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Should pathological gambling and obesity be considered addictive disorders? A factor analytic study in a nationally representative sample

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Abstract

Objective—Pathological gambling (PG) is now aligned with substance use disorders in the DSM-5 as the first officially recognized behavioral addiction. There is growing interest in examining obesity as an addictive disorder as well. The goal of this study was to investigate whether epidemiological data provide support for the consideration of PG and obesity as addictive disorders.

Method—Factor analysis of data from a large, nationally representative sample of US adults (N=43,093), using nicotine dependence, alcohol dependence, drug dependence, PG and obesity as indicators. It was hypothesized that nicotine dependence, alcohol dependence and drug use dependence would load on a single factor. It was further hypothesized that if PG and obesity were addictive disorders, they would load on the same factor as substance use disorders, whereas failure to load on the addictive factor would not support their conceptualization as addictive disorders.

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Contributors

Carlos Blanco designed the study and wrote the initial draft of the manuscript. Shuai Wang and Melanie Wall undertook the statistical analysis. All authors contributed to and have approved the final manuscript.

Conflict of Interest

All the authors declare that they have no conflicts of interest.

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Results—A model with one factor including nicotine dependence, alcohol dependence, drug dependence and PG, but not obesity, provided a very good fit to the data, as indicated by CFI=0.99, TLI=0.99 and RMSEA=.01 and loadings of all indicators >0.4.

Conclusion—Data from this study support the inclusion of PG in a latent factor with substance use disorders but do not lend support to the consideration of obesity, as defined by BMI, as an addictive disorder. Future research should investigate whether certain subtypes of obesity are best conceptualized as addictive disorders and the shared biological and environmental factors that account for the common and specific features of addictive disorders.

Keywords

Addictive Disorders; substance use disorders; pathological gambling; gambling disorder; obesity

1. INTRODUCTION

Inclusion of pathological gambling (renamed as gambling disorder) in the Substance-Related and Addictive Disorders chapter of the fifth edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has brought to the fore considerations of which disorders or behaviors are best conceptualized as addictive disorders. This debate is important because the nosological status of a disorder is an important determinant of its lines of research (Blanco et al., 2009; Moreyra et al., 2002), treatment models (Greene et al., 2011; Petry et al., 2006) and approaches to policy and funding for treatment and research (Petry and Blanco, 2013).

Among the potential candidates that were considered to join the substance related and addictive disorders category in DSM-5, few garnered more attention than pathological gambling (PG) and obesity, possibly due to their devastating impact and recent increases in prevalence (Swinburn et al., 2011; Ziauddeen et al., 2012). Clinical experience and research evidence have suggested that PG shares many features with substance use disorders. These include high levels of comorbidity in clinical (Black and Moyer, 1998; Ibanez et al., 2001) and epidemiological samples (Kessler et al., 2008; Petry et al., 2005), shared genetic variance (Blanco et al., 2012; Slutske et al., 2000), high levels of impulsivity (Blanco et al., 1996, 2009; Petry, 2001), performance in biobehavioral studies (Leeman and Potenza, 2012) and brain imaging studies (Leeman and Potenza, 2012; van Holst et al., 2010). Furthermore, both pharmacological (Grant et al., 2006; Kim et al., 2001) and psychotherapy treatments for PG have been often based on its conceptualization as an addictive disorder (Hodgins et al., 2001; Petry et al., 2006).

Data supporting the consideration of obesity as an addictive disorder have been more mixed. Some empirical studies and reviews have supported the conceptualization of obesity as an addiction based on data suggesting overlapping molecular and cellular mechanisms and reward brain circuits as well as shared genetic vulnerabilities (Galanti et al., 2007; Grucza et al., 2010; Kenny, 2011a, 2011b; Lilenfeld et al., 2008; Volkow et al., 2011; Weller et al., 2008), but other studies have provided countervailing evidence (Fernandez-Castillo et al., 2010; Haltia et al., 2007; Munafo et al., 2009; Smith et al., 2008; Ziauddeen et al., 2012).

We sought to contribute to those lines of research by conducting a factor analysis using data from the National Epidemiologic Survey on Alcohol and Related Conditions, a large, nationally representative sample of US adults. We hypothesized that substance use disorders, i.e., nicotine dependence, alcohol dependence and drug use dependence, would load on a single factor, consistent with prior analyses (Kessler et al., 2011, 1998). Furthermore, we hypothesized that if PG and obesity were addictive disorders, they would load on the same factor as substance use disorders. By contrast, failure to load on the addictive factor would not support their conceptualization as addictive disorders, at least not as defined within this dataset.

2. METHODS AND MATERIALS

2.1 NESARC Sample

The 2001–2002 NESARC is a survey of a representative sample of the United States sponsored by the NIAAA (Grant et al., 2003b; Grant and Kim, 2002). The target population was individuals age 18 years and older in the civilian non-institutional population residing in households and group quarters. The survey included those residing in the continental United States, District of Columbia, Alaska and Hawaii. Face-to-face personal interviews were conducted with 43,093 respondents. The survey response rate was 81%. Blacks, Hispanics, and young adults (ages 18 to 24) were oversampled. The research protocol, including informed consent procedures, received full human subjects review and approval from the U.S. Census Bureau and the U.S. Office of Management and Budget.

Data were weighted to reflect the design characteristics of the NESARC survey and to account for oversampling. Adjustment for non-response across numerous variables, including age, race/ethnicity, sex, region and place of residence, was performed at the household and person level. Weighted data were then adjusted to be representative of the civilian population of the United States on a variety of socioeconomic variables including region, age, race/ethnicity and sex based on the 2000 Decennial Census.

2.2 DSM-IV Assessment of SUD, PG and obesity

All diagnoses in this study refer to 12-month diagnoses at the time of the interview. The diagnostic interview was the NIAAA Alcohol Use Disorder and Associated Disabilities Interview Schedule-DSM-IV Version (AUDADIS-IV), a state-of-the-art structured diagnostic interview designed for use by experienced lay interviewers (Grant and Hasin, 2001). As described in detail elsewhere, the AUDADIS-IV included an extensive list of symptom questions that operationalized DSM-IV criteria for nicotine dependence, alcohol dependence and drug-specific dependence for ten classes of drugs: sedatives, tranquilizers, opiates (other than heroin or methadone), stimulants, hallucinogens, cannabis, cocaine, inhalants/solvents, heroin, and other drugs (Grant et al., 2003a, 2004a, 2004b, 2004c). As in previous reports (Compton et al., 2007), dependence on any one or more of the other drugs outlined were aggregated into a single category of drug dependence to increase statistical power and ensure the stability of the estimates.

To assess the presence of PG, all respondents who had gambled 5 or more times in at least one year of their lives were asked about the symptoms of DSM-IV PG. Specifically, the

gatekeeping question asked, “Now I’d like to ask you a few questions about gambling. By gambling I mean playing cards for money, betting on horses or dogs or sports games, playing the stock or commodities market, buying lottery tickets or playing bingo or Keno or gambling at a casino, including playing the slot machines. Have you ever gambled at least 5 times in any one year of your life?” Those who responded affirmatively were asked the DSM-IV pathological gambling questions. Consistent with DSM-IV, lifetime AUDADIS-IV diagnoses of PG required the respondent to meet at least five of the ten DSM-IV criteria. Fifteen symptom items operationalized the 10 PG criteria with high test-retest reliability, validity and internal consistency (Petry et al., 2005). Furthermore, because substantial evidence from clinical (Strong et al., 2004), genetic (Blanco et al., 2012; Slutske et al., 2000) and epidemiological (Strong and Kahler, 2007) studies suggest that PG forms a continuum, for the purpose of sensitivity analyses, we defined a broader category of disordered gambling (DG). Consistent with prior research we defined DG as requiring at least three criteria be met (Shaffer et al., 1999). We also examined whether the results held when using in the analyses gambling disorder, as defined by DSM-5, i.e., meeting four out of nine criteria (with the “illegal acts criteria” no longer being included among the diagnostic criteria).

We used the body mass index (BMI) to measure obesity as recently recommended (Mooney et al., 2012). Consistent with the National Institutes of Health (NIH), BMI-based definition for obesity (National Institutes of Health, 2004), we defined obesity as a BMI ≥ 30 . Heights and weights were self-reported.

Test-retest reliability (Canino et al., 1999; Chatterji et al., 1997; Grant et al., 1995), internal consistency and validity (Chatterji et al., 1997; Grant et al., 2004a, 2004b, 2004c; Hasin et al., 2003) of the AUDADIS-IV measures are well documented in psychometric studies conducted in clinical and general population samples. In addition, reliability and validity of AUDADIS substance use disorders were found to be good to excellent in several countries participating in the National Institutes of Health/World Health Organization Reliability and Validity Study (Chatterji et al., 1997; Cottler et al., 1997; Pull et al., 1997; Ustun et al., 1997).

2.3 Statistical analysis

We conducted two sets of analyses. First, raw odds ratio (ORs) as well as ORs adjusted for age and gender (AORs) and their 95% confidence intervals (95% CIs) were used to estimate the bivariate association between pairs of disorders. In accord with longstanding conventions (Agresti, 2002), we consider as significant those ORs whose 95% CIs do not cross 1.

Second, factor analysis was used to test the latent structure of the disorders considered jointly. Factor analyses were selected over other approaches such as latent class analyses or hybrid models (e.g., factor mixture modeling) because prior work has consistently shown that the latent structure of substance use disorders is best represented as a continuum (Markon and Krueger, 2005). To test whether nicotine dependence, alcohol dependence, drug dependence, PG and obesity loaded in the same factor, we conducted a series of confirmatory factor analyses (CFA), using the disorders as binary indicators. First, to

confirm we fitted a one-factor model for nicotine dependence, alcohol dependence and drug use dependence. Next, we fitted two models by adding (separately) PG and obesity to the previous model, to prevent PG and obesity from influence testing each other. An additional model was fitted that included only alcohol dependence, drug dependence and obesity in case inclusion of nicotine dependence in the model could conceal associations among the other three variables. Finally, we fitted a model with all five indicators in the model. As a sensitivity analysis, we re-fitted this model excluding individuals with BMI<18.5, to ensure that individuals with low BMI did not suppress potential associations between obesity and the other indicators. With only 3 indicators, a one factor model is fully saturated with zero degrees of freedom and thus common fit indices are not useful. For the models with 4 and 5 indicators, we used goodness of fit measures including the comparative fit index (CFI), Tucker-Lewis index (TLI), and root mean squared error of approximation (RMSEA). Hu and Bentler recommended CFI and TLI values above .95, and RMSEA values below .06, as representing good model fit (Hu et al., 1992). More important than the goodness of fit for our current question is the magnitude and statistical significance of the factor loadings. If a factor loading between the latent factor and an indicator is not significantly different from zero and not at least moderately large, e.g., >0.40, then there is no evidence the disorder relates to the common factor. To test the robustness of our results, we repeated our analyses using DG and gambling disorder, rather than PG as the indicators in our analyses, as well as excluding from the sample individuals with low-weight, operationalized as BMI<18.5.

All analyses were conducted in Mplus Version 6.1 (Muthen and Muthen, 1998–2012), which takes into account the NESARC sampling weights and design effects in all analyses, including parameter as well as standard error estimation and model fit calculations. Because all variables were binary, we used as the default estimator for the analysis the variance-adjusted weighted least squares (WLSMV), a robust estimator which does not assume normally distributed variables and provides the best option for modeling categorical or ordered data (Muthén, 1984).

3. RESULTS

The 12-month prevalence of obesity, nicotine dependence, alcohol dependence, drug dependence, and pathological gambling were 21.8 (95% CI=21.0–22.6), 12.8% (95% CI=12.0–13.6), 3.8% (95% CI=3.5–4.1), 0.6% (95% CI=0.4–0.8), and 0.16% (95% CI=0.12–0.20), respectively. The mean BMI in the sample was 26.7 (95% CI=26.6–26.9). Bivariate analyses indicated that PG was significantly and positively associated to nicotine, alcohol and drug dependence, with ORs ranging from 2.82 for drug dependence to 8.17 for nicotine dependence. By contrast, obesity was significantly and negatively associated alcohol and drug dependence, and was not significantly associated with nicotine dependence or PG (Table 1). After adjusting for age and gender, results remained unchanged, except for the association between obesity and drug use disorders, which was no longer significant (Table 2).

The results of the series of CFAs indicated that a model with one factor including nicotine dependence, alcohol dependence, drug dependence had all significant loadings >.4. Inclusion of PG in the model resulted in very good fit (CFI=0.99, TLI=0.99 and RMSEA=.

01) also with loadings of all indicators $>.4$. By contrast, although all models including obesity provided good fit to the data (in all cases CFI = 0.98, TLI = 0.95 and RMSEA = 0.02), in none of the models did obesity have a loading $>.4$ indicating that obesity did not load on that factor (Table 3).

When DG or gambling disorder, rather than PG, were included as indicators in the analysis, a one-factor model including nicotine dependence, alcohol dependence, drug dependence and DG (or gambling disorder), but not obesity, provided a good fit for the data. In all cases, the loading for obesity was negative and with an absolute value <0.1 , again indicating that it did not load on that factor (Table 4).

4. DISCUSSION

In a large, nationally representative sample of US adults, nicotine, alcohol and drug dependence and pathological gambling loaded on the same factor. By contrast, the loading of obesity on the addictive factor was very low. Furthermore, the findings were robust to model specification as indicated by the results of sensitivity analyses that used disordered gambling and DSM-5 gambling disorder as indicators of the factor.

Consistent with our first hypothesis, we found that nicotine, alcohol and drug dependence loaded on the same factor, which could be broadly characterized as an addiction factor. Our results are in accord with prior analyses from the NESARC and other studies documenting the high levels of comorbidity and shared variance of addictive disorders (Agrawal et al., 2007). Several overlapping mechanisms, including shared genetic variance (Kendler et al., 2011), neurocircuitry (Koob and Volkow, 2010; Yalachkov et al., 2012), psychological development (Verdejo-Garcia et al., 2012), and environmental risk factors (Kendler et al., 2003) contribute to explain the existence of this factor and suggest opportunities for joint treatment and prevention interventions. To the extent that disorders share common causes or etiological mechanism, acting on those common causes may lead to simultaneous improvements in several disorders (Blanco et al., 2013).

In support of our second hypothesis, PG also had high loadings on the addiction factor. Our results are consistent with a broad range of research on neurobiological (Blanco et al., 2006; Slutske et al., 2000), clinical (Blanco et al., 2009; Grant et al., 2006; Hodgins et al., 2001; Kim et al., 2001; Petry et al., 2006) and epidemiological (Blanco et al., 2012; Leeman and Potenza, 2012; Petry, 2001) studies, as well as with the recommendations of the DSM-5 task force to include gambling disorder in the Substance Related and Addictive Disorders category (Petry, 2010). We also performed analyses using the new diagnosis of gambling disorder (meeting 4 of the 9 criteria, eliminating the illegal acts criterion) and results were similar. However, although the loading of PG was high (>0.4), it was slightly lower than those of alcohol and drug dependence. Our data suggest that although it has important commonalities with substance use disorders, PG also has sources of unique variance, which indicate the existence of specific risk factors for this disorder. Similarly, the fact that the loadings of nicotine, alcohol and drug dependence were substantially below 1 indicates that all those disorders have common but also unique source of variance (i.e., specific risk factors). Future research should determine whether those risk factors involve some specific

genetic predisposition or other biological determinant, particular personality traits, or unique environmental factors, such as sociocultural acceptance of substance use or gambling behaviors, availability or exposure to drugs or gambling.

By contrast, we found that obesity did not load on the addiction factor. This finding suggests that obesity, as currently defined by NIH in terms of BMI, may not be best understood as an addiction. Our data are in contrast with findings from several clinical (Gearhardt et al., 2009), neuroimaging (Stice et al., 2008), animal (Kenny, 2011b) and human laboratory studies (Kenny, 2011a; Volkow et al., 2011), but in accord with a recent comprehensive review of the evidence of the addiction model of obesity that found only limited clinical overlap between obesity and substance dependence (Ziauddeen et al., 2012), meta-analyses that have failed to replicate findings of shared vulnerability between obesity and substance dependence (Fernandez-Castillo et al., 2010; Munafo et al., 2009; Smith et al., 2008) and inconsistent findings in both PET and functional imaging studies (Haltia et al., 2007; Ziauddeen et al., 2012). One explanation for these findings may relate to the differential categorizations of these conditions in the study; that is, substance dependence and PG were classified by behavioral criteria while obesity was assessed based on BMI alone. Obesity is multi-determined and perhaps certain subtypes of obesity that are more behaviorally determined (Gearhardt et al., 2011) may constitute a useful heuristic and load on an addiction factor. Another possibility for the lack of association is that food and drugs may compete for similar neural receptors (Gearhardt and Corbin, 2009) or substitute for each other. However, that explanation is not fully consistent with the fact that drugs and alcohol, which also compete for the same receptors, do tend to co-occur (Compton et al., 2007) and remission from one substance use disorder tends to decrease, rather than increase, the risk of new onset or relapse of another substance use disorder (Blanco et al., 2014). A potential avenue for future research would be to focus on food-related behaviors that may have addictive-like qualities, rather than obesity per se. This approach may help circumvent the limitations of BMI and facilitate comparison with other addictive behaviors, which are generally described in behavioral terms. There is also a need to investigate whether other validators such as shared genetic liability, similar risk factors and clinical course, laboratory studies or response to treatment support the conceptualization of obesity or certain food-related behaviors as addictions.

Our results have additional limitations. First, as all large epidemiological surveys, data collected by the NESARC, including height and weight, were based on self-report and not subject to independent verification, raising the possibility of social desirability and recall bias. However, those biases should have resulted in increased error variance, weakening associations and biasing results towards less strong associations among disorders. Second, to decrease respondent burden, the NESARC restricted the assessment of gambling behaviors to individuals who had gambled at least 5 times in one of their lives. It is possible that this gating question may have excluded some individuals who met DSM-IV criteria for PG, but this seems unlikely (Blanco et al., 2006; Petry et al., 2005). Third and as noted earlier, whereas substance use disorders and PG were diagnosed based on a structured interview, obesity was diagnosed based on the BMI. However, BMI is currently the recommended measure to diagnose obesity (Mooney et al., 2012) and the basis for the NIH definition of obesity and overweight (NIH, 2004). There are no alternative established approaches to the

diagnoses of obesity or addictive disorders. Furthermore, a method effect is unlikely to explain, per se, the aggregation of PG and alcohol and drug dependence, as numerous studies have shown that not all psychiatric disorders, even when assessed by the same method or interview, are indicators of the same factor, but rather are indicators of factors that are theoretically coherent and give clues about their etiology. Fourth, the NESARC did not assess eating disorders, and thus could not be included in this study. Data from both the National Comorbidity Study Adolescent Supplement (Kessler et al., 2012) and the Norwegian Institute of Public Health Twin Panel (Roysamb et al., 2011) indicate that eating disorders and substance use disorders load on different factors, although it is possible that certain subtypes of eating disorders may have behavioral or biological similarities with addictive disorders.

In summary, our data strongly support the inclusion of pathological gambling in a latent factor with substance use disorders and its consideration as an addictive disorder. By contrast, our data are less supportive of the consideration of obesity, as defined by BMI, as an addictive disorder. Future research should investigate whether certain subtypes of obesity or eating disorders are best conceptualized as an addictive disorder and to investigate the shared biological and environmental factors that account for the common and specific features of addictive disorders.

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Highlights

1. An addiction factor includes substance use disorders and pathological gambling
2. Obesity does not load into the addiction factor
3. The data are supportive of pathological gambling as addictive disorder.
4. Future research should investigate whether certain obesity subtypes are addictions

Table 1

Odds ratio (OR) for past year substance use disorders, pathological gambling, and obesity.

	Nicotine Dependence			Alcohol Dependence			Drug Dependence			Pathological Gambling			Obesity		
	OR	95% CI		OR	95% CI		OR	95% CI		OR	95% CI		OR	95% CI	
Nicotine Dependence	.	.		4.36	3.91	4.87	8.10	6.67	9.83	8.17	4.77	13.99	0.97	0.89	1.05
Alcohol Dependence	4.36	3.91	4.87	.	.	.	15.17	12.60	18.28	7.26	4.12	12.80	0.81	0.72	0.91
Drug Dependence	8.10	6.67	9.83	15.17	12.60	18.28	.	.	.	2.82	0.97	8.19	0.75	0.61	0.93
Pathological Gambling	8.17	4.77	13.99	7.26	4.12	12.80	2.82	0.97	8.19	.	.	.	1.38	0.71	2.65
Obesity	0.97	0.89	1.05	0.81	0.72	0.91	0.75	0.61	0.93	1.38	0.71	2.65	.	.	.

Adjusted Odds ratio (OR) for past year substance use disorders, pathological gambling, and obesity.

Table 2

	Nicotine Dependence			Alcohol Dependence			Drug Dependence			Pathological Gambling			Obesity		
	AOR	95%	CI	AOR	95%	CI	AOR	95%	CI	AOR	95%	CI	AOR	95%	CI
Nicotine Dependence	.	.	.	3.77	3.37	4.22	6.67	5.47	8.13	7.29	4.16	12.80	0.97	0.89	1.06
Alcohol Dependence	3.74	3.34	4.19	.	.	.	9.57	7.84	11.69	6.34	3.40	11.81	0.87	0.78	0.98
Drug Dependence	6.63	5.44	8.09	9.58	7.84	11.72	.	.	.	2.13	0.71	6.37	0.89	0.71	1.10
Pathological Gambling	7.31	4.17	12.82	6.36	3.39	11.94	2.07	0.69	6.17	.	.	.	1.38	0.71	2.66
Obesity	0.97	0.89	1.05	0.87	0.77	0.98	0.88	0.71	1.10	1.37	0.71	2.66	.	.	.

Table 3

Confirmatory Factor Analyses of past year substance use disorders, pathological gambling, and obesity.^a

Fit indices	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6 ^b
CFI ^c	1.00	1.00	0.99	0.99	0.99	0.99
TLI	1.00	1.00	0.99	0.99	0.99	0.98
RMSEA	0.00	0.00	0.01	0.00	0.00	0.01
Disorder	Factor loadings					
Nicotine Dependence	0.58	-	0.59	0.58	0.59	0.59
Alcohol Dependence	0.72	0.78	0.73	0.73	0.73	0.73
Drug Dependence	0.85	0.79	0.84	0.85	0.84	0.84
Pathological Gambling	-	-	0.46	-	0.46	0.47
Obesity	-	-0.08	-	-0.06	-0.06	-0.07

^a Empty cells indicate that the item was not included in that particular analysis, to allow examining the fit of different models. We note that because the models are not nested within each other, it is not possible to compare them directly using formal statistical tests.

^b Excluding individuals with BMI<18.5.

^c Abbreviations: CFI, Comparative Fit Index; RMSEA, root mean square error of approximation; TLI, Tucker-Lewis Index.

Table 4

Confirmatory Factor Analyses of past year substance use disorders, gambling disorder, disordered gambling and obesity.^a

Fit indices	Model 1	Model 2	Model 3	Model 4	Model 5	Model 6
CFI ^b	1.00	1.00	0.99	0.99	0.99	0.99
TLI	1.00	1.00	0.99	0.99	0.99	0.99
RMSEA	0.00	0.00	0.01	0.00	0.00	0.01
Disorder	Factor loadings					
Nicotine Dependence	0.60	0.60	0.59	0.60	0.59	0.59
Alcohol Dependence	0.72	0.73	0.73	0.73	0.73	0.74
Drug Dependence	0.83	0.82	0.82	0.83	0.82	0.82
Obesity	-	-	-	-0.05	-0.05	-0.04
Gambling Disorder ^c	0.48	-	-	0.47	-	-
Disordered Gambling ^d	-	0.43	-	-	0.43	-

^a Empty cells indicate that the item was not included in that particular analysis, to allow examining the fit of different models. We note that because the models are not nested within each other, it is not possible to compare them directly using formal statistical tests.

^b Abbreviations: CFI, Comparative Fit Index; RMSEA, root mean square error of approximation; TLI, Tucker-Lewis Index.

^c Gambling disorder defined, in accord with DSM-5, as meeting 4 out of 9 criteria but not including "committing illegal acts" as one of the nine criteria.

^d Disordered gambling defined as (3–10) criteria.