmol	53.8 mmol	185 %
Fiber, Crude		3.4 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 3 3rd Recall

SEX: Female AGE: 40 years FRAME SIZE: Medium

WEIGHT: 71.7 kg (158 lbs.) HEIGHT: 162 cm (5 ft, 4 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	66.7	32.0%	at least 50%
Protein	51.6	24.7%	10-15%
Total Fat	37.6	40.5%	30-35%
Essential Fatty Acid	6.4	6.9%	3%
Saturated Fatty Acid	11.5	12.4%	
Polyunsaturated F.A.	6.4	6.9%	
Monounsaturated F.A.	16.6	17.9%	
Alcohol	2.8	2.3%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		3,491.0 kj	
Protein	53.0 g	51.7 g	97 %
Vitamin A	800.0 RE	216.7 RE	27 %
Vitamin C	45.0 mg	99.3 mg	221 %
Vitamin D	2.5 ug	0.9 ug	37 %
Thiamin	0.8 mg	0.6 mg	71 %
Riboflavin	1.0 mg	0.8 mg	79 %
Niacin	14.4 NE	24.6 NE	171 %
Vitamin B6	0.8 mg	0.8 mg	104 %
Folacin	165.0 ug	106.8 ug	65 %
Vitamin B12	2.0 ug	1.7 ug	85 %
Pantothenic Acid	1.9 mg	3.0 mg	157 %
Calcium	700.0 mg	407.0 mg	58 %
Phosphorus	700.0 mg	581.2 mg	83 %
Iron	14.0 mg	5.7 mg	40 %
Potassium	53.7 mmol	33.8 mmol	63 %
Sodium	28.7 mmol	46.9 mmol	164 %
Fiber, Crude		1.6 g	

DATE: 85/10/25

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

NAME: SUBJECT 4 1st Recall

SEX: Female AGE: 33 years FRAME SIZE: Medium

WEIGHT: 54.4 kg (120 lbs.) HEIGHT: 157 cm (5 ft, 2 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	120.5	33.5%	at least 50%
Protein	48.9	13.6%	10-15%
Total Fat	53.9	33.7%	30-35%
Essential Fatty Acid	10.4	6.5%	3%
Saturated Fatty Acid	17.8	11.1%	
Polyunsaturated F.A.	10.4	6.5%	
Monounsaturated F.A.	21.2	13.3%	
Alcohol	41.6	20.2%	

NUTRIENT BREAKDOWN

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		6,009.0 kj	
Protein	40.3 g	49.0 g	122 %
Vitamin A	800.0 RE	1,971.3 RE	246 %
Vitamin C	45.0 mg	102.0 mg	227 %
Vitamin D	2.5 ug	1.4 ug	54 %
Thiamin	0.8 mg	0.6 mg	71 %
Riboflavin	1.0 mg	1.1 mg	111 %
Niacin	14.4 NE	18.0 NE	125 %
Vitamin B6	0.7 mg	0.8 mg	108 %
Folacin	165.0 ug	110.4 ug	67 %
Vitamin B12	2.0 ug	4.0 ug	199 %
Pantothenic Acid	3.3 mg	3.3 mg	100 %
Calcium	700.0 mg	629.8 mg	90 %
Phosphorus	700.0 mg	782.8 mg	112 %
Iron	14.0 mg	9.0 mg	64 %
Potassium	40.8 mmol	35.9 mmol	88 %
Sodium	21.8 mmol	66.6 mmol	306 %
Fiber, Crude		1.5 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 4 2nd Recall

SEX: Female AGE: 33 years FRAME SIZE: Medium

WEIGHT: 52.7 kg (116 lbs.) HEIGHT: 157 cm (5 ft, 2 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/08/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
	-----	-----	-----
Carbohydrate	139.0	38.6%	at least 50%
Protein	47.1	13.0%	10-15%
Total Fat	46.5	29.0%	30-35%
Essential Fatty Acid	6.2	3.8%	3%
Saturated Fatty Acid	18.6	11.6%	
Polyunsaturated F.A.	5.7	3.6%	
Monounsaturated F.A.	17.8	11.1%	
Alcohol	42.7	20.8%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		6,022.0 kj	
Protein	38.9 g	47.1 g	121 %
Vitamin A	800.0 RE	758.6 RE	95 %
Vitamin C	45.0 mg	18.1 mg	40 %
Vitamin D	2.5 ug	0.3 ug	11 %
Thiamin	0.8 mg	0.6 mg	79 %
Riboflavin	1.0 mg	0.7 mg	69 %
Niacin	14.4 NE	20.8 NE	145 %
Vitamin B6	0.7 mg	0.8 mg	110 %
Folacin	165.0 ug	64.7 ug	39 %
Vitamin B12	2.0 ug	1.6 ug	80 %
Pantothenic Acid	3.3 mg	1.7 mg	50 %
Calcium	700.0 mg	340.9 mg	49 %
Phosphorus	700.0 mg	663.1 mg	95 %
Iron	14.0 mg	7.8 mg	56 %
Potassium	39.5 mmol	34.3 mmol	87 %
Sodium	21.0 mmol	72.0 mmol	342 %
Fiber, Crude		1.6 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 4 3rd Recall

SEX: Female AGE: 34 years FRAME SIZE: Medium

WEIGHT: 53.1 kg (117 lbs.) HEIGHT: 160 cm (5 ft, 3 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	111.8	39.2%	at least 50%
Protein	51.4	18.0%	10-15%
Total Fat	39.2	31.0%	30-35%
Essential Fatty Acid	6.3	4.9%	3%
Saturated Fatty Acid	12.4	9.8%	
Polyunsaturated F.A.	6.1	4.8%	
Monounsaturated F.A.	15.2	12.0%	
Alcohol	21.5	13.2%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		4,763.0 kj	
Protein	39.3 g	51.5 g	131 %
Vitamin A	800.0 RE	758.4 RE	95 %
Vitamin C	45.0 mg	60.6 mg	135 %
Vitamin D	2.5 ug	1.5 ug	62 %
Thiamin	0.8 mg	0.8 mg	104 %
Riboflavin	1.0 mg	1.0 mg	98 %
Niacin	14.4 NE	16.3 NE	113 %
Vitamin B6	0.8 mg	1.3 mg	166 %
Folacin	165.0 ug	115.3 ug	70 %
Vitamin B12	2.0 ug	1.7 ug	84 %
Pantothenic Acid	2.6 mg	3.1 mg	119 %
Calcium	700.0 mg	331.0 mg	47 %
Phosphorus	700.0 mg	706.3 mg	101 %
Iron	14.0 mg	8.5 mg	60 %
Potassium	39.8 mmol	48.0 mmol	121 %
Sodium	21.2 mmol	102.1 mmol	481 %
Fiber, Crude		2.9 g	

DATE: 85/10/25

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

=====

NAME: SUBJECT 5 1st Recall

SEX: Female AGE: 36 years FRAME SIZE: Medium

WEIGHT: 64.4 kg (142 lbs.) HEIGHT: 167 cm (5 ft, 6 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	108.4	32.5%	at least 50%
Protein	69.8	20.9%	10-15%
Total Fat	62.9	42.4%	30-35%
Essential Fatty Acid	12.1	8.1%	3%
Saturated Fatty Acid	24.2	16.3%	
Polyunsaturated F.A.	12.0	8.0%	
Monounsaturated F.A.	22.0	14.8%	
Alcohol	10.4	5.4%	

NUTRIENT BREAKDOWN

=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		5,585.0 kj	
Protein	47.7 g	69.9 g	147 %
Vitamin A	800.0 RE	563.6 RE	70 %
Vitamin C	45.0 mg	186.8 mg	415 %
Vitamin D	2.5 ug	2.9 ug	115 %
Thiamin	0.8 mg	1.0 mg	131 %
Riboflavin	1.0 mg	1.4 mg	139 %
Niacin	14.4 NE	23.4 NE	162 %
Vitamin B6	1.0 mg	1.4 mg	129 %
Folacin	165.0 ug	174.8 ug	106 %
Vitamin B12	2.0 ug	2.8 ug	138 %
Pantothenic Acid	3.1 mg	4.8 mg	157 %
Calcium	700.0 mg	685.9 mg	98 %
Phosphorus	700.0 mg	1,052.9 mg	150 %
Iron	14.0 mg	9.9 mg	71 %
Potassium	48.3 mmol	64.4 mmol	133 %
Sodium	25.8 mmol	74.7 mmol	290 %
Fiber, Crude		4.1 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA
=====

NAME: SUBJECT 5 2nd Recall

SEX: Female AGE: 36 years FRAME SIZE: Medium

WEIGHT: 66.4 kg (146 lbs.) HEIGHT: 167 cm (5 ft, 6 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/09/01 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL
=====

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	122.4	31.1%	at least 50%
Protein	80.0	20.3%	10-15%
Total Fat	78.0	44.6%	30-35%
Essential Fatty Acid	8.1	4.6%	3%
Saturated Fatty Acid	34.7	19.8%	
Polyunsaturated F.A.	7.7	4.4%	
Monounsaturated F.A.	30.0	17.1%	
Alcohol	9.2	4.1%	

NUTRIENT BREAKDOWN
=====

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		6,582.0 kj	
Protein	49.0 g	80.0 g	163 %
Vitamin A	800.0 RE	620.1 RE	78 %
Vitamin C	45.0 mg	37.4 mg	83 %
Vitamin D	2.5 ug	2.4 ug	95 %
Thiamin	0.8 mg	1.2 mg	152 %
Riboflavin	1.0 mg	1.4 mg	144 %
Niacin	14.4 NE	25.1 NE	175 %
Vitamin B6	1.2 mg	1.0 mg	83 %
Folacin	165.0 ug	129.0 ug	78 %
Vitamin B12	2.0 ug	3.7 ug	184 %
Pantothenic Acid	3.6 mg	3.4 mg	93 %
Calcium	700.0 mg	1,072.3 mg	153 %
Phosphorus	700.0 mg	1,310.1 mg	187 %
Iron	14.0 mg	11.4 mg	82 %
Potassium	49.7 mmol	55.0 mmol	111 %
Sodium	26.5 mmol	137.2 mmol	518 %
Fiber, Crude		4.0 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

NAME: SUBJECT 5 3rd Recall

SEX: Female AGE: 36 years FRAME SIZE: Medium

WEIGHT: 68.5 kg (151 lbs.) HEIGHT: 167 cm (5 ft, 6 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	177.2	36.8%	at least 50%
Protein	109.6	22.8%	10-15%
Total Fat	88.2	41.2%	30-35%
Essential Fatty Acid	13.1	6.1%	3%
Saturated Fatty Acid	36.1	16.9%	
Polyunsaturated F.A.	11.5	5.4%	
Monounsaturated F.A.	33.2	15.5%	
Alcohol	3.6	1.3%	

NUTRIENT BREAKDOWN

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		8,047.0 kj	
Protein	50.7 g	109.7 g	216 %
Vitamin A	800.0 RE	4,374.1 RE	1,797 %
Vitamin C	45.0 mg	107.8 mg	239 %
Vitamin D	2.5 ug	6.3 ug	253 %
Thiamin	0.8 mg	2.1 mg	258 %
Riboflavin	1.0 mg	6.3 mg	628 %
Niacin	14.4 NE	52.0 NE	361 %
Vitamin B6	1.6 mg	2.2 mg	136 %
Folacin	165.0 ug	399.1 ug	242 %
Vitamin B12	2.0 ug	67.6 ug	3,381 %
Pantothenic Acid	4.4 mg	11.1 mg	251 %
Calcium	700.0 mg	1,483.3 mg	212 %
Phosphorus	700.0 mg	1,935.7 mg	277 %
Iron	14.0 mg	22.4 mg	160 %
Potassium	51.4 mmol	81.2 mmol	158 %
Sodium	27.4 mmol	224.5 mmol	819 %
Fiber, Crude		6.5 g	

DATE: 85/10/25

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

NAME: SUBJECT 6 1st Recall

SEX: Female

AGE: 37 years

FRAME SIZE: Medium

WEIGHT: 65.8 kg (145 lbs.)

HEIGHT: 175 cm (5 ft, 9 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/04/14 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	110.0	60.5%	at least 50%
Protein	28.8	15.8%	10-15%
Total Fat	20.6	25.5%	30-35%
Essential Fatty Acid	3.3	4.1%	3%
Saturated Fatty Acid	4.8	6.0%	
Polyunsaturated F.A.	3.2	4.0%	
Monounsaturated F.A.	8.0	9.9%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN

Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		3,041.0 kj	
Protein	48.7 g	28.9 g	59 %
Vitamin A	800.0 RE	438.5 RE	55 %
Vitamin C	45.0 mg	135.9 mg	302 %
Vitamin D	2.5 ug	0.8 ug	30 %
Thiamin	0.8 mg	0.7 mg	90 %
Riboflavin	1.0 mg	0.8 mg	80 %
Niacin	14.4 NE	10.0 NE	70 %
Vitamin B6	0.4 mg	0.6 mg	131 %
Folacin	165.0 ug	176.0 ug	107 %
Vitamin B12	2.0 ug	0.4 ug	20 %
Pantothenic Acid	1.7 mg	2.1 mg	128 %
Calcium	700.0 mg	431.3 mg	62 %
Phosphorus	700.0 mg	634.1 mg	91 %
Iron	14.0 mg	5.2 mg	37 %
Potassium	49.3 mmol	40.9 mmol	83 %
Sodium	26.3 mmol	67.4 mmol	256 %
Fiber, Crude		3.0 g	

DATE: 85/10/28

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

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NAME: SUBJECT 6 2nd Recall

SEX: Female

AGE: 37 years

FRAME SIZE: Medium

WEIGHT: 66.2 kg (146 lbs.)

HEIGHT: 175 cm (5 ft, 9 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/09/01 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

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	Your Intake (g)	% of Total Energy	Recommended
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Carbohydrate	178.7	53.7%	at least 50%
Protein	53.4	16.0%	10-15%
Total Fat	48.9	33.0%	30-35%
Essential Fatty Acid	3.8	2.6%	3%
Saturated Fatty Acid	23.4	15.8%	
Polyunsaturated F.A.	7.3	4.9%	
Monounsaturated F.A.	18.3	12.3%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN

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Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		5,569.0 kj	
Protein	49.0 g	53.5 g	109 %
Vitamin A	800.0 RE	626.3 RE	78 %
Vitamin C	45.0 mg	111.8 mg	248 %
Vitamin D	2.5 ug	1.0 ug	42 %
Thiamin	0.8 mg	0.7 mg	91 %
Riboflavin	1.0 mg	1.1 mg	113 %
Niacin	14.4 NE	18.5 NE	129 %
Vitamin B6	0.8 mg	1.0 mg	123 %
Folacin	165.0 ug	118.0 ug	72 %
Vitamin B12	2.0 ug	2.1 ug	105 %
Pantothenic Acid	3.1 mg	3.1 mg	102 %
Calcium	700.0 mg	852.4 mg	122 %
Phosphorus	700.0 mg	990.6 mg	142 %
Iron	14.0 mg	8.6 mg	61 %
Potassium	49.7 mmol	56.3 mmol	113 %
Sodium	26.5 mmol	67.5 mmol	255 %
Fiber, Crude		4.0 g	

DATE: 85/11/26

* * * N U T R I P R O F I L E * * *

SUMMARY OF PERSONAL DATA

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NAME: SUBJECT 6 3rd Recall

SEX: Female AGE: 37 years FRAME SIZE: Small

WEIGHT: 64.4 kg (142 lbs.) HEIGHT: 175 cm (5 ft, 9 in)

Results refer to an average of 3 day(s) intake.

This data was recorded on 85/11/23 .

PERCENT ENERGY FROM NUTRIENTS AND ALCOHOL

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	Your Intake (g)	% of Total Energy	Recommended
Carbohydrate	162.8	50.8%	at least 50%
Protein	63.9	19.9%	10-15%
Total Fat	41.9	29.4%	30-35%
Essential Fatty Acid	5.2	3.6%	3%
Saturated Fatty Acid	15.9	11.1%	
Polyunsaturated F.A.	5.0	3.5%	
Monounsaturated F.A.	18.1	12.7%	
Alcohol	0.0	0.0%	

NUTRIENT BREAKDOWN

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Nutrient	Recommended Amount	Your Intake	Percent Value
Energy		5,362.0 kj	
Protein	47.7 g	63.9 g	134 %
Vitamin A	800.0 RE	278.0 RE	35 %
Vitamin C	45.0 mg	85.2 mg	189 %
Vitamin D	2.5 ug	0.9 ug	36 %
Thiamin	0.8 mg	0.9 mg	112 %
Riboflavin	1.0 mg	1.4 mg	142 %
Niacin	14.4 NE	21.3 NE	148 %
Vitamin B6	1.0 mg	1.2 mg	123 %
Folacin	165.0 ug	127.8 ug	77 %
Vitamin B12	2.0 ug	3.1 ug	155 %
Pantothenic Acid	2.9 mg	3.3 mg	110 %
Calcium	700.0 mg	741.4 mg	106 %
Phosphorus	700.0 mg	977.9 mg	140 %
Iron	14.0 mg	8.9 mg	63 %
Potassium	48.3 mmol	52.4 mmol	108 %
Sodium	25.8 mmol	64.7 mmol	251 %
Fiber, Crude		2.3 g	