THE EFFECT OF THE MENSTRUAL CYCLE ON ENERGY INTAKE AND DIETING HABITS OF ADOLESCENTS

by

Suzanne Marilyn Cole

A Thesis Submitted to the Faculty of the

DEPARTMENT OF NUTRITIONAL SCIENCES

In Partial Fulfillment of the Requirements

For the Degree of

MASTER OF SCIENCE

In the Graduate College

THE UNIVERSITY OF ARIZONA

1 9 9 5

UMI Number: 1376004

UMI
300 North Zeeb Road
Ann Arbor, MI 48103

STATEMENT BY AUTHOR

This thesis has been submitted in partial fulfillment of requirements for an advanced degree at The University of Arizona and is deposited in the University Library to be made available to borrowers under rules of the Library.

Brief quotations from this thesis are allowable without special permission, provided that accurate acknowledgment of source is made. Requests for permission for extended quotation from or reproduction of this manuscript in whole or in part may be granted by the head of the major department or the Dean of the Graduate College when in his or her judgement the proposed use of the material is in the interests of scholarship. In all other instances, however, permission must be obtained from the author.

SIGNED: Suzanne M. Cole

APPROVAL BY THESIS DIRECTOR

This thesis has been approved on the date shown below:

Cheryl K. Ritenbaugh
Associate Professor
of Family and Community Medicine

July 26, 1995
Date

ACKNOWLEDGEMENTS

I would like to extend to my advising professor, Dr. Cheryl Ritenbaugh my heartfelt gratitude and thanks for her guidance and support with this study. Her encouragement and direction through each step of the project was very much appreciated. A special thanks to my committee members, Dr. Teufel and Dr. Taren for their time and assistance with this thesis. I am grateful to Dr. Aickin for his guidance and suggestions with the statistical analysis.

I dedicate this thesis to my husband, Dan Corrigan; my parents, Arlene and Phil Cole, and my brother, Brian Cole. I want to thank my parents for their loving support and their timely words of encouragement as I pursued this degree. A special thanks to my brother for being such an inspiration to me. Lastly, I am indebted to my husband for his willingness to move to the Southwest so that I could realize my dream. His enthusiasm and support have been unfaltering and for that I am grateful. Thank you Dan for sharing this experience with me.

TABLE OF CONTENTS

TABLE OF CONTENTS - Continued

LIST OF TABLES

LIST OF FIGURES

LIST OF FIGURES - Continued

ABSTRACT

The effect of the menstrual cycle on energy intake and the dieting habits of adolescents was examined retrospectively for three years in 64, eighth and ninth grade girls. Dieting episodes were found to be evenly distributed across the five menstrual phases with no greater proportion of dieting occurring during the follicular phase. Media, peers, family members, and social pressures have a larger impact on adolescent dieting behaviors as opposed to the menstrual cycle. Comparisons of energy intake between the pre- and postovulatory phases revealed no significant differences in any year. Fifty to eighty percent of the girls' cycles may have been anovulatory the first two years of the study. Variations in energy intake are not observed in anovulatory cycles due to low ovarian hormone levels. Changes in food consumption that correspond to menstrual phases may be observed in girls who are six years or more beyond menarche, when cycles are predominantly ovulatory.

FOREWORD

While working at a weight management clinic in a small community hospital, I had the opportunity to observe about 150 patients, predominately women, during a period of two years. The majority of the patients, attempting to lose between 50 and 125 pounds followed a weight loss program that required clinic attendance once a week for six to seven months. During the first 13 weeks, the patients consumed a liquid supplement that provided between 500 and 800 kcal per day. After week 13, the packets of liquid supplement were gradually replaced with solid foods and the total calories per day were increased in small increments per week.

One of my responsibilities was to meet with each individual when they came in for their weekly clinic visit and discuss their progress and adherence to the dietary protocols. One question that was posed by women on more than one occasion was, "I went off my diet when I got my period... can I reduce the number of packets I am taking for a few days until I am back on track?". After about ten women asked similar questions over the course of two years, I began to wonder if restricting food consumption after a menstrual period was a common behavior among dieters in the general population. Were women consciously restricting their intake after menses in response to altered eating habits when premenstrual or menstrual?

INTRODUCTION

There is evidence that the menstrual cycle may influence such behaviors as dieting and eating in premenopausal women. Numerous studies have demonstrated significant changes in energy intake in relation to different menstrual phases (Barr, Janelle, and Prior, 1995; Martini et al., 1994; Johnson et al., 1994; Tarasuk and Beaton, 1991; Lyons et al., 1989; Gong, Garrel, and Calloway, 1989; Lissner et al., 1988; Manocha, Ghoudhuri, and Tandon, 1986; Davit-McPhillips, 1983). Results from animal studies indicate the ovarian hormones, estrogen and progesterone, may be interacting directly or indirectly with physiologic and metabolic pathways that regulate food intake (Bonavera et al., 1994; Wade and Schneider, 1992; Bielert and Busse, 1983; Czaja, 1978).

While researchers have not specifically examined whether there is a relationship between dieting and the menstrual cycle phases in women, several sources of evidence suggest a relationship may exist. My observations at the clinic revealed that women may wish to diet after menses if during this period of time they consumed foods not usually found in their daily eating habits or they consumed greater quantities of food. These behaviors may be reflecting the effect of ovarian hormones on food intake discussed above.

Another factor that may promote dieting after menses is water retention, a premenstrual symptom that effects roughly 30-50% of women (Huerta-Franco and Malacara, 1993; Woods et al., 1982). Carr-Nangle (1994) and Altabe (1990) found that women experienced increased anxiety pertaining to physical appearance and body size around menses. Water retention has been associated with a dissatisfaction of body image as well as a perception that body size has increased. Women may be using dieting as a strategy to reduce a perceived or real weight gain either from water retention or a change in eating habits.

The purpose of this study is to explore whether these findings observed in adult women could be extended to adolescent girls. To date, researchers have not examined the role of the menstrual cycle in food consumption in this population. However, if estrogen and progesterone are influencing the regulation of food consumption in women then it is plausible that similar patterns might also be observed in menstruating adolescents.

Given that dieting is a common behavior among adolescents, several of the factors (water retention and altered eating habits) observed pre- or perimenstrually in women may be influencing the dieting habits of teenage girls (Serdula et al., 1993; Moore, 1993; Wardle and Marsland, 1990). Changes in food intake as well as water retention appear to be regulated directly or indirectly by estrogen and progesterone. Therefore, menstruating girls could experience these factors as well as the associated anxieties that have been observed in women.

Current research on the teenage girls indicates that media, peers, family, and society influence dieting practices (Nichter and Vuckovic, 1994; Killen et al., 1992; Moore, 1993; Casper and Offer, 1990). Determining if the menstrual cycle also influences the dieting practices in adolescents will contribute to our understanding of the motivations behind this behavior. Lastly, assessing whether the cycle effects food intake in girls will provide further insight into the role of ovarian hormones on the regulation of food consumption.

Chapter 1

REVIEW OF LITERATURE

Menstrual Cycle and Dieting

There is some evidence to support the idea that women may be dieting after menstruation. As mentioned earlier, Carr-Nangle (1994) and Altabe (1990) found that women experience a greater number of negative thoughts about their physical appearance and body image when they are pre- and perimenstrual. One of the factors that contributed significantly to the anxiety was water retention, the most common perimenstrual symptom reported (Logue and Moos, 1986). A perceived shift in body size has been correlated with water retention. Women experiencing this side-effect indicate that their waist and body feel larger. Altabe (1990) reported that some women who expressed that their waists were larger found waist sizes to be the same size when pre- and postmeasurements were compared. Although measurements may not necessarily reflect a shift to a larger size, Woods (1982) found that approximately 48% (n=839) of women experience a weight gain when premenstrual and 33% when menstrual. Boyle (1987) and Halbreich (1982) reported 81 - 83% of women experience an increase in weight around menses.

Given our society's obsession for thin figures, it is reasonable to suggest that women may respond to these anxieties by dieting. The women I observed at the clinic expressed a desire to reduce their calories further, because they had consumed foods that were not prescribed on the diet (cake, brownies, ice cream, and lasagna). However, if they were also experiencing water retention, this side-effect could have magnified their concerns of gaining versus losing weight.

Dieting and Adolescent Girls

Among adolescent girls, dieting is a common phenomenon. Numerous studies have shown that between 44 - 61% of high school girls are dieting (Serdula et al., 1993; Wardle and Marsland, 1990; Desmond, 1986; Dwyer, Feldman, and Mayer, 1967). The National Adolescent Student Health Survey, which surveyed over 11,000 eighth and tenth graders, found that 61% of the female students had dieted in the past year (American School Health Association, 1988).

Dieting behaviors are not limited to Caucasian girls. Serdula (1993) reported among high school girls attempting to lose weight, 47% were Caucasian, 39% were Hispanic, and 30% were African-American. Nichter et al. (in press) found in a sample of Caucasian, Mexican-, Asian-, and Native-American adolescent girls, 63% had dieted. Among 6697 Native-American girls, 48% reported dieting (Story et al.; 1994). Although dieting appears to occur across racial groups, Casper (1990) and Wardle (1989) found that African-

American girls and to a lesser extent, Asian girls were less likely to be concerned about weight and body size when compared with Caucasian girls.

The primary reason for dieting is that girls are dissatisfied with their physical appearance and weight (Casper and Offer, 1990; Wardle and Marsland, 1990; Moore, 1988). Desmond (1986) found that 43% of normal-weight Caucasian girls perceived themselves to be heavy. Sixty percent of 248 racially diverse, urban high school girls responded that they were "terrified" of gaining weight (Casper and Offer, 1990). Concerns about weight gain and shape appear to increase as adolescents become older (Wardle and Marsland, 1990). This may reflect the increase of adiposity that occurs during puberty.

Nichter and Vuckovic (1994) reported that 44% of eighth and ninth grade girls wanted to be thin in order to be more appealing to the boys. Maternal attitudes about size and shape can also contribute to adolescent perceptions of an "ideal" weight and size.

Given that girls are concerned about their weight and appearance, they may experience anxieties similar to those observed in women around menses. Teenage girls may even be dieting more frequently after menses. Their anxieties about body image may be compounded around menstruation if they perceive a shift in body size (due to water retention) or a change in eating habits that promotes weight gain.

Menstrual Cycle and Food Intake

Researchers have demonstrated a relationship between the menstrual cycle and food intake in premenopausal women. Variations in food consumption corresponding to phases of the menstrual cycle have been demonstrated in numerous human studies (Barr, Janelle, and Prior, 1995; Tarasuk and Beaton, 1991; Gong, Garrell, and Calloway, 1989; Lyons et al., 1989; Lissner et al., 1988; Dalvit-McPhillips, 1983). Energy and macronutrient intakes have been shown to decrease during the ovulatory phase when estrogen levels are surging and progesterone levels are very low (Lyons et al., 1989). During the luteal phase, intakes rise coinciding with an increase in progesterone and a drop in estrogen levels (Barr, Janelle, and Prior, 1995; Tarasuk and Beaton, 1991; Lissner et al., 1988; Dalvit-McPhillips, 1983). Figure 1 depicts the changes in estradiol (the most biologically active estrogen secreted by the ovaries) and progesterone during the menstrual cycle, whereas, Table 1 reviews the menstrual phases.

These patterns in energy intake throughout the menstrual cycle have also been reported in primates (Bielert and Busse, 1983; Rosenblatt et al., 1980; Czaja, 1978). In two separate studies using adult female Rhesus monkeys, food intake was greater during the luteal phase as compared to the follicular phase. Rosenblatt's (1980) study also found that food consumption was depressed around ovulation.

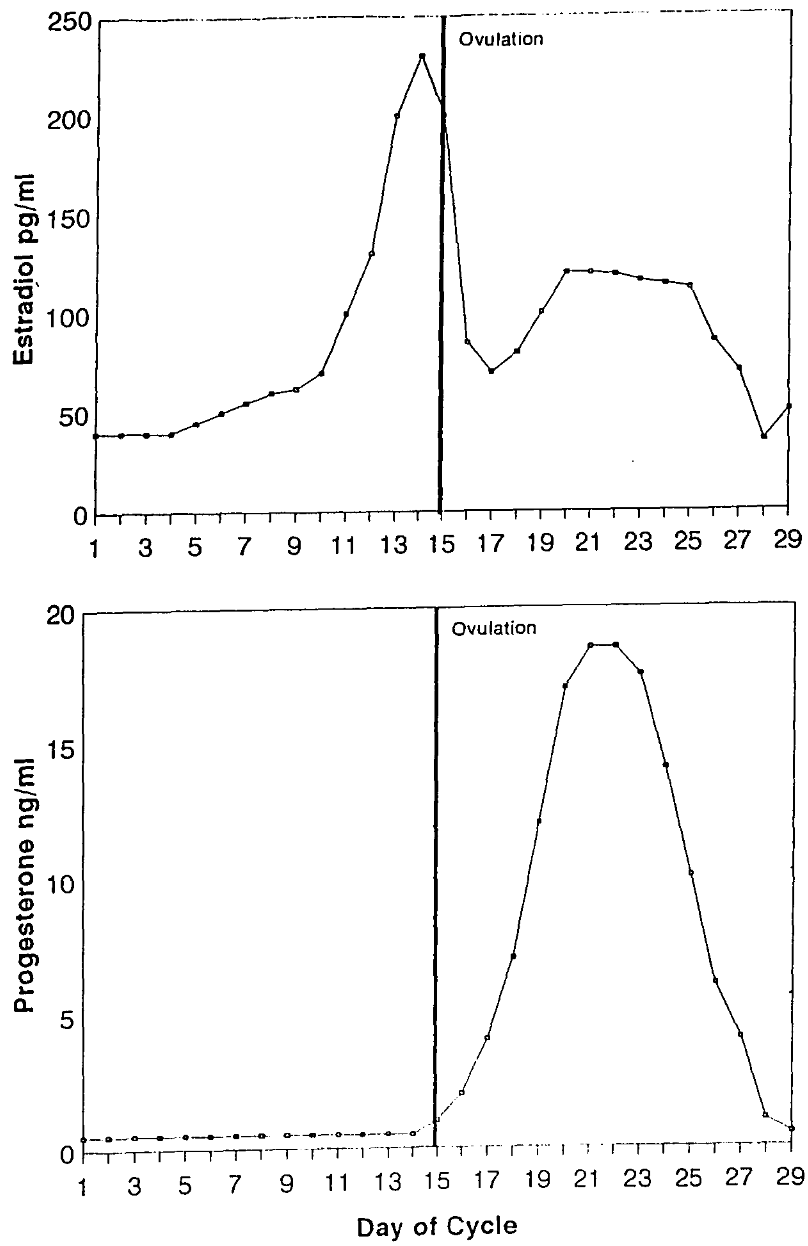

Figure 1. Ovarian hormone changes during the menstrual cycle. (Adapted from Thorneycroft IH, Boyers SP. (1983). The human menstrual cycle: correlation of hormonal patterns and clinical signs and symptoms. Obstet Gynecol 12:199-225.)

Table 1. Phases of a 28-day menstrual cycle.

Phase	Menstrual cycle days
Menses	Days 1 - 4 from beginning of menses
Follicular	Days between menses and ovulation (5 - 12)
Ovulation	4 days around ovulation (13 - 16)
Luteal	All days after ovulation and before the onset of menses. (17 - 28)

Bielert (1983) observed that wild baboons spend less time foraging and feeding during ovulation. Tables 2 and 3 summarize the results of human and primate studies measuring food intake during the menstrual cycle. Comparisons among the phases varied between studies, therefore the tables are a simplification of the results illustrating the trends.

Changes in food intake during the menstrual cycle have not been studied in menstruating adolescent girls. It would be interesting to determine if teenage girls also exhibit a food consumption pattern that corresponds to ovarian hormone levels. Such findings could further our understanding of the effect of ovarian hormones on the regulation of food intake and eating behaviors.

The underlying premise is that the intake patterns are occurring because of cyclical changes in estrogen and progesterone levels that are directly or indirectly modifying the regulation of satiety and eating. Evidence from animal studies strongly suggests that estrogen is an appetite suppressant.

Estrogen and the Regulation of Food Intake

Animal Studies

Food consumption is regulated by a complex network of metabolic pathways each influenced by numerous behavioral and physiological factors. There is a wealth of information about food cravings and the environmental and behavioral

Table 2. Summarizes the changes in human food consumption during the menstrual cycle.

		Ovulation				Luteal			
Study	SS	CHO	Prot	Fat	Energy	CHO	Prot	Fat	Energy
Barr	29							⇑	⇑
Fong	9	↓		↓	↓	⇑		↑	⇑
Lyons	18	⇓	⇓		⇓	↑			↑
Gong	7				↓				⇑
Tarasuk	14					↑		⇑	⇑
McPhillips	8					⇑			⇑
Lissner	23								⇑

SS = sample size
⇑⇓ = significant results
↑↓ = indicates a trend

Table 3. Summarizes the changes in primate food consumption during the menstrual cycle.

		Ovulation				Luteal			
Study	SS	CHO	Prot	Fat	Energy	CHO	Prot	Fat	Energy
Bielert	17	na	na	na		na	na	na	⇑
Czaja	6	na	na	na	⇑	na	na	na	⇑
Rosenblatt	14	na	na	na	⇑	na	na	na	⇑

SS = sample size
⇑⇓ = significant results
na = macronutrients were not examined in these studies.

factors that influence eating. For this study, I will focus on the physiologic and metabolic elements that contribute to the regulation of eating that are reportedly affected by estrogen.

There are several classic animal studies using ovariectomized rats and monkeys that clearly demonstrate the effect of estrogen and progesterone on feeding. Using ovariectomized rats, Wade (1976) found that the animals increased their food intake and body weight after the procedure. When the same animals were treated with physiologic levels of estrogen, their food intake and body weight decreased to pre-operative levels. Progesterone administered to the ovariectomized rats had no effect on food consumption or body weight. However, when progesterone was administered to rats who had intact ovaries, the hormone caused an increase in food intake and weight (Wade, 1992). These findings were also observed in ovariectomized monkeys (Kemnitz, 1989; Bielert, 1983).

Based on these results, progesterone treatment alone does not appear to elicit any changes in food intake. Researchers have demonstrated that either estrogen or a combination of progesterone and estrogen stimulated changes in levels of food intake. The characteristics of progesterone receptors necessitates that both hormones be present for progesterone to exert its effects on tissues. When a greater concentration of progesterone receptors are required (during the luteal phase, for example) a minimum level of estrogen is necessary to stimulate the synthesis of more receptors.

These studies also suggest that progesterone is inhibiting the anorectic effects of estrogen. Although the exact mechanism is not known, progesterone has been shown to reduce the number of estrogen receptors, thus decreasing the actions of estrogen (Greenspan and Baxter, 1994).

Estrogen and the Central Nervous System

The effect of estrogen on food intake has also been demonstrated in brain implant and lesion studies. An important control center for food intake and satiety is located in two regions of the hypothalamus, the ventromedial hypothalamus (VMH) and the lateral hypothalamus. The lateral hypothalamus has been termed the feeding center because studies have shown that lesions in that region caused hypophagia and decreased body weight in rats. The VMH is generally called the satiety center. Animal studies have shown that when the VMH was stimulated, food consumption decreased even if the animal was starving (Wade, 1986).

In the 1970's, researchers suggested that the effect of estrogen on food intake occurred because the hormone was stimulating the VMH which in turn, caused satiety (Wade, 1976; Jankowiak and Stern, 1974). When estrogen levels decreased, the stimulatory effect was removed from the VMH and the lateral hypothalamus could promote feeding.

More recent studies have shown that the paraventricular nucleus (PVN), a region of the hypothalamus adjacent to the VMH, is a critical regulatory site for feeding (Butera and Beikirch, 1989; Palmer and Gray, 1989). In addition, the PVN appears to be a major site for estrogen binding based on the number of estrogen containing neurons located in the region (Butera, 1992; Wade, 1992).

In separate studies, Wade and Butera demonstrated that ovariectomized rats treated with estradiol implants in the PVN decreased their food consumption. Lesions to the PVN have been shown to cause hyperphagia and obesity. When ovariectomized rats with PVN lesions were implanted with estrogen, no change in feeding was observed. Butera (1992) has proposed that estrogen stimulation of the PVN modifies the neurochemical components of PVN neurons that extend into the brain stem triggering satiety messages.

Estrogen and Neuropeptide Y

Neuropeptide Y, a member of the pancreatic polypeptide family, is one of the most abundant peptides in the central nervous system with high concentrations found in the hypothalamus (White, 1993). Functioning as a neuro-transmitter, the peptide causes a profound, long-lasting stimulation of food intake in animals (Bonavera, 1994; Sahu, 1992). In fact, there is a strong correlation between neuropeptide Y levels and feeding. Injections of the peptide into the PVN of rats causes a substantial increase in food intake, specifically carbohydrates and to a lesser extent, fat (Stanley et al., 1989). Very low levels of neuropeptide Y administered to satiated rats caused feeding, whereas a continuous infusion of the peptide caused hyperphagia and obesity (White, 1993).

A recent study confirmed the results of earlier work that estrogen decreases neuropeptide Y concentrations in the hypothalamus, thus causing a reduction in food consumption (Bonavera, 1994). Ovariectomized rats treated with estradiol exhibited decreased food intake and decreased levels of neuropeptide Y as compared to the control rats. The findings suggest that estrogen inhibits the secretion of the peptide in the PVN, however, a reduction in the synthesis or an increase in the degradation of neuropeptide Y should not be ruled out.

Given that estrogen levels are the highest around ovulation, the observed reduction in food intake in women may occur in part, due to the inhibitory effect of estrogen on the generation of neuropeptide Y. However, once estrogen levels are lowered and inhibited by progesterone during the luteal phase, neuropeptide Y would no longer be suppressed by the hormone, enabling the neurotransmitter to stimulate carbohydrate and fat intake.

Estrogen and Norepinephrine

Norepinephrine-stimulated feeding occurs primarily in the PVN of the hypothalamus. It has been shown in rats that injections of norepinephrine into the PVN stimulates carbohydrate consumption (Leibowitz, 1985). In fact, norepinephrine and neuropeptide Y are found in the same neurons in the PVN and are released simultaneously when the nerves are stimulated (Leibowitz and Stanley, 1986).

One of the effects estrogen may have on norepinephrine is a reduction in the synthesis of the neurotransmitter. The catabolism of estrogen produces a product called catecholestrogen. This compound inhibits an enzyme, tyrosine hydroxylase, which is the critical enzyme for the synthesis of norepinephrine (Rosenberg, 1991). When estrogen levels are low, tyrosine hydroxylase is actively synthesizing norepinephrine. However, when estrogen levels are quite high, the concentration of catecholestrogen increases, suppressing the activity of tyrosine hydroxylase. Theoretically, during the ovulatory phase when estrogen levels are surging, norepinephrine levels might be suppressed causing a reduction in carbohydrate intake. During the luteal phase when estrogen levels are at a lower concentration, norepinephrine could be synthesized, increasing carbohydrate consumption.

Considering that both neuropeptide Y and norepinephrine stimulate carbohydrate (and fat to a lesser extent) intake, these two neurotransmitters may be partly responsible for the increased consumption of carbohydrates and fat observed in women during the luteal phase.

Estrogen and Cholecystokinin

The ability of the peptide hormone, cholecystokinin (CCK) to induce satiety has been documented in animal and to a lesser extent in human studies (Morley, 1987). It can inhibit feeding, peripherally through the vagus nerve or act directly on the hypothalamus. Estrogen appears to effect CCK by enhancing the ability of CCK to inhibit food intake (Geary et al, 1994; Butera et al., 1993). Ovariectomized rats exhibited decreased food intake when given CCK injections, however ovariectomized rats treated with both estrogen and CCK exhibited an even greater decrease in food consumption. Although the exact mechanism is not known, it appears that by binding to the PVN, estrogen can magnify the incoming peripheral receptor signals from CCK (Geary et al., 1994).

Estrogen and Lipid Metabolism

Estrogen may alter food intake peripherally through its actions on lipid metabolism. The effect of estrogen on adipose tissue has been studied extensively in rats. Estradiol decreases fat stores in rats by mobilizing free fatty acids from adipocytes as well as decreasing fatty acid uptake by the adipose tissue (Giudicelli et al., 1993; Wade, Gray, and Bartness, 1985). Studies using ovariectomized rats showed an increase in their fat stores, whereas subsequent treatment with estradiol caused a decrease in fat stores (Giudicelli et al, 1993; Palmer and Gray, 1986).

The mechanisms by which estradiol alters lipid metabolism appears to be multiple. Estrogen stimulates lipolysis of triglycerides in adipocytes by increasing the activity of adenylate cyclase which enhances the cell's responsiveness to catecholamines (Giudicelli et al, 1993). Estrogen also inhibits the activity of adipose tissue lipoprotein lipase (LPL), causing a decrease in the uptake of fatty acids from chylomicrons or very-low density lipoproteins (VLDL) (Palmer and Gray, 1986; Kim and Kalkhoff, 1978). Finally, estrogen may act distally by altering the production of apolipo-proteins affecting the ability of LPL to hydrolyze triglycerides from chylomicrons or VLDLs (Deeley, Tam, and Archer, 1985; Kim and Kalkhoff, 1978).

The combined effects of estrogen on fatty acid uptake and release from the adipose tissue is elevated triglyceride levels. A plausible explanation for this outcome is that estrogen is redirecting a fuel source to be used by reproductive tissues. Estrogen does not affect the LPL activity in other tissues such as the heart, lung and the mammary glands, consequently these tissues can take up fatty acids.

The relevancy of elevated triglycerides levels to food intake is that fatty acid oxidation by tissues utilizing the available fuel source may be triggering an afferent signal via the autonomic nervous system causing the central nervous system to induce satiety (Langhans and Scharrer, 1992). A reduction in food intake during ovulation (when estrogen levels are surging) has been observed in animals and women, and may be mediated by the effects of estrogen on lipid metabolism.

Menarche

Menarche, occurring during the later stages of puberty, signifies the reproductive maturation of an adolescent girl. This maturation is characterized by the development of a hormonal feedback loop system between the hypothalamus, pituitary, and the ovaries, that regulates menstruation and ovulation (Figure 2). A brief discussion of pubertal events that lead to the onset of menarche and the role of the feedback loop system in stimulating ovulation will follow.

At the onset of puberty, the hypothalamus begins to secrete increased levels of gonadotropin-releasing hormone (GnRH) which in turn stimulates the pituitary to secrete follicular-stimulating hormone (FSH) and luteinizing hormone LH). FSH induces the development of the follicles and the secretion of estrogen from the ovaries. Higher

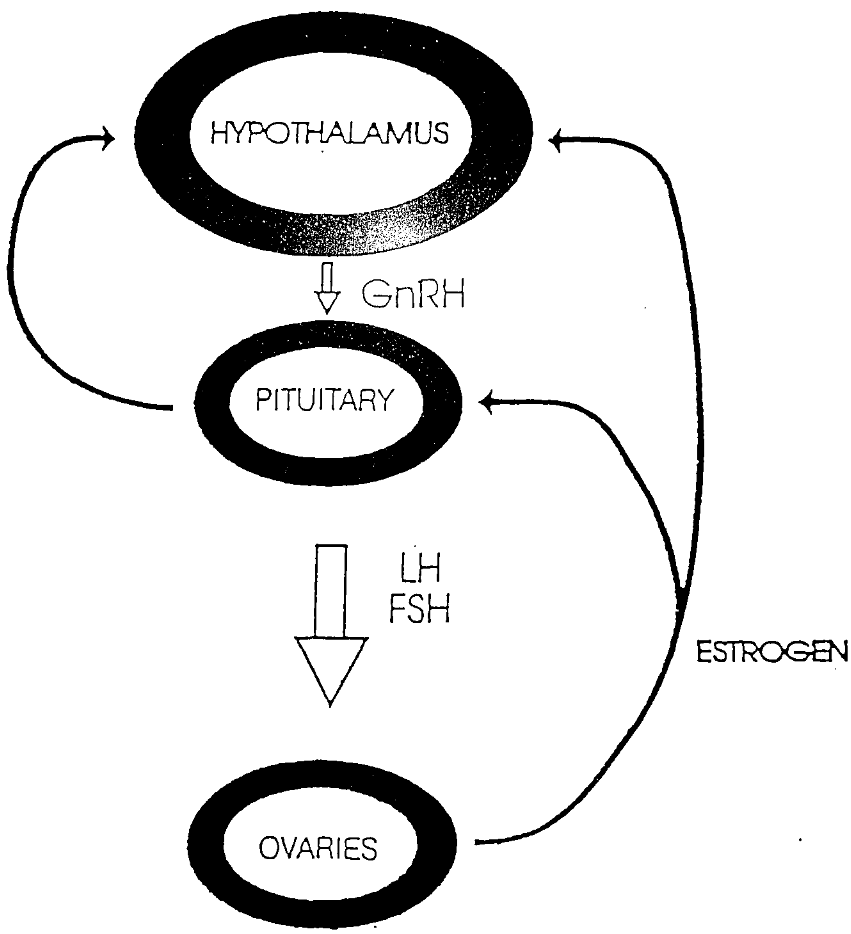

Figure 2. Regulation of gonadotropin release via feedback-loop system. GnRH = Gonadotropin-releasing hormone; LH = Luteinizing hormone; FSH = Follicular-stimulating hormone. (Adapted from Ferin M; The Menstrual Cycle, Oxford University Press, New York, 1993)

concentrations of estrogen stimulates the proliferation of the endometrium causing menstruation.

Up to this point, the primary function of the feedback loop system is to prevent excess production of GnRH. The hypothalamus secretes GnRH which stimulates the pituitary to release FSH and LH. These two hormones target the ovaries causing folliculogenesis and estrogen production. The secretion of estrogen feedback inhibits the release of GnRH from the hypothalamus. The significance of the feedback system is evident during the induction of ovulation. The rapid rise of estrogen levels causes a positive (stimulatory) feedback effect on the hypothalamus and pituitary triggering a surge in LH levels. This surge of LH causes ovulation.

The relevancy of this feedback loop system pertains to the continuing reproductive maturity of adolescents after menarche. Initially, the menstrual cycles of teenage girls are irregular in length and anovulatory. These cycles occur because the positive feedback effect of estrogen that stimulates the LH surge is not fully developed. Several researchers have found that the occurrence of anovulatory cycles decreases as postmenarcheal years increase (Metcalf et al, 1983; Apter 1980). The number of ovulatory cycles the first year of menarche ranged from 7-22%, 25% for the second year, 45-50% by the third postmenarcheal year, and 80-90% by the sixth year.

The length of adolescent menstrual cycles are also effected by the immaturity of the hypothalamic-pituitary-ovarian feedback system. Figure 3 summarizes the amount of variability in the length of the menstrual cycle in menarcheal, reproductively mature, and menopausal subjects. The average cycle length in sexually mature women is 28 days with a range of 24 to 33 days (Thorneycroft and Boyers, 1983). If menarcheal and menopausal subjects are added to the sample, the average cycle length remains 28 days, however the range increases to 22-41 days.

The occurrence of anovulatory cycles in adolescents may be important when trying to establish a relationship between the menstrual cycle and food intake. Barr (1995) found that energy intake did not change in relation to menstrual phases in women who experienced anovulatory cycles.

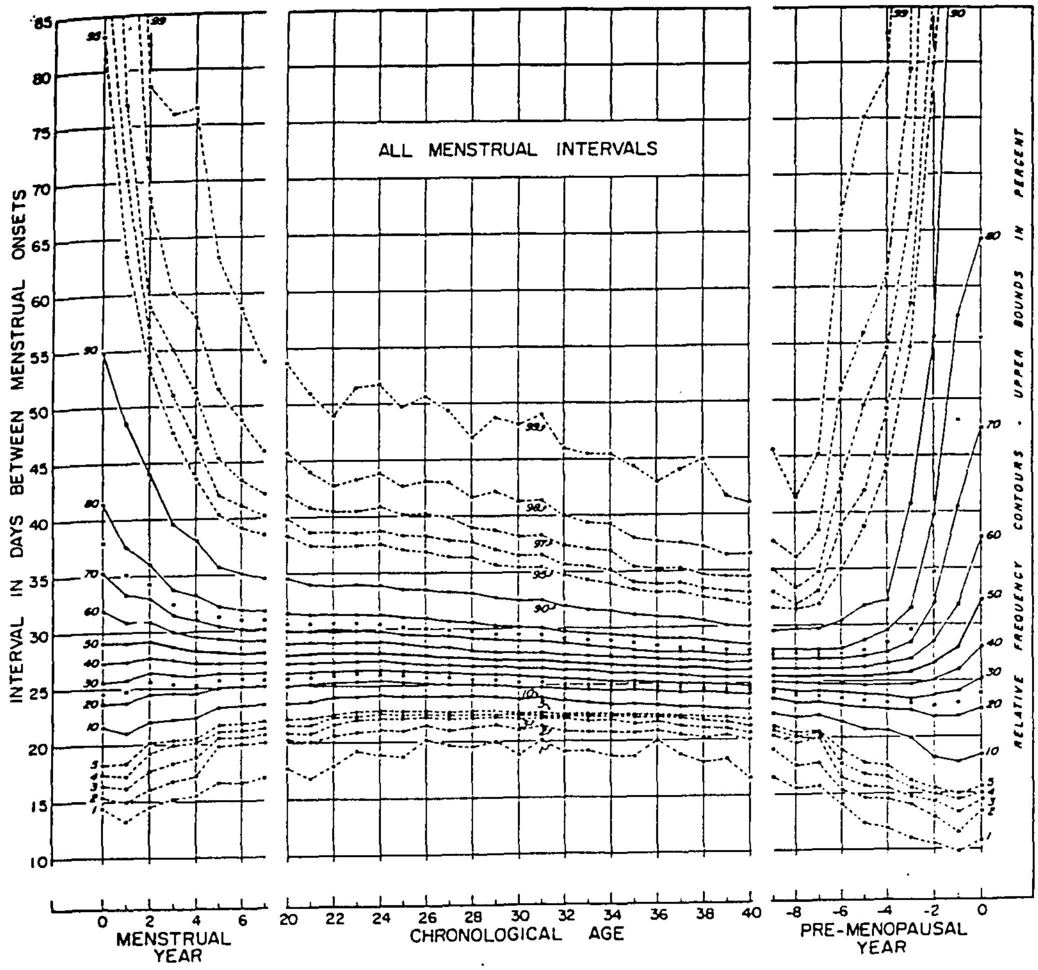

Figure 3. Frequency Distribution of menstrual cycle intervals. (From Treloar AE, Boynton RE, Borghild GB, Byron WB. (1967). Variation of the human menstrual cycle through reproductive life. Int J Fertil 12:77-126.)

Chapter 2

METHODOLOGY

Study Design

This retrospective study evolved from a three-year longitudinal study called the Teen Lifestyle Project. The purpose of the project was to examine dieting and smoking behaviors and body image perceptions in teenage girls. The project began during the summer of 1989 and continued until the spring of 1992. Utilizing the data of a subset of the study participants from the Teen Lifestyle Project, the present study was designed to assess the effect of the menstrual cycle on food intake and the dieting habits of adolescent girls. The following hypotheses were tested:

Hypothesis 1: Adolescent girls are dieting more frequently during the follicular phase.

Hypothesis 2: Food consumption will be increased during the luteal phase and decreased during ovulation.

Study Sample

The subjects used in this study were drawn from a cohort of 257 girls who participated in the Teen Lifestyle Project. Selection of the study population for this sub-study was based on the criteria that the subject had indicated on a food journal an attempt to lose weight at least once in the three years of study. Each time a girl recorded her diet in a food journal, she also answered "yes" or "no" to the question "Were you trying to lose weight today?". If a girl answered "yes" to the question in one or more food journals, then she was included in the study sample. Out of the 257 girls in the Teen Lifestyle Project, 64 (25%) met that requirement. Upon entry into the study, 60% of the girls were eighth graders and 40% were ninth graders.

The study population was predominantly Caucasian (61%) with 23% Mexican-Americans, 5% Asian, 3% Native-Americans, 3% African-Americans, and 5% having an ethnicity other than the categories provided (Table 4).

Recruitment

Three hundred eighth and ninth grade female students from four urban junior and senior high schools in the Amphitheater School District in Tucson, Arizona volunteered to participate in the Teen Lifestyle Project. The study began the fall of 1989 and followed the cohort of girls until the spring of 1992. Forty-three of the participants dropped out of the study, because of their families moving out of the area and decreased time to commit to the project.

Recruitment was held in physical education classes where the Teen Lifestyle Project staff introduced and described the study to the students. In order to be selected as a study subject, the girls had to attend one of

Table 4. Distribution of ethnicity among the subjects.

	Frequency Count (n=61)	(Percent)
Caucasian	37	(60.66)
Native-American	2	(3.28)
Mexican-American	14	(22.95)
African-American	2	(3.28)
Asian	3	(4.92)
Other	3	(4.92)

the participating schools, be in eighth or ninth grade, understand English, and complete the informed consent process. 82% of the eligible girls participated in the project.

The girls consented to interviews, surveys, height and weight measurements, recording their diet in food journals, and providing dates of their last menstruation. Informed consents were signed by both the student and a parent. Permission to recruit was obtained from the Amphitheater School District. The Teen Lifestyle Project was reviewed and accepted by the Human Subjects Committee, University of Arizona.

Data Collection and Management

Dietary Intake Data

The subjects recorded their dietary intake on a form called the Teen Lifestyle Project Food Journal. The journal was designed specifically for an adolescent population with a format that maximized the quality of dietary recording while providing simplified instructions and examples for estimating portions. The girls received instructions and practice in completing the 24-hr food journal and estimating portion sizes. Training classes were conducted in the fall of 1989 by project staff members.

Subjects were requested to complete six food journals each year for three years. Approximately every six months (during the fall and spring semester) three food journals were randomly assigned over a three-week period with only one journal to be completed in a given week. The three days included two weekdays and one weekend day. A journal was mailed to each girl several days before her assigned date with a postage-paid envelope enclosed for returning the completed journal. Girls were called the night of their assigned day to assure that they had recorded their diets, to answer any questions, and to remind them to mail the journals when they were completed.

Dietary Data Entry

Nutritionist III v.6 software was used for coding and entering dietary data from the food journals. School lunch recipes and portion sizes were added to Nutritionist III to customize the foods database for the project. Snack foods and fast foods were also added to the software to increase the quality and quantity of data entry of reported food items. A limitation of Nutritionist III was that it underestimated dietary fat, reportedly as much as 20% (Mattes and Gabriel, 1988). To overcome this limitation, food codes and gram weights generated from Nutritionist III were matched to a USDA's CSFII-85 food composition data base. This produced a nutrient analysis that was based on USDA food composition tables.

To assure the quality of the dietary data entry, 10% of the diet records were randomly selected for re-entry into Nutritionist III by a second coder. The duplicate records were reviewed and discrepancies were reconciled.

Assessment of Menstrual Phases

Most researchers divide the menstrual cycle into four phases that correspond to specific reproductive processes and ovarian hormone levels. To determine the effects of the menstrual cycle on food intake, studies may compare intakes between the four standard phases: menses, follicular, ovulation, and luteal. Other studies have compared intakes by dividing the menstrual cycle into two phases, follicular and luteal. The importance of using the four-phase method over the two-phase method for comparing dietary intakes relates to the evidence that was discussed earlier that intake may decrease during ovulation. A decrease in food consumption during ovulation would be missed if intakes were compared between the follicular and luteal phases.

A third method of assessing energy changes across the menstrual cycle utilizes a fifth phase called postovulation (Lyons, 1989). The postovulatory phase, which is four days in length, occurs just after the ovulatory phase and is characterized by low estrogen levels and steadily rising progesterone levels. The purpose of this phase is to examine whether the coinciding changes in progesterone and estrogen levels are effecting food consumption. Since a goal of the present study was to assess whether ovarian hormones were effecting the food intake and dieting habits of adolescent girls, five phases (menses, follicular, ovulatory, postovulatory, and premenses) were used for comparisons.

Determination of Phases

Determining in which of the five phases the subjects' dietary intake occurred was accomplished by using menstrual data obtained from each of the girls' food journals. On the days that the girls recorded their diet in a food journal, they were also instructed to circle the first day of their last menstruation on a calendar provided in the journal. If they had not reached menarche, they answered "no" to the question, "Have you ever had a menstrual period?". Using the date of a subject's last menstrual period and the date of the food journal, the following steps were used to calculate phase information:

1) If a girl filled out her food journal up to nine days after the onset of menses, then menses (day 1-4) or the follicular phase (day 5 - 9) could be assigned to that day of dietary intake. If she recorded her diet 10 days or more after the first day of her menstrual period, then no phase designation was assigned (see Figure 4 for an example). The reason for not assigning a phase under these conditions is that whereas the luteal phase is the most constant phase

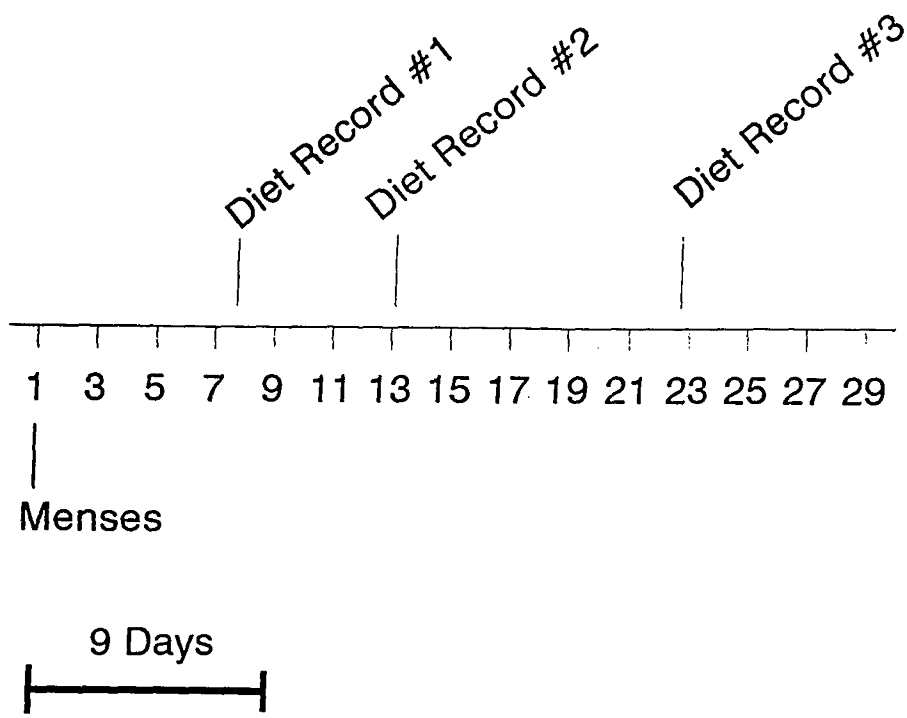

Figure 4. An example of assigning phases by counting forward nine days.

Diet Record 1.	Count forward from menses 8 days 8 days=Follicular Phase
Diet Record 2.	Count forward from menses 13 days 13 days=No phase assignment
Diet Record 3.	Count forward from menses 23 days 23 days=No phase assignment

in length (12 $\pm$ 1 days in a 28-day cycle), the duration of the follicular phase varies in accordance with changes in the length of the menstrual cycle (Thorneycroft and Boyers, 1983). For this reason, counting forward from menses beyond day 10 could produce an erroneous phase if a subject had anything but a 28-day standard cycle length. By counting forward nine days, it was assumed that cycle lengths would not be less than 25 days. Eighty-one percent of 103 cycles were greater than 24 days in length (Figure 5).

2) If a subject got her menstrual period again before the last assigned food journal in the three-week time frame, that period date could be used to calculate phase information for the third journal by counting forward no greater than nine days. The date of menses might also be used to determine the phases for food journals #1 and #2 by counting backwards from menses up to 20 days (see Figure 6 for an example). A diet record that occurred on day -1 to -7 would be considered premenses, -8 to -12 is considered postovulation, -13 to -16 ovulation, and -17 to -20 are four days into the follicular phase. Counting beyond 20 days may again produce an inaccurate phase since the follicular phase length is so variable. Using these coding rules, menstrual cycle phases could be determined for 51% of the 680 food journals completed by the subjects.

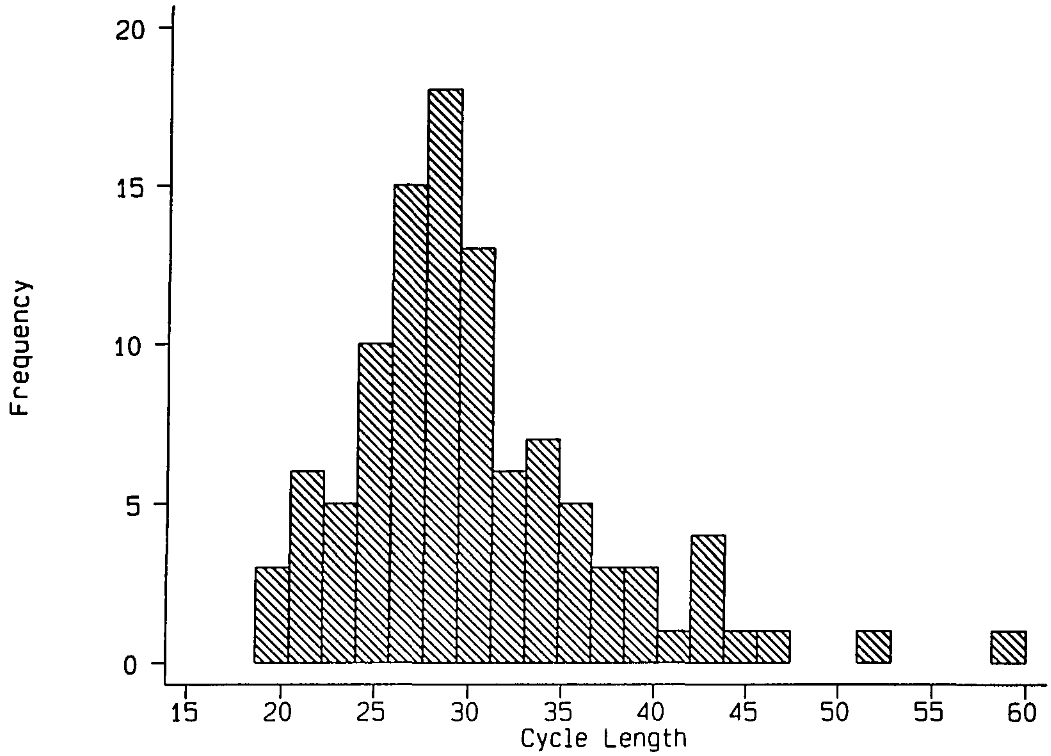

Figure 5. Frequency distribution of 103 menstrual cycle lengths.

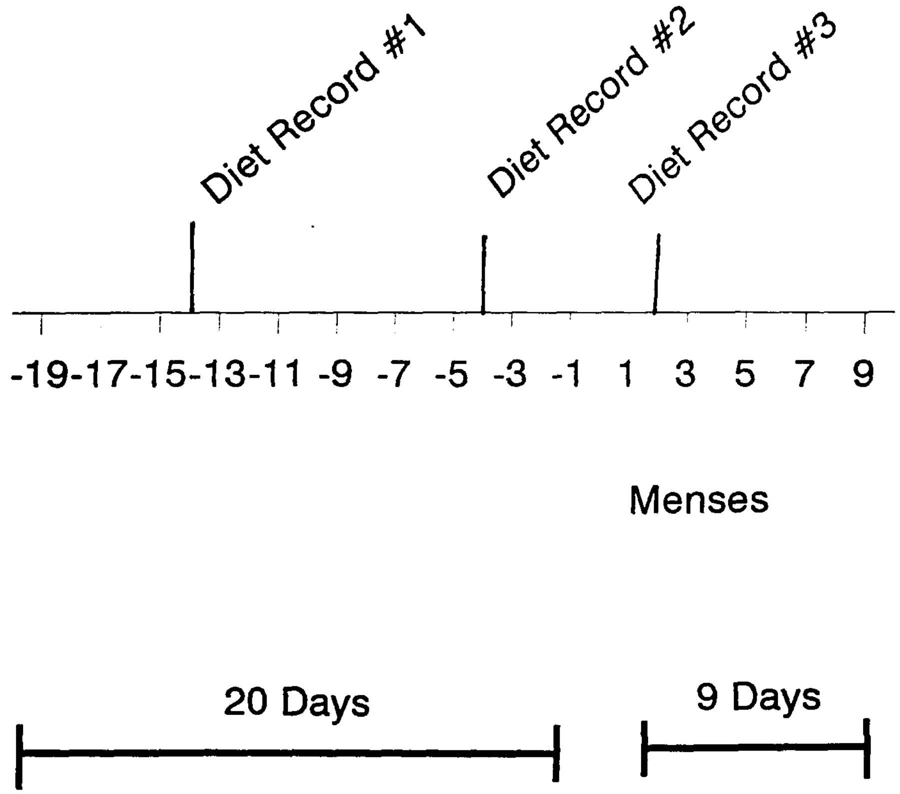

Figure 6. An example of assigning phases by counting forward nine days and backwards 20 days.

Diet Record 1. Count backwards from menses -14 days
-14 days=Ovulatory Phase

Diet Record 2. Count backwards from menses -4 days
-4 days=Premenses Phase

Diet Record 3. Count forward from menses 2 days
2 days=Menses Phase

Dieting Data

Responding "yes" to the food journal question, "Were you trying to lose weight today?" designated that particular diet record as a "dieting day". Of the 680 food journals returned by the girls, 205 (30%) were considered dieting days. It was possible to designate one of the five phases to 126/205 food journals.

Menarche Data

Menarcheal age was provided by the subjects through a baseline survey and again during an in-depth interview. Their responses from the survey and interview were then reconciled to assure accuracy.

The number of years elapsed from the onset of menarche (postmenarcheal years) was calculated by subtracting the age of a girl on June 1, 1992 from her menarcheal age. Given that eight of the girls did not begin menstruating until after entry into the study, a date was chosen at the end of the third year in order to include as many menarcheal years as possible.

Postmenarcheal years were also assigned to individual diet records for the purposes of analysis. This was accomplished by subtracting the date of the diet record from a subject's date of birth. Postmenarcheal years could then be calculated by subtracting a girl's age at menarche from the age produced in the previous step.

Height and Weight Data

Study subjects were measured for height and weight by Teen Lifestyle Project (TLP) staff during each spring semester for three years. The girls also reported their height and weight in surveys collected each year. Reported height and weight measurements were used rather than the measurements collected by TLP staff because the data was more complete over the three years. The measurements reported by the girls were considered reliable as there were no statistically significant differences between the measured and reported height and weight data (Tables 5 and 6).

During the first year, the subjects under-reported their weight by 4.8 pounds. At this time, many of the girls were only 1.5 to 3.5 years from menarche and still experiencing some growth in height as well as an increase in adiposity. It is possible that they were not weighing themselves frequently enough to observe the increase.

It is interesting to note that although the reported and measured height and weights were not significantly different, the reported measurements were consistently lower. Similar results was also observed by Fortenberry (1992) and Tienboon (1992) who found that adolescent girls underestimated height by 0.5 - 1.1 cm and weight by 0.8 - 1.5 kg.

Table 5. Comparison of reported and measured heights (cm) of study population (x ± SD).

Year	n	Reported	n	Measured	T†	P‡
1990	51	1.62 ± .07	35	1.63 ± .06	0.79	.437
1991	39	1.64 ± .06	43	1.64 ± .06	1.56	.129
1993	45	1.66 ± .06	35	1.67 ± .06	-0.50	.619

† T value from paired t-test.
‡ P value from paired t-test.

Table 6. Comparison of reported and measured weights (kg) of study population (x ± SD).

Year	n	Reported	n	Measured	T†	P‡
1990	51	58.2 ± 9.2	35	60.4 ± 9.9	2.46	.020
1991	39	60.8 ± 9.7	43	61.0 ± 9.9	2.21	.035
1992	45	62.8 ± 11.2	35	63.0 ± 9.4	1.54	.134

† T value from paired t-test.
‡ P value from paired t-test.

Body Mass Index Data

Body mass index (BMI) was calculated using height and weight measurements reported by the subjects as an indirect method of estimating fatness. The following equation was computed:

$$BMI = weight\ (kg)/height(m)2$$

In the female adolescent population, body mass index is considered an acceptable measure of fatness when assessing body composition (Buckler, 1990; Roche et al., 1981).

Statistical Analysis

All of the data analyses were conducted using STATA analysis software (Stata Corp, 1993). Chi-square was used to test for a significant proportion of dieting days during the follicular phase, and the distribution of diet records among the menstrual cycle phases. Paired t-tests (one-sided) were used to compare energy and macronutrient intakes across the phases. The Wilcoxon signed-rank test was used to compare the intake differences between the phases for each of the three years.

For these analyses, a reclassification of the phases was necessary. The ability to perform within-girl comparisons of energy intake across the five phases was limited due to the following reasons: 1) Only four of the subjects had dietary intake data that represented all five phases. 2) The majority of the girls had two to three diet records that were designated the same phase. 3) Only 15/345 diet records were assigned to the ovulatory phase. In order to improve the statistical power within each phase, the five phases were consolidated. Menses and the follicular phase were combined to create a new phase called preovulation (Figure 7). It is generally not a standard procedure to combine these two phases; however, several researchers have shown no significant difference between the dietary intake during menses and the follicular phase (Fong and Kretsch, 1993, Gong et al.,1989, Lyons et al., 1989).

The postovulatory and premenses phases were similarly combined to produce a phase which will be designated "postovulation". The new phase is synonymous with the luteal phase which is commonly used in similar dietary assessment studies. The ovulatory phase was dropped from analysis because so few diet records occurred in this phase.

Conditional logistic regression was used to predict menstrual phases using kcal, grade, and postmenarcheal years as explanatory variables. Results were considered statistically significant if $P < .05$.

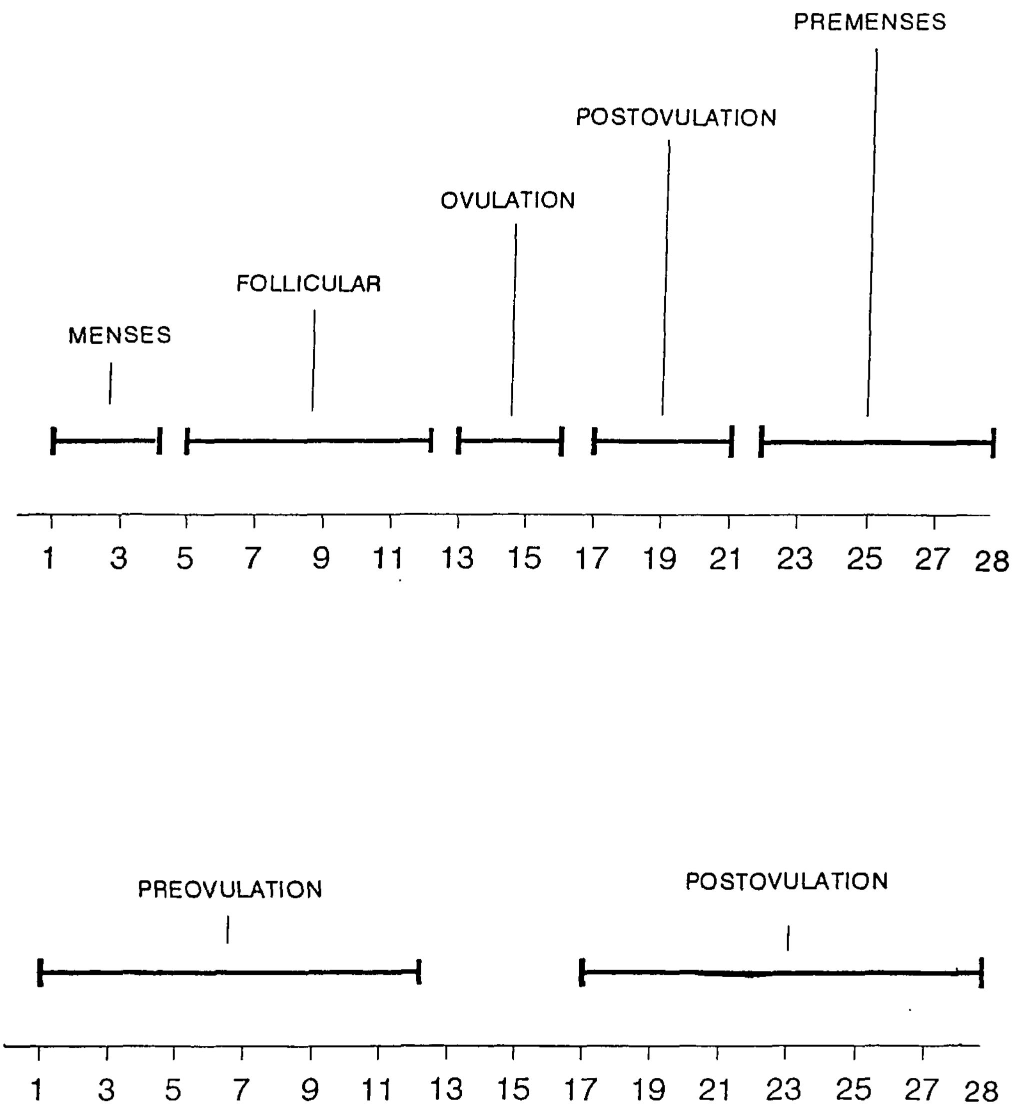

Figure 7. Reclassification of five menstrual cycle phases to two phases.

Chapter 3

RESULTS

Characteristics of Sample

The study sample included a total of 64 girls for analysis (one subject was dropped from the menarcheal analysis because her age at menarche was missing). Table 7 shows the means, standard deviations, and ranges of age, age at menarche and the postmenarcheal years (the number of years that have elapsed since menarche). The girls' mean age at menarche (12.6) was similar to the menarcheal ages of U.S. girls (12.8 and 12.9) reported by Zacharias (1976) and Frisch (1971). The range of menarcheal ages found in this present study appear to be within normal limits when compared to a standard range of 9-15 years of age for the onset of menarche in U.S. populations (Dooley and Brincat, 1994; Zacharias, 1976). Upon completion of the study, all 63 girls had reached menarche. The distribution of post-menarcheal years among the subjects is shown in Figure 8.

The mean and standard deviations of height, weight, and body mass index for each of the three years is listed in Table 8. A comparison of the third year body mass index with a standard population of similar age revealed that the study subjects were slightly heavier (22.7 $\pm$ 3.5 vs 21.8 $\pm$ 3.6), although the difference was not statistically significant ($P = 0.132$) (NHANES II, 1987). However, when

Table 7. Age, age at menarche, and postmenarcheal years at the end of year 03.

	n	$\bar{x} \pm SD$	Range
Age (y)	64	16.7 ± .65	(15.7 - 18.2)
Age at Menarche (y)	63	12.6 ± .86	(10.8 - 14.9)
Postmenarcheal Years	63	4.2 ± 1.1	(1.48 - 5.87)

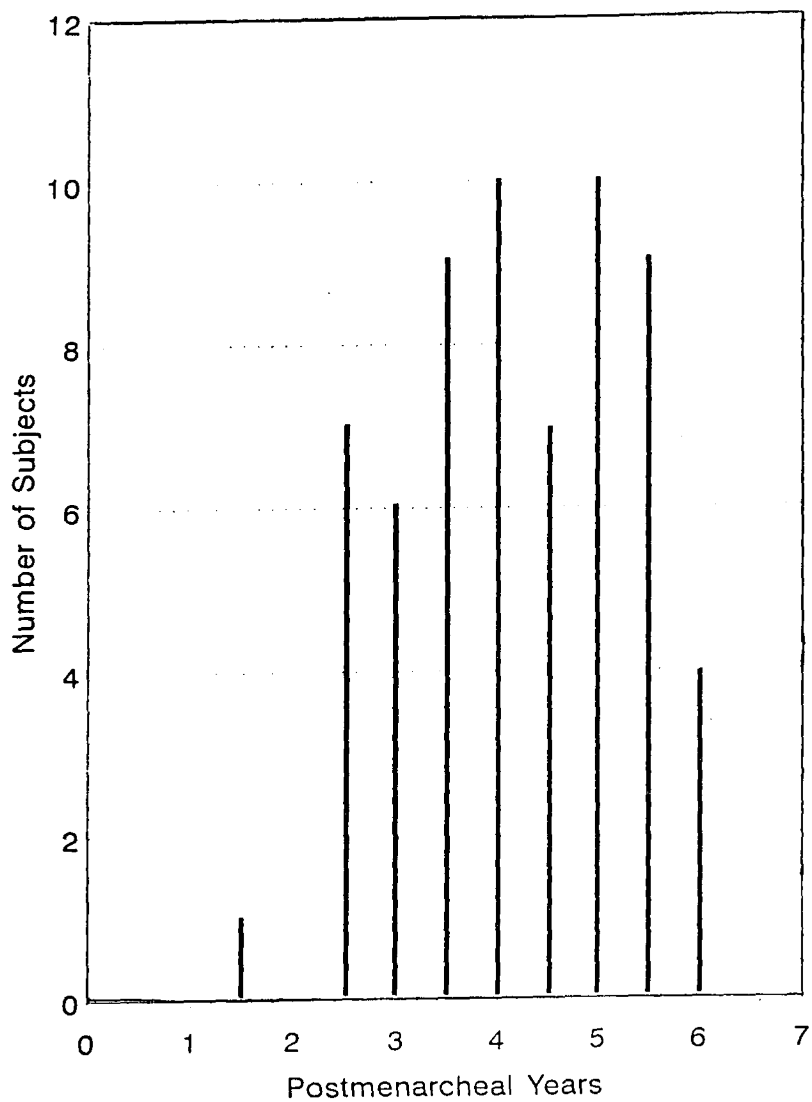

Figure 7. Distribution of postmenarcheal years (based on reference date: June 1, 1992).

Table 8. Reported height, weight, and body mass index of subjects for three years (x ± SD).

Year	n	Height (m)	Weight (kg)	BMI
1990	51	1.62 ± .07	58.2 ± 9.7	22.1 ± 3.4
1991	39	1.64 ± .06	60.8 ± 9.7	22.3 ± 3.2
1992	45	1.66 ± .06	62.8 ± 11.2	22.7 ± 3.5

the body mass indices were compared between the dieting population of this study to the non-dieters in the Teen Lifestyle Project (n=193), it was discovered that the dieters were significantly heavier for each of the three years (Table 9).

Dieting Habits

As mentioned in the methodology section, this study sample consists of subjects who responded "yes" to a question in their food journal, "Were you trying to lose weight today?". Out of 257 girls, 64 (25%) had at least one dieting day. Figure 9 shows the distribution of dieting days among the subjects. The prevalence of dieting in the Teen Lifestyle Project population (25%)was considerably less when compared to the percentages of girls dieting in other studies (44-60%) or to a U.S. national survey (61%) (Serdula et al., 1993, Wardle and Marsland, 1990; Desmond, 1986; American School Health Association, 1988).

Menstrual Phases

Menstrual cycle phases were assigned to food journals using the procedures described in the methodology section. Of the 680 food journals completed by the subjects, 345 (51%) were assigned to one of the five phases. Figure 10 compares the distribution of diet records among the girls with the distribution of diet records with phase

Table 9. Comparison of body mass index in dieting and non-dieting subjects for three years (x ± SD).

Year	n	Dieters	n	Non-dieters	T*	P‡
1990	51	22.1 ± 3.4	192	20.4 ± 3.6	3.13	.002
1991	39	22.3 ± 3.2	172	20.7 ± 2.7	2.90	.006
1992	45	22.7 ± 3.5	139	21.2 ± 3.9	2.43	.017

* T value from t-test.
‡ P value from t-test.

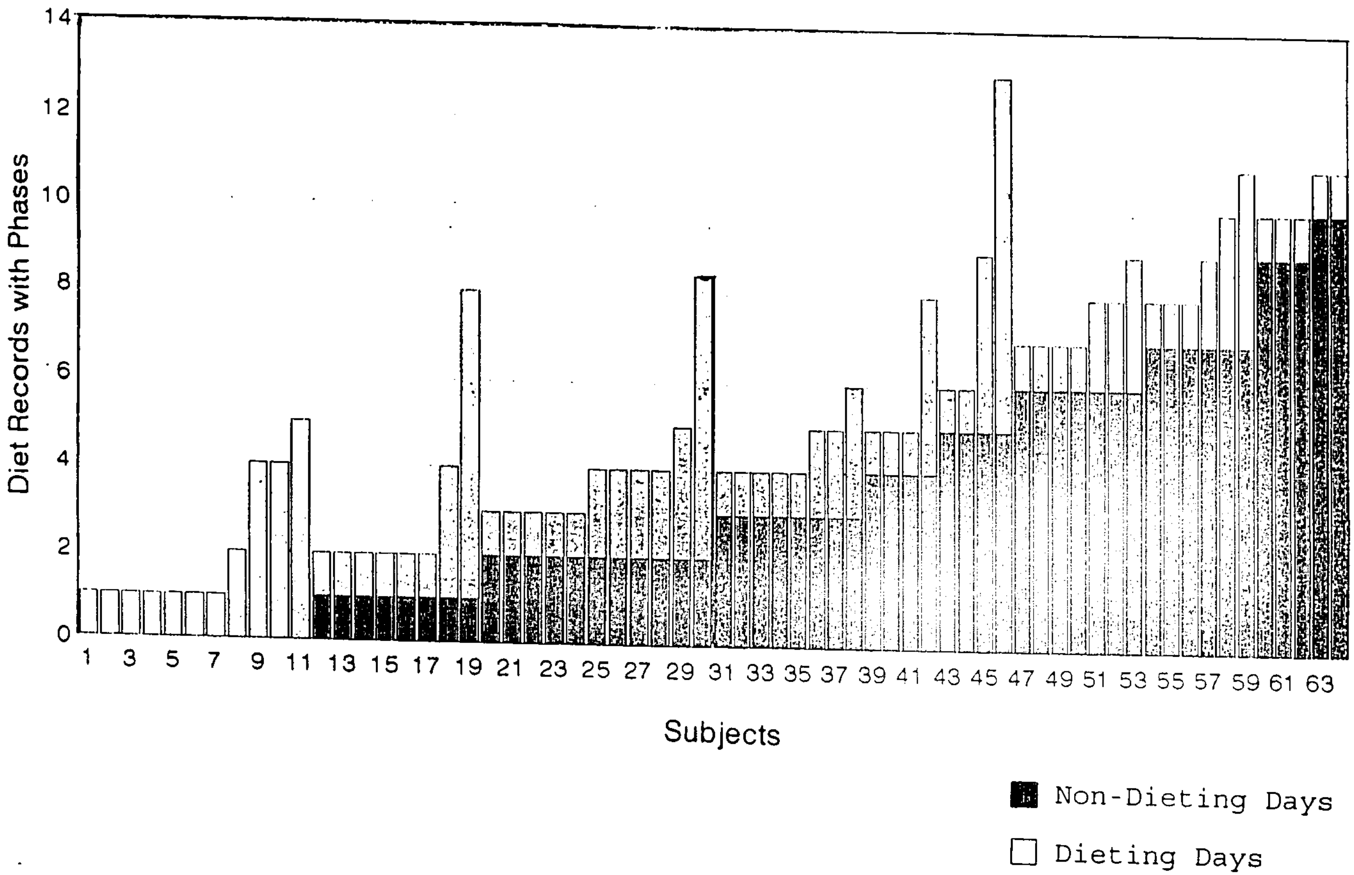

Figure 9. Distribution of dieting days among diet records with phase assignments for all subjects (n=64).

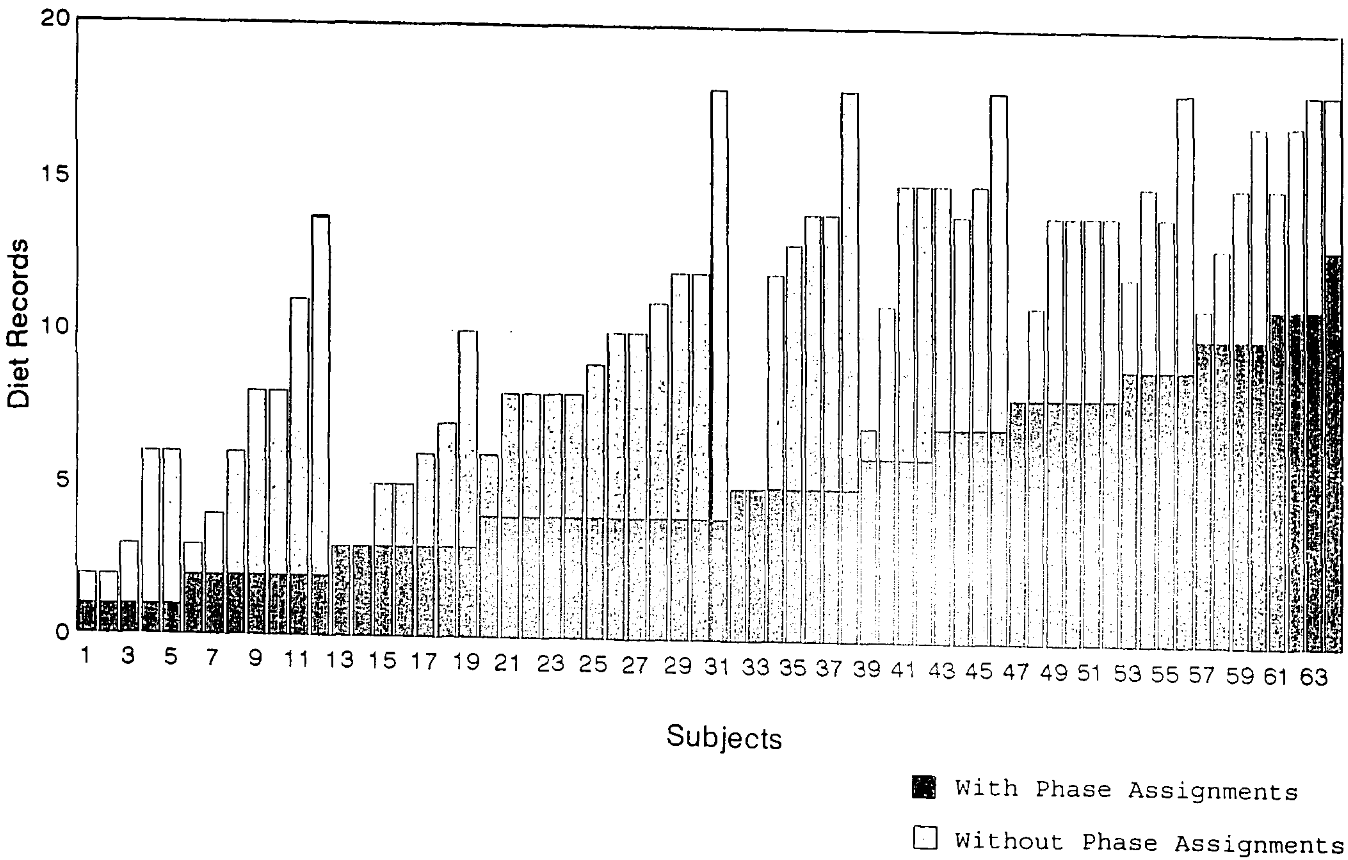

Figure 10. Distribution of diet records with and without phase assignments for all subjects (n=64).

assignments. The distribution of the journals among the five phases were as follows: 27% of the food journals were assigned to the menses phase, 35% to the follicular phase, 4% to ovulation, 14% to postovulation, and 20% to premenses.

Using chi-square, independence between diet record and menstrual phase was tested. The results revealed a significant difference ($p < .001$) between the expected and observed distributions (Figure 11). To determine if missing diet records contributed to the difference observed, the distribution of subjects with all 18 diet records (n=6) was compared with the remaining subjects. Figure 12 demonstrates that there was no significant difference between the two samples ($p = .997$).

One explanation for the non-random distribution of diet record by phase is found in the procedure for determining the phases in which a food journal occurs. The follicular phase has twice the opportunity of being assigned to a diet record because it can be reached by counting forward nine days from menses or backwards 20 days from menses. The other four phases can only be determined by counting in just one direction. Figure 13 shows the high proportion of diet records assigned to menses and the follicular phase.

The non-random distribution of diet records is not adequately explained by the coding procedure, however. Figure 14 demonstrates that a greater number of diet records

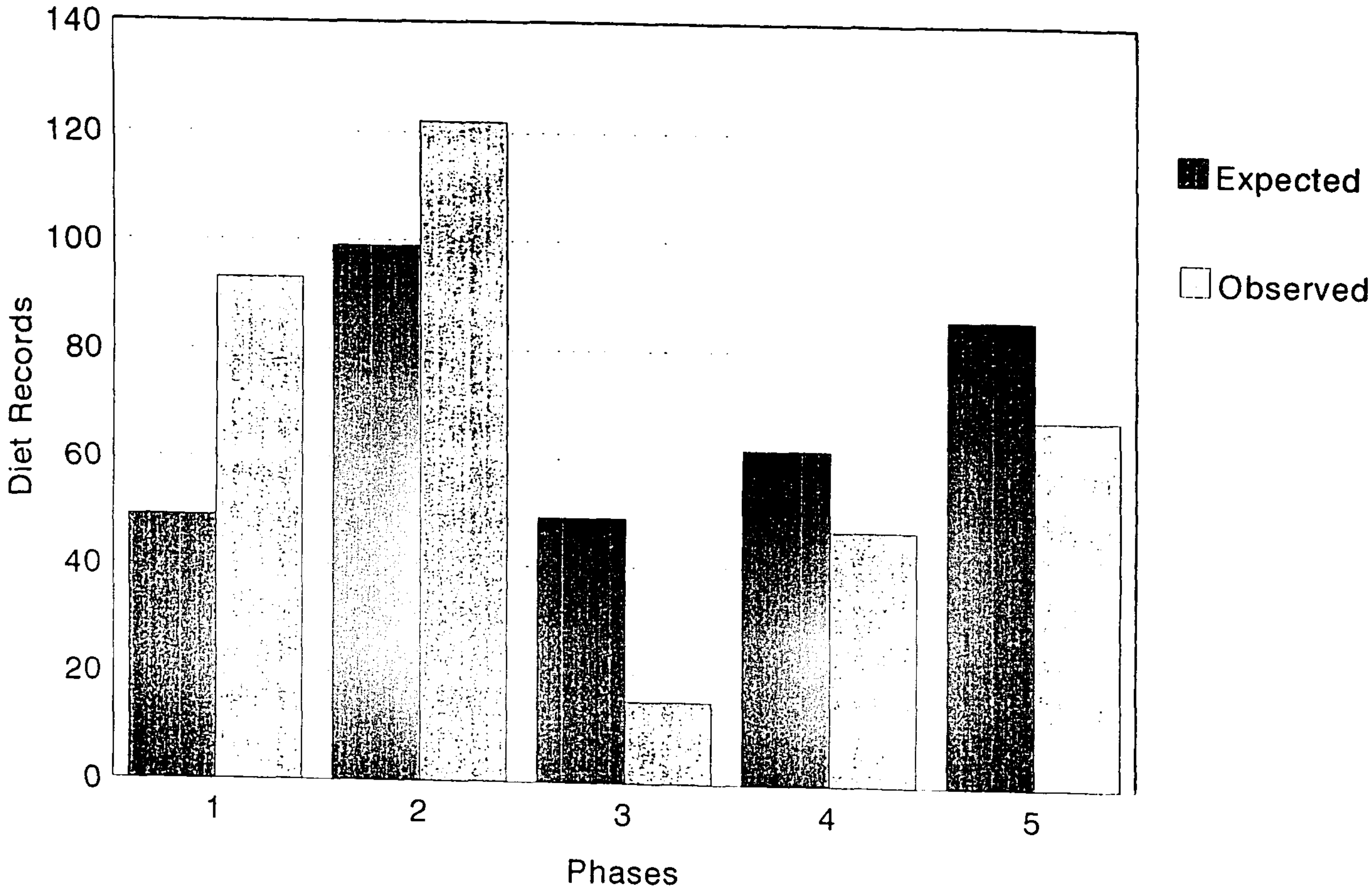

Figure 11. Distribution of expected versus observed number of diet records across the five phases (chi-square, P < .001).

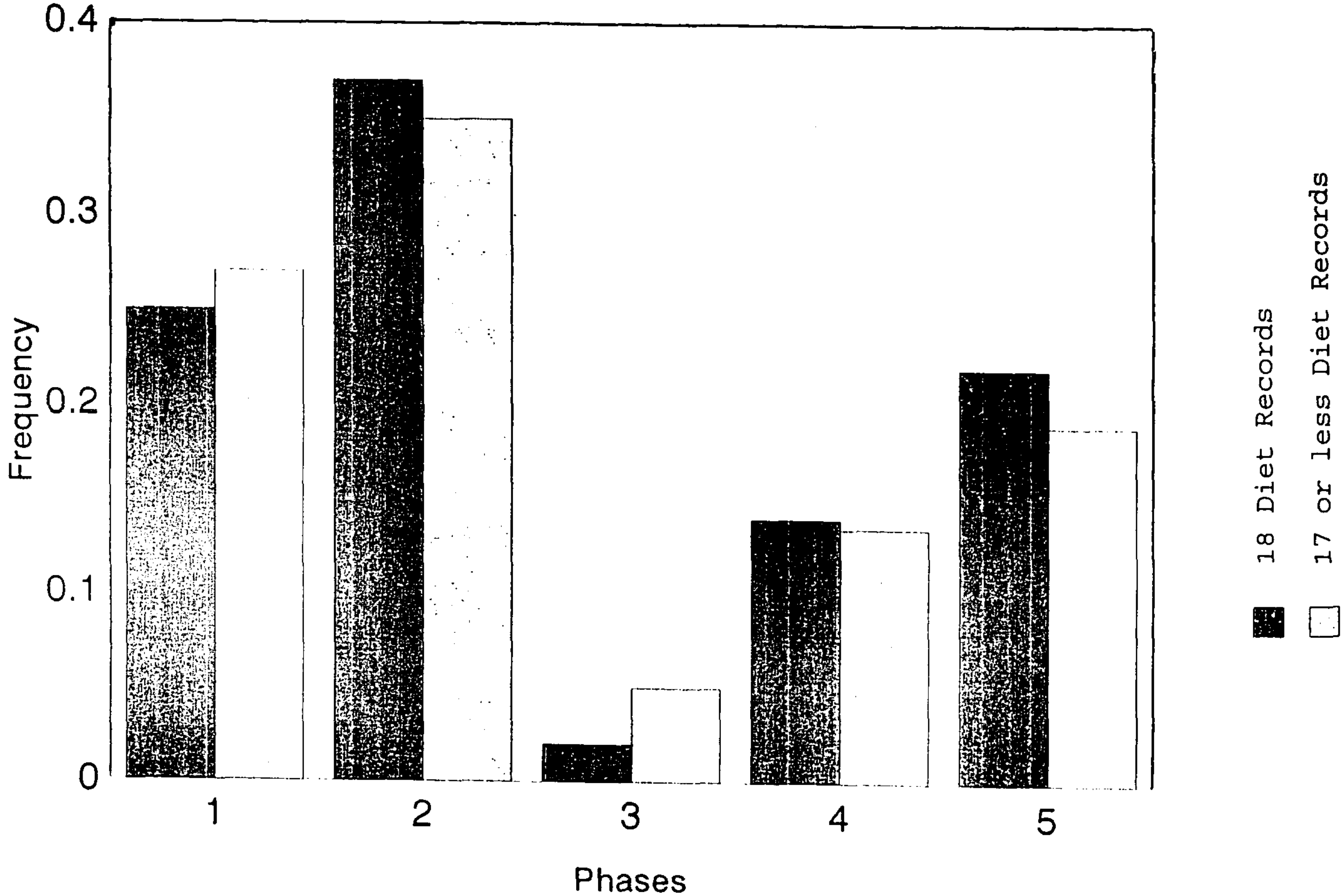

Figure 12. Distribution of six subjects with 18 diet records versus subjects with less than 18 diet records

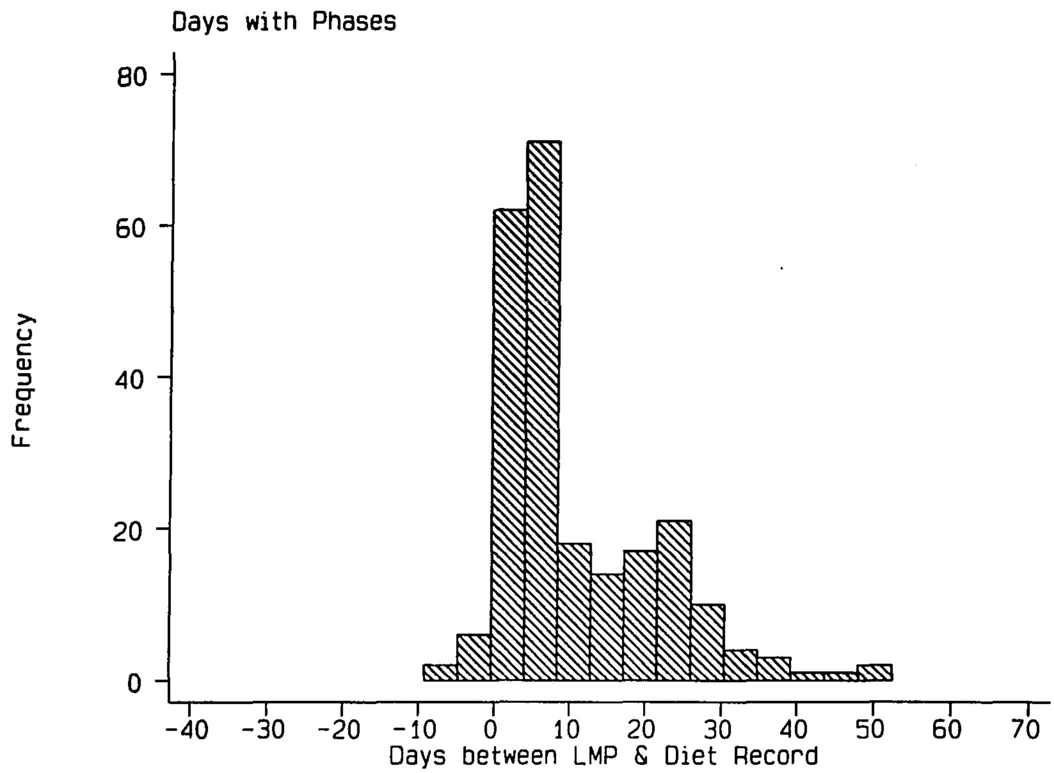

Figure 13. Frequency distribution of days (with phase assignments) between the last menstrual period (LMP) and the diet record for years 01 and 03 (raw data).

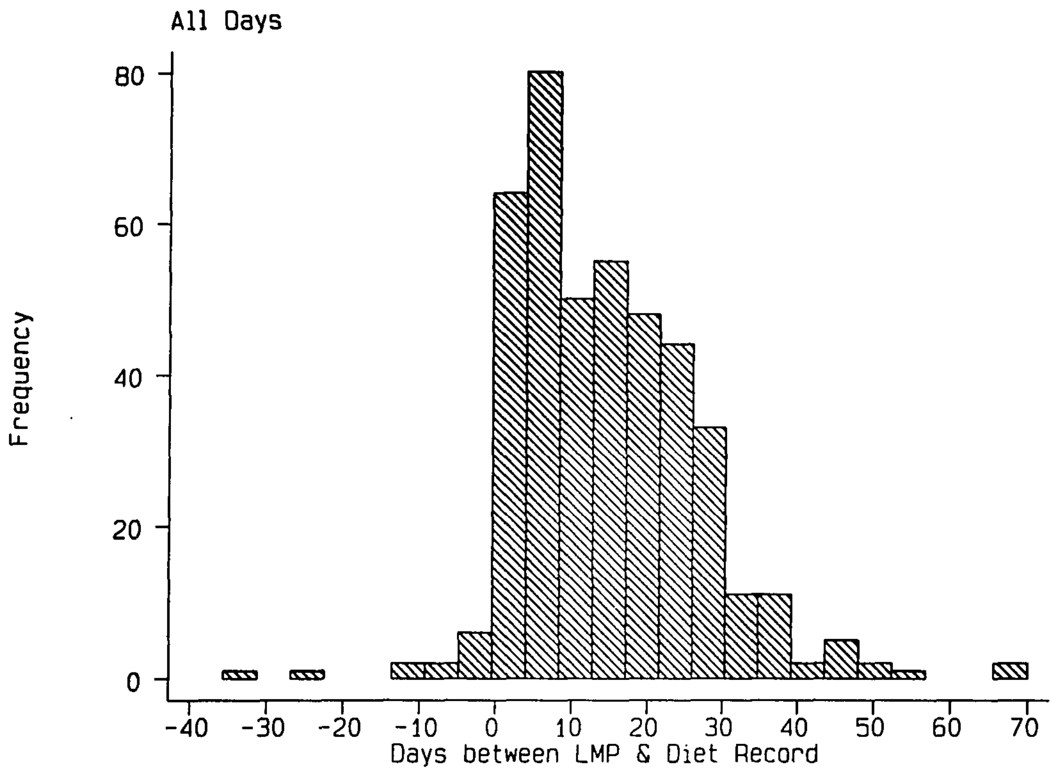

Figure 14. Frequency distribution of all days between the last menstrual period (LMP) and the diet record for years 01 and 03 (raw data).

occurred in the menses and follicular phase before the coding rules were applied. This indicates that the coding rules were not causing the non-random distribution. Why this phenomenon occurred could not be determined.

To establish if a greater proportion of dieting days occurred during the follicular phase, the distribution of dieting days among the five phases was analyzed using chi-square (Figure 15). The results revealed that dieting was evenly distributed across the menstrual cycle phases with no significant proportion ($P = .763$) of dieting days occurring in any phase.

Food Intake Patterns

Table 10 presents the means and standard deviations of energy and macronutrient intakes (includes intake data from diet records without phase assignments) for each of the three years. When the mean energy intakes of this sample were compared to the non-dieting subjects from the Teen Lifestyle Project, it was found that non-dieters were eating between 77-213 kcal more than the dieters (Table 11). This suggests that the dieters report more restrictive food consumption than the non-dieters.

To determine if adolescent girls increase food intake during the luteal or postovulatory phase, energy and macronutrient intakes were compared between pre- and postovulatory phases for each of the three years

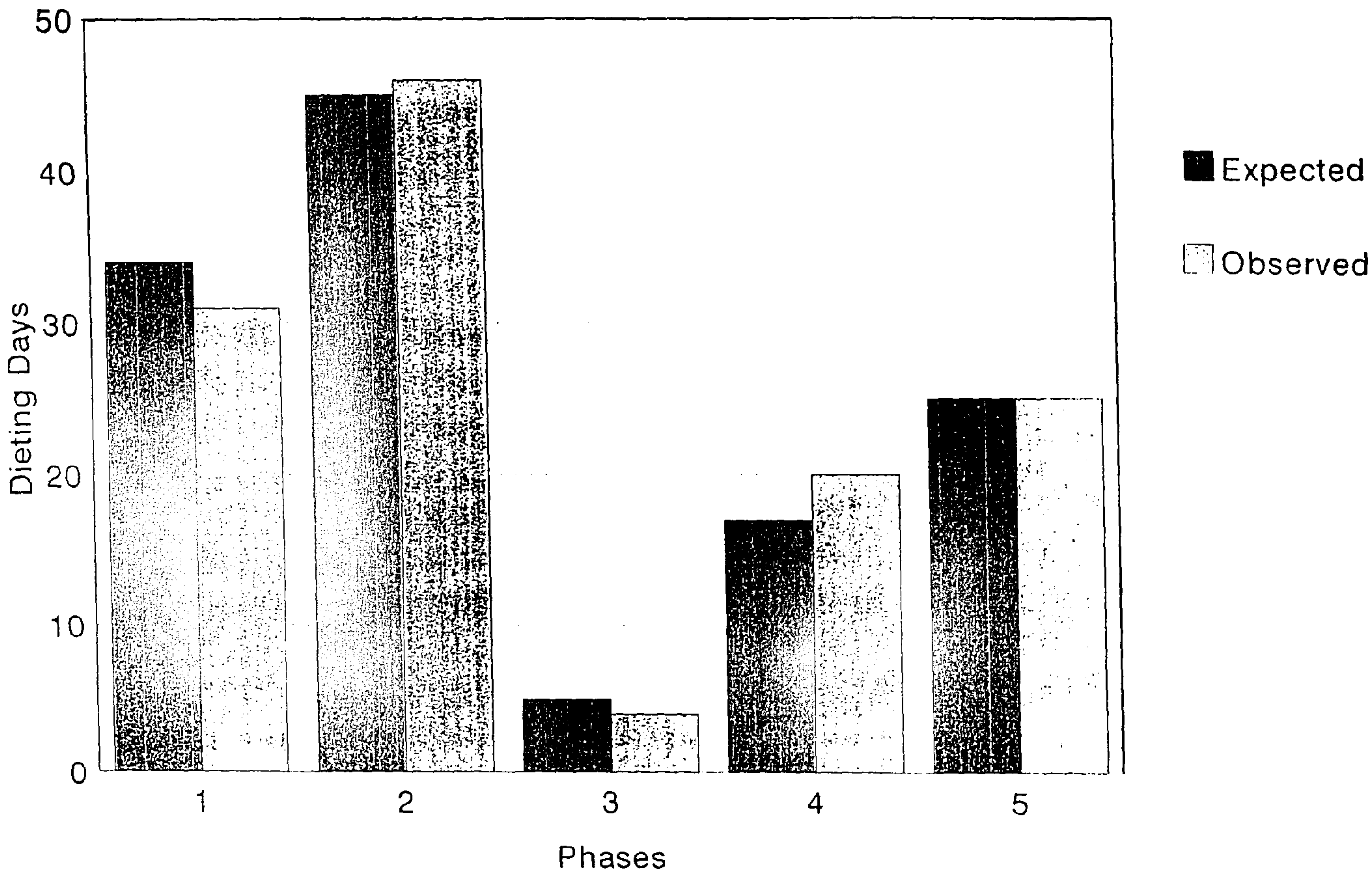

Figure 15. Distribution of expected versus observed number of dieting days across the five phases (chi square, P = .763).

Table 10. Average energy and macronutrient intake* over three years (x ± SD).

	1990 (n=57)	1991 (n=55)	1992 (n=52)
Energy			
kcal/d	1674 ± 533	1757 ± 653	1768 ± 655
Carbohydrate			
g/d	221 ± 68	233 ± 92	227 ± 84
% of kcal	51%	52%	50%
Protein			
g/d	57 ± 20	61 ± 25	61 ± 24
% of kcal	14%	14%	14%
Fat			
g/d	65 ± 25	67 ± 28	71 ± 30
% of kcal	35%	34%	36%

* Includes all intake data with and without phase assignments.

Table 11. Comparison of energy intake between dieting and non-dieting subjects* (x ± SD).

Year	n	Dieters	n	Non-dieters	T†	P‡
1990	57	1674 ± 533	200	1887 ± 524	-2.70	.007
1992	55	1757 ± 653	170	1815 ± 553	-0.65	.518
1993	52	1768 ± 655	188	1845 ± 584	-0.82	.413

* Non-dieting subjects are the remaining girls in the Teen Lifestyle Project who had not indicated dieting during the study.
† T value from t-test.
‡ P value from t-test.

(Table 12). No significant difference was detected in energy or macronutrient intakes in any year (Table 13).

To examine the possibility that the mean intakes for the two phases during the third year may resemble the patterns observed in adult women to a greater degree than shown in the first year, the differences between the phases for each year were compared across all three years. The Wilcoxon signed-rank test revealed no significant difference between year 01 and 02 (P = .241), year 02 and 03 (P = .650), and year 01 and 03 (P = .441).

Though not statistically significant, during the third year there was a 90 kcal increase in energy consumption during postovulation as compared to preovulation. A trend of increased energy intake during postovulation is in agreement with the studies that have demonstrated increased energy intake during the luteal (or postovulatory) phase in women (refer to Table 2).

Using conditional logistic regression, kcal, grade, and postmenarcheal years were found to have no significant contribution of predicting pre- and postovulation over time (Table 14).

Table 12. Comparison of average energy and macronutrient intake during pre- and postovulation over three years ($\bar{x} \pm$ SD).

	1990 (n=33)	1991 (n=25)	1992 (n=21)
Energy (kcal/d)			
Pre	1661 ± 729	1763 ± 729	1463 ± 624
Post	1701 ± 587	1670 ± 712	1553 ± 572
Carbohydrate (g/d)			
Pre	217 ± 80	236 ± 89	179 ± 82
Post	230 ± 93	212 ± 88	197 ± 61
Protein (g/d)			
Pre	59 ± 33	59 ± 25	51 ± 22
Post	58 ± 21	58 ± 31	54 ± 25
Fat (g/d)			
Pre	65 ± 37	67 ± 37	62 ± 30
Post	64 ± 26	68 ± 32	63 ± 35

* No pre- or postovulation comparisons were significant

Table 13. Mean energy and macronutrient differences between pre- and postovulation over three years.

	n	Difference*	T†	P‡
Energy (kcal/d)				
1990	33	40 ± 613	0.38	.708
1991	25	-93 ± 759	-0.61	.546
1992	21	90 ± 712	0.58	.566
Carbohydrate (g/d)				
1990	33	12 ± 87	0.85	.404
1991	25	-24 ± 107	-1.14	.267
1992	21	18 ± 90	0.92	.366
Protein (g/d)				
1990	33	-0.84 ± 34	-0.14	.887
1991	25	-6.00 ± 21	-1.38	.180
1992	21	3.26 ± 27	0.55	.586
Fat (g/d)				
1990	33	-1.01 ± 33	-0.17	.863
1991	25	1.45 ± 35	0.21	.837
1992	21	0.33 ± 33	0.05	.964

* x ± SD. Difference=postovulatory mean intake-preovulatory mean intake.
† T value from paired t-test.
‡ P value from paired t-test.

Table 14. Prediction of pre- and postovulation by energy intake, grade, and postmenarcheal years.*

Explanatory Variables	Odds Ratio	β	SE	P
Kcal	1.00	.0007	.0004	.872
Grade	1.12	.1125	.5817	.829
Postmenarcheal Years	1.00	-.0001	.0001	.783

* Conditional logistic regression; data stratified by subjects.

Chapter 4

DISCUSSION

The purpose of this study was to assess whether a relationship existed between the menstrual cycle and the dieting habits and food consumption of adolescent girls. A discussion of the findings of study by examining each of the two hypotheses will follow.

Dieting Habits

Hypothesis 1: Adolescent girls will choose to diet more frequently after menses, specifically, during the follicular phase.

Results showed that the proportion of dieting days among the five phases was roughly the same. The girls did not exhibit an increased number of dieting days during any one phase. This finding is not consistent with my observation in a weight management clinic that some of the women wished to reduce their caloric intake after menses because they had consumed foods that were not on their prescribed diet. Given that weekly weight losses were fairly constant on the liquid diets (patients generally lost 3-5 pounds each week), it is possible that the women felt under pressure to maintain their rate of weight loss in order to reach their "goal". Therefore, their desire to diet after menses may have just reflected the need to "stay on track".

The results of this study also conflicts with the findings of others (Carr-Nangle, 1994; Altabe and Thompson, 1990) who demonstrated that women are dissatisfied with their body size and appearance around menses. Water retention appears to be an important factor contributing to those feelings, as the side-effect elicits from women a perception that their body has become larger. Dieting after menses may be one strategy for coping with the anxiety of a shift to a larger body size

During the first year of the study, 58 of the girls were experiencing menstrual cycles that were between 50-80% anovulatory. By the second year, the percentage of anovulatory cycles was 50% or less. The high rate of anovulatory cycles indicates that the reproductive processes of this study population were still maturing. Girls who had predominantly anovulatory cycles would have had lower estrogen levels. The significance of this relates to water retention and negative body images. Estrogen is responsible for the edema that women experience when they are premenstrual. If estrogen levels are lower in adolescents due to developing reproductive cycles, then water retention may be minimal or only a sporadic occurrence during the early postmenarcheal years. Consequently, the negative perceptions of appearance and body size that surround bloating might be reduced. The girls in this study did not exhibit an increase in the frequency of dieting after menses suggesting that factors other than the menstrual cycle may play a more predominant role in influencing their dieting behaviors.

Adolescent girls are very preoccupied with their appearance and body shape and often report the reason for dieting was to reduce their stomach, hips, thighs, or bottom or to "look better" (Moore 1993; Casper and Offer, 1990; Wardle and Marsland, 1990). Although both women and girls are experiencing similar feelings about body image, the cause of these concerns may be different due to their ages. Teenage girls appear to be particularly susceptible to the influence of peers, family members, and mass media (Nichter and Vuckovic, 1994; Moore, 1993; Farthing, 1991). Several researchers have also found that as girls progress through puberty their dissatisfaction with their body increases (Killen et al., 1992; Gross and Duke, 1980). During sexual maturation, girls will increase their body fat from 15% to approximately 22%. The development of fuller hips and thighs during puberty probably contradicts the slim image most girls consider "ideal". An increase in adiposity coupled with the societal pressures to be thin may have a greater impact on the dieting habits of adolescents rather than the hormonal stimuli received during the luteal and menses phase of the menstrual cycle.

Food Intake Patterns

Hypothesis 2: Adolescent food consumption will be increased during the luteal (or the postovulatory) phase and decreased during ovulation.

Contrary to adult studies demonstrating fluctuations in food consumption, this study found no significant differences in energy intake when the pre- and postovulatory phases were compared. However, during the third year, the postovulatory mean intake was 90 kcal greater than the mean intake during the preovulatory phase. The change in energy intake between the pre- and postovulatory phases was within the range of 87-504 kcal found significant in adult studies (Dalvit, 1981; Lissner et al., 1988).

One explanation for the nonsignificant results in the third year pertains to the sample size. Although the study population contained 64 girls, the samples sizes for analysis were often reduced to 20-30 subjects. To compare intakes between the pre- and postovulatory phases, a girl had to have dietary intake data for either menses or the follicular phase <u>and</u> dietary data for the preovulatory or premenses phase. Many of the girls had dietary intake data for either preovulation or postovulation but not both. One significant limitation of this retrospective study is that food intake was not measured across all five phases.

Reproductive immaturity may also explain why the mean difference in energy intake during the first and second year were either minimal or contrary to adult food patterns. By the second year, most of the girls were only experiencing ovulatory cycles 50% of the time. The rise in progesterone during the luteal or post- ovulatory phase is caused by the secretion of the hormone from the corpus luteum. The corpus luteum exists only after ovulation has occurred. Consequently, ovulation must take place in order for variations of food intake to be observed. Since the girls may have had 50-80% anovulatory cycles the first year and 50% anovulatory cycles the second year, the effect of the menstrual cycle on food consumption was apparently minor. These findings are consistent with the results reported by Barr (1995) in which food intake did not change significantly in women with anovulatory cycles.

By the third year however, over 50% of the girls were 4-6 years beyond menarche. The frequency of ovulatory cycles occurring 4-5 years after menarche is about 60%, whereas, the percentage of ovulatory cycles 5-6 years after menarche ranges from 70-90% (Metcalf et al, 1983; Apter, 1980). The 90 kcal increase observed in the postovulatory phase during the third year may indicate that food patterns were developing as the girls reach the end-stage of puberty. Dalvit (1983) measured pre- and postovulatory intakes of college students (18-22 years of age) across two cycles and found postovulatory intakes to be consistently higher. The mean difference for the first cycle was 504 kcal and the difference for the second cycle was 496 kcal. The significance of Dalvit's study is that the college students were only 2-6 years older than the girls in this study. Although cycles can still be variable during the early twenties, this study indicates that food intake patterns develop soon after sexual maturity.

Summary and Conclusions

The effect of the menstrual cycle on energy intake and the dieting habits of adolescents was examined retrospectively for three years in 64 girls. All of the subjects in the study population had indicated dieting at least once throughout the three years.

When the distribution of dieting days across the menses, follicular, ovulatory, postovulatory, and premenses phases was examined, no relationship was demonstrated. Girls were not dieting with any greater frequency during the follicular phase when compared with the other four phases. The results of the study revealed no significant differences in energy intake between the pre- and postovulatory phases for each of the three years or overall. However, a 90 kcal increase in energy intake during the postovulatory phase in the third year is consistent with an increase in food consumption observed in adult women.

Although there is a high prevalence of dieting and anxiety pertaining to weight control in adolescents, the findings of this study suggest that the menstrual cycle is not contributing to those behaviors. Dieting practices among teenage girls appears to be driven in part by a desire to be thin in order to be popular among the boys and accepted into social groups. Media, in the form of television and magazines, also contributes to the motivation to diet by portraying a thin woman as a successful member of society or that a particular body shape is necessary to be attractive to males. Lastly, the development of adiposity which increases from 15% to 22% as girls progress through puberty probably influences dieting behaviors even further as their body shapes become fuller.

These issues appear to be much more central to the dieting practices of adolescents as opposed to water retention and altered eating habits that have been suggested to influence adult dieting behaviors. Although water retention and changes in food intake may become more evident as a girl matures, her concern as how she fits into her social environment may override any effect of the ovarian hormones on body size and appearance.

A consistent increase in energy intake during the luteal phase may not have been observed due to the immaturity of the girls' reproductive cycles. In order for the anorectic effects of estrogen to be inhibited by progesterone, an ovulatory cycle must occur. Given that the majority of the subjects were experiencing roughly 50-80% anovulatory cycles until the third year of the study, the influence of estrogen and progesterone on the regulation of food intake would have been theoretically altered or minimized due to low concentrations of each hormone.

A plausible explanation for the observed 90 kcal increase in the postovulatory phase during the third year is that the girls were experiencing a greater number of ovulatory cycles. Sufficient estrogen levels will trigger ovulation which produces a corpus luteum that can secrete progesterone. Progesterone can then inhibit effects of estrogen and promote increased food intake.

In conclusion, the purpose of the study was to examine whether the effects of the menstrual cycle on food consumption and dieting habits exhibited in women could be extended to adolescents. Prior to this study, the role of ovarian hormones on food intake and dieting had not been examined in this population.

An effect of the menstrual cycle on dieting in adolescent girls was not established in this study. Dieting behaviors among teenage girls appears to be greatly influenced by size comparisons of other females, dieting attitudes of their mothers, a desire to be attractive to boys, and pressures exerted by media. Any influence ovarian hormones might have on dieting choices appears to be masked by societal and cultural ideals. It is possible that when a girl matures into an adult and her social environment changes to due to work and/or a family, the various pressures to diet also shift in emphasis. The menstrual cycle phases may have more of an impact on dieting behaviors in an emotionally and reproductively mature woman.

The findings of this study suggest that ovarian hormones may exert a greater influence on energy intake when a girl is at least six years beyond menarche. That would place a girl at the end-stage of puberty when her reproductive cycle would be more fully developed and the corresponding hormone levels would resemble levels exhibited in adults. At this stage of development, the frequency of ovulatory cycles would be roughly 80-90%.

Future research examining the effects of changing ovarian hormone levels on food intake in adolescents should document ovulation using basal body temperature or salivary progesterone measurements. In order to determine any effect the menstrual phases may have on food consumption, an ovulatory cycle needs to be established, particularly when girls are less than six years from menarche.

APPENDIX A

TEEN LIFESTYLE PROJECT FOOD JOURNAL

ID# ________

TEEN
LIFESTYLE
PROJECT

FALL 1991
FOOD
JOURNAL

Dear ____________________

Please complete this journal on

____

If you have any questions or problems, please call us at
626-2886.

Lydia Melinda Melissa Tracy

EXAMPLE OF A RECORD

TIME	LOCATION	AMOUNT	FOOD/DRINK/GUM/CIG	DESCRIPTION/ PREPARATION
11:15a	class	1 pc	gum	extra
12:30p	cafeteria	1	pretzel	w/cheese
		1 can	diet coke	
		1	apple	red
2:20p	Hall	3 sips	water	
3:30p	Burger king	1 lg	frenchfries	
		2 pkg	ketchup	
		1 sm	ice tea w/sugar	
4:00	Bus stop	1	cigarette	Marlboro 100
4:30p	Home	1 glass	milk	2%
		3	oreos	

Use measurements like ounces, grams, etc. that you find on wrappers and packages, or compare the size to something like a tennis ball or a quarter.

Please be as specific as possible and include EVERYTHING you eat, drink and smoke. (Even sips of water, vitamins, medications, and diet aids like Slimfast.)

MORNING

(ANYTHING CONSUMED BEFORE 11:00 A.M.)

TIME	LOCATION	AMOUNT	FOOD/DRINK/GUM/CIG	DESCRIPTION/ PREPARATION

AFTERNOON

(ANYTHING CONSUMED BETWEEN 11:OO A.M. AND 5:OO P.M.)

TIME	LOCATION	AMOUNT	FOOD/DRINK/GUM/CIG	DESCRIPTION/ PREPARATION

FAMILY = **FAM**
FRIENDS = **FRS**
ALONE = **ALO**

EVENING

(ANYTHING CONSUMED AFTER 5:00 P.M.)

TIME	LOCATION & WHO WITH	AMOUNT	FOOD/DRINK/GUM/CIG	DESCRIPTION/ PREPARATION

PLEASE TAKE A FEW MINUTES TO ANSWER THE FOLLOWING QUESTIONS.

(CIRCLE ONE)

1. Did you take any vitamin/mineral pills today? YES NO
   if YES, please list the brand name(s) and amount(s) here:

2. Did you eat/drink any diet aids today? (diet pills, drinks like Slimfast, or foods like Nutrisystems.) YES NO
   if YES, please list the brand name(s) and amount(s) here:

3. Were you trying to lose weight today? YES NO

4. Please circle the FIRST day of your last period on the calendar below.

1991

JANUARY

S	M	T	W	T	F	S
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

FEBRUARY

S	M	T	W	T	F	S
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28		

MARCH

S	M	T	W	T	F	S
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24/31	25	26	27	28	29	30

APRIL

S	M	T	W	T	F	S
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30				

MAY

S	M	T	W	T	F	S
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30	31	

JUNE

S	M	T	W	T	F	S
						1
2	3	4	5	6	7	8
9	10	11	12	13	14	15
16	17	18	19	20	21	22
23/30	24	25	26	27	28	29

JULY

S	M	T	W	T	F	S
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

AUGUST

S	M	T	W	T	F	S
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

SEPTEMBER

S	M	T	W	T	F	S
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30					

OCTOBER

S	M	T	W	T	F	S
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

NOVEMBER

S	M	T	W	T	F	S
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

DECEMBER

S	M	T	W	T	F	S
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31				

5. Can you usually predict when your period will start? YES NO
   if YES, please tell us the DATE that you are expecting your next period to begin:____________

NOTES/COMMENTS:

WAS IT A GOOD DAY TODAY?

ANYTHING UNUSUAL?

THANKS AGAIN!!

TEEN
LIFESTYLE
PROJECT

800 E. University Blvd. • Suite 300 • Tucson, Arizona 85719 • (602) 626-2886

REFERENCES

Altabe M, Thompson JK. (1990). Menstrual cycle, body image, and eating disturbance. Int J Eat Dis 9:395-401.

American School Health Association, Association for the Advancement of Health Education, Society for Public Health Education. The National Adolescent Student Health Survey: A Report on the Health of America's Youth. Oakland, CA, Third Party Publishing Co, 1989.

Bancroft J, Backstrom T. (1985). Premenstrual syndrome. Clin Endocrinol 22:313-336.

Barr SI, Janelle C, Prior JC. (1995). Energy intakes higher during the luteal phase of ovulatory menstrual cycles. Am J Clin Nutr 61:39-43.

Bielert C, Busse C. (1983). Influences of ovarian hormones on the food intake and feeding of captive and wild female Chacma baboons. Physiol Behav 30:103-111.

Bonavera JJ, Dube MG, Kalra PS, Kalra SP. (1994). Anorectic effects of estrogen may be mediated by decreased neuropeptide Y release in the hypothalamic paraventricular nucleus. Endocrinology 134:2367-2370.

Borsos A, Lampe L, Balogh A, Csoknyay J, Ditroi F, Szekely P. (1988). Ovarian function after the menarche and hormonal contraception. Int J Gynecol Obstet 27:249-253.

Boyle CA, Berkowitz GS, Kelsey JL. (1987). Epidemiology of premenstrual symptoms. Am J Public Health 77:349-350.

Buckler J. Fat. A Longitudinal Study of Adolescent Growth, 70-75. Springer-Verlag, New York, 1990.

Butera PC, Beikirch, RJ. (1989). Central implants of diluted estradiol: independent effects on ingestive and reproductive behaviors of ovariectomized rats. Brain Res 491:266-273.

Butera PC, Willard DM, Raymond SA.(1992). Effects of PVN lesions on the responsiveness of female rats to estradiol. Brain Res 576:304-310.

Butera PC, Bradway DM, Cataldo NJ. (1993). Modulation of the satiety effect of cholecystokinin by estradiol. Physiol Behav 53:1235-1238.

Carr-Nangle RE, Johnson WG, Bergeron KC, Nangle D. (1994). Body image changes over the menstrual cycle in normal women. Int J Eat Dis 16:267-273.

Casper RC, Offer D. (1990). Weight and dieting concerns in adolescents, fashion or symptom? Pediatrics 86:384-390.

Czaja JA. (1978). Ovarian influence on primate food intake: assessment of progesterone actions. Physiol Behav 21:923-928.

Dalvit SP. (1981). The effect of the menstrual cycle on patterns of food intake. Am J Clin Nutr 34:1811-1815.

Dalvit-McPhillips SP. (1983). The effect of the human menstrual cycle on nutrient intake. Physiol Behav 31:209-212.

Deeley RG, Tam SP, Archer TK. (1985). The effects of estrogen on apolipoprotein synthesis. Can J Biochem Cell 63:882-889.

Desmond SM, Price JH, Gray N, O'Connell JK. (1986). The etiology of adolescents' perceptions of their weight. J Youth Adolesc 15:461-474.

Farthing, MC. (1991). Current eating patterns of adolescents in the United States. Nutr Today 35-39.

Ferin M, Jewelewicz R, Warren M. Hypothalamic-pituitary-ovarian-communication. The Menstrual Cycle, 42-61. Oxford University Press, New York, 1993.

Fong AKH, Kretsch MJ. (1993). Changes in dietary intake, urinary nitrogen, and urinary volume across the menstrual cycle. Am J Clin Nutr 57:43-46.

Fortenberry JD. (1992). Reliability of adolescents' report of height and weight. J Adolesc Health 1992:114-117.

Frisch RE, Revelle R. (1971). Height and weight at menarche and a hypothesis of menarche. Arch Dis Child 46:695-701.

Geary N, Trace D, McEwen B, Smith GP. (1994). Cyclic estradiol replacement increases the satiety effect of CCK-8 in ovariectomized rats. Physiol Behav 56:281-289.

Giudicelli Y, Dieudonne MN, Lacasa D, Pasquier YN, Pecquery R. (1993). Modulation of sex hormones of the membranes transducing system regulating fatty acid mobilization in adipose tissue. Prostaglandins Leukot Essent Fatty Acids 48:91-100.

Gong EJ, Garrel D, Calloway DH. (1989). Menstrual cycle and voluntary food intake. Am J Clin Nutr 49:252-258.

Greenspan FS, Baxter JD. Ovaries. Basic & Clinical Endocrinology, 419-470. Appleton & Lange, Norfolk, 1994.

Gross RT, Duke PM. (1980). The effect of early versus late physical maturation on adolescent behavior. Pediatr Clin North Am Adolesc Med 27:71-78.

Hannan WJ, Wrate RM, Cowan SJ, Freeman CP. (1995). Body mass index as an estimate of body fat. Int J Eat Dis 18:91-97.

Harlan WR, Harlan EA, Grillo GP. (1980). Secondary sex characteristics of girls 12 to 17 years of age: The U.S. Health Examination Survey. J Pediatr 96:1074-1078.

Huerta-Franco MR, Malacara JM. (1993). Association of physical and emotional symptoms with the menstrual cycle and life-style. J Reprod Med 38:448-454.

Jankowiak R, Stern JJ. (1974). Food intake and body weight modifications following medial hypothalamic hormone implants in female rats. Physiol Behav 12:875-879.

Johnson WG, Corrigan SA, Lemmon CR, Bergeron KB, Crusco AH. (1994). Energy regulation over the menstrual cycle. Physiol Behav 56:523-527.

Kemnitz JW, Gibber JR, Lindsay KA, Eisele SG. (1989). Effects of ovarian hormones on eating behaviors, body weight, and glucoregulation in Rhesus monkeys. Horm Behav 23:235-250.

Killen JD, Hayward C, Litt I, Hammer LD, Wilson DM, Miner B, Taylor CB, Varady A, Shisslak C. (1992). Is puberty a risk factor for eating disorders? AJDC 146:323-325.

Langhans W, Scharrer E. (1992). Metabolic control of eating. World Rev Nutr Diet 70:1-67.

Leibowitz SF, Stanley BG. Neurochemical controls of appetite. Feeding Behavior, 191-227. Academic Press, San Diego, 1986.

Lissner L, Stevens J, Levitsky DA, Rasmussen KM, Strupp BJ. (1988). Variation in energy intake during the menstrual cycle: implications for food-intake research. Am J Clin Nutr 48:956-962.

Logue CM, Moos RH. (1986). Perimenstrual symptoms: prevalence and risk factors. Psychosom Med 48:388-411.

Lyons PM, Truswell AS, Mira M, Vizzard J, Abraham SF. (1989). Reduction of food intake in the ovulatory phase of the menstrual cycle. Am J Clin Nutr 49:1164-1168.

Manocha S, Ghoudhuri G, Tandon BN. (1986). A study of dietary intake in pre-and post-menstrual period. Human Nutr Appl Nutr 40A:213-216.

Martini MC, Lampe JW, Slavin JL, Kurzer MS. (1994). Effect of the menstrual cycle on energy and nutrient intake. Am J Clin Nutr 60:895-899.

Mattes RD, Gabriel SJ. (1988). A comparison of results from two microcomputer nutrient analysis software packages and a mainframe system. J Nutr Educ 20:70-76.

Metcalf MG, Skidmore DS, Lowry GF, Mackenzie JA. (1983). Incidence of ovulation in the years after the menarche. J Endocrinol 97:213-219.

Moore DC. (1993). Body image and eating behavior in adolescents. J Am Col Nutr 12:505-510.

Morley JE. (1987). Neuropeptide regulation of appetite and weight. Endocrine Rev 8:256-287.

Nichter M, Vuckovic N. Fat Talk: Body image among adolescent girls, ed. Nicole Sault. Rutgers University Press, New Bruinswick, 1994.

Nichter M, Ritenbaugh C, Nichter M, Vuckovic N, Aickin M. (1995). Dieting and watching behaviors among adolescent females: report of a multimethod study. J Adolesc Health (in press).

Palmer K, Gray JM. (1986). Central vs. peripheral effects of estrogen on food intake and lipoprotein lipase activity in ovariectomized rats. Physiol Behav 37:187-189.

Roche AF, Siervogel RM, Chumlea WC, Webb P. (1981). Grading body fatness from limited anthropometric data. Am J Clin Nutr 34:2831-2838.

Rosenblatt H, Dyrenfurth I, Ferin M, Vandewiele RL. (1980). Food intake and the menstrual cycle in Rhesus monkeys. Physiol Behav 24:447-449.

Serdula MK, Collins ME, Williamson DF, Anda RF, Pamuk E, Byers TE. (1993). Weight control practices of U.S. adolescents and adults. Ann Intern Med 119:667-671.

Speroff L, Glass RH, Kase NG. Abnormal puberty and growth problems. Clinical Gynecologic Endocrinology and Infertility, 361-399. Williams and Wilkins, Baltimore, 1994.

Stanley BG, Anderson KC, Grayson MH, Leibowitz SF. (1989). Repeated hypothalamic stimulation with neuropeptide Y increases daily carbohydrate and fat intake and body weight gain in female rats. Physiol Behav 46:173-177.

Story M, Hauck FR, Broussard BA, White LL, Resnick MD, Blum RW. (1994). Weight perceptions and weight control practices in American Indian and Alaska Native adolescents. Arch Pediatr Adolesc Med 148:567-571.

Thorneycroft IH, Boyers SP. (1983). The human menstrual cycle: correlation of hormonal patterns and clinical signs and symptoms. Obstet Gynecol Ann 12:199-225.

Tienboon P, Wahlqvist ML, Rutishauser IHE. (1992). Self-reported weight and height in adolescents and their parents. J Adolesc Health 13:528-532.

Tarasuk V, Beaton GH. (1991). Menstrual-cycle patterns in energy and macronutrient intake. Am J Clin Nutr 53:442-447.

Treloar AE, Boynton RE, Behn BG, Brown BW. (1967). Variation of the human menstrual cycle through reproductive life. Int J Fertil 12:77-126.

Vuorento, T, Huhtaniemi I. (1992). Daily levels of salivary progesterone during menstrual cycle in adolescent girls. Fertil Steril 58:685-690.

Wade, GN. Hormones and body weight. Advances in the Study of Behavior, 201-279. Academic Press, New York, 1976.

Wade GN, Gray JM, Bartness TJ. (1985). Gonadal influences on adiposity. Int J Obesity 9:83-92.

Wade GN, Schneider JE.(1992). Metabolic fuels and reproduction in female mammals. Neurosci Biobehav Rev 16:235-272.

Wardle J, Marsland L. (1990). Adolescent concerns about weight and eating; a social-developmental perspective. J Psychosom Res 34:377-391.

White JD. (1993). Neuropeptide Y: a central regulator of energy homeostasis. Regul Pept 49:93-107.

Woods NF, Most A, Dery GK. (1982). Prevalence of perimenstrual symptoms. Am J Public Health 72:1257-1264.

Zacharias L, Rand WM, Wurtman RJ. (1976). A prospective study of sexual development and growth in American girls: the statistics of menarche. Obstet Gynecol Survey 31:326-337.