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Authors

Greenwood, Eleni A
Pasch, Lauri A
Cedars, Marcelle I
et al.

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Obesity and depression are risk factors for future eating disorder-related attitudes and behaviors in women with polycystic ovary syndrome

Eleni A. Greenwood, M.D., M.Sc., Lauri A. Pasch, Ph.D., Marcelle I. Cedars, M.D., and Heather G. Huddleston, M.D.

Department of Obstetrics, Gynecology and Reproductive Sciences, University of California-San Francisco, San Francisco, California

Objective(s): To identify clinical predictors of future eating disorder symptoms in women with polycystic ovary syndrome (PCOS).
Design: Prospective cohort study.
Setting: University center.
Patient(s): One hundred sixty-four women with PCOS by the Rotterdam criteria.
Intervention(s): Participants were characterized at a baseline visit between 2006 and 2017. A questionnaire including the validated Eating Disorder Examination-Questionnaire (EDE-Q) was self-administered at follow-up.
Main Outcome Measure(s): EDE-Q global score (0–6, higher scores indicate more severe symptoms).
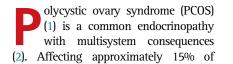**Result(s):** One hundred sixty-four women completed the follow-up survey an average of 5.3 years after the baseline visit. Compared with a normative population, women with PCOS had higher EDE-Q global scores (2.3 vs. 1.5) and scored higher on all subscales. Within the PCOS cohort, the following baseline clinical characteristics were independently predictive of scoring in the highest EDE-Q global score tertile: body mass index, waist circumference, hyperandrogenemia, high sensitivity C-reactive protein, and depression scores. Obesity at baseline conferred a 6.9-fold increase in the odds of elevated EDE-Q score (adjusted odds ratio = 6.89; 95% confidence interval, 2.70, 17.62), while a positive depression screen conferred 3.6-fold increased odds (adjusted odds ratio = 3.58; 95% confidence interval, 1.74-7.35). Compared with white women, nonwhite women were at risk of higher EDE-Q scores.
Conclusion(s): Women with PCOS are at risk of disordered eating attitudes and behaviors, which may interfere with attempts at lifestyle interventions. Clinicians should screen women with PCOS for eating disorder psychopathology, especially those with obesity or depression. An exclusive focus on weight loss may have unintended consequences. (Fertil Steril® 2020;113:1039–49. ©2020 by American Society for Reproductive Medicine.)
El resumen está disponible en Español al final del artículo.

Key Words: Polycystic ovary syndrome, eating disorders, depression, obesity, quality of life

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Polycystic ovary syndrome (PCOS) (1) is a common endocrinopathy with multisystem consequences (2). Affecting approximately 15% of reproductive-age women (3–5), PCOS is characterized by ovulatory dysfunction and hyperandrogenism and recognized by characteristic polycystic ovarian morphology on transvaginal ultrasound (6). Metabolic dysfunction is a critical clinical correlate of PCOS, with an increased risk of overweight, obesity, and insulin resistance (7–9). Consequently, women with PCOS are at increased risk of diabetes, hypertension, dyslipidemia, and metabolic syndrome (2).

Lifestyle interventions including diet and exercise are first-line treatments to offset cardiometabolic risk in PCOS (2, 10). Weight loss has been

shown to improve ovulatory function and reduce hyperandrogenism and hyperinsulinemia (11, 12). However, efforts at behavioral changes may be hampered by dysfunctional eating attitudes and behaviors. Furthermore, a single focus on weight loss might provoke weight-related stigma and impair the therapeutic alliance between clinician and patient.

Indeed, body image issues are prevalent in PCOS (13, 14), and disturbances in body image cluster with eating disorders (EDs) such as anorexia nervosa (AN), bulimia nervosa (15), or binge eating disorder (BED) (16, 17). AN is characterized by severely restricted energy intake resulting in low body weight, coupled with an intense fear of weight gain and distorted perception of body shape (17). BN involves recurrent episodes of binge eating followed by inappropriate compensatory behaviors such as purging (17). BED varies from BN in that binge eating episodes occur in absence of regular use of inappropriate compensatory behaviors (17). "Disordered eating" reflects the behavioral, emotional, and cognitive symptoms associated with EDs relating to eating and body weight, short of meeting diagnostic criteria for a particular ED. Disordered eating is far more prevalent than EDs (18).

Early evidence has suggested an increased prevalence of disordered eating (19–23) and EDs (21, 24) in women with PCOS. Binge eating and BED are most frequently implicated in PCOS, while restrictive behaviors are less commonly reported. In the general population, BED is linked with obesity (25).

Although an increased prevalence of disordered eating has been demonstrated in women with PCOS (19), critical gaps in the literature remain. It is unknown whether certain clinical parameters predict or protect from disordered eating attitudes and behaviors several years after an initial PCOS evaluation. An understanding of these risk factors would help clinicians identify women who should receive targeted, multidisciplinary interventions. Further, data regarding differences in ED symptomatology based on PCOS phenotype and race are lacking (26).

We hypothesized that body mass index (BMI) and depression would be clinical predictors of future ED symptomatology years after initial PCOS evaluation and designed a study to test this hypothesis. Understanding the relationship between BMI and ED symptoms might inform patient-centered care, enhancing clinician sensitivity of weight loss counseling. We further sought to explore whether PCOS phenotype and race predicted ED symptoms several years after baseline evaluation.

MATERIALS AND METHODS

This is an observational study of women with PCOS diagnosed by the Rotterdam criteria (6) enrolled in a longitudinal PCOS research cohort at a single academic center. Institutional review board approval was granted before all study activities. Subjects provided informed, written consent to participate.

Subjects

Over the course of 11 years (2006–2017), women were recruited to participate in the PCOS research cohort during a baseline visit to a multidisciplinary PCOS clinic at an academic institution. Details of the clinic protocol and cohort recruitment have been published elsewhere (27, 28).

Inclusion criteria were PCOS diagnosed by Rotterdam criteria (6), age between 16 and 45 years, and completion of a follow-up survey in 2018 examining interval updates in medical history, medication use, and ED attitudes and behaviors. The study cohort included 164 women.

Baseline Clinical Evaluation

Prior to the baseline clinic visit, patients completed a battery of self-administered questionnaires addressing medical history, gynecologic history, family history, review of systems, health behaviors, and mood. The International Physical Activity Questionnaire (29) was used to ascertain weekly exercise engagement. The seven-item Beck Depression Inventory Fast-Screen (BDI-FS) (30) was used to determine depression risk using a cutoff score of >4 (31). The BDI-FS was derived from the 21-item Beck Depression Inventory (BDI-II) (32), incorporating items that emphasize anhedonia, dysphoria, and cognitive aspects of depression (30).

Serum testing was performed at the university laboratory or one of two large commercial labs on the basis of insurance networks. Endocrine and metabolic assays were obtained, including a 2-hour 75-g oral glucose tolerance test. Homeostatic model assessment of insulin resistance, a correlate of the euglycemic clamp test to measure insulin resistance, was calculated from fasting glucose and insulin according to the method described by Matthews et al. (33). Blood was collected following an overnight fast and after abstaining from hormonally active medications for 1 month. Biochemical hyperandrogenism was established on the basis of serum androgen(s) exceeding lab-specific cutoff values; free and total T, androstenedione, and dehydroepiandrosterone sulfate (DHEAS) were individually considered. Upper limits of normal varied by laboratory: free T, 4.2–6.4 pg/mL; total T, 45–70 ng/dL; androstenedione, 235–285 ng/dL (cycle-dependent); DHEAS, 279-432 μg/dL (age-dependent).

During the series of two visits comprising the baseline clinic examination, women underwent complete history review and physical examination including anthropometric evaluation, transvaginal ultrasound, and dermatologic evaluations. Oligomenorrhea was defined as fewer than eight menses annually. Transvaginal ultrasounds were performed by one of two attending reproductive endocrinologists. Polycystic ovarian morphology was established on the basis of Rotterdam criteria, comprising an antral follicle count ≥12 or ovarian volume ≥10 mg in either ovary. One attending dermatologist performed skin examinations; modified Ferriman-Gallwey scores >8 established a diagnosis of hirsutism. Patients were counseled to remain off hormonally active medications for the month before ultrasonography (often aligning with lab testing to minimize lifestyle interruptions).

Follow-up Survey

In 2017, the follow-up survey was distributed to consenting PCOS cohort participants who had been evaluated in clinic

>6 months before. The survey was completed online via a secure REDCap platform (Vanderbilt University).

The survey featured the Eating Disorder Examination-Questionnaire (EDE-Q). The EDE-Q was derived from the Eating Disorder Examination interview (EDE) (34), an investigator-based semistructured interview of 45–75 minutes' duration. Based on the Diagnostic and Statistical Manual of Mental Disorders, 4th edition (35), the EDE is commonly used as the principal tool for diagnosing EDs (36).

Like the EDE, the EDE-Q generates data regarding frequency of behavioral features of EDs (e.g., "Over the past 28 days, how many times have you made yourself sick [vomit] as a means of controlling your shape or weight?"), as well as data about the severity of attitudinal aspects of ED psychopathology, via four subscale scores: restraint, eating concern, shape concern, and weight (e.g., "Has your weight influenced how you think [judge] yourself as a person?") (37). The EDE-Q enables assessment of EDE content in a 36-item self-report format. Overall agreement between EDE and EDE-Q has been established, with a possibly enhanced sensitivity in assessing the severity of complex features such as binge eating in the self-report EDE-Q form (38). The EDE-Q has been validated in the general population (39) as well as in clinical populations of individuals afflicted by EDs (40, 41). The reliability (42) and temporal stability (43) of the EDE-Q have been demonstrated.

Focusing on the past 28 days, the EDE-Q offers seven-point Likert-type response categories. Items are graded on a scale from 0 to 6, with 6 reflecting greatest severity and/or frequency of disordered eating attitudes and behaviors. Scoring of the four EDE-Q subscales involves averaging scores of items within that topical subscale; the EDE-Q global score is a numerical mean of the four subscale scores (i.e., 0–6).

To assess the frequencies of dysfunctional (binge eating and compensatory) ED behaviors, subjects are asked to indicate the number of episodes of each behavior they experienced over the prior 4 weeks. These items are separate from attitudinal subscale scores. Specific behaviors interrogated include self-induced vomiting, laxative misuse, diuretic misuse, objective overeating episodes, subjective overeating episodes, extreme dietary restraint, and excessive exercise. As per the EDE, in the EDE-Q objective overeating episodes are distinguished from subjective episodes if others would regard their amount of food intake as "unusually large," as opposed to a subjective sense of "having lost control and eaten too much," without actually having "eaten an unusually large amount of food given the circumstances." Extreme dietary restraint was indicated by self-report of going "for long periods of time (8 hours or more) without eating anything in order to influence [one's] shape or weight." Behavioral frequencies were reported as those activities that occurred at least weekly in accordance with prior literature (36). An exception to this was self-induced vomiting, which was considered if any episodes were reported in the prior 28 days, given the low frequency of this behavior. The EDE-Q was not administered at baseline.

The follow-up survey also queried updates in medical history, current medication use, and current height and weight, from which BMI was calculated. The seven-item BDI-FS was repeated to assess depression symptoms (30).

Statistical Analysis

Data were tested for normality, and descriptive statistics were provided. Subjects were divided into EDE-Q tertiles of equal size based on global EDE-Q scores, with the following cutoffs between the first and second tertile and second and third tertile, respectively: 1.48 and 2.98. Characteristics of subjects were compared across tertiles using Kruskal-Wallis testing for continuous variables and χ^2 or Fisher's exact test for categorical variables as appropriate. Correction for multiple comparisons was not performed given the exploratory nature of the endeavor.

EDE-Q scores and behaviors were compared across PCOS phenotypes. Similar comparisons were made across racial groups, categorized as white compared with nonwhite, and in an exploratory analysis further broken down into the following racial backgrounds: "white," "black," "Asian," "Hispanic," or "mixed." Persons reporting Hispanic ethnicity were considered in the Hispanic group, regardless of whether they further reported a white or black racial background.

EDE-Q scores and behavioral frequencies were compared with a reference population (36), published for the purposes of establishing a normative comparison for researchers using the EDE-Q. We used two-sided t tests, χ^2, or Fisher's exact as appropriate. In this circumstance, parametric testing was reported given the format of the available published data (36). The normative population comprised a sample of 5,255 women ages 18–42 years in the Australian Capital Territory region of Australia enrolled in an epidemiological study. The EDE-Q was self-administered, and an identical scoring methodology was used.

Logistic regression modeling was used to identify precedent (i.e., baseline) and concomitant (i.e., follow-up) characteristics associated with scoring in the highest tertile of EDE-Q global scores. A univariate model was first explored, with subsequent addition of potential confounders in two multivariate models. Multivariate model 1 was adjusted for baseline age and follow-up interval. Multivariate model 2 was adjusted for the same covariates as multivariate model 1, with the addition of baseline BMI. In a sensitivity analysis, we alternatively considered global EDE-Q as a dichotomous outcome using a cutoff score ≥ 4 to indicate an abnormal result.

Finally, the relationship between EDE-Q scores and depression risk at time of follow-up (concurrent with EDE-Q testing) was explored. Subjects were divided into those at risk for depression (i.e., BDI-FS >4) and those not at risk for depression. EDE-Q subscale and global scores were compared between groups using Kruskal-Wallis testing; behavioral frequencies were compared using χ^2 or Fisher's exact testing.

Statistical analyses were performed with STATA, version 14.2.

RESULTS

Of the PCOS cohort participants evaluated during the baseline study period (n = 478), 450 women had available email addresses, 423 of which were functional at the time of survey

distribution. The survey response rate was 43% (179/423); 164 women completed the EDE-Q and were included in the study cohort.

Responders Versus Nonresponders

Compared with the 286 women who either did not respond to the survey or complete the EDE-Q, the 164 women in the study cohort were slightly older at baseline clinic examination (28.9 vs. 27.6 years, $P=.02$), and had been seen more recently in clinic (4.9 vs. 5.9 years since baseline examination, $P=.04$). Respondents did not differ from nonrespondents on the basis of baseline BMI (28.1 vs. 27.7 kg/m^2, $P=.53$) or baseline depression score (3 vs. 4, $P=.34$).

Baseline Characteristics

At baseline clinical examination, median age was 28.9 years, 66% of women were white, and average BMI was 28.1 kg/m^2 (Table 1). Additional sociodemographic, endocrine, and metabolic parameters of the study cohort at baseline are shown in Table 1. Further breaking down the study cohort by tertile of EDE-Q global score, completed with the follow-up survey, we observed that a variety of patient baseline characteristics varied by EDE-Q scores. Baseline BDI-FS depression scores were higher in women with the highest EDE-Q scores (i.e., most severe ED symptomatology; tertile 3), and the prevalence of positive depression screens increased with EDE-Q tertile, from 15% in tertile 1 to 52% in tertile 3 ($P<.001$; Table 1). Endocrine parameters, such as hirsutism, serum T, and oligomenorrhea, did not differ by EDE-Q tertile.

Several baseline metabolic features varied by EDE-Q tertile. BMI increased progressively with EDE-Q symptom burden, from 23.5 kg/m^2 in tertile 1, to 29.9 kg/m^2 and 31.2 kg/m^2 in tertiles 2 and 3, respectively ($P<.001$). Waist circumference had a corresponding pattern. Concomitantly, high-density lipoprotein cholesterol decreased while triglycerides, fasting and 2-hour insulin, 2-hour glucose, homeostatic model assessment of insulin resistance, and high-sensitivity C-reactive protein (hsCRP) all increased with increasing EDE-Q symptom tertile (Table 1).

Follow-up Characteristics

Median follow-up interval (time between baseline clinic evaluation and follow-up survey administration) was 4.9 years (Supplemental Table 1). Patients were 34.3 years old on average at this time, with an average BMI of 28.1 kg/m^2. Further scrutinizing by EDE-Q tertile, patients scoring in the highest tertile (i.e., greatest disordered eating psychopathology) had higher BMI, higher depression scores, and prevalence of positive depression screens and gained more weight (average change +0.76 kg/m^2) compared with women with EDE-Q scores in lower tertiles (Supplemental Table 1).

EDE-Q Scores by PCOS Phenotype

The majority of women met all three Rotterdam criteria (Table 2). EDE-Q global and subscale scores did not vary as a function of PCOS phenotype. Of all the queried ED behav-

iors, only objective overeating episodes varied by phenotype, with more women in phenotype C reporting regular objective binge eating episodes (Table 2).

EDE-Q Scores by Race

Comparing white versus nonwhite women with PCOS, several differences in EDE-Q scores emerged. Nonwhite women had higher global EDE-Q scores (2.8 vs. 2.0, $P=.02$), reflecting higher ED symptom burden (Table 2). All subscale scores except restraint were higher in nonwhite women than among white women. Meanwhile, we did not observe variation in ED behavior frequencies between racial categories.

In an exploratory analysis, we further broke down race by the following categories: white, black, Asian, Hispanic, and mixed racial groups. Although nonwhite group sizes were small, overall black, Hispanic and mixed women emerged as having higher EDE-Q global and subscale scores compared with white women, while Asian women scored lower than white women (Supplemental Table 2).

Comparison with Normative Population

Compared with the normative figures from a community-based sample of Australian women ages 18–42, women in our PCOS cohort had higher global and subscale scores in all EDE-Q domains (Table 3). Behaviors also varied compared with this control cohort, with patients with PCOS endorsing more regular diuretic misuse, objective overeating episodes, extreme dietary restraint, and excessive exercise. Self-induced vomiting, laxative misuse, and subjective overeating episodes did not vary between the study cohort and control set (Table 3). Notably, the two cohorts varied significantly in terms of average age and BMI.

Correlates of Elevated EDE-Q Scores

Using logistic regression models, we investigated clinical features associated with scoring in the highest tertile of EDE-Q global scores.

Baseline clinical predictors associated with high EDE-Q scores appear in Table 4. In a univariate analysis, BDI-FS depression scores were associated with odds of elevated EDE-Q scores. Women with positive depression screens (BDI-FS > 4) at baseline had greater than a three-fold increase in odds of elevated EDE-Q scores (odds ratio [OR] = 3.62; 95% confidence interval [CI], 1.77, 7.41; $P<.01$). Increasing baseline BMI was associated with odds of aberrant EDE-Q attitudes; women who were obese at baseline had 6.5 times the odds of scoring in the highest tertile compared with lean women (OR = 6.52; 95% CI, 2.58, 16.47; $P<.01$). Notably, weight gain over the study period was also associated with high EDE-Q scores; each 1 kg/m^2 increase in BMI corresponded to a 15% increased odds of scoring in the highest tertile (OR = 1.15; 95% CI, 1.03, 1.29; $P=.01$; Table 4).

Higher 2-hour serum glucose after oral glucose challenge was associated with increased odds of high EDE-Q scores, as was baseline hsCRP. There was a suggestion of an association between biochemical hyperandrogenism and elevated EDE-Q scores; however, this did not meet statistical significance.

TABLE 1

Patient characteristics at baseline, by global EDEQ score.

	Overall (n = 164)	Tertile 1 (n = 55)	Tertile 2 (n = 55)	Tertile 3 (n = 54)	P
Sociodemographic					
Age	28.9 (25.3, 32.4)	29.3 (25.3, 32.2)	28.9 (25.5, 33.2)	28.7 (24.5, 32.6)	.98
Caucasian, %	66%	82%	57%	61%	.02
Education					.08
High school	5%	2%	6%	6%	
College	61%	49%	63%	71%	
Postgraduate	34%	59%	31%	23%	
Income					.89
<$50,000	36%	33%	37%	37%	
$50,000–100,000	35%	39%	29%	37%	
$100,000–200,000	22%	24%	24%	28%	
>$200,000	78%	4%	10%	8%	
Parous, %	12%	8.3%	16%	12%	.60
Smoker, %	10%	8.2%	10%	13%	.75
BDI-FS score[a]	3 (1, 7)	1 (0, 3)	3 (0.5, 7)	6 (2, 9)	<.001
At risk for depression[a]	36%	15%	35%	52%	<.001
Antidepressant use, %	7.8%	6.5%	11%	5.4%	.71
Exercise, minutes/week	210 (60, 360)	210 (90, 355)	220 (60, 45)	180 (40, 300)	.52
Endocrine					
Oligomenorrhea, %	86%	80%	91%	89%	.20
Polycystic ovarian morphology, %	89%	89%	89%	90%	.99
Modified Ferriman-Gallwey score	8 (4, 13)	7 (4, 11)	8.5 (4, 13)	8.5 (4, 14)	.29
Hirsute, %	57%	51%	65%	56%	.33
Biochemical hyperandrogenism, %	67%	61%	62%	77%	.16
Total T, ng/dL	47 (36, 63)	46 (34, 60)	47 (33, 68)	52 (38, 62)	.58
Free T, ng/dL	4.9 (2.6, 7.5)	4.2 (2.2, 7.2)	4.9 (2.9, 8.1)	5.7 (2.1, 7.4)	.56
AntiMüllerian hormone, ng/mL	7.3 (4.6, 10.9)	8.3 (7.5, 11.4)	6.0 (4.5, 8.8)	6.3 (3.2, 13.6)	.26
Metabolic					
Body mass index, kg/m^2	28.1 (23.8, 35.2)	23.5 (20.9, 27.8)	29.9 (26.0, 19.8)	31.2 (27.2, 37.5)	<.001
Waist, inches	34 (29, 40)	29 (27, 34)	35 (31, 41)	38 (31, 42)	<.001
Systolic blood pressure, mm Hg	112 (102, 122)	109 (100, 122)	113 (102, 122)	115 (105, 121)	.13
Total cholesterol, mg/dL	181 (165, 207)	181 (157, 203)	179 (157, 206)	186 (171, 216)	.37
Low-density lipoprotein cholesterol, mg/dL	107 (86, 133)	98 (83, 135)	104 (86, 130)	116 (98, 133)	.24
High-density lipoprotein cholesterol, mg/dL	55 (47, 68)	63 (53, 72)	52 (45, 63)	52 (44, 64)	<.01
Triglycerides, mg/dL	76 (53, 124)	68 (49, 86)	78 (55, 128)	101 (71, 136)	<.01
Fasting glucose, mg/dL	87 (81, 92)	87 (79, 93)	88 (82, 92)	85 (81, 91)	.54
Fasting insulin, mg/dL	8 (4, 16)	5 (2, 14)	8 (4, 17)	13 (6, 19)	<.01
2-hour glucose, mg/dL	94 (80, 112)	87 (75, 99)	97 (78, 115)	102 (85, 130)	<.01
2-hour insulin, mg/dL	40 (18, 98)	21 (6, 41)	47 (25, 110)	58 (26, 132)	<.01
Homeostatic model assessment of insulin resistance	1.6 (0.8, 3.6)	1.1 (0.5, 3.0)	1.7 (0.8, 3.2)	2.6 (1.2, 4.7)	.01
High-sensitivity C-reactive protein, mg/L	1.1 (0.2, 3.2)	0.2 (0.1, 1.8)	1.2 (0.4, 2.0)	1.6 (0.7, 7.8)	.01

Note: Tertiles 1–3 go from the lowest tertile (1, least symptomatology) to the highest (3, most symptomatology). Data are shown as median (interquartile range) or %. P values are from Kruskal-Wallis (nonparametric), χ^2, or Fisher's exact test as indicated.
[a] Beck Depression Inventory Fast-Screen (BDI-FS) > 4.

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Other endocrine measures, such as oligomenorrhea, polycystic ovarian morphology, and hirsutism, were not associated with EDE-Q global score (Table 4).

After adjusting for baseline age and follow-up interval, the above correlates of high EDE-Q scores (including positive depression screens, BMI, weight gain, 2-hour glucose, and hsCRP) remained with similar effect sizes (Table 4, multivariate model 1). Upon further adjustment for baseline BMI, we observed an attenuation of the link between 2-hour glucose and hsCRP levels with EDE-Q scores. Conversely, the association of biochemical hyperandrogenism with EDE-Q scores became statistically significant, with hyperandrogenic women at baseline having twice the odds of elevated EDE-Q scores (adjusted OR [aOR] = 2.24; 95% CI, 1.00-5.03; P = .05; Table 4).

In a sensitivity analysis, EDE-Q global scores were dichotomized using a cutoff of ≥ 4. Sixteen women (10%) exceeded this threshold, indicating clinically severe ED symptoms. With this approach, the association between BMI and elevated EDE-Q scores was minimally diminished (aOR = 1.04; 95% CI, 0.82-1.00; P = .06), after adjustment for baseline age and follow-up interval. Considered categorically, BMI was no longer significantly associated (P = .12). Baseline BDI-FS scores, meanwhile, remained robustly associated with elevated EDE-Q scores in this model (aOR = 6.18; 95% CI, 1.85-20.60; P < .01).

TABLE 2

EDE-Q scores, by PCOS phenotype and race.

	Overall (n = 164)	PCOS-A (n = 107)	PCOS-B (n = 19)	PCOS-C (n = 19)	PCOS-D (n = 15)	P	White (n = 106)	Nonwhite (n = 58)	P
EDE-Q Attitudes									
EDE-Q global score	2.3 (1.2, 3.2)	2.3 (1.1, 3.2)	2.7 (1.3, 3.5)	1.5 (1.3, 3.1)	2.8 (1.2, 3.5)	.82	2.0 (1.1, 3.1)	2.8 (1.9, 3.3)	.02
Restraint	2.0 (0.6, 3.4)	2.0 (0.8, 3.4)	1.6 (0.8, 2.8)	2.0 (0.6, 3.4)	3.0 (1.4, 4.0)	.84	2.0 (0.4, 3.2)	2.0 (0.8, 3.6)	.24
Eating concern	0.6 (0.2, 2.2)	0.6 (0.2, 2.2)	0.8 (0.2, 2.6)	0.6 (0.2, 2.2)	1.2 (0.2, 2.8)	.96	0.6 (0.2, 1.8)	1.1 (0.4, 2.4)	.01
Shape concern	3.0 (1.6, 4.3)	2.9 (1.8, 4.3)	3.5 (1.6, 4.5)	1.8 (1.3, 3.8)	3.8 (1.2, 4.8)	.64	2.6 (1.3, 4.1)	3.8 (2.4, 4.5)	<.01
Weight concern	2.8 (1.4, 4.0)	3.0 (1.4, 4.0)	3.4 (1.6, 4.2)	2.6 (1.4, 4.2)	3.6 (1.2, 5.2)	.79	2.6 (1.2, 3.8)	3.2 (2.2, 4.0)	.03
EDE-Q Behaviors									
Self-induced vomiting (any), %	1.2%	1.9%	0%	0%	0%	1.00	0.9%	1.8%	1.00
Laxative misuse	2.4%	2.8%	0%	0%	6.7%	.52	1.9%	3.5%	.62
Diuretic misuse	1.2%	1.9%	0%	0%	0%	1.00	0%	3.5%	.12
Overeating episodes, objective	20%	19%	16%	21%	27%	.87	21%	17%	.59
Overeating episodes, subjective	16%	12%	11%	42%	13%	.01	16%	16%	.93
Extreme dietary restraint	18%	19%	21%	16%	13%	.93	16%	21%	.46
Excessive exercise	24%	18%	12%	12%	9.4%	.10	23%	26%	.64

Note: Data are presented as median (interquartile range) or %. *P* values are from Kruskal-Wallis (nonparametric), χ^2, or Fisher's exact test as indicated. PCOS phenotypes are as follows: A, oligomenorrhea, hyperandrogenism, and polycystic ovarian morphology; B, oligomenorrhea and hyperandrogenism; C, hyperandrogenism and polycystic ovarian morphology; D, oligomenorrhea and polycystic ovarian morphology. All behavior prevalence data are for regular occurrence of that behavior (at least weekly) unless otherwise indicated. EDE-Q = Eating Disorder Examination-Questionnaire; PCOS = polycystic ovarian syndrome.

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Depression Risk and EDE-Q Scores

We additionally probed the relationship between EDE-Q global scores and BDI-FS depression risk scores at the time of follow-up survey (Supplemental Table 3). In women screening positive for depression, we observed increased EDE-Q scores across all subscales except restraint. The frequency of binging behaviors was increased in women with positive depression screens; compensatory behaviors that were increased in women with positive depression screens included laxative misuse and extreme dietary restraint, but not vomiting, diuretic misuse, or excessive exercise (Supplemental Table 3).

DISCUSSION

In a longitudinal study, we found that women with PCOS had higher EDE-Q scores than a normative cohort and report that BMI, hyperandrogenism, and depression symptoms at a baseline evaluation predict disordered eating attitudes and behaviors ascertained an average of 5 years later. Concerningly, we also found that disordered eating was associated with persistent weight gain over the follow-up period.

The concurrence of obesity and binge ED has been characterized in the general population (25). In PCOS, we observed a strikingly similar relationship between BMI and abnormal EDE-Q scores as a prior study including 148 women with PCOS by Lee et al. (19). We extended this finding to confirm that baseline BMI, years before EDE-Q assessment, is a predictor of abnormal EDE-Q scores with a similar effect size. We further examined BMI as a categorical predictor for facile clinical translation. We found that women who were obese at baseline had 6.9 times adjusted odds of elevated EDE-Q scores compared with lean women. Moreover, an increase in BMI from baseline to follow-up assessment also predicted EDE-Q scores, with each

TABLE 3

EDE-Q PCOS scores versus normative population.

	PCOS (N = 164)	Control population (N = 5,225)	P
Age	34.51 (6.63)	30.26 (7.22)	<.001
BMI, kg/m^2	29.81 (8.04)	24.52 (5.25)	<.001
EDE-Q Attitudes			
EDE-Q score (global)	2.33 (1.47)	1.52 (1.25)	<.001
Restraint	2.06 (1.50)	1.30 (1.40)	<.001
Eating concern	1.26 (1.32)	0.76 (1.06)	<.001
Shape concern	2.97 (1.66)	2.23 (1.65)	<.001
Weight concern	2.80 (1.64)	1.79 (1.51)	<.001
EDE-Q Behaviors			
Self-induced vomiting, %	0.0%	1.4%	.13
Laxative misuse, %	2.4%	1.0%	.06
Diuretic misuse, %	1.2%	0.3%	.05
Overeating episodes, objective, %	20%	10.6%	<.001
Overeating episodes, subjective, %	16%	13%	.23
Extreme dietary restraint, %	18%	3.4%	<.001
Excessive exercise, %	24%	4.9%	<.001

Note: Mean (SD) or percent (%) as indicated. Data are presented as median (##) or %. *P* values are from two-sided *t* test, χ^2, or Fisher's exact as appropriate. All behavior prevalence data are for regular occurrence of that behavior (at least weekly). EDE-Q = Eating Disorder Examination-Questionnaire; PCOS = polycystic ovarian syndrome.

Greenwood. BMI, depression, disordered eating in PCOS. Fertil Steril 2020.

TABLE 4

Baseline characteristic predictors of scoring in the highest tertile of global Eating Disorder Examination-Questionnaire—logistic regression models.

	Univariate OR	P	Multivariate 1 aOR	P	Multivariate 2 aOR	P
Sociodemographic						
Age	1.00 (0.94, 1.05)	.87	—	—	—	—
Caucasian	0.71 (0.35, 1.45)	.35	0.69 (0.33, 1.42)	.31	0.95 (0.43, 2.08)	.89
Education						
High school	Ref	.13	Ref	.09	Ref	.17
College	0.83 (0.18, 3.98)	.26	0.70 (0.14, 3.47)	.66	0.87 (1.01, 1.11)	.87
Postgraduate	0.39 (0.07, 1.99)	.71	0.30 (0.05, 1.64)	.16	1.06 (1.01, 1.11)	.29
Income						
<$50,000	Ref	.91	Ref	.51	Ref	.93
$50,000–100,000	1.03 (0.46, 2.32)	.46	1.00 (0.44, 2.27)	1.00	1.28 (0.54, 3.06)	.57
$100,000–200,000	0.74 (0.28, 1.93)	.28	0.73 (0.28, 1.92)	.52	1.23 (0.43, 3.52)	.70
>$200,000	1.08 (0.28, 4.18)	.91	1.03 (0.26, 4.02)	.51	1.49 (0.36, 6.13)	.93
Parous	0.99 (0.25, 2.81)	.98	1.09 (0.35, 3.46)	.88	1.01 (0.31, 3.28)	.98
Smoker	1.41 (0.47, 4.23)	.54	1.47 (0.49, 4.44)	.50	1.43 (0.46, 4.45)	.54
BDI-FS score	1.19 (1.09, 1.31)	<.01	1.19 (1.09, 1.31)	<.01	1.17 (1.07, 1.29)	<.01
At risk for depression[a]	3.62 (1.77, 7.41)	<.01	3.58 (1.74, 7.35)	<.01	3.33 (1.56, 7.08)	<.01
Antidepressant use	0.60 (0.12, 2.97)	.53	0.59 (0.12, 2.95)	.52	0.42 (0.08, 2.25)	.31
Exercise, hours/week	0.95 (0.88, 1.03)	.20	0.94 (0.86, 1.03)	.18	0.95 (0.86, 1.04)	.24
Endocrine						
Oligomenorrhea	1.36 (0.50, 3.71)	.55	1.49 (0.53, 4.17)	.45	1.18 (0.41, 3.43)	.76
Polycystic ovarian morphology	1.10 (0.36, 3.31)	.87	1.09 (0.36, 3.28)	.88	1.07 (0.33, 3.48)	.91
Modified Ferriman-Gallwey score	1.03 (0.97, 1.09)	.33	1.02 (0.97, 1.08)	.40	1.00 (0.94, 1.06)	.92
Hirsute	0.92 (0.47, 1.79)	.81	0.88 (0.44, 1.72)	.70	0.59 (0.28, 1.25)	.17
Biochemical hyperandrogenism	2.08 (0.98, 4.44)	.06	2.09 (0.98, 4.46)	.06	2.24 (1.00, 5.03)	.05
Total T, ng/dL	1.01 (0.99, 1.02)	.39	1.01 (0.99, 1.02)	.40	1.01 (0.99, 1.03)	.25
Free T, ng/dL	1.00 (0.91, 1.10)	1.00	1.00 (0.91, 1.10)	.99	0.95 (0.84, 1.06)	.34
Antimüllerian hormone, ng/mL	1.05 (0.89, 1.22)	.57	1.08 (0.91, 1.27)	.38	1.15 (0.94, 1.42)	.18
Metabolic						
Body mass index, kg/m^2	1.07 (1.02, 1.12)	<.01	1.07 (1.03, 1.12)	<.01	—	—
Body mass index category						
Lean	Ref	<.01	Ref	<.01	Ref	
Overweight	3.24 (1.17, 8.98)	.02	3.22 (1.16, 8.97)	.03		
Obese	6.52 (2.58, 16.47)	<.01	6.89 (2.70, 17.62)	<.01	—	—
Waist, inches	1.06 (1.01, 1.12)	.01	1.07 (1.02, 1.12)	.01	0.98 (0.87, 1.10)	.70
Systolic blood pressure, mm Hg	1.01 (0.99, 1.04)	.27	1.01 (0.98, 1.04)	.43	0.00 (0.96, 1.02)	.55
Total cholesterol, mg/dL	1.00, (0.99, 1.01)	.34	1.01 (0.99, 1.02)	.33	1.00 (0.99, 1.02)	.44
Low-density lipoprotein cholesterol, mg/dL	1.01 (1.00, 1.02)	.21	1.01 (1.00, 1.02)	.21	1.00 (0.99, 1.02)	.58
High-density lipoprotein cholesterol, mg/dL	0.99 (0.97, 1.01)	.46	0.99 (0.97, 1.01)	.40	1.01 (0.99, 1.04)	.32
Triglycerides, mg/dL	1.01 (1.00, 1.01)	.05	1.01 (1.00, 1.01)	.05	1.00 (1.00, 1.01)	.43
Fasting glucose, mg/dL	1.00 (0.97, 1.04)	.77	1.00 (0.97, 1.04)	.80	0.99 (0.95, 1.02)	.48
Fasting insulin, mg/dL	1.00 (0.99, 1.02)	.62	1.00 (0.99, 1.02)	.63	1.00 (0.98, 1.01)	.72
2-hour glucose, mg/dL	1.02 (1.00, 1.03)	<.01	1.02 (1.00, 1.03)	<.01	1.01 (1.00, 1.02)	.18
2-hour insulin, mg/dL	1.00 (1.00, 1.01)	.27	1.00 (1.00, 1.01)	.32	1.00 (0.99, 1.01)	.80
Homeostatic model assessment of insulin resistance	1.01 (0.96, 1.05)	.77	1.01 (0.96, 1.05)	.78	0.99 (0.93, 1.04)	.62
High-sensitivity C-reactive protein, mg/L	1.17 (1.02, 1.34)	.02	1.16 (1.01, 1.34)	.03	1.13 (0.98, 1.31)	.09
Change in body mass index, kg/m^2	1.15 (1.03, 1.29)	.01	1.16 (1.04, 1.30)	.01	1.10 (0.98, 1.24)	.10

Note: Odds ratios (ORs) for chances of scoring in the highest symptom tertile (i.e., tertile 3) compared with the lower two tertiles. Multivariate model 1 adjusted for baseline age and follow-up interval (for baseline predictors), adjusted for age at survey completion (for follow-up predictors). Multivariate model 2 adjusted for covariates in model 1, plus body mass index at corresponding time point. aOR = adjusted odds ratio.
[a] Beck Depression Inventory Fast-Screen (BDI-FS) > 4.

Greenwood. BMI, depression, disordered eating in PCOS. Fertil Steril 2020.

1 kg/m^2 gain increasing the odds of high global EDE-Q scores by 10%–15%. This is of paramount clinical importance, as women with PCOS who experience ongoing weight gain face worsening metabolic health. Disordered eating may be a marker for women at high risk of ongoing weight gain and should be considered a red flag for clinical management.

The link between overweight/obesity and disordered eating may originate early in life. Overweight adolescents are more likely than peers to use inappropriate weight-control behaviors such as vomiting or laxatives than their peers (44), and bulimia and obesity are frequently comorbid in adolescents and young adults (45). Shared mechanisms predisposing to both EDs and obesity include body image dissatisfaction, media exposure, dieting, weight-related teasing, child abuse and neglect, and an external locus of control (46, 47). Indeed, obesity and EDs have been conceptualized to co-occur along a spectrum of "weight-related disorders" (46). Targeted interventions to prevent or treat obesity and EDs should ideally acknowledge these myriad root sources and recognize that risk may emerge in young girls and women.

Women with PCOS are at increased risk of overweight/ obesity as well as interrelated ED psychopathology, presenting a complex management challenge for clinicians. A singular focus on weight loss, emphasized as paramount to obviate metabolic sequelae in PCOS (2, 10), may be counterproductive in the context of unrecognized disordered eating attitudes and behaviors. Repeated recommendations to lose weight may exacerbate underlying body image issues, while dietary restriction may trigger ensuing disinhibition and bulimic pathology (48, 49). Obesity and BED are frequently comorbid; in this situation it is suggested that the most effective intervention strategy is to first address the BED psychopathology before attempts at weight loss, through measures such as cognitive behavioral therapy (50). Clearly, a one-size-fits all "diet and exercise" approach for weight loss among overweight women with PCOS may be inadequate in actualizing positive metabolic change. Women with PCOS should be screened for ED attitudes and behaviors to contextualize weight-related issues and tailor effective treatments.

We identified an association between hyperandrogenemia at baseline and EDE-Q global scores, which was statistically significant in the fully adjusted multivariate model (aOR = 2.24; 95% CI, 1.00-5.03). Biologic plausibility is supported by various lines of evidence. Testosterone (T) is involved in stimulating food intake (51). Elevated circulating T levels have been demonstrated in women with bulimia nervosa (52, 53), while T levels are reduced in those with anorexia (53). T has been implicated in aggressive and impulsive behavior, which may contribute to binge eating (51). Notably, examined continuously, neither total nor free serum T levels were associated with EDE-Q scores, indicating a possible threshold effect.

Depression scores were strongly linked with EDE-Q scores. This is consistent with prior literature linking mood disorders and EDs (19, 24). The causal direction between depression and ED psychopathology is complex; while it has been proposed that binge eating develops as a coping mechanism for underlying psychological distress (54), it is also possible the EDs exacerbate mood dysfunction or that shared psychiatric risk factors predispose to both disorders (45). In our cohort, we are unable to disentangle these likely interdependent pathways; although depression at baseline was predictive of EDE-Q scores at follow-up, it is probable that EDE-Q scores were also higher in these individuals at baseline.

EDE-Q attitudes, but not reported disordered behaviors, varied as a function of race. Nonwhite women had higher (i.e., worse) scores across all subscales except restraint when compared with white women (Table 2). This is the first report of racial differences in EDE-Q scores among women living in the United States with PCOS. Even in the general population, there is a significant gap in knowledge regarding the impact of race on ED attitudes and behaviors (55), with one study suggesting a greater prevalence of AN and BN among white women compared with black women (56), one study suggesting BED is more common among black women (57), and others failing to observe any racial variation in EDs (58, 59). It is possible that cultural differences differentially contribute to ED risk, via the media, customs related to eating, or ideals of beauty. The impact of race may also be confounded by interrelated socioeconomic factors. To tailor culturally competent interventions in the future, additional studies are required to decipher whether and how race might modulate ED symptom risk in PCOS.

Strengths, Limitations, and Future Directions

Our study has several important limitations. We were unable to track the trajectory of EDE-Q scores over time, as this was not a component of the baseline clinical testing battery. BMI and depression scores both at baseline and follow-up were associated with EDE-Q scores (ascertained at follow-up) and with similar effect sizes, suggesting EDE-Q symptoms were likely present over the course of many years. The cutoff for our highest symptom tertile was 2.98, which is in the normal range (<4); the clinical implications of scores from 3 to 4 and the likelihood of score progression over time are unknown. Yet, cutoffs are necessarily somewhat arbitrary by nature, and the increasing BMI and BDI scores observed in the higher EDE-Q tertiles corroborate the plausibility of our main findings. Another limitation is that our control cohort was derived from published literature rather than matched in clinic; however, the overall results of increased scores in PCOS are consistent with prior literature. Sociocultural factors may differ between the Australian women and our Northern California–based cohort; however, many such risk factors for EDs are shared across the populations, including the Western beauty ideal of thinness, societal pressure to succeed, peer pressure, and teasing or bullying (60). Finally, as a multidisciplinary PCOS specialty clinic at a university center, our cohort may reflect a particularly motivated and/or medically complex set of individuals; our results may not be extrapolated accurately to all populations of women with PCOS.

Strengths of our study include an overall relatively large sample of thoroughly characterized women followed for several years. We were able to compare clinical parameters at two time points and interval changes in certain predictors such as BMI. The availability of baseline data, including diagnostic criteria, sociodemographic data, anthropometrics, and serum metabolic and endocrine panel testing enabled us to identify clinical risk factors that predict EDE-Q psychopathology several years down this road, enabling clinicians to target high-risk individuals. To our knowledge, this is the first study to further evaluate differences in EDE-Q scores on the basis of PCOS phenotype and racial subgroups.

Future efforts should focus on prospectively cataloguing EDE-Q scores over time in a larger sample of multiethnic

women with PCOS diagnosed by a variety of phenotypic features. Population-based studies of ED symptoms among women with PCOS would add to our understanding of the frequency and severity of these symptoms in a broader cohort. Furthermore, interventions that address the potential co-occurrence of disordered eating behaviors and overweight/obesity must be examined to identify tailored treatments that most effectively combat long-term metabolic sequelae of PCOS while enhancing quality of life.

Conclusions

Women with PCOS are at risk of disturbances in ED-related attitudes and behaviors. Elevated BMI and depressed mood are predictors of ED symptoms. Lifestyle interventions such as diet and exercise are recommended as first-line therapies to counteract metabolic risk in PCOS; however, such recommendations may be ineffective or potentially exacerbate harm in the setting of unrecognized ED symptoms. Women with PCOS should thus be screened for ED symptoms. A multidisciplinary approach addressing body and mind should be undertaken in treatment of PCOS women with comorbid weight and disordered eating issues.

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La obesidad y la depresión son factores de riesgo que predisponen en el futuro comportamientos relacionados con trastornos de la alimentación en mujeres con síndrome de ovario poliquístico

Objetivo: identificar predictores clínicos de síntomas de futuros trastorno de la alimentación en mujeres con síndrome de ovario poliquístico (PCOS).

Diseño: estudio de cohorte prospectiva.

Entorno: centro universitario.

Paciente(s): 164 mujeres con PCOS según los criterios de Rotterdam.

Intervención(es): las participantes fueron categorizadas en una visita inicial entre 2006 y 2017. En el seguimiento se utilizó un cuestionario autocompletado que incluía el validado Eating Disorder Examination-Questionnaire (EDE-Q).

Principal(es) medida(s) de resultado(s): puntuación global del EDE-Q (0–6, puntuaciones mayores indican síntomas más severos).

Resultado(s): 164 mujeres completaron la encuesta de seguimiento en una media de 5.3 años tras la visita inicial. Las mujeres con PCOS, comparadas con una población normalizada, tuvieron una mayor puntuación global en EDE-Q (2.3 vs. 1.5) y tuvieron una puntuación mayor en todas las subescalas. Dentro de la cohorte PCOS, las siguientes características clínicas basales fueron independientemente predictivas para puntuar en el tercil mayor del EDE-Q: índice de masa corporal, circunferencia de la cintura, hiperandrogenemia, proteína C-reactiva de alta sensibilidad y marcadores de depresión. La obesidad en el momento basal confirió un riesgo de 6.9 veces de obtener una puntuación EDE-Q elevada (odds ratio ajustada = 6.89; intervalo de confianza del 95%, 2.70–17.62), mientras que un cribado positivo para depresión confirió un riesgo de 3.6 veces (odds ratio ajustadas = 3.58; intervalo de confianza del 95% 1.74–7.35). Comparadas con las mujeres de raza blanca, las de raza no blanca tuvieron mayor riesgo de obtener mayores puntuaciones en el EDE-Q.

Conclusión(es): las mujeres con PCOS tienen un mayor riesgo de predisposición a comportamientos de trastornos alimentarios, que podrían interferir sobre los intentos para cambios de estilo de vida. Los clínicos deben cribar la psicopatología de los trastornos de la alimentación en las mujeres con PCOS, especialmente en aquellas con obesidad o depresión. Un enfoque exclusivo en la pérdida de peso podría tener consecuencias indeseadas.