Nutritional characteristics, eating pathology, and hormonal status in young women

Cheryl L. Rock, Daniel W. Gorenflo, Adam Drewnowski, and Mark A. Demitrack

ABSTRACT Ovulatory dysfunction is common in patients with eating disorders. However, many women engage in pathologic eating behaviors without meeting the current diagnostic criteria for anorexia or bulimia nervosa. Clinical eating disorders are only the most extreme form of pathologic eating attitudes and behaviors that are present in many young women. Specific food choices and nutrient intakes may be associated with altered gonadal hormone status of these dieters. This cross-sectional study was conducted to describe the nutritional characteristics of college-aged women defined by their eating attitudes and behaviors with a previously described questionnaire. We evaluated dietary intake, body composition, and selected biochemical indicators in 76 undergraduate women. Serum concentrations of estradiol, progesterone, and carotenoids were measured on days 6, 21, and 28 of one menstrual cycle. Dietary assessment was based on food records at two 3-d intervals during the cycle. Ovulatory status was definitively determined on the basis of biochemical data for 46 of the women. Increased degree of pathologic dieting was associated with a significantly lower intake of dietary fat ($P < 0.02$), despite similar mean body mass index and body composition across the eating pathology groups. Serum concentration of $\alpha$-carotene was significantly greater ($P < 0.005$) in association with a greater degree of eating pathology. With ovulation as a between-group factor, serum lutein concentration and dietary intake of energy and fat differed significantly between groups ($P < 0.003$). Nutritional characteristics associated with pathologic eating behavior may also be associated with menstrual irregularities in young women. Am J Clin Nutr 1996;64:566–71.

KEY WORDS Eating disorders, women, carotenoids, diet, ovulatory dysfunction

INTRODUCTION

The prevalence of anorexia nervosa and bulimia nervosa in the female population has been estimated at <1% and <3%, respectively (1, 2). However, $\geq 30\%$ of women of reproductive age have been shown to be dieting, and $15\%$ are regularly engaging in bulimic behaviors, including both binge eating and purging (3, 4). In these women, altered eating patterns and food choices may result in alterations in nutrient intake, body composition, and serum concentrations of dietary constituents such as carotenoids.

The eating disorders anorexia and bulimia nervosa have long been associated with ovulatory dysfunction. Adequate nutritional status is a critical determinant of the onset and maintenance of normal reproductive function. Abundant evidence shows that menstrual function regresses to a prepubertal hormonal architecture in patients with anorexia nervosa, other extremely underweight women, and normal subjects consuming diets that promote weight loss (5–8). However, menstrual irregularities have been observed in the absence of weight loss, notably in normal-weight patients with bulimia nervosa (9).

Survey studies have also shown that a greater degree of eating pathology in nonclinical populations is associated with menstrual irregularities (4), possibly as a result of short-term physiologic adaptations to intermittent food restriction. In addition, dieting behavior may influence the consumption of fat, fiber, phytoestrogens, and carotenoids—specific dietary constituents that have been reported to influence gonadal hormone status in women or to affect fertility in animals (10–19).

The purpose of this cross-sectional study was to describe the nutritional characteristics of college-aged women across a range of eating behaviors, including dietary intake, body composition, and serum concentrations of carotenoids, cholesterol, and triacylglycerols. Relations between these nutritional characteristics and ovulatory status were also examined. We hypothesized that nutritional features characteristic of eating disorders would occur in association with increased degree of pathologic eating attitudes and behavior in a nonclinical population of young women.

SUBJECTS AND METHODS

Subjects

This investigation was part of a larger project examining the relation between eating behavior, neuroendocrine function, and fertility in women. Seventy-six subjects were recruited from among the female undergraduate students of a major midwestern university. Inclusion criteria were as follows: a history of regular menstrual cycles, normal weight-for-height [body mass index (BMI; in kg/m$^2$) between 18 and 25], and 18–20 y of age.

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To remove excessive exercise as a confounding variable, all subjects who reported exercising for > 60 min/d or > 7 h/wk were excluded. Other exclusion criteria were pregnancy, use of any oral medications (including oral contraceptives in the preceding 3 mo), and any evidence of renal, hematologic, cardiovascular, neurologic, or hepatic disease. Each subject underwent a complete medical history and physical examination, and routine screening clinical laboratory tests were conducted. Procedures for this study were approved by the Institutional Review Board of the University of Michigan School of Medicine and all subjects gave informed consent.

Procedures

Subjects were admitted to the Clinical Research Center (CRC) for 24-h periods at three intervals during one menstrual cycle (approximately on days 6, 21, and 28). While in the CRC, pulsatile secretory characteristics of hypothalamic-pituitary-gonadal axis function were measured. Results of the pulsatility studies will be presented elsewhere. A 12-h fasting blood sample was obtained at 0800 during each visit for measurement of serum cholesterol, triacylglycerols, and carotenoids. Body composition was estimated by using caliper measurements (Lange, Cambridge, MD) of skinfold thickness, and percentage body fat was calculated by the method of Durnin and Womersley (20). Height and weight were obtained by standard procedures (21), and BMI was calculated. Subjects were instructed to maintain their usual dietary patterns throughout the study. General historical data, such as usual menstrual cycle length, were collected.

Subjects completed a self-administered questionnaire that addresses weight history, binge eating, purging, and a range of diet-related attitudes and behaviors. Mutually exclusive eating pathology categories were defined and subjects were grouped before nutritional assessment and data analysis. Development and use of this instrument was described in previous reports (22, 23). Briefly, the questionnaire included items that approximated the Diagnostic and Statistical Manual of Mental Disorders, third edition, revised (DSM-III-R) diagnostic criteria for bulimia nervosa (24). Questions addressed the frequency of dieting (described in general terms), binge eating, and use of fasting, laxatives, diuretics, diet pills, and self-induced vomiting for weight control. Further questions probed current and desired body weight, as well as self-rated depression and stress. Categorization into an eating pathology or dieting group was based on three domains: binge-eating behavior, purging and excessive dieting, and dysfunctional attitudes (24). Three mutually exclusive categories across the continuum were defined as nondieters, intense dieters, and dieters at risk.

Analysis of hormonal status

On each of the scheduled test days (approximately days 6, 21, and 28 of the menstrual cycle), eight blood samples were obtained between 0800 and 2100, and the mean values of serum estradiol and progesterone concentrations in these samples were calculated and used in this analysis. Estradiol and progesterone were quantified with enzyme-linked immunosorbent assays by using the Boehringer Mannheim Automated ES 300 immunoassay system (Indianapolis). With this system, the CV for estradiol was 4.5% and for progesterone it was 2.1%. From the midfollicular to late luteal phases of the cycle under study subjects were instructed to collect daily urine samples for measurement of luteinizing hormone (LH) concentration. The CIBA-Corning ACS 180 LH Chemiluminoassay (Pittsburgh) was used to quantify serum and urinary LH. The CV for LH when using this system was 2.2%. Positive ovulatory function was determined on the basis of the hormonal measurements, with a review of all available data.

Dietary assessment

At the first and second CRC visits (approximately days 6 and 21 of the menstrual cycle), subjects were trained by a dietitian to record all food and beverages consumed for a 3-d period during the subsequent days including two weekdays and one weekend day, to reflect intake during the follicular and luteal phases of the menstrual cycle. All subjects were assured complete confidentiality of dietary data and nonjudgmental review of the records, with the importance of complete and accurate records being stressed when instructions were provided. Data obtained from these food records were analyzed with a microcomputer dietary analysis system based on US Department of Agriculture food content data, supplemented with manufacturer information, and processed with the NUTRITIONIST III interface software (N-Squared Computing, San Bruno, CA). The two sets of 3-d records were used to compare nutrient intakes during the follicular and luteal phases and to compare nutrient intakes across the continuum of eating pathology.

Serum carotenoids and lipids

Blood samples were protected from light throughout processing and handling. After collection by venipuncture, blood was allowed to clot and was separated by centrifugation at 2300 × g at 4°C for 10 min to obtain sera. Samples were stored at −70°C in cryogenic tubes until lipid extraction and HPLC analysis, which was conducted with a Varian Star 9010, 9050 system with a variable wavelength UV/VIS detector (Varian Analytical Instruments, Walnut Creek, CA) with the wavelength set at 450 nm. The column used was a Supelco (Bellefonte, PA) Supelcosil LC-18 (25 cm × 4.6 mm × 5 μm). The method of Bieri et al (25), as modified by Craft et al (26, 27), was used with a mobile phase of acetonitrile:methanol: methylene chloride (70:10:20, by vol), with 0.13% triethylamine (by vol) and 0.01% ammonium acetate (wt:vol) modifiers used to enhance recovery. This analytical method measures 90% of the total plasma carotenoids present and permits quantification of the predominant carotenoids (α-carotene, β-carotene, lycopene, and β-cryptoxanthin, and lutein plus zeaxanthin). With this acetonitrile-based method, the peak designated lutein is assumed to also contain the isomerically related carotenoid zeaxanthin. Accuracy was assessed by periodic analysis of National Institute of Standards and Technology (NIST) standard reference material SRM986, fat-soluble vitamins, and a pooled plasma reference sample was analyzed concurrent with batches of study samples to monitor analytical precision. Values for carotenoid concentrations were within 10% of NIST values with this analytic method, and CVs during analysis of study samples were < 5%.

Determination of serum cholesterol and triacylglycerols was performed with the Kodak Ektachem analyzer system (Eastman Kodak Company, Rochester, NY) (28). Standard reference materials from the manufacturer were used to validate analytical precision of these procedures.
Statistical analysis

Initially, descriptive statistics were calculated for all variables. Those variables not normally distributed were log transformed to achieve normal distribution before analysis. Correlations among the variables were examined by using Pearson product-moment correlations. Carotenoid data were corrected to ratios with cholesterol concentration, as previously described for tocopherols (29). One-way analysis of variance (ANOVA) with Scheffé’s post hoc test was conducted to compare baseline (early follicular) serum concentrations with dietary variables during the follicular and luteal menstrual phases. Differences between groups were also examined by using univariate techniques (eg, chi square) when appropriate. Based on the results of the univariate tests, multiple-regression models were developed for serum hormone concentrations at midluteal and late luteal phases as dependent variables and nutritional factors as independent variables. Each model was established by using forward and backward stepwise selection with the likelihood-ratio test to determine variables to be added or removed from the model.

Repeated-measures ANOVA was conducted to test for a main time effect and for interactions between time and ovulatory status for serum concentrations of gonadal hormones, lipids, and carotenoids, and for dietary variables. All analyses were performed by using SPSS PC+ (version 6.1, SPSS Inc, Chicago). For descriptive purposes means ± SEMs are reported.

RESULTS

Study subjects

The 76 subjects were distributed as 33 nondieters, 28 intense dieters, and 15 subjects with the most severe eating pathology, termed dieters at risk, according to results of the questionnaire. All subjects were of normal weight-for-height, as defined by the inclusion criteria, with no differences in BMI or percentage body fat observed across the groups (Table 1). Unequivocal hormonal data (including luteal phase progesterone concentration and a urinary LH surge) indicated that 35 subjects ovulated during the study cycle under observation. Among subjects with unequivocal hormonal data (n = 46), 100% of the nondieters, 83% of the intense dieters, and 27% of the dieters at risk had ovulated during the study cycle under observation. Ovulatory status (positive versus negative) was significantly associated with eating pathology (chi square, P < 0.0001); subjects having a greater degree of eating pathology were less likely to have ovulated during the menstrual cycle under observation.

Dietary intake

Energy and selected nutrient intakes of the three groups are shown in Table 2. During the follicular phase, intake of dietary fat differed significantly as a function of eating pathology (P < 0.02). Dietary fat contributed 30%, 29%, and 23% of energy intake in the nondieters, intense dieters, and dieters at risk, respectively. In the luteal phase, increased energy intake was also associated with reduced dietary fat intake (P = 0.05), and total energy intake tended to be different (P = 0.06). Women who were consuming a diet with ≤ 20% of energy from fat were most likely to be dieters at risk (P < 0.01).

Intake of energy and macronutrients was not significantly different in the follicular compared with the luteal menstrual cycle phases in the total group or in the subgroup of subjects who had ovulated. However, a significantly higher intake of energy, carbohydrate, and fat (P < 0.002, 0.008, and 0.003, respectively), as well as the proportion of energy from fat (P < 0.02), was observed in subjects who ovulated compared with subjects who did not ovulate during the menstrual cycle. Overall, ovulatory subjects obtained ≈ 30% of energy from fat, compared with 23% of energy from fat in subjects who were anovulatory.

Table 2

Dietary intake of selected dietary components for the study subjects in two phases of the menstrual cycle

<table>
<thead>
<tr>
<th>Dietary component</th>
<th>Nondieters (n = 33)</th>
<th>Intense dieters (n = 28)</th>
<th>Dieters at risk (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy (kJ)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>8124 ± 483</td>
<td>6985 ± 385</td>
<td>6558 ± 925</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>8068 ± 385</td>
<td>7385 ± 455</td>
<td>6218 ± 792(^{2})</td>
</tr>
<tr>
<td><strong>Carbohydrate (g)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>282.2 ± 23.5</td>
<td>234.9 ± 14.0</td>
<td>238.7 ± 39.2</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>264.3 ± 13.1</td>
<td>257.5 ± 17.5</td>
<td>226.4 ± 33.0</td>
</tr>
<tr>
<td><strong>Protein (g)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>60.4 ± 3.2</td>
<td>61.3 ± 5.3</td>
<td>55.1 ± 8.4</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>66.1 ± 4.4</td>
<td>68.0 ± 4.7</td>
<td>49.8 ± 6.2</td>
</tr>
<tr>
<td><strong>Fat (g)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>65.0 ± 4.0</td>
<td>55.7 ± 3.9</td>
<td>42.7 ± 7.3(^{f})</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>68.8 ± 5.5</td>
<td>65.9 ± 6.0</td>
<td>44.7 ± 7.6(^{e})</td>
</tr>
<tr>
<td><strong>Dietary fiber (g)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>10.2 ± 1.0</td>
<td>9.2 ± 0.7</td>
<td>11.8 ± 1.4</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>10.7 ± 0.8</td>
<td>10.4 ± 0.7</td>
<td>11.2 ± 1.2</td>
</tr>
<tr>
<td><strong>Cholesterol (mg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>178 ± 16</td>
<td>165 ± 22</td>
<td>156 ± 32</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>210 ± 30</td>
<td>204 ± 24</td>
<td>116 ± 22</td>
</tr>
<tr>
<td><strong>Calcium (mg)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Follicular phase</td>
<td>679 ± 45</td>
<td>622 ± 47</td>
<td>531 ± 78</td>
</tr>
<tr>
<td>Luteal phase</td>
<td>704 ± 51</td>
<td>635 ± 57</td>
<td>623 ± 82</td>
</tr>
</tbody>
</table>

\(^{f}\) Significantly different from other groups, P < 0.02. (ANOVA with Scheffé post hoc test).

\(^{e}\) P = 0.05 (ANOVA).
nadal hormones at baseline (early follicular phase) are summarized in Table 3. The serum α-carotene concentration increased significantly with a greater degree of eating pathology (P < 0.005), both corrected and uncorrected for serum lipids. The serum lycopene concentration tended to be lower if uncorrected for serum lipids with a greater degree of eating pathology (P = 0.083), but was unassociated with eating pathology if corrected for serum lipids. Baseline (early follicular) estradiol and progesterone concentrations were similar across the groups.

As anticipated, the serum estradiol concentration increased significantly (P < 0.0001) and the serum progesterone concentration tended to increase (P = 0.0539) over the menstrual cycle, although ovulation was not significant as a between-group factor. Serum cholesterol decreased significantly (P < 0.02) and triacylglycerols tended to decrease over the menstrual cycle (P = 0.074), irrespective of ovulation status. With ovulation as a between-group factor, the lutein concentration decreased over the menstrual cycle (P < 0.01) but was significantly higher (P < 0.0003) in the anovulatory women.

Several correlations between serum carotenoid concentrations and dietary variables were observed. The serum α-carotene concentration was inversely associated with intake of fat (r = −0.3, P < 0.05) and directly associated with intake of fiber (r = 0.4, P < 0.05). Serum β-carotene was inversely associated with intake of fat (r = −0.3, P < 0.05) and energy intake (r = −0.3, P < 0.05). The multiple-regression model for midluteal serum estradiol concentration identified luteal phase energy and fiber intakes as significant independent predictors (r² = 0.2, P < 0.03 and 0.006, respectively). Approximately 24% of the variation in late luteal phase serum estradiol concentrations was explained by serum α-carotene (P < 0.001) and β-carotene (P < 0.03) concentrations. Independent predictors of luteal phase serum progesterone concentrations in multiple-regression models were not identified, in part because of numerous interactions among the dietary and serum variables.

**DISCUSSION**

Women who engage in pathologic eating behavior without meeting the full diagnostic criteria for an eating disorder exhibit nutritional characteristics that are similar to those observed in patients with clinical eating disorders. Eating pathology in young women is typified by a chronic or intermittent restriction of energy intake, driven primarily by the avoidance of dietary fat. Concomitant with this dietary pattern is an increase in serum carotenoids obtained from vegetables, a biochemical profile that is common in both low-weight patients with anorexia nervosa (30, 31) and normal-weight patients with bulimia nervosa (32).

Concern with body weight and dieting is considered normal behavior among young women in the United States. The clinical eating disorders, which are characterized by severe eating pathology and dieting (33, 34), are only the pathologic extreme of the broader continuum of weight-related attitudes and behaviors. A particular avoidance of dietary fat (in nonbinge meals) has been observed in clinical studies of anorexic and bulimic patients (35–37). We found a reduction in fat intake to be the dietary feature most strongly associated with increased degree of eating pathology in young women. None of the subjects in the present study considered herself to have an eating disorder or had sought treatment for disordered eating patterns. None of these subjects was overweight, so dieting behavior may be considered inappropriate.

Results from the present study indicate an association between eating pathology in a nonclinical population and dietary patterns that have been suggested to affect reproductive health. In several (5, 10, 11, 38, 39) but not all (40) previous studies, a reduction in dietary fat intake (to < 25% of energy), typically associated with reduced total energy and increased dietary fiber intakes, has been observed to promote reduced circulating estrogen concentrations and clinical indications of altered menstrual cycle hormonal patterns. Increased dietary fiber has also been shown to reduce serum estrogen concentrations in premenopausal women (11, 13).

Of the carotenoids measured in the present study, all except lycopene reflect dietary intake of carotenoid-containing fruits and vegetables (41–43). We found the serum α-carotene concentration to be increased in association with greater degrees of eating pathology and the serum lutein concentration to be higher in anovulatory than ovulatory women when corrected for concentrations of cholesterol-carrying lipoproteins that transport carotenoids and are influenced by gonadal hormone concentrations (44, 45). The serum α-carotene concentration is typically lower than that of several other carotenoids but has been found to be the one most highly correlated with total

**TABLE 3**

<table>
<thead>
<tr>
<th>Nutritional Variable</th>
<th>Nondieters (n = 33)</th>
<th>Intense dieters (n = 28)</th>
<th>Dieters at risk (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol (mmol/L)</td>
<td>3.89 ± 0.15</td>
<td>3.95 ± 0.15</td>
<td>4.06 ± 0.35</td>
</tr>
<tr>
<td>Triacylglycerols</td>
<td>0.89 ± 0.12</td>
<td>0.99 ± 0.10</td>
<td>1.03 ± 0.18</td>
</tr>
<tr>
<td>α-Carotene (μmol/L)</td>
<td>0.062 ± 0.008</td>
<td>0.097 ± 0.022</td>
<td>0.172 ± 0.040</td>
</tr>
<tr>
<td>β-Carotene (μmol/L)</td>
<td>0.295 ± 0.026</td>
<td>0.301 ± 0.032</td>
<td>0.402 ± 0.067</td>
</tr>
<tr>
<td>β-Cryptoxanthin</td>
<td>0.198 ± 0.019</td>
<td>0.206 ± 0.018</td>
<td>0.176 ± 0.032</td>
</tr>
<tr>
<td>Lycopene (μmol/L)</td>
<td>0.808 ± 0.072</td>
<td>0.803 ± 0.082</td>
<td>0.537 ± 0.058</td>
</tr>
<tr>
<td>Lutein (μmol/L)</td>
<td>0.471 ± 0.051</td>
<td>0.532 ± 0.053</td>
<td>0.388 ± 0.067</td>
</tr>
<tr>
<td>Estradiol (pmol/L)</td>
<td>34.75 ± 3.38</td>
<td>42.95 ± 7.01</td>
<td>46.23 ± 10.08</td>
</tr>
<tr>
<td>Progesterone (nmol/L)</td>
<td>0.250 ± 0.023</td>
<td>0.261 ± 0.046</td>
<td>0.266 ± 0.051</td>
</tr>
</tbody>
</table>

1. ± = SEM.
2. Significantly different from other groups, P < 0.005, uncorrected; P < 0.002, corrected for serum lipids (ANOVA with Scheffé post hoc test).
3. P = 0.083, uncorrected for serum lipids (ANOVA).
vegetable intake (41), especially carotenoid-rich vegetables (43). In contrast, the serum lycopene concentration reflects intake of mixed dishes containing tomato sauce rather than vegetables and fruits (41, 43). In the present study, the lycopene concentration tended to be inversely associated with eating pathology and a difference across the groups was not apparent when corrected for serum lipids. Hypercarotenemia is a well-known clinical characteristic of low-weight anorexia nervosa patients (30, 31), who also (by definition of the disorder) are amenorrheic (24). However, hypercarotenemia has also been observed in normal-weight patients with bulimia nervosa (32), and in women who had been dieting to lose weight and presented with hypothalamic amenorrhea (18). Additionally, normal-weight anovulatory women who were hypercarotenemic from eating a diet rich in raw vegetables have also been identified and described in the literature (19).

The energy and nutrient intakes observed in the present study were comparable with those described previously for young women in the midwestern United States in studies using similar methods of diet assessment (46). Significant differences in energy and macronutrient intakes in the follicular compared with the luteal phase were not observed, although the women who ovulated consumed significantly more energy and fat, and a greater proportion of energy from fat, than did anovulatory women. The effect of menstrual cycle phase on energy intake is presumably hormonally mediated, which explains why it was not observed in a group exhibiting both typical and atypical hormonal patterns over the cycle. Barr et al (47) reported that mean energy and macronutrient intakes of anovulatory women did not vary over the menstrual cycle, but found significantly greater energy intakes during the luteal phase in women in an ovulatory cycle (based on diet records at three points). Martini et al (46) also observed significantly greater energy and macronutrient intakes in the luteal than in the follicular phase among women confirmed to have been ovulatory according to urinary LH concentrations, who were evaluated over four to six ovulatory cycles.

A limitation of the present study is that the subjects were observed for one menstrual cycle, with diet records at two time points. The degree of hormonal assessment and overall observation was comparable, however, with that reported for previous studies of this nature (15, 38, 46, 47). A more detailed assessment, analysis, or control of subject intake would have enhanced examination of the role of dietary factors, especially because these might be associated with ovulatory dysfunction, but these inadequacies reflect limitations in the depth and scope of the investigation as it was originally conceived. It is possible that inaccuracies in the approach to estimating ovulatory status might have resulted in misclassification of subjects, although the likelihood of false-positive results is low. Because of the cross-sectional nature of the design, the results should be cautiously interpreted as associations rather than as established cause and effect relations.

The limitations of dietary records, as an approach to examining nutrient intake, must also be recognized (48). The most notable weakness is that usual dietary intake as reported on diet records may be underestimated because subjects may modify food choices or underreport their actual food intake (48). Requiring subjects to record only a few days over the menstrual cycle (to reduce the burden on subjects), as in the present study, is a strategy to increase the likelihood of completeness of the diet record. However, the power to detect small or subtle differences, such as changes over the menstrual cycle, is reduced by fewer days of diet records. Underreporting in one or more subgroups in the present study is also possible. However, even in the observational studies of patients with clinically diagnosed bulimia nervosa, reduced energy intake (compared with that of normal control subjects) has been found (49, 50), which supports our findings of reduced energy and fat intakes among those subjects with greater degrees of eating pathology in a nonclinical population.

Also, findings from this study may not apply to all age groups of women. Anovulatory menstrual cycles are known to be more common in adolescents (14–18 y of age) than in older age groups (51), such as those in the present study. The proportion of women < 25 y of age with irregular cycles may be higher than in older women, although simple maturational effects are much less likely to exert an effect on reproductive function than are higher rates of sports participation and eating pathology among those ≥ 18 y of age, as addressed in this study. However, age-related changes in the reproductive cycle have been observed in women in age groups spanning 18–39 y, including the timing of ovulation (52) and follicular phase length (53), so these results may not be extrapolated across all age groups.

In conclusion, nutritional traits of those with clinical eating disorders appear to also be characteristic of young women with subclinically pathologic eating attitudes and behaviors. In comparison with the clinical eating disorders (which are relatively rare), some degree of eating pathology is common in a substantial proportion of young women. Nutritional characteristics associated with pathologic dieting behavior may also be associated with menstrual irregularities in young women.

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