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Obesity and the three-year longitudinal course of bipolar disorder

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Abstract

Objectives—Despite substantial cross-sectional evidence that obesity is associated with increased medical and psychiatric burden in bipolar disorder (BD), few longitudinal studies have examined this topic.

Methods—Subjects with BD (n = 1,600) who completed both Wave 1 and Wave 2 of the National Epidemiologic Survey on Alcohol and Related Conditions were included. Analyses examined the association between obesity at Wave 1, and the subsequent course of BD, and of psychiatric and medical comorbidities, between Wave 1 and Wave 2.

Results—BD subjects with obesity (n = 506; 29.43%), compared to BD subjects without obesity (n = 1,094; 70.57%) were significantly more likely to have a major depressive episode and to receive counseling for depression during follow-up, more likely to report a lifetime suicide attempt, and less likely to develop new onset alcohol use disorders. These differences were no longer significant, however, after controlling for baseline demographic variables. No significant differences in new episodes or treatment of mania/hypomania were observed. After controlling for demographic variables, obese subjects remained significantly more likely to report any new-onset medical condition [odds ratio (OR) = 2.32, 95% confidence interval (CI): 1.63–3.30], and new-onset hypertension (OR = 1.81, 95% CI: 1.16–2.82) and arthritis (OR = 1.64, 95% CI: 1.07–2.52). Obese subjects were significantly more likely to report physician-diagnosed diabetes (OR = 6.98, 95% CI: 4.27–11.40) and hyperlipidemia (OR = 2.32, 95% CI: 1.63–3.30) (assessed in Wave 2 only). The incidence of heart attacks was doubled among obese subjects, although this difference was not statistically significant.

Conclusions—The association between obesity and increased prospective depressive burden appears to be explained by baseline demographic variables. In contrast, obesity independently predicts the accumulation of medical conditions among adults with BD. Treatment of obesity could potentially mitigate the psychiatric and medical burden of BD.

Keywords

bipolar disorder; epidemiologic; longitudinal; obesity

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Bipolar disorder (BD) is a neuropsychiatric disorder that is characterized by a high degree of symptom severity and episode recurrence, and by its frequent association with psychiatric and medical comorbidities (1–3). One comorbidity that has generated substantial interest is obesity. In recent years, clinical and epidemiologic studies have yielded several convergent findings regarding the association between BD and obesity. First, the prevalence of obesity is significantly increased in BD, whether subjects are ascertained from community samples or treatment-seeking samples (4–6). Explanations for this association are numerous, and include the effect of psychotropic medications, excessive carbohydrate consumption, low rate and intensity of exercise, reduced fat oxidation, substance misuse, and maladaptive efforts at self-modulation of mood by over eating (7–13).

Second, it appears that obesity is associated with several proxies for illness severity in BD, including increased number of manic and depressive episodes, increased depressive symptom severity, treatment-resistance, suicidality, and treatment utilization (14–21). A natural question that emerges from these cross-sectional associations is whether obesity is a predictor of prospective illness course in BD.

Recently, we examined the prevalence and correlates of obesity in Wave 1 (2001–2002) of the National Survey on Alcohol and Related Conditions (NESARC), a large representative sample of the United States population (4). We found that obesity is significantly more prevalent among adults with BD as compared to the general population. We also found that obesity is associated with greater depressive severity and increased prevalence of anxiety disorders, but decreased prevalence of past-year substance use disorder (SUD). However, by virtue of the cross-sectional methodology employed, the direction of the observed associations is uncertain.

We therefore set out to examine the hypothesis that obesity at baseline is associated with a more severe longitudinal course of BD, determined prospectively, as defined by greater burden of mood symptoms, new-onset psychiatric and medical comorbidity, and increased functional impairment. We hypothesized that obese subjects with BD, as compared to non-obese subjects with BD, would experience more mood episodes, have higher rates of psychiatric treatment utilization, would more frequently develop comorbid conditions, and would experience greater increases in functional impairment during the three years between Wave 1 and Wave 2 (2004–2005) of the NESARC.

Methods

Sample and procedures

Subjects were identified from among respondents who completed both Wave 1 of the NESARC and Wave 2 interviews three years (36.6 ± 2.62 months) later (22). The NESARC is a representative sample of the United States conducted by the National Institute on Alcoholism and Alcohol Abuse (NIAAA). A detailed description of Wave 1 of NESARC can be found elsewhere (22). Briefly, 43,093 non-institutionalized civilian respondents, 18 years and older, completed face-to-face computer-assisted personal interviews. Blacks, Hispanics, and adults ages 18–24 were oversampled. The overall Wave 1 response rate was 81% (22). After excluding respondents who were ineligible for Wave 2 (e.g., deceased), 86.7% of respondents ($n = 34,653$) were re-interviewed, and sample weights were developed to additionally adjust for Wave 2 non-response. The weighted data were then adjusted to represent the United States civilian population based on the 2000 census. After adjustment, comparisons between Wave 2 respondents and the target population (comprising Wave 2 respondents and eligible non-respondents) indicated that there were no significant differences in terms of a number of baseline (Wave 1) sociodemographic measures or the presence of any lifetime substance, mood, anxiety or personality disorder (23).

Assessments

Psychiatric diagnoses—The NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV version (AUDADIS-IV) was used to generate the diagnoses presented in this report. Consistent with DSM-IV, *primary* AUDADIS-IV diagnoses excluded disorders that are substance-induced or due to general medical conditions. Subjects were considered to have BD if, at Wave 1, they had a lifetime manic or mixed episode that was not secondary to substance use or a medical condition [bipolar I disorder (BD-I)], or lifetime history of at least one hypomanic episode and at least one major depressive episode [bipolar II disorder (BD-II)] (22). This study examines subjects with a *lifetime* diagnosis of BD because selecting solely subjects with 12-month prevalence of BD would introduce a selection bias into the analysis, as not all individuals with BD have mood episodes in a given year.

The anxiety disorders included in the present study are social anxiety disorder, panic disorder, and generalized anxiety disorder (posttraumatic stress disorder was ascertained only in Wave 2). The NESARC provides information regarding daily nicotine use and regarding abuse and dependence of alcohol and illicit drugs. For the purpose of this report, subjects with a lifetime prevalence of abuse or dependence of alcohol or any illicit drugs were considered to have lifetime SUD. The AUDADIS-IV diagnoses of SUD and of mood and anxiety disorders have demonstrated adequate reliability and validity (22, 24, 25). For analyses, subjects were divided into two groups depending on whether or not they were obese at Wave 1.

Obesity—Self-reported height and weight were systematically obtained in NESARC Wave 1. Obesity was defined as having a body mass index [(BMI); weight in kilograms divided by square of height in meters] of $\geq 30 \text{ kg/m}^2$ (26).

Medical diagnoses—Wave 1 and Wave 2 NESARC respondents were asked about the presence of 11 medical conditions in the past year: arthritis, hypertension, gastritis, stomach ulcer, liver cirrhosis, other liver disease, angina, arteriosclerosis, heart attack, tachycardia, and other heart disease. In addition, Wave 2 respondents were asked about the lifetime presence of diabetes, high cholesterol, stroke, and sexually-transmitted infections. Only diagnoses reported to have been made by physicians or health professionals were considered present for the purpose of this study.

Functioning—The NESARC also includes among its measures the Short-Form 12 version 2 (SF-12), a reliable and valid measure of health-related quality of life used in large population surveys that yields component and profile scores assessing various dimensions of physical and mental disability and impairment (27).

Information regarding early neglect, physical abuse, and sexual abuse was introduced in Wave 2.

Statistical analyses

Chi-square tests for contingency tables were computed to compare the distribution of the proportions across groups and regression analyses was used to compare these groups on dimensional measures. Regression analyses for demographic variables with between-group differences in univariate analyses ($p < 0.10$), regression analyses were computed controlling for demographic variables with between-group differences ($p < 0.10$) in univariate analyses. The latter analyses are reported using adjusted ORs. All analyses were conducted SUDAAN to adjust for the design effects of the NESARC.

Results

The prevalence of obesity at Wave 1 among BD subjects in the current study is 29.43%, whereas the prevalence of obesity was 29.82% among all Wave 1 subjects with BD (i.e., including those who did not complete Wave 2). This suggests that the current sample is representative of the overall Wave 1 sample with regard to obesity. Table 1 summarizes the demographic characteristics of obese and non-obese subjects. Between-group differences were noted in race, age, gender, income, and smoking status, which were included in multivariable analyses. Of note, neglect, physical abuse, and sexual abuse were not significantly associated with obesity among subjects with BD. Between-group differences in demographic and clinical variables in Wave 1 of the NESARC have been reported previously (4).

Table 2 summarizes the course of BD for obese and non-obese subjects. Obese subjects were significantly more likely to have a major depressive episode during follow-up (51.13% versus 44.42%, $p = 0.03$), and were significantly more likely to receive counseling/psychotherapy for depression (60.31% versus 50.10%, $p = 0.04$), compared to non-obese subjects. These differences were no longer significant, however, after controlling for baseline demographic variables. Lifetime suicide attempt history was ascertained in Wave 2 only, and was significantly more prevalent among obese (29.78%) versus non-obese (20.90%, $p = 0.03$) subjects. No significant differences in new episodes or treatment of mania/hypomania were observed.

Table 3 summarizes the course of psychiatric and medical comorbidities and functioning for obese and non-obese subjects. Obesity was not significantly associated with new-onset SUD or new-onset anxiety disorders. To examine the possibility of differential associations between specific substances (e.g., cannabis, stimulants) and obesity, substances were examined separately. New-onset alcohol use disorders were less common among obese versus non-obese subjects (11.81% versus 19.24%, respectively; $p = 0.03$). This difference was no longer significant after controlling for baseline demographic variables. Because this topic was not previously examined, Table 4 presents baseline associations between specific substances and obesity among BD subjects in NESARC. Obese BD subjects were significantly less likely than non-obese BD subjects to have lifetime cannabis use disorders at Wave 1 (24.29% versus 30.64%, respectively; $p = 0.047$). This difference was no longer significant after controlling for baseline demographic variables.

Obesity was significantly associated with new-onset medical conditions. After controlling for baseline race, age, gender, income, and smoking status, obese subjects remained significantly more likely to report any new-onset medical condition (OR = 2.32, 95% CI: 1.63–3.30). In terms of specific medical conditions, obese subjects were significantly more likely to develop new-onset hypertension (OR = 1.81, 95% CI: 1.16–2.82) and arthritis (OR = 1.64, 95% CI: 1.07–2.52). Obese subjects were significantly more likely to report lifetime physician-diagnosed diabetes (OR = 6.98, 95% CI: 4.27–11.40), and hyperlipidemia (OR = 2.32, 95% CI: 1.63–3.30). The incidence of heart attacks was doubled among obese subjects (2.34% versus 0.99%), however this study was not adequately powered to detect as statistically significant differences in infrequent events that may be clinically significant. Controlling for baseline demographic characteristics, obese subjects were significantly more likely to have a regular provider of medical care than non-obese subjects (OR = 1.40, 95% CI: 1.03–1.89). We therefore conducted sensitivity analyses regarding the development of new-onset medical conditions, controlling for having a regular provider, and findings remained unchanged. In terms of functioning, obese subjects demonstrated significantly greater reductions in the general health subscale of the SF-12 (controlling for demographic variables), but not other subscales, as compared to non-obese subjects.

Exploratory analyses examined for changes in obesity status between Wave 1 and Wave 2. A relatively small percentage of subjects shifted from the non-obese to obese category (8.39%) or from the obese to non-obese category (3.58%).

Discussion

This study examines the association of obesity with the prospectively-ascertained longitudinal course of BD in a large representative sample of adults. As expected, obesity predicted the accumulation of medical comorbidities. Although obesity predicted significantly greater likelihood of new-onset depressive episodes, greater likelihood of receiving counseling for those episodes, and lower likelihood of new-onset alcohol use disorders, these associations were attenuated when controlling for demographic variables. To our knowledge, this is the first epidemiologic study that focuses on the association of obesity with the prospectively-determined longitudinal course of BD, and the first study to examine longitudinally the impact of obesity on comorbidity in BD.

These findings add to an exceedingly limited literature on the impact of obesity on the longitudinal course of BD. Fagiolini and colleagues (18), using a clinical sample, previously reported that obese subjects with BD-I had significantly increased risk of mood episode recurrence, and significantly shorter time to recurrence than non-obese subjects with BD-I. Examination of the mood polarities separately indicated that this association was accounted for by greater propensity toward depressive recurrence but not manic/mixed recurrence. The association between BMI and BD-I outcomes remained significant after controlling for previous number of episodes and baseline depressive symptoms; however, demographic characteristics were not included as covariates. This is noteworthy in light of the fact that demographic correlates of obesity in that study differed from those in the present study. In the study by Fagiolini and colleagues, obesity was not associated with race or gender. In the present study, obesity is associated with Black race and female gender. Further, Kemp and colleagues (28) found that obesity was a significant negative predictor of remission of depressive symptoms among 225 adults with rapid-cycling BD enrolled in studies of lithium and valproate treatment. Finally, Bond and colleagues (29) found that the likelihood of mood episode recurrence was similar among overweight/obese and non-overweight/obese subjects with first-episode mania followed prospectively for 12 months.

Previous longitudinal findings from NESARC indicate that obesity at Wave 1 was modestly but significantly associated with new-onset major depressive disorder (OR = 1.2, 95% CI: 0.02–1.51) among women (30). Indeed, a recent meta-analysis of longitudinal studies provides robust evidence that obesity increases the risk of incident depression (31). Interestingly, recent neuroimaging findings indicate that obesity among adults with first-episode mania is associated with decreased white matter volume and decreased temporal lobe volume (32). Those findings, evident essentially at the onset of BD, suggest that the association between obesity and increased severity of BD may be in part explained by proximal neurobiological factors, rather than being solely the long-term consequence of a pernicious variant of the illness.

The question arises as to why the association of obesity with proxies for BD severity were somewhat less pronounced in the current study than in previous cross-sectional, retrospective studies, including our previous findings from Wave 1 of NESARC (4). Fagiolini and colleagues (17) found that most weight gain occurred during acute treatment as opposed to during maintenance treatment of BD, that the amount of increase in BMI during acute treatment was positively associated with baseline depressive symptom severity and negatively associated with manic symptom severity, and that higher BMI at study intake was associated with lesser weight gain during treatment. Taken together, these findings

suggest the possibility that the association between obesity and increased burden of BD is strongest during the process of significant weight gain and that obese subjects are less likely to experience significant weight gain. The present study specifically examined subjects who are already obese, and it is possible that some of the association between obesity and increased illness severity is especially evident earlier in the illness.

Obese subjects demonstrated greater likelihood of incident medical problems, particularly hypertension and arthritis, and greater reductions in general health. Although not statistically significant, the risk of heart attack during follow-up was doubled among obese subjects (2.34% versus 0.99%). Concerns about the medical burden of BD are increasingly salient as convergent data accumulates (6, 33–37). Recently, large-scale treatment studies have demonstrated that integrated treatment focusing simultaneously on psychiatric and medical outcomes may offer substantial advantages over usual care, which is often fragmented (38, 39). Preliminary treatment models for adults with BD specifically have been articulated, with promising initial findings (40, 41).

The limitations of this study are important to consider when interpreting these findings. First, although this study provides categorical data regarding mood episodes during the follow-up interval, more fine-grained data regarding the weekly symptomatic burden of BD cannot be gleaned from the AUDADIS as they can from instruments such as the Longitudinal Interval Follow-up Evaluation (3). Because sub-threshold symptoms account for much of the burden of BD, this study does not rule out the possibility that obesity is associated with greater mood symptom burden over time. Second, obesity was determined based on BMI, and information regarding central or visceral adiposity was not available. Previous findings suggest that visceral adiposity may be more strongly associated with mood symptoms than overall obesity (42). Third, the groups examined in this study were determined based on obesity status at intake. Subjects who developed obesity, or who became non-obese, during follow-up may have contributed to an under-estimation of the impact of obesity on the variables examined. Fourth, information regarding quality of medical and psychiatric care was not available in the NESARC. Because obese subjects were significantly more likely to have a regular physician, it is possible that they also received better medical and/or psychiatric care than non-obese subjects, which may have attenuated the between-group differences. This could also potentially explain in part the greater likelihood of receiving new medical diagnoses during follow-up. However, sensitivity analyses controlling for this variable suggest otherwise. Finally, NESARC did not include information regarding specific medications. Therefore we could not examine to what degree different medication regimens may have confounded the observed findings. For example, more severely ill subjects may have been treated with more medications associated with weight gain, which may have confounded the univariate association between obesity and depressive burden.

In summary, the association between obesity and increased prospective depressive burden appears to be explained by baseline demographic variables. In contrast, obesity independently predicts the accumulation of medical conditions among adults with BD. Treatment of obesity could potentially mitigate the psychiatric and medical burden of BD. Increasing evidence suggests that integrating models of care focusing simultaneously on psychiatric and medical outcomes may optimize global outcomes in BD.

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Table 1

Demographic characteristics

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value
	%	SE	%	SE			
Personal income, US\$					2.32	3	0.0840
0–19,999	63.79	2.54	56.39	1.84			
20,000–34,999	20.35	2.16	23.03	1.46			
35,000–69,999	12.91	1.59	16.76	1.29			
70,000	2.95	0.85	3.83	0.71			
Race					2.80	4	0.0328
White	70.27	2.77	71.33	2.05			
Black	15.44	1.87	10.27	0.99			
Native American	3.04	1.02	4.60	0.92			
Asian	1.38	0.66	3.44	1.05			
Hispanic	9.87	1.77	10.36	1.40			
Age, years					9.39	3	< 0.0001
18–29	26.91	2.41	39.29	2.18			
30–44	35.46	2.56	35.07	1.87			
45–64	33.65	2.31	21.66	1.40			
65+	3.98	0.92	3.98	0.62			
Sex					10.96	1	0.0015
Male	37.38	2.51	47.44	1.67			
Female	62.62	2.51	52.56	1.67			
Employment status (Wave 1/Wave 2)					2.28	3	0.0871
Employed/Employed	49.32	2.74	56.10	1.87			
Employed/Unemployed	9.50	1.53	9.97	1.18			
Unemployed/Employed	10.81	1.93	11.06	1.13			
Unemployed/Unemployed	30.37	2.70	22.87	1.60			
Marital Status (Wave 1/Wave 2)					1.19	3	0.3197

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value
	%	SE	%	SE			
Married/Married	50.09	2.58	44.73	1.89			
Married/Not married	3.04	0.90	4.56	0.80			
Not married/Married	8.30	1.57	7.49	0.88			
Not married/Not married	38.57	2.47	43.22	1.70			
Daily smoker (Wave 1/Wave 2)					5.88	3	0.0013
Smoker/Smoker	32.44	2.53	43.70	1.96			
Smoker/Non-smoker	24.86	2.18	16.64	1.33			
Non-smoker/Smoker	1.59	0.58	1.82	0.55			
Non-smoker/Non-smoker	41.11	2.70	37.84	1.80			
Early physical abuse (Wave 2)	12.59	1.80	11.63	1.07	0.23	1	0.6311
Early neglect (Wave 2)	11.54	1.75	9.39	0.88	1.22	1	0.2734
Early sexual abuse (Wave 2)	22.54	2.22	18.06	1.47	3.02	1	0.0868

BD = bipolar disorder; SE = standard error.

Table 2

Bipolar disorder characteristics

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value	AOR ^a	95% CI	$(\chi^2$ or F) ^a	df ^a	p-value ^a
	% or Mean	SE	% or Mean	SE								
Any episodes of depression, mania, or hypomania (since Wave 1)	61.50	2.35	55.05	1.78	4.87	1	0.0308	1.22	0.95–1.55			
Depression (since Wave 1)												
Any episodes	51.13	2.54	44.42	1.85	4.72	1	0.0335	1.19	0.92–1.54			
No. of episodes	3.19	0.26	3.06	0.18	0.17	1	0.6842			0.23	1	0.6295
Length of depression (weeks) ^b	9.87	1.17	11.62	1.22	0.99	1	0.3241			0.45	1	0.5046
Attempted suicide	6.52	1.70	7.91	1.62	0.36	1	0.5483	0.89	0.45–1.75			
Suicidal ideation	33.08	3.76	30.30	2.39	0.39	1	0.5361	1.13	0.74–1.74			
Recovery from depression	71.72	6.01	80.45	4.46	1.58	1	0.2132	0.65	0.29–1.43			
Counselor for depression ^c	60.31	3.70	50.10	3.19	4.22	1	0.0440	1.39	0.92–2.11			
Hospitalized for depression ^c	15.54	2.88	12.54	2.36	0.65	1	0.4221	1.20	0.64–2.25			
Emergency room for depression ^c	18.61	3.58	12.45	2.18	2.34	1	0.1309	1.46	0.77–2.77			
Medication for depression ^c	59.31	4.05	52.45	3.07	1.83	1	0.1806	1.22	0.79–1.89			
Sought any treatment ^c	69.11	3.88	60.24	2.87	3.19	1	0.0786	1.39	0.90–2.17			
Lifetime suicide attempt ^d	29.78	3.39	20.90	2.18	5.06	1	0.0279	1.48	0.95–2.30			
Earliest suicide attempt ^d	22.96	1.38	24.40	1.66	0.46	1	0.5008			1.96	1	0.1660
Recent suicide attempt ^d	29.95	1.87	31.74	2.12	0.39	1	0.5331			1.43	1	0.2362
Mania/hypomania (since Wave 1)												
Any manic/hypomanic episodes	36.00	2.48	34.72	1.81	0.16	1	0.6863	1.04	0.80–1.36			
No. of episodes	3.22	0.29	3.28	0.27	0.02	1	0.8807			0.04	1	0.8356
Length of mania (weeks) ^b	5.26	0.98	6.25	0.94	0.51	1	0.4773			0.32	1	0.5715
Recovery from mania	81.06	5.00	73.45	4.60	1.18	1	0.2822	1.28	0.55–2.98			
Counselor for mania ^e	36.44	5.24	37.28	3.87	0.01	1	0.9053	0.76	0.39–1.49			
Hospitalized for mania ^e	5.65	2.06	8.85	2.19	1.14	1	0.2888	0.56	0.20–1.53			

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value	AOR ^a	95% CI	(χ^2 or F) ^a	df ^a	p-value ^d
	% or Mean	SE	% or Mean	SE								
Emergency room for mania ^e	4.51	1.84	8.67	2.31	1.78	1	0.1874	0.50	0.16–1.57			
Medication for mania ^e	36.34	5.00	37.83	3.72	0.05	1	0.8254	0.77	0.41–1.46			
Sought any treatment ^e	46.92	5.49	45.38	3.88	0.04	1	0.8337	0.95	0.50–1.81			

BD = bipolar disorder; SE = standard error; CI = confidence interval.

^a Adjusted odds ratio (AOR) is adjusted by race, age, gender, income, and past-year smoking status (all in Wave 1).

^b Longest duration.

^c Among subjects with depression between Wave 1 and Wave 2 (n = 214 obese, n = 397 non-obese).

^d Assessed at Wave 2 only.

^e Among subjects with hypo/mania between Wave 1 and Wave 2 (n = 134 obese, n = 268 non-obese).

Table 3

Psychiatric and medical comorbidity

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value	AOR ^a	95% CI	(χ^2 or F) ^a	df ^a	p-value ^a
	% or Mean	SE	% or Mean	SE								
Comorbidities												
New onset substance use disorder	9.13	2.30	12.54	2.00	1.09	1	0.3003	0.78	0.36–1.70			
New onset cocaine and/or amphetamine use disorder	0.96	0.45	1.70	0.51	1.01	1	0.3179	0.77	0.28–2.09			
New onset cannabis use disorder	2.07	0.73	3.83	0.90	2.03	1	0.1591	0.70	0.28–1.75			
New onset other drug use disorder ^b	2.84	0.87	2.99	0.60	0.02	1	0.8833	1.05	0.53–2.05			
New onset alcohol use disorder	11.81	2.30	19.24	1.96	5.08	1	0.0275	0.73	0.41–1.29			
New onset anxiety disorder (not specific phobia)	38.07	2.86	32.00	1.62	3.52	1	0.0653	1.27	0.94–1.69			
Medical burden												
<i>New physician-diagnosed medical conditions</i>												
Any	75.69	2.54	52.64	2.13	42.06	1	<0.0001	2.32	1.63–3.30			
Arteriosclerosis	3.19	0.85	1.88	0.49	1.82	1	0.1823	1.21	0.49–3.01			
Hypertension	16.44	2.29	8.95	1.11	10.43	1	0.0019	1.81	1.16–2.82			
Diabetes ^c	22.12	2.19	3.51	0.69	84.39	1	<0.0001	6.98	4.27–11.40			
Cirrhosis	0.00	0.00	0.21	0.16	0.00	1	1.0000	–	–			
Liver disease	2.28	0.83	0.65	0.25	5.64	1	0.0205	2.15	0.79–5.84			
Angina	10.29	1.83	7.66	1.12	1.64	1	0.2046	1.30	0.79–2.14			
Tachycardia	11.20	1.84	8.94	1.20	1.11	1	0.2965	1.05	0.63–1.75			
Heart attack	2.34	0.84	0.99	0.46	2.06	1	0.1556	1.72	0.55–5.41			
High cholesterol ^c	26.79	2.24	15.85	1.46	15.97	1	0.0002	1.55	1.07–2.23			
Other heart disease	2.93	0.90	1.35	0.43	3.54	1	0.0643	1.74	0.71–4.25			
Stomach ulcer	4.90	1.18	3.58	0.71	1.00	1	0.3212	1.19	0.61–2.34			
HIV ^c	1.00	0.48	0.70	0.25	0.35	1	0.5534	1.15	0.29–4.57			
AIDS ^c	0.00	0.00	0.29	0.16	0.00	1	1.0000	–	–			
STD ^c	2.04	0.61	1.76	0.48	0.20	1	0.6593	1.06	0.52–2.16			

	BD obese (n = 506; 29.43%)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value	AOR ^a	95% CI	(χ^2 or F) ^a	df ^a	p-value ^d
	% or Mean	SE	% or Mean	SE								
Gastritis	8.76	1.70	5.06	0.88	5.15	1	0.0265	1.49	0.86–2.57			
Arthritis	17.71	2.56	10.08	1.20	10.17	1	0.0022	1.64	1.07–2.52			
Stroke ^c	0.91	0.46	0.41	0.22	1.18	1	0.2812	1.50	0.33–6.78			
Total	1.14	0.06	0.64	0.04	49.65	1	< 0.0001			29.88	1	< 0.0001
<i>Change in Short-Form 12 norm-based functioning scores</i>												
Physical disability	-1.24	0.58	-0.38	0.30	1.80	1	0.1846			2.25	1	0.1389
Mental disability	1.18	0.82	1.04	0.44	0.02	1	0.8764			0.03	1	0.8627
Physical functioning	-0.86	0.63	-0.10	0.33	1.18	1	0.2807			1.25	1	0.2675
Role physical	-1.35	0.65	-1.35	0.37	0.00	1	1.0000			0.12	1	0.7257
Bodily pain	0.04	0.77	1.30	0.45	2.05	1	0.1569			1.92	1	0.1701
General health	-0.61	0.62	0.82	0.38	3.92	1	0.0519			4.04	1	0.0486
Vitality	-0.34	0.68	-0.99	0.45	0.66	1	0.4207			0.49	1	0.4882
Social functioning	1.41	0.74	0.92	0.53	0.29	1	0.5931			0.43	1	0.5136
Emotional	0.21	0.72	-0.01	0.47	0.07	1	0.7870			0.00	1	0.9533
Mental health	0.88	0.79	2.03	0.43	1.74	1	0.1913			1.64	1	0.2054
No. of hospitalizations ^c	0.36	0.05	0.24	0.03	4.37	1	0.0405			1.53	1	0.2211
Days in hospital ^c	6.72	3.60	4.34	1.65	0.73	1	0.3947			0.35	1	0.5563
Emergency room visits ^c	0.99	0.15	0.60	0.05	5.90	1	0.0179			5.57	1	0.0213
Regular provider ^c	77.02	2.14	68.26	1.74	9.18	1	0.0035	1.40	1.03–1.89			

BD = bipolar disorder; SE = standard error; CI = confidence interval.

^a Adjusted odds ratio (AOR) is adjusted by race, age, gender, income, and past-year smoking status (all in Wave 1).

^b Including sedatives, tranquilizers, opioids, hallucinogens, inhalant/solvents, heroine, and other drugs.

^c Only has Wave 2 data.

Table 4

Obesity and Wave 1 substance use disorders

	BD obese (n = 506; 29.43% ⁰)		BD not obese (n = 1,094; 70.57%)		χ^2 or F	df	p-value	AOR ^a	95% CI
	% or Mean	SE	% or Mean	SE					
Wave 1 substance use disorders									
Any lifetime substance use disorder	65.58	2.38	70.09	1.75	2.74	1	0.1024	1.05	0.78–1.42
Cocaine and/or amphetamine use disorder	13.18	1.98	16.10	1.46	1.30	1	0.2576	0.92	0.60–1.42
Cannabis use disorder	24.29	2.42	30.64	1.87	4.11	1	0.0467	0.86	0.61–1.22
Other drug use disorder ^b	13.30	1.65	15.70	1.47	1.11	1	0.2955	0.97	0.66–1.43
Alcohol use disorder	54.28	2.59	57.78	1.82	1.39	1	0.2429	1.09	0.83–1.42

BD = bipolar disorder; SE = standard error; CI = confidence interval.

^a Adjusted odds ratio (AOR) is adjusted by race, age, gender, income, and past-year smoking status (all in Wave 1).

^b Including sedatives, tranquilizers, opioids, hallucinogens, inhalant/solvents, heroin, and other drugs.