Menstrual Cycle Abnormalities and Subclinical Eating Disorders: A Preliminary Report

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Menstrual dysfunction is a common concomitant of anorexia nervosa and bulimia. Initial investigations emphasized the role of weight loss and lean/fat ratio in amenorhea. Subsequent studies suggest a more complex interaction between eating disorders and menstrual status. However, in past investigations, menstrual abnormalities have been confounded with low weight. We conducted two studies to ascertain the prevalence of menstrual abnormalities in a group of women with subclinical eating pathology versus an age-, education-, and weight-matched group of normal controls. In Study 1, 93.4% of the subclinical subjects reported a history of menstrual abnormality as compared to 11.7% of the normal controls. In Study 1, 100% of the subclinical subjects, versus 15.0% of the controls, reported an abnormal menstrual history. These data suggest that menstrual dysfunction often occurs in women with abnormal eating attitudes but without weight loss or diagnosable eating pathology. Several hypotheses for this finding are proposed.

INTRODUCTION

This paper concerns a serendipitous finding. In the course of collecting control subjects for a study on anorexia nervosa and bulimia, we encountered a subset who scored high on the Eating Attitudes Test yet did not evidence diagnosable eating pathology. The psychological features of this group, which we labeled "subclinical eating disorders," were quite interesting. However, we were most struck by a physiological finding: these women reported a history of significant menstrual disruption. Understanding the relationship between menstrual cycle abnormal-

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ities and subclinical eating pathology is the focus of this paper.

Amenorrhea, or the cessation or inhibition of menstrual periods, has long been associated with anorexia nervosa (1). In fact, the DSM-III-R (2) now includes amenorrhea as a necessary condition for the diagnosis of anorexia nervosa in females. Researchers have also noted the prevalence of menstrual abnormalities in other types of eating disorders. In a study of normal-weight bulimics, Johnson et al. (3) detected amenorrhea in 20% and menstrual irregularity in 51% of their subjects. Similar findings have been reported in other bulimic samples (4).

Menstrual abnormalities have also emerged in high-risk, nonclinical samples. Garner and Garfinkel (5) reported elevated rates of amenorrhea, menstrual irregularity, and pathological eating attitudes among low-weight ballet dancers. Gadpaille et al. (6) noted increased eating pathology and major affective disorder in amenorrheic runners. However, in

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both of these studies, menstrual function was confounded with low weight; amenorrheic subjects weighed significantly less than their menstruating counterparts.

Weight is clearly important in regulating menstrual status, yet recent reports (e.g., Refs. 7–10) indicate that eating disorders are associated with menstrual dysfunction in ways that cannot be accounted for on the basis of weight or weight change alone. Past research has focused exclusively on clinical or highrisk samples, and menstrual dysfunction has been confounded by low weight. This report presents two preliminary studies which examine the extent to which menstrual abnormalities are associated with subclinical eating pathology in normalweight, nonclinical populations.

STUDY I

Method

Subjects for Study I were white females, aged 16-31, who weighed no more than 110% of ideal body weight (IBW). Eighty participants comprised three groups: 48 with diagnosed eating disorders (including 17 with restrictive anorexia nervosa, 14 with bulimic anorexia nervosa, and 17 with normalweight bulimia nervosa), 15 with subclinical eating disorders (SEDs), and 17 normal controls (C).

The SED and C groups were recruited from introductory psychology classes and from signs posted at the University of Rochester Medical Center advertising a study entitled "Thoughts and Feelings about Body Image." The Eating Attitudes Test (EAT-26) [11] was used to screen for undiagnosed eating pathology. The 17 subjects who scored below the recommended cut-off score (19 points) were considered Cs. The 16 subjects scoring above this level were interviewed to determine whether they had ever met DSM-III criteria for either anorexia nervosa or bulimia nervosa.' One subject was diagnosed with restrictive anorexia nervosa. The remaining 15 subjects had never fulfilled the criteria for either eating disorder and were considered SEDs. All subclinical subjects reported frequent dieting and preoccupation with weight and shape. In addition, two patterns of pathological eating were discernible. The first pattern was characterized by excessive starvation, exercise, vomiting, or laxative abuse, without binging or significant weight loss. The second pattern involved occasional binging and purging.

All three groups were indistinguishable on age ($\tilde{x} = 21.2$, SD = 3.7), education ($\tilde{x} = 13.3$, SD = 2.0), marital status (84.5% single), and age of menarche ($\tilde{x} = 13.1$, SD = 1.4). In addition, the SED and C groups were matched for current ($\tilde{x} = 91.2$, SD = 3.7) and lowest ($\tilde{x} = 83.6$, SD = 6.8) percentage of IBW (12). The groups did differ, however, in terms of their highest percentage of IBW ($\tilde{x} = 102.1\%$ and 96.33%, SD = 4.6 and 8.8, for subclinicals and controls, respectively; t = 2.36, p < 0.03) and the difference between their highest and lowest percentages of IBW ($\tilde{x} = 20.6\%$ and 10.6%, SD = 8.1 and 5.0, respectively; t = 4.12, p < 0.001).

After signing a consent form, subjects completed a battery of self-report measures including the EAT-26 and provided basic clinical data, including weight history and menstrual functioning.² Amenorrhea was defined as the cessation of menses for at least four consecutive months. Menstrual disruption due to physical conditions, such as diagnosed polycystic ovary syndrome or pregnancy, was excluded from consideration.

Results

As shown in the upper portion of Table 1, the three groups differed dramatically on menstrual history ($\chi^2 = 94.5$, p < 0.0001). None of the SEDs or Cs was currently amenorrheic. However, 66.7% of the SEDs had a history of secondary amenorrhea; none of the Cs had such a history. The mean duration of secondary amenorrhea in SEDs was 11.9 months. An

¹ The 17 C subjects were also interviewed, and all denied current or past eating problems.

² Information about the self-report measures is available from Jaine Strauss.

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TABLE 1. Menstrual Abnormalities in Diagnosed Eating Disorders, Subclinical Eating Disorders, and					
Normal Controls					

	Diagnosed eating disorders	Subclinical eating disorders	Normal controls
	n = 48	n = 15	n = 17
Study I			
Amenorrhea	37 (77.0) ^a	0	0
Current			
Past	5 (10.5)	10 (66.7)	0
Oligomenorrhea	3 (6.2)	4 (26.7)	2 (11.7)
Total	45 (93.7)	14 (93.4)	2 (11.7)
		n = 7	n = 20
Study II			
Amenorrhea		0	0
Current			
Past		2 (28.6)	1 (5.0)
Oligomenorrhea		5 (71.4)	2 (10.0)
Total		7 (100 0)	3 (15.0)

" Numbers in parentheses are percentages.

additional 26.7% of the SEDs had experienced oligomenorrhea, as compared to 11.8% of the Cs. One SED subject reported a pregnancy resulting in a stillbirth and one C subject had two uncomplicated, full-term deliveries; no other subjects had been pregnant. Overall, abnormal menstrual function characterized 93.4% of the SEDs but only 11.8% of the Cs.

To clarify the relationship between weight change and menstrual abnormalities, we tabulated the frequency of menstrual disruption by weight change separately from the SED and C groups. All 15 SED subjects had experienced weight fluctuations of at least 10% IBW, including the only subject who reported no history of menstrual irregularities. Seven subjects (46.7%) had weight fluctuations of greater than 20% IBW, and all had a history of secondary amenorrhea. Thus, there appears to be a strong relationship between weight shifts and menstrual disruption among subclinical subjects. The pattern was much less clear among con-

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trol subjects. The two C subjects (11.8%) with a history of menstrual disruption (oligomenorrhea) had not experienced appreciable weight fluctuations. Conversely, subjects with a history of weight shifts did note menstrual disturbances; nine subjects (52.4%) with weight changes of 10-20% IBW and one subject (5.9%) with a fluctuation of more than 20% IBW reported no menstrual disruptions.

Discussion

Clearly, subjects with dysfunctional eating attitudes reported significant menstrual disruption. However, it is likely that these subjects—while indistinguishable from the controls in terms of their current or lowest percentage of IBW—had indeed experienced weight pathology. Their higher weights and weight fluctuations suggest that the subclinical subjects may have been below their physiological "normal" weight; even though they were at "normal" weight based on standardized tables, these women may have been underweight relative to their natural setpoints.

Alternatively, the subclinical subjects may have gone on more "crash diets," resulting in menstrual disruption due to sudden and dramatic weight fluctuations (13).³ While it is noteworthy that alterations in weight do not appear to be associated with menstrual changes among control subjects, the confounding of prior weight fluctuations and group membership attenuates the clarity of these findings.

An additional difficulty in interpreting these data arises from the title of the study. It is likely that "Thoughts and Feelings about Body Image" inadvertently attracted a biased sample. We undertook Study II to examine whether the relationship between menstrual disruption and subclinical eating pathology would persist in the absence of any weight pathology in a less skewed sample of women.

STUDY II

Method

Subjects for Study II were students in a large, upper-level psychology course who elected to participate in a project entitled "Construct validation" in exchange for extra credit. Ninety-nine female participants completed a large battery of psychological instruments including the Eating Disorders Inventory (EDI) (13) and a menstrual history form.⁴ As in Study I, amenorrhea was defined as cessation of menstruation for at least four consecutive months and did not include menstrual disruption due to physical conditions.

Our analyses focus on 27 subjects, all of whom weighed between 90 and 110% of IBW. We classified 7 subjects as SEDs and 20 as Cs based on their EDI profiles. The EDI consists of eight subscales with empirically derived norms; three of the subscales assess eating disturbances. SED subjects had to score above the 99% confidence interval for controls on all three eating disorder subscales. In an attempt to ensure that these subjects did not, in fact, suffer from diagnosable eating pathology, we required that they also score below the 99% confidence interval for anorexia nervosa on at least one eating disorder subscale.5 To be classified as Cs, subjects had to score within or below the 99% confidence interval for controls on all three eating disorder subscales.6 The resulting groups did not differ on their current $(\bar{x} = 93.3, SD = 7.6)$, lowest $(\bar{x} = 87.3, SD = 6.7)$, or highest ($\bar{x} = 98.0$, SD = 9.1) percentage of IBW, nor did they differ in the magnitude of their weight fluctuations ($\hat{x} = 10.7\%$, SD = 5.5). The groups also did not differ on age of menarche ($\bar{x} = 12.5$, SD = 1.6), and none of the subjects reported a history of pregnancy.

Results

As shown in the lower portion of Table 1, the SED and C subjects did indeed manifest differences in menstrual function ($\chi^2 = 16.09$, p < 0.0001). None of the subjects was currently amenorrheic. However, 28.6% of the SEDs vs. 5.0% of the Cs had a history of secondary amenorrhea. The mean duration of secondary

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³ We are indebted to an anonymous reviewer for this explanation and reference.

⁴ Information about the additional measures in the battery can be obtained from Richard Ryan.

⁵ Because of confidentiality concerns, we were unable to interview subjects to determine more accurately the nature of their eating pathology.

⁶ Seventy-two subjects were excluded from analyses because: a) they could not be clearly classified as SED or C, b) they would not provide weight information, or c) they weighed more than 110% of IBW.

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amenorrhea was 8.0 months in the SEDs and 4.0 months in the Cs. An additional 71.4% of the SEDs vs. 10.0% of the Cs had experienced menstrual irregularity. Thus, 100% of the SEDs and 15.0% of the Cs reported abnormal menstrual histories.

Of the 99 original participants, 11 (11.1%) reported having stopped menstruating for three consecutive months or more. Of these 11, 10 (90.9%) scored above the normal range on at least one of the EDI eating disorder subscales.

Discussion

The subjects in study II, unlike those in Study I, were not explicitly recruited to participate in an investigation of eating attitudes. Thus, it is unlikely that the subject population was skewed toward women with particular weight-related concerns. In addition, the subclinical group in Study II did not report any weight pathology relative to the controls. Yet the results of Study II largely replicate the findings obtained in Study I. Women with disturbed eating attitudes had experienced considerably more menstrual abnormalities than their weight-matched peers.

GENERAL DISCUSSION

There are obvious shortcomings to both of the studies reported here. Both relied on small subject pools and involved retrospective recall of menstrual history. Neither measured lean/fat ratio, exercise level, gonadotropic hormone levels, or dietary intake, and Study II did not include subject interviews. Neither study examined the temporal relationship between

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the eating and menstrual disturbances. However, despite their limitations, these are among the first investigations in which differences in menstrual status were not confounded with differences in weight. Moreover, both studies demonstrated that disturbances of the hypothalamic-pituitary-gonadal axis were in the histories of women with subclinical eating disorders.

There are several ways to account for this co-occurrence. First, the menstrual irregularities noted here, like the "psvchogenic" amenorrhea of earlier years (14), may result from generalized emotional stress. According to this view, the menstrual dysfunction and the eating disturbance would be parallel symptoms of the same psychological process. Second, the menstrual irregularity might be the result of a specific behavioral pattern, such as erratic dietary intake or excessive exercise. Third, the endocrine mechanism underlying the menstrual disturbance might also interfere with appetite regulation. Finally, the hypothalamic-pituitary-gonadal axis might be more causally linked to the etiology of eating pathology than has been previously suspected. A poorly regulated hypothalamicpituitary-gonadal axis, reflected in menstrual irregularity, might provide the pathophysiologic substrate necessary for the genesis and maintenance of eating pathology. The viability of these speculations awaits further research.

SUMMARY

Weight loss has often been implicated in the etiology of menstrual abnormalities in eating disordered women. Recent research suggests that weight or weight change may not be sufficient to explain the complex relationship between pathological eating attitudes and menstrual dysfunction, yet, in past investigations, menstrual abnormalities have been confounded with low weight. The two studies reported here examined the prevalence of amenorrhea and oligomenorrhea in women with abnormal eating attitudes but without diagnosable eating pathology or weight pathology (subclinical eating disorders). We compared these women with age-, education-, and weightmatched control subjects (controls). In Study I, 93.4% of the subclinical subjects reported a history of menstrual abnormality, as compared to only 11.7% of the controls. Similarly, in Study II, 100% of the subclinical subjects and 15.0% of the controls had experienced menstrual dysfunction. Despite methodological problems, these data suggest that this elevated prevalence of oligomenorrhea and amenorrhea in subclinical eating disorders cannot be explained solely on the basis of weight or weight loss.

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