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SCIENTIFIC INVESTIGATIONS

Association of obstructive sleep apnea risk with depression and anxiety symptoms in women with polycystic ovary syndrome

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Study Objective: To determine whether obstructive sleep apnea (OSA) risk is associated with depression and anxiety symptoms in women with polycystic ovary syndrome (PCOS).

Methods: This is a cross-sectional study of women with PCOS, by the Rotterdam criteria, seen at a single academic center between June 2017 and June 2020. Depression symptoms, anxiety symptoms, and OSA risk were assessed with the self-administered Patient Health Questionnaire-9 (PHQ-9), Generalized Anxiety Disorder-7 (GAD-7), and Berlin questionnaires, respectively. Univariate and multivariate logistic regression analyses were used to determine the odds of moderate/severe symptoms of depression (PHQ-9 ≥ 10) and anxiety (GAD-7 ≥ 10) in the high-risk vs low-risk OSA groups. The primary multivariate model adjusted for age, body mass index, free testosterone, and insulin resistance.

Results: Of the 200 participants, the mean age was 28.0 years and 38% screened high risk for OSA. Women who screened high-risk OSA had > 3 times the odds of moderate/severe depression (odds ratio [OR]: 3.19; 95% confidence interval [CI]: 1.76–5.78; $P < .001$) and > 2 times the odds of having moderate/severe anxiety (OR: 2.49; 95% CI: 1.34–4.64; $P = .004$). These associations were only slightly attenuated in the adjusted models: the adjusted OR for moderate/severe depression was 3.06 (95% CI: 1.36–6.88; $P = .01$) and the aOR for moderate/severe anxiety was 2.39 (95% CI: 1.03–5.59; $P = .04$).

Conclusions: Among women with PCOS, those at high risk of OSA experienced elevated depression and anxiety symptoms compared with those at low risk for OSA, independent of the effects of age, body mass index, hyperandrogenism, and insulin resistance.

Keywords: obstructive sleep apnea, depression, anxiety, polycystic ovary syndrome

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BRIEF SUMMARY

Current Knowledge/Study Rationale: Women with polycystic ovary syndrome are disproportionally impacted by obstructive sleep apnea, depression, and anxiety. Evidence suggests a link between the obstructive sleep apnea and psychiatric comorbidities in the general population, but these relationships are understudied and less well understood in this relevant population.

Study Impact: We found that being high risk for obstructive sleep apnea was associated with more severe depression and anxiety symptoms after controlling for important potential confounders, highlighting the interrelatedness of these highly prevalent comorbidities in women with polycystic ovary syndrome. Although confirmatory studies are needed, the results raise the possibility of obstructive sleep apnea treatment having additional psychiatric benefits in this population.

INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine disorder affecting 6–10% of reproductive-aged women.[1] The condition is characterized by irregular menstrual cycles, hyperandrogenism, and polycystic ovarian morphology. Affected women experience diverse comorbidities, including higher rates of psychiatric disorders. A recent meta-analysis showed that women with PCOS had 4.2 times the odds of having moderate/severe depressive symptoms and 5.6 times the odds have having moderate/severe anxiety symptoms compared with control women without PCOS.[2] Potential explanatory models for adverse mental health outcomes in PCOS have been proposed, including obesity, hyperandrogenism, and insulin resistance; however, evidence suggests that there are other contributing factors.

Women with PCOS are also at disproportionate risk for obstructive sleep apnea (OSA), with 35% affected,[3] which is substantially greater than the estimated prevalence of 6–19% for women in the general population[4] and 3% in reproductive-aged women.[5] Although women with PCOS experience greater rates of obesity, which is one of the main risk factors for OSA, the increased risk for OSA persists even after accounting for obesity.[6] OSA is commonly comorbid with psychiatric disorders in the general population, with a 23% prevalence of clinical depression in patients with OSA.[7] By comparison, the 12-month prevalence of major depressive episodes among adults was approximately 6% in a large, population-based study.[8] International PCOS guidelines recommend screening for OSA to identify and alleviate related symptoms, including the potential for fatigue to contribute to mood disorders.[9] Yet, there is a paucity of literature

addressing psychiatric disorders, and OSA specifically, in the PCOS population.

A better understanding of the role of OSA and its association with depression and anxiety may aid in maximizing therapies for women with PCOS. Behavioral interventions for OSA such as weight loss and exercise coincide with recommended treatments for the obesity and metabolic derangements that are often seen in women with PCOS. Medical devices such as continuous positive airway pressure and mandibular repositioning devices have been shown to decrease depression symptoms, particularly with optimal treatment adherence.[10–13] Although the evidence is less robust and more conflicting with regard to the impact of OSA treatment on anxiety, recent studies have also demonstrated continuous positive airway pressure therapy to have a positive impact on anxiety symptoms.[10,14]

In this cross-sectional study, we examined the association between OSA risk and depression and anxiety symptoms among women with PCOS engaged in care at a large academic health care system. OSA risk was determined by the Berlin questionnaire, while depression and anxiety symptoms were similarly assessed with validated instruments. We hypothesized that being high risk for OSA is associated with elevated depression and anxiety symptoms in this population. By elucidating the connection between these highly prevalent comorbidities, this study may inform counseling and interventions to improve both sleep and psychiatric profiles within this vulnerable population.

METHODS

Study design and population

This was a cross-sectional study of women with PCOS, by the Rotterdam criteria, seen at a single university-based multidisciplinary PCOS clinic between June 2017 and June 2020. The revised 2003 Rotterdam criteria required 2 out of 3 of the following features: (1) oligo- or anovulation, (2) clinical and/or biochemical hyperandrogenism, and (3) polycystic ovaries.[15] Participants were excluded if they did not complete all the required questionnaires. Study approval was obtained from the University of California, San Francisco, Institutional Review Board.

Measures

Patients received an email link to a set of validated questionnaires via the REDCap online platform at the time that their visits were scheduled, usually several months in advance, and were required to complete the questionnaires prior to their initial clinical visit. OSA risk was assessed with the Berlin questionnaire, which consists of 11 items divided into the 3 domains of snoring/apneas, fatigue/sleepiness, and obesity/hypertension.[16] No positive scores or a positive score in 1 domain is considered low risk for OSA. A positive score in 2 or 3 domains is considered high risk for OSA. The sensitivity and specificity of the Berlin questionnaire in the general adult population for diagnosis of moderate to severe OSA (apnea-hyponea index ≥ 15 events/h) using the gold standard of polysomnography were 77% (95% confidence interval [CI]: 73–81%) and 44% (95% CI: 38–51%), respectively.[17]

Depressive symptoms were assessed using the 9-item Patient Health Questionnaire-9 (PHQ-9). Scores of 5, 10, 15, and 20 correspond to mild, moderate, moderately severe, and severe symptoms of depression. A PHQ-9 score ≥ 10 had a sensitivity of 88% and a specificity of 88% for major depression using structured mental health professional interviews as the criterion standard.[18] Anxiety symptoms were assessed using the 7-item Generalized Anxiety Disorder-7 (GAD-7) questionnaire. Scores of 5, 10, and 15 correspond to mild, moderate, and severe symptoms of anxiety. A GAD-7 score ≥ 10 had a sensitivity of 89% and specificity of 82% using structured mental health professional interviews as the criterion standard.[19]

Laboratory evaluation was completed, on average, 1 to 2 months prior to presentation to the clinic. Patients were asked to discontinue any hormonally active medications for a minimum of 1 month prior and metformin 2 weeks prior. Testing was performed at the university laboratory or 1 of 2 large commercial laboratories, with the exception of a small percentage of patients who had testing performed at other laboratories as determined by insurance coverage. Biochemical hyperandrogenism was present if ≥ 1 of the following androgens were elevated per laboratory specific thresholds: free testosterone, total testosterone, androstenedione, and dehydroepiandrosterone sulfate (DHEAS). A fasting lipid panel and 2-hour, 75-g oral-glucose-tolerance test were performed to assess for metabolic abnormalities. Insulin resistance was represented by the HOMA-IR (homeostatic model assessment for insulin resistance) score, as calculated from the fasting glucose and insulin levels.[20] C-reactive protein (CRP) and anti-müllerian hormone (AMH) were obtained.

Patients completed their PCOS evaluation and counseling over the course of 2 clinic visits within the same calendar month. During the first visit, the PCOS diagnosis by the Rotterdam criteria was confirmed with a thorough history and physical exam. Anthropomorphic measurements were collected. Ovulatory dysfunction was characterized as < 8 periods per year. Transvaginal ultrasounds were performed by 1 of 2 reproductive endocrinologists, with the threshold for polycystic ovarian morphology being ≥ 12 follicles per ovary and/or ovarian volume ≥ 10 mL in either ovary. A single dermatologist evaluated patients for hirsutism, as defined by a modified Ferriman-Gallwey (MFG) score ≥ 8. At the second visit, the patient received personalized counseling regarding her PCOS diagnosis and recommended treatment from the reproductive endocrinologist, and had the opportunity to meet with a dietitian and psychologist for additional counseling. All aspects of the study were conducted in conjunction with the standard clinical care provided in our PCOS clinic.

Statistical analysis

Data were assessed for normality. Differences in participant characteristics between the low-risk and high-risk OSA groups were assessed with 2-sided *t* tests, chi-square tests, and Fisher's exact tests, as appropriate. Univariate and multivariate logistic regression analyses were used to determine the odds of moderate/severe symptoms of depression and anxiety defined by PHQ-9 score ≥ 10 and GAD-7 score ≥ 10 in the high-risk vs low-risk OSA

groups. Multivariate model 1 adjusted for a priori–selected possible confounders: age, body mass index (BMI), free testosterone level, and insulin resistance as measured by the HOMA-IR. Multivariate model 2 adjusted for variables significant at $P < .10$ in univariate models in addition to the variables adjusted for in model 1. A 2-tailed P value < .05 was considered statistically significant. All analyses were conducted with Stata version 15.1 (StataCorp, College Station, TX).

RESULTS

A total of 212 women with PCOS were seen during the study period of whom 12 were excluded for having incomplete questionnaires. Of the remaining 200 participants, 124 (62%) screened low risk for OSA and 76 (38%) screened high risk for OSA. The mean age of all participants was 28.0 years (standard deviation [SD]: 6.2) with a range of 14.3 to 43.7 years. The majority of the participants were White, with Asian being the next most prevalent race. There were no differences in age and racial/ethnic makeup between the low-risk OSA and high-risk OSA groups.

The mean BMI was 30.9 kg/m^2 (SD: 9.0) among all participants, 38.1 kg/m^2 (SD: 8.2) in the high-risk OSA group and 26.5 kg/m^2 (SD: 6.3) in the low-risk OSA group ($P < .001$). The high-risk OSA group also had a significantly higher mean waist circumference and systolic blood pressure. There were no differences in mean diastolic blood pressure between groups. Both OSA groups were similar in their PCOS features, with the exception that the high-risk OSA group had a higher rate of ovulatory dysfunction as compared with the low-risk OSA group (86.5% vs 70.7%, $P = .01$). The majority of participants were phenotype A with all 3 general features of PCOS present.

Significant differences in laboratory results were noted depending on OSA risk. Although the level of total testosterone was similar between groups, participants who were high risk for OSA had a significantly higher level of free testosterone compared with those who were low risk for OSA (6.5 vs 4.5 ng/dL, $P < .001$). The high-risk OSA group in comparison to the low-risk OSA group had higher levels of fasting glucose (102.7 vs 88.0 ng/dL, $P < .001$) and fasting insulin (25.3 vs 12.5 mg/dL, $P < .001$), and a resulting higher HOMA-IR (7.0 vs 2.9, $P < .001$).

The high-risk OSA group in comparison to the low-risk OSA group also had a less favorable lipid profile, with higher levels of triglycerides (126.2 vs 82.4 mg/dL, $P < .001$) and lower levels of high-density lipoproteins (49.5 vs 61.4 mg/dL, $P < .001$). CRP levels were higher in the high-risk vs low-risk OSA groups (6.7 vs 2.3 mg/L, $P < .001$). AMH levels were lower in the high-risk vs low-risk groups (5.9 vs 8.9 ng/mL, $P < .001$) (**Table 1**).

Depression

Participants in the high-risk OSA group had a higher mean PHQ-9 score as compared with the low-risk OSA group (12.0 vs 8.4, $P < .001$) (**Figure 1**), and also had a higher rate of current antidepressant use (22.4% vs 8.9%, $P < .01$). Selective serotonin reuptake inhibitors were the most common type of antidepressant used, with a significantly higher percentage of patients in the high-risk OSA group taking these antidepressants (14.5% vs 5.7%, $P = .03$). In the univariate logistic regression, women in the high-risk OSA group had increased odds of moderate or severe depression symptoms (odds ratio [OR]: 3.19; 95% CI: 1.76–5.78; $P < .001$). This association was only slightly attenuated in the multivariate logistic regressions. In multivariate model 1 adjusting for age, BMI, free testosterone, and HOMA-IR, the adjusted OR (aOR) for moderate or severe depression symptoms was 3.06 (95% CI: 1.36–6.88; $P = .01$). In multivariate model 2, which additionally adjusted for the variables significant at $P < .10$ in univariate models of MFG score, triglycerides, high-density lipoproteins, and CRP, the aOR was 2.76 (95% CI: 1.15–6.63; $P = .02$) (**Table 2**).

Anxiety

The high-risk OSA group had a higher mean GAD-7 score as compared with the low-risk OSA group (8.9 vs 6.4, $P = .002$) (**Figure 1**). In the univariate logistic regression, women in the high-risk OSA group had increased odds of moderate or severe anxiety symptoms (OR: 2.49; 95% CI: 1.34–4.64; $P = .004$). This association was minimally changed in the multivariate regression models. In multivariate model 1 adjusting for age, BMI, free testosterone, and HOMA-IR, the aOR for moderate or severe anxiety symptoms was 2.39 (95% CI: 1.03–5.59; $P = .04$). In multivariate model 2, which additionally adjusted for MFG score, which was significant at $P < .10$ in the univariate model, the aOR was 2.53 (95% CI: 1.05–6.09, $P = .04$) (**Table 3**).

DISCUSSION

In this clinic-based study of women with PCOS performed at a large academic center, we found that being high risk for OSA is indeed associated with increased rates of moderate/severe symptoms of depression and anxiety, confirming that what has previously been seen in the general population also extends to this special population. Given the increased prevalence of OSA and psychiatric comorbidities in women with PCOS, the importance of screening and treating these disorders in this population has previously been acknowledged, but as separate entities.[9,21] This study brings to light the importance of considering how these comorbidities may be interrelated.

Obesity and insulin resistance, common features of PCOS, are also proposed risk factors for OSA.[1,22] Obesity, in particular, is one of the strongest predictors for OSA due to associated changes in upper airway anatomy. Obesity and insulin resistance have also been linked to mental health comorbidities in PCOS.[23] However, we found that the association between OSA risk and depression and anxiety symptoms persisted after adjustment for these potential confounders. Similarly, hyperandrogenism, a cardinal feature of PCOS, has been linked to OSA in some but not all studies.[24] Nevertheless, adjustment for hyperandrogenism did not alter the associations found between OSA and depression and anxiety.

Although the directionality of the association cannot be assessed in this cross-sectional study, prior longitudinal studies of sleep disturbances and depression and anxiety in the general population suggest that the relationship is bidirectional.[25,26] OSA is characterized by sleep fragmentation, which, in itself,

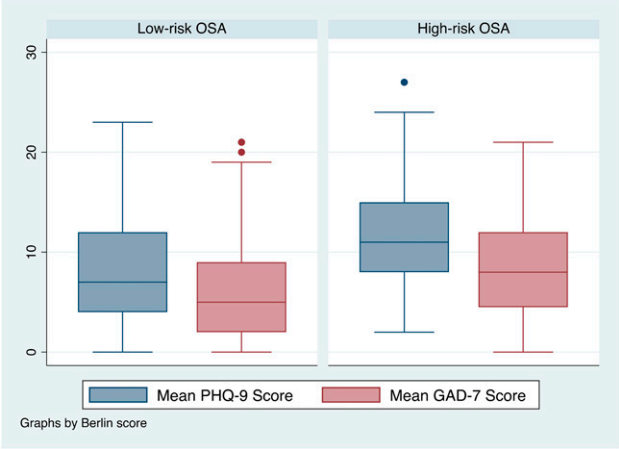
Table 1—Participant demographics, clinical findings, and laboratory results.

	Total (n = 200)	Low-Risk OSA (n = 124)	High-Risk OSA (n = 76)	*P*
Demographics				
Age, y (SD)	28.0 (6.2)	27.8 (6.0)	28.4 (6.5)	.53
Race/ethnicity				.61
White	54.2%	56.8%	50.0%	
Asian	20.5%	22.0%	18.1%	
Hispanic	4.7%	4.2%	5.6%	
Black	2.6%	2.5%	2.8%	
Native American	1.6%	1.7%	1.4%	
Multiracial	16.3%	12.7%	22.2%	
Clinical findings				
Body mass index, kg/m^2 (SD)	30.9 (9.0)	26.5 (6.3)	38.1 (8.2)	<.001
Waist circumference, cm (SD)	88.6 (25.1)	80.3 (20.1)	102.2 (26.5)	<.001
SBP, mm Hg (SD)	118.2 (12.4)	115.5 (11.5)	122.7 (12.4)	<.001
DBP, mm Hg (SD)	71.1 (10.6)	70.0 (10.0)	73.0 (11.4)	.06
MFG score (SD)	10.1 (6.0)	9.6 (5.7)	10.8 (6.5)	.19
Hirsutism	64.4%	65.0%	63.5%	.83
Hyperandrogenemia	58.0%	54.8%	63.2%	.25
Polycystic ovaries	95.4%	97.5%	91.9%	.08
Ovulatory dysfunction	76.7%	70.7%	86.5%	.01
Phenotype A	53.0%	49.2%	59.2%	.17
Phenotype B	4.5%	2.4%	7.9%	.09
Phenotype C	22.5%	28.2%	13.2%	.01
Phenotype D	16.0%	16.9%	14.5%	.65
Laboratory results				
Total testosterone, ng/dL (SD)	47.1 (19.9)	48.1 (19.9)	45.4 (19.9)	.35
Free testosterone, ng/dL (SD)	5.2 (3.7)	4.5 (2.7)	6.5 (4.7)	< .001
Fasting glucose, mg/dL (SD)	93.6 (30.4)	88.0 (15.4)	102.7 (43.8)	< .001
Fasting insulin, mg/dL (SD)	17.3 (22.2)	12.5 (18.3)	25.3 (25.6)	< .001
HOMA-IR score (SD)	4.5 (6.9)	2.9 (4.8)	7.0 (8.7)	< .001
Triglycerides, mg/dL (SD)	100.3 (60.4)	82.4 (48.7)	126.2 (66.4)	< .001
HDL, mg/dL (SD)	56.8 (15.7)	61.4 (14.6)	49.5 (14.6)	< .001
CRP, mg/L (SD)	4.0 (5.1)	2.3 (3.5)	6.7 (5.1)	< .001
AMH, ng/mL (SD)	7.8 (5.9)	8.9 (6.0)	5.9 (5.3)	< .001
Current antidepressant use				
Overall	14.0%	8.9%	22.4%	.01
SSRIs	9.0%	5.7%	14.5%	.03
SNRIs	2.5%	0.9%	5.3%	.05
Atypical	3.0%	3.2%	2.6%	.81
TCA	0.5%	0.0%	1.3%	.20

Data indicate mean (SD) or %. *P* values determined by *t* tests or Fisher's exact tests, as appropriate. CRP = C-reactive protein, DBP = diastolic blood pressure, HDL = high-density lipoproteins, HOMA-IR = homeostatic model assessment of insulin resistance, MFG = modified Ferriman-Gallwey, OSA = obstructive sleep apnea, PHQ-9 = Patient Health Questionnaire-9, SBP = systolic blood pressure, SD = standard deviation, SNRI = serotonin-norepinephrine reuptake inhibitor, SSRI = selective serotonin reuptake inhibitor, TCA = tricyclic antidepressant.

can lead to psychiatric deterioration.[27] The accompanied hypoxemia also causes sympathetic hyperactivity, and increased reactive oxygen species and inflammation, which are known to contribute to the pathophysiology of depression and anxiety. On the other hand, psychiatric disorders can also lead to oxidative stress and inflammation, which, along with neurotransmitter imbalances, can alter neurobiological and endocrine function to increase OSA risk.[28] Consistent with these proposed

Figure 1—PHQ-9 and GAD-7 scores stratified by OSA risk.



GAD-7 = Generalized Anxiety Disorder-7, OSA = obstructive sleep apnea, PHQ-9 = Patient Health Questionnaire-9. Filled circles represent outliers.

mechanisms, significantly higher levels of CRP were observed in both those who were high risk for OSA and those with moderate/severe depression symptoms in this study.

This study contributes to the small body of existing literature on OSA in women with PCOS and is the largest study to our knowledge to specifically address the relationship between OSA risk and psychiatric comorbidities in this population. Additional strengths of this study include the systematic characterization of patients seen in our multidisciplinary PCOS clinic, allowing us to adjust for potential confounders of the observed relationship.

Several limitations of this study should also be acknowledged. We utilized self-administered questionnaires to assess OSA risk, as well as depression and anxiety symptoms. Formal assessment of OSA by polysomnography and depression and anxiety by clinical interview should be considered in future investigations. Furthermore, it is important to recognize that the Berlin questionnaire has not previously been proven reliable for identifying OSA specifically in women with PCOS, as the pathophysiology and manifestations of OSA in this population may differ from that of the general population.

This initial exploration highlights the need for further investigation. Future directions should include confirmation of the association in larger studies in which OSA, depression, and anxiety have all been clinically confirmed. Furthermore, longitudinal studies would also help to better understand the intricacies of the bidirectional relationship.

Perhaps the most clinically impactful aspect of further study is to understand the impact of OSA treatment on depression and anxiety in women with PCOS. Evidence supports that continuous positive airway pressure treatment improves depression symptoms in the general population,[12] and that the improvement is greater in the setting of elevated baseline depression symptoms[13] and continuous positive airway pressure adherence.[10] In addition to symptomatic improvement, successful OSA treatment may ultimately help to clarify symptoms due to OSA as opposed to primary psychiatric disorders, and potentially decrease the need for psychotropic medications.[29]

The impact of depression on the overall well-being of women with PCOS is significant. Unfortunately, prior studies evaluating the effect of PCOS-related treatments on depression have shown no or some improvement in symptoms.[21] If treatment of OSA is shown to improve mental health outcomes in women with PCOS, it would expand the arsenal of available tools to use when OSA and depression are comorbid.

In conclusion, among those with PCOS, women at high risk of OSA experienced elevated levels of depression and anxiety symptoms compared with those at low risk for OSA, independent of the effects of age, BMI, hyperandrogenism, and insulin resistance. The complex comorbidities of

Table 2—Logistic regressions examining the impact of OSA risk on moderate/severe depressive symptoms.

	Univariate		Multivariate Model 1		Multivariate Model 2	
	OR (95% CI)	*P*	aOR (95% CI)	*P*	aOR (95% CI)	*P*
High-risk OSA	3.19 (1.76–5.78)	< .01	3.06 (1.36–6.88)	.01	2.76 (1.15–6.63)	.02
Age	0.98 (0.93–1.02)	.32	0.95 (0.91–1.01)	.08	0.93 (0.88–0.99)	.03
BMI	1.04 (1.00–1.07)	.03	0.99 (0.95–1.04)	.78	0.95 (0.89–1.02)	.15
Free testosterone	1.03 (0.95–1.11)	.45	0.98 (0.90–1.07)	.68	0.97 (0.88–1.08)	.58
HOMA-IR	1.03 (0.99–1.08)	.15	1.01 (0.95–1.06)	.79	1.03 (0.97–1.11)	.31
MFG score	1.05 (1.01–1.11)	.02			1.05 (0.98–1.12)	.14
Triglycerides	1.01 (1.00–1.01)	.02			1.00 (1.00–1.01	.29
HDL	0.98 (0.96–1.00)	.02			1.02 (0.99–1.05)	.28
CRP	1.07 (1.00–1.14)	.05			1.07 (0.98–.16)	.15

ORs and aORs of moderate/severe depressive symptoms (PHQ-9 score ≥ 10). Model 1 adjusted for age, BMI, free testosterone, and HOMA-IR. Model 2 adjusted for MFG score, triglycerides, HDL, and CRP in addition to the variables adjusted for in model 1. aOR = adjusted odds ratio, BMI = body mass index, CI = confidence interval, CRP = C-reactive protein, HDL = high-density lipoproteins, HOMA-IR = homeostatic model assessment of insulin resistance, MFG = modified Ferriman-Gallwey, OR = odds ratio, OSA = obstructive sleep apnea.

Table 3—Logistic regressions examining the impact of OSA risk on moderate anxiety symptoms (GAD-7 score ≥ 10).

	Univariate		Multivariate Model 1		Multivariate Model 2	
	OR (95% CI)	*P*	aOR (95% CI)	*P*	aOR (95% CI)	*P*
High-risk OSA	2.49 (1.34–4.64)	<.01	2.39 (1.03–5.59)	.04	2.53 (1.05–6.09)	.04
Age	1.0 (0.95–1.05)	.95	0.97 (0.92–1.03)	.30	0.96 (0.91–1.02)	.21
BMI	1.04 (1.00–1.07)	.04	1.02 (0.97–1.08)	.49	1.00 (0.95–1.06)	.95
Free testosterone	1.00 (0.93–1.10)	.88	0.95 (0.86–1.05)	.32	0.93 (0.83–1.04)	.21
HOMA-IR	1.03 (0.99–1.07)	.18	1.00 (0.94–1.05)	.86	1.00 (0.95–1.06)	.92
MFG score	1.08 (1.03–1.14)	<.01			1.08 (1.01–1.14)	.01

ORs and aORs of moderate/severe anxiety symptoms (GAD-7 score ≥ 10). Model 1 adjusted for age, BMI, free testosterone, and HOMA-IR. Model 2 adjusted for MFG score in addition to the variables adjusted for in model 1. BMI = body mass index, CI = confidence interval, CRP = C-reactive protein, GAD-7 = Generalized Anxiety Disorder-7, HDL = high-density lipoproteins; HOMA-IR = homeostatic model assessment of insulin resistance, MFG = modified Ferriman-Gallwey, OSA = obstructive sleep apnea.

OSA and psychiatric conditions in women with PCOS allow for multiple targets of intervention. Routine OSA screening in women with PCOS should be undertaken, particularly in the setting of existing depression and anxiety. Referral for OSA diagnosis and treatment may have added psychiatric benefits in this population.

ABBREVIATIONS

AMH, anti-müllerian hormone
BMI, body mass index
CRP, C-reactive protein
GAD-7, Generalized Anxiety Disorder-7
HOMA-IR, homeostatic model assessment for insulin resistance
MFG, modified Ferriman-Gallwey
OSA, obstructive sleep apnea
PCOS, polycystic ovary disease
PHQ-9, Patient Health Questionnaire-9

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