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Original article

Examining the construct of depression in obstructive sleep apnea syndrome

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Abstract

Background and purpose: Depression is commonly reported by patients suffering from Obstructive Sleep Apnea Hypopnea Syndrome (OSAS). We used the factor structure of the Beck Depression Inventory–2nd edition (BDI-II) to examine the unique contributions of OSAS severity and obesity to depressive symptoms in OSAS. We predicted that the Somatic and Cognitive dimensions of the BDI-II would be more strongly associated with apnea severity and obesity, respectively.

Patients and methods: Ninety-three moderate to severe OSAS patients (61 men, 32 women) were seen for psychological testing prior to initiating CPAP treatment. They completed the BDI-II and Epworth Sleepiness Scale (ESS). Measures of apnea severity (RDI, percent of sleep time below 90% oxygen saturation) and BMI were also collected.

Results: RDI was significantly related to BMI and BDI-II total score, but not to ESS score. BMI was related to BDI-II total score and ESS score. Partial correlations indicated that RDI was independently related to the Somatic dimension on the BDI-II. By contrast, BMI was uniquely associated with the Cognitive dimension. Although there was no difference in depression scores between men and women, the relationship between the Somatic factor and apnea severity was significant in men, whereas obesity and the Cognitive factor were significantly associated in women.

Conclusions: OSAS severity and obesity contribute differentially to symptoms of depression in OSAS. In addition, symptoms of depression in OSAS manifest differently in men than in women.

Keywords: Sleep apnea; Depression; Obesity; Sleepiness

1. Introduction

Depressive symptoms are widely regarded as typical clinical sequelae of Obstructive Sleep Apnea Syndrome (OSAS) [1–3]. Most case–control studies have reported increased prevalence rates of depression in OSAS compared to controls [4–7], but other studies indicate no differences [8–10]. Further, the relationship between OSAS and depressive symptoms may be moderated by factors such as gender and OSAS severity [11]. Many studies, however, have been limited by varying sample characteristics, small sample sizes, and their choice of measures to assess depressive symptomatology.

OSAS can lead to feelings of fatigue, tiredness, lack of energy, and irritability. These somatic symptoms are also hallmarks of depression [11]. Thus, one possibility for elevated depression scores in OSAS patients is that they may frequently endorse such items on depression scales. Rather than being related to a distinct psychiatric disorder, however, affirmative responses may more accurately reflect the consequences of apnea severity. Indeed, this view that depression is largely an epiphenomenon of OSAS has led some to conclude that the relationship should be

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conceptualized as a mood disorder secondary to a medical disorder [12]. Support for this hypothesis comes largely from studies showing reduced depression following CPAP treatment [13–16], even when treatment adherence is poor [17]. Improvement in depression following CPAP therapy is not a universal finding, however, [18] and treatment studies with short-term follow-ups have found that symptomatic improvement may reflect a placebo-response [19,20].

An alternative explanation is that the relationship between OSAS and depression is indirect, mediated by a correlate of OSAS, such as obesity. Obesity is the strongest risk factor for the development of OSAS [21,22]. In morbidly obese patients (BMI $\geq$ 40 kg/m^2), the rates of OSAS range from 69 to 98% [23,24]. One study found that a 10% increase in body weight increased the relative risk of developing moderate to severe OSAS six-fold [25]. Moreover, several studies have shown an increased prevalence of depression among obese subjects even without consideration for the presence of OSAS [26–28]. One theory to explain this relationship suggests that obese individuals suffer body image dissatisfaction, discrimination, guilt from past failures to lose weight, and psychosocial distress [29]. Moreover, body image dissatisfaction has been shown to mediate partially the relationship between obesity and depression [30,31]. Experimental studies also support this view, showing that changes in body image associated with significant weight loss are associated with significant reductions in depressed mood [32]. These studies suggest that obese patients may endorse a different, more cognitive, aspect of depression than the more fatigue-related, somatic aspect that is likely associated with sleep problems.

The primary aim of this study was to determine the degree to which apnea severity and obesity independently contribute to the relationship between depressive symptoms and OSAS. We examined depression using the Beck Depression Inventory–2nd edition (BDI-II), a measure designed to detect depressive symptoms in an outpatient population [33]. We specifically chose the BDI-II because of its consistent use in previous studies and its factor structure. The BDI-II can be divided into two primary factors. One factor measures depressive symptoms that are predominantly associated with the somatic dimension of depression (Somatic factor), including items measuring apathy, loss of energy, and irritability. The second factor is associated with the cognitive dimension of depression (Cognitive factor), including items measuring pessimism, feelings of failure, and self-dislike. A secondary aim of this study was to examine the degree to which the above relationships are moderated by gender. We hypothesized that the interrelatedness between OSAS severity, sleepiness, depression, and obesity would be high. We also expected that the Somatic dimension would be more strongly associated with apnea severity independent of obesity, whereas the Cognitive dimension would be more strongly associated with obesity independent of apnea severity.

2. Methods

2.1. Participants

Patients between 25 and 85 years of age with OSAS were recruited from the Lifespan Sleep Disorders Center as part of a large-scale study investigating the effects of CPAP adherence on psychological functioning. Two hundred sixty-two consecutive patients were screened for initial eligibility by their treating physician between 9/1/2002 and 12/1/2003. Initial eligibility included the following: (a) age between 25 and 85 years; (b) diagnosed with OSAS; (c) fluent in English; (d) no other diagnosable sleep disorder; (e) no serious medical disorder that would interfere with treatment (e.g. kidney failure, neurological illness, or major psychiatric condition); and (f) no previous treatment with CPAP. One hundred thirty patients met these initial criteria and were referred to the project coordinator for a formal request to participate in the study. After further explanation, 37 participants declined invitation into the study, leaving 93 active participants (61 men and 32 women). Only pre-treatment, baseline data are presented for the purposes of this study. Participants who reported major depression that was treated prior to the diagnosis of sleep apnea ($N=22$) were included in the study. It was intended that patients with untreated depression would be excluded from the study and referred to receive mental health treatment. This condition was, however, not encountered. All participants provided informed consent before entering into the study. This study was approved by the Institutional Review Boards at Rhode Island Hospital and Brown University.

2.2. Procedures and measures

All participants were seen for psychological testing prior to beginning treatment for OSAS. Testing was completed by trained research assistants in our laboratory who were blind to apnea severity.

OSAS was diagnosed with a full night of in-laboratory, clinical polysomnography (PSG) evaluating the following physiological and respiratory variables: central and occipital EEG, oblique EOG, submental and tibialis EMG activity, EKG, nasal and oral airflow via nasal pressure transducer and thermister, thoracic and abdominal excursions with peizo belts and continuous oxygen saturation. Sleep stage was scored by trained technicians using standard criteria [34]. Apneas and hypopneas were scored using recommended guidelines [35]. Apnea severity measures included the Respiratory Disturbance Index (RDI), an index of apneas and hypopneas per hour of sleep, and the percentage of total sleep time spent below 90% blood oxygenation during the overnight PSG (Sa90). Measurements of height and weight were recorded and body mass index (BMI) was derived (kg/m^2). Subjective sleepiness was measured using the Epworth Sleepiness Scale (ESS) [36,37]. This widely used 8-item self-report scale requires participants to rate their

Table 1
Item content by BDI-II factor

Somatic dimension	Cognitive dimension
Item 4: loss of pleasure	Item 1: sadness
Item 10: crying	Item 2: pessimism
Item 11: agitation	Item 3: past failure
Item 12: loss of interest	Item 5: guilty feelings
Item 13: indecisiveness	Item 6: punishment feelings
Item 15: loss of energy	Item 7: self-dislike
Item 16: changes in sleeping pattern	Item 8: self-criticalness
Item 17: irritability	Item 9: suicidal thoughts
Item 18: changes in appetite	Item 14: worthlessness
Item 19: concentration difficulty	
Item 20: tiredness or fatigue	
Item 21: loss of interest in sex	

likelihood of dozing off in various situations on a 0 (would never doze) to 3 (high chance of dozing) scale. Scores range from 0 to 24, with scores greater than 10 indicative of significant daytime sleepiness. The ESS is psychometrically sound with adequate reliability [37,38]. Depressive symptoms were assessed using the BDI-II, a 21-item self-report scale that evaluates symptoms of depression over the past week. Respondents rate the severity of each symptom on a 0 (absent) to 3 (most severe) scale. Total scores range from 0 to 63. The BDI-II has demonstrated adequate reliability and validity for assessing the construct of depression [33]. The BDI-II was separated into the two factors based on the factor analysis presented in the published manual: a Somatic factor and a Cognitive factor [33]. The items related to each resulting factor are presented in Table 1. Items for each factor were summed to provide Somatic and Cognitive factor scores that were entered along with the total score into the statistical analyses described below.

2.3. Analyses

Data analyses were conducted using SPSS 11.5 (SPSS, Inc., Chicago, IL). Significance level was set at 0.05 for all analyses. Pearson product moment correlation coefficients were used to examine the relationships between apnea severity, obesity, subjective sleepiness, and depression. One-tailed partial correlation coefficients were employed to address specific hypotheses about the relative contribution of either apnea severity (RDI) or obesity (BMI) to the constructs of depression. Independent *t*-tests were employed to examine gender differences in BDI scores. The relative contribution of gender to these specific hypotheses was also examined using partial correlations.

3. Results

3.1. Sample characteristics

Table 2 shows the demographic information for the 93 study participants. In general, participants were middle-aged men with severe OSAS, clinically significant subjective sleepiness, high BMI and minimal depression. Table 3 shows the number and percentage of participants reaching BDI-II cutoffs for various levels of depression at baseline.

3.2. Effects of apnea severity and obesity on depression

Table 4 shows the relationships (Pearson product moment correlations) between the OSAS severity measures (RDI and Sa90), BMI, total depression score on the BDI-II, and subjective sleepiness on the ESS. RDI was significantly related to BMI and to total BDI-II score, but not to subjective sleepiness. Percent time below 90% oxygen saturation was related to RDI and BDI-II score. In addition to being related to RDI, BMI was related to both the total BDI-II score and to subjective sleepiness.

We employed partial correlation coefficients to delineate the degree to which apnea severity (RDI) and obesity (BMI) contributed independently to different aspects of depression. Fig. 1 shows the partial correlations between RDI and BMI and the two factors of the BDI-II (Somatic factor, and Cognitive factor) using a one-tailed significance criterion to test our directional hypotheses ($P < .05$). Each correlation for RDI represents its relationship with the dependent variable after controlling for the variance in RDI associated with BMI. Similarly, BMI correlations control for the variance associated with RDI.

RDI was significantly related to the Somatic factor of the BDI-II independently from BMI, but was not related to the Cognitive dimension. BMI was significantly related to the Cognitive dimension independently from RDI, but not to the Somatic factor.

3.3. Gender as a moderator

Table 5 shows the comparison between men and women on the total depression score of the BDI-II, the Somatic factor score, and the Cognitive factor score. Men and women did not differ significantly on any of the depression scores. In Table 6, we examined the relative effects of gender on the relationships between apnea severity and depression. There was no correlations between obesity and depression in men. Apnea severity, however, did correlate with the total depression score ($P < .05$) and with the Somatic factor ($P < .05$) in men, independent of obesity. This relationship did not exist for the Cognitive factor score ($P > .05$). Women showed no relationship between apnea severity and depression, independent of obesity ($P > .05$). They did, however, show a significant relationship between obesity and the Cognitive factor score on the depression scale ($P < 0.05$), independent of apnea severity. There was no relationship between obesity and the Somatic factor score independent of apnea severity in women.

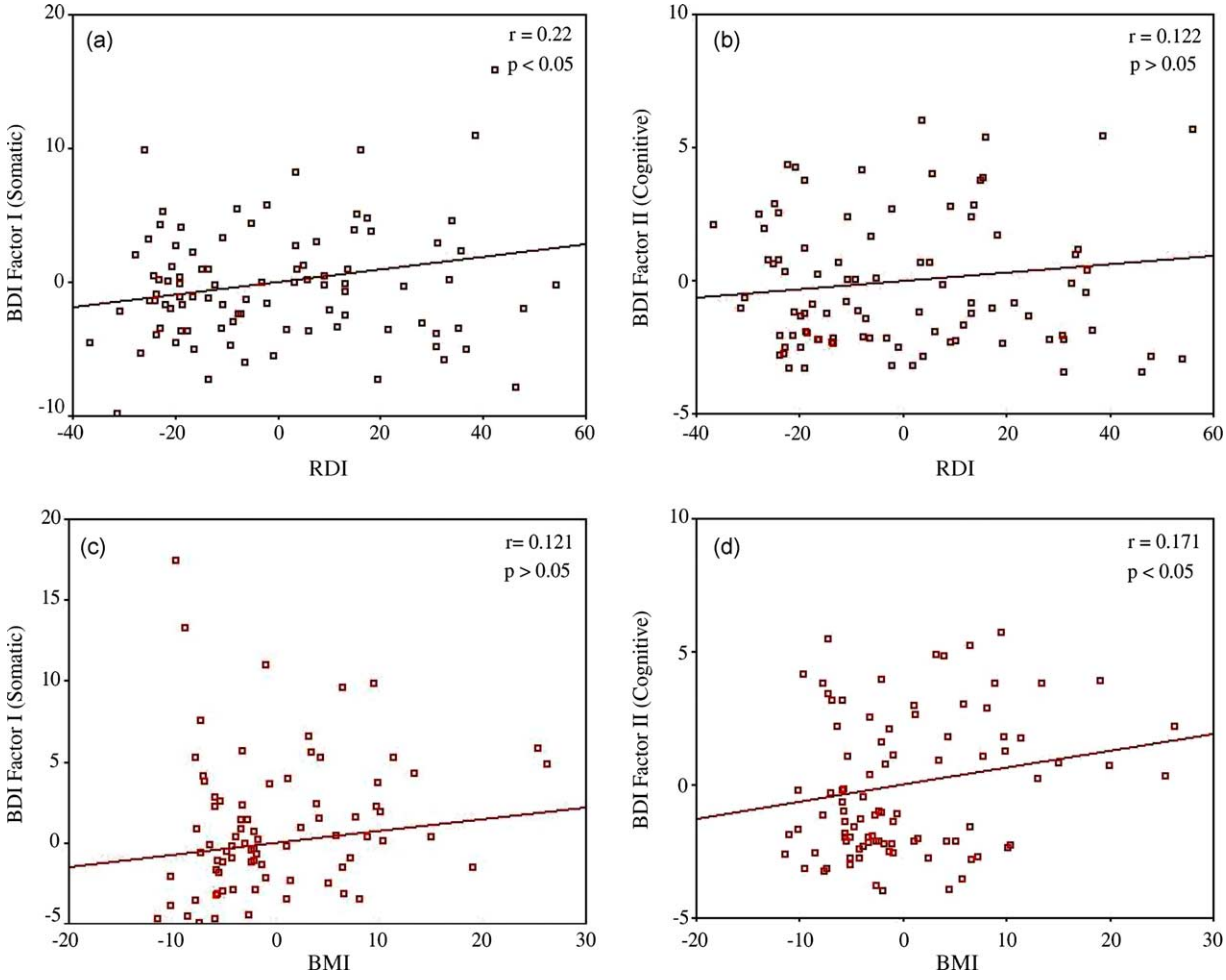

Fig. 1. Partial correlations between apnea severity or obesity and factor scores on the BDI-II. Note: (a) Partial correlation between RDI and Factor I (somatic); (b) Partial correlation between RDI and Factor II (cognitive); (c) Partial correlation between BMI and Factor I (somatic); (d) Partial correlation between Factor II (cognitive).

4. Discussion

Depression is commonly reported as one of the daytime sequelae of OSAS, and many studies have found increased rates of depression among OSAS patients [1–3]. The authors of some of these studies make mention of the likely complex nature of this relationship, highlighting multiple potential etiologies. In this study, we examined two likely contributors to the increased report of depressive symptoms in OSAS, apnea severity and obesity, and the moderating effects of gender on these relationships. We designed the analyses for this study to identify the degree to which depression, in OSAS, could be separated by these potential etiological factors, both of which have been independently related to depression and OSAS in previous studies [12,26–28]. We employed the natural factor structure of the BDI-II to determine whether or not two logical dimensions of depression would relate differently to these etiologies.

Although two-thirds of the participants fell below any cutoff for depression, a full one-third scored in the mild to severe range of BDI-II depression. The percentages of

Table 2
Demographics for study sample

Variable	Mean	SD	Min	Max
Age (yrs)	52.2	11.1	26	81
Education (yrs)	14.3	3.2	6	20
RDI (number/h)	41.5	24.0	11.2	99.9
SA90 (%)	22.6	27.6	0	100
BMI (kg/m^2)	34.8	8.4	23.4	65.4
Epworth (/24)	12.1	5.0	0	23
BDI-II (/63)	11.2	7.4	1	42

Table 3
Participants meeting published cutoffs for various levels of depression on the BDI-II

Level of depression	Number (%) of sample
Normal–minimal (0–13)	62 (66.7)
Mild (14–19)	22 (23.6)
Moderate (20–28)	5 (5.4)
Severe (29 or greater)	4 (4.3)

Table 4
Pearson correlations between apnea severity, obesity, sleepiness, and depression

	RDI	Sa90[a]	BMI	Epworth	BDI-II total score
RDI	–	.563***	.328***	.146	.255**
Sa90		–	.118	.106	.219*
BMI			–	.272**	.223*
Epworth				–	.301**

*$P<.05$, **$P<.01$, ***$P<.001$.
[a] Minutes spent below 90% oxygenation during diagnostic PSG.

patients falling in the mild, moderate, and severe categories of depression in our sample were comparable to those reported in at least one other study using the BDI in patients with OSAS (36% depressed) [17]. OSAS severity, as measured by the RDI, was related to depression and obesity in our study, with more severe participants showing a greater tendency toward obesity and the endorsement of depressive symptomatology. Obesity itself was also related to depressive symptoms, with more obese participants scoring higher on the BDI-II. The relationships between RDI and obesity, RDI and depression, and obesity and depression have all been reported in the literature [4,21,28]. Our finding that patients reporting higher daytime sleepiness were more likely to report higher depression has also been reported previously [39].

Our primary aim in this study was to separate the relative contributions of apnea severity and obesity to the prediction of different aspects of depressive symptoms, somatic and cognitive. To do this we first separated the shared variance between our two independent variables of interest, RDI and BMI. After removing variance associated with obesity, RDI was only related to the Somatic dimension of depression as we had hypothesized. This suggests that the primary load in the association between apnea severity and depression comes from an association with the subset of depressive symptoms that are somatic in nature. Although it has been suggested that having a chronic medical condition like OSAS would result in greater endorsement of cognitively-laden symptoms of depression [17], we did not find a linear relationship between OSAS severity and the Cognitive factor. Obesity, on the other hand, has been associated with symptoms of self-dislike and guilt over past failures [29–31], items more consistently measured by the Cognitive factor of the BDI-II. In support of this hypothesis, BMI was related to the Cognitive and not the Somatic factor in this study. It should be noted that although statistically supportive of our findings, the difference between the significant relationships and those not significant was relatively small.

Table 5
Depression scores (means and SDs) by gender

Variable	Men	Women	t	P
BDI-II total score (/63)	10.6 (7.8)	12.3 (6.5)	1.0	ns
Somatic factor score (/36)	8.2 (5.1)	9.3 (4.5)	1.0	ns
Cognitive factors score (/27)	2.4 (3.2)	3.0 (2.8)	0.86	ns

Table 6
Partial correlations between apnea severity, obesity, and factors on the BDI-II by gender

	Factor I (somatic)		Factor II (cognitive)	
	Men	Women	Men	Women
RDI	.299	.145	.186	.014
	$P=.01$	$P=.22$	$P=.08$	$P=.47$
BMI	.051	.128	.050	.296
	$P=.35$	$P=.25$	$P=.35$	$P=.05$

Correlations and P-values are presented.

One unexpected finding was the absence of an association between sleepiness and apnea severity. This finding, however, is consistent with at least one other large scale study, which found the ESS to be poorly related to an objective measure of daytime sleepiness, the Multiple Sleep Latency Test (MSLT) [40]. Interpretations of the ESS should therefore be made cautiously. However, other questions are raised by our ESS findings. First, ESS was related to BMI, even though it was not related to RDI. This relationship has been seen in other studies reporting that obese patients without OSAS had objectively poorer sleep and shorter sleep onset latencies on MSLT [41]. Therefore, other forms of sleep disturbance may account for higher ESS scores in obese patients. Second, ESS was related to depression in our study. It is possible that some of the correlation between ESS and depression is accounted for by method variance. Self-report measures often correlate with each other, especially when they are taken in the same testing session. There may, however, be other reasons to believe that depression and sleepiness should be related. The relationship could be pulled primarily by the somatic symptoms of depression, which could be related to subjective sleepiness in this sleep-disordered sample. In general, the findings associated with subjective sleepiness in our study raise several questions that call for further investigation into the construct validity of the ESS and its relationship with other subjective and objective measures.

Gender appears to moderate the relationships between apnea severity, obesity, and depression. Men only showed a relationship between apnea severity and somatic complaints associated with depression, independent of obesity. Women, on the other hand, only showed a relationship between obesity and the Cognitive factor of depression, independent of apnea severity. Each of these relationships follows the direction of our original predictions, although we did not expect each relationship to exist primarily in one gender. These findings suggest that men and women with apnea manifest depressive symptoms differently. Pillar et al. [11] found that women with OSAS scored higher on depression and anxiety scales than did men with OSAS. They attributed these findings in part to basic gender differences in

personality, suggesting that women tend to focus more on their symptoms than do men. We additionally found an association between apnea severity and certain aspects of depression (e.g., somatic complaints) in men. Somatic versus cognitive depressive symptoms were not examined in their study. There are several alternative explanations for our findings. It is possible that the somatic aspects of depression are easier for men to identify than are the cognitive aspects. In other words, men may be more reluctant to acknowledge feelings of guilt, sadness, and worthlessness than women or they may indeed not be cognizant of these feelings. It is also possible that the differential societal values regarding body habitus for men and women affected the gender findings on our study. Specifically, men may focus less on their weight, resulting in fewer thoughts of self-criticalness, fewer failures with past attempts to lose weight, and less overall concern about their obesity. Alternatively, women may focus more on their weight, thus, reducing the significance of apnea severity in the prediction of depression in our study. Given these findings, the gender-specific manifestation of depression and the mechanisms underlying these relationships deserves closer attention.

This study highlights the complex nature of the construct of depression as it relates to apnea using only two simple dimensions of depression. There may be other dimensions of depression that are yet to be explored. Future clinical and experimental studies should address the complex nature of this construct in OSAS and the effects of treatment, especially where gender-specific manifestations are concerned. Our study has several limitations. First, based on the nature of our correlational analyses, causation cannot be inferred. Second, the current study also focuses on patients with moderate to severe OSAS and findings cannot be generalized to less severe patients. Third, it could be argued that obesity itself may also contribute to somatic complaints; however, we did not find this association in our sample of OSAS patients (see Fig. 1c). We conclude that obesity and apnea severity differentially contribute to depressive symptoms in a population of patients with moderate to severe obstructive sleep apnea, and that these contributions are moderated by gender.

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