



# Anhedonia in obsessive-compulsive disorder: Beyond comorbid depression



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## ABSTRACT

Obsessive-compulsive disorder (OCD) has been linked to reward dysfunctions, highlighting a possible role of anhedonia in OCD. Surprisingly, anhedonia in OCD has never been evaluated. Moreover, although nicotine typically has anti-anhedonic effects, anecdotal reports suggest low prevalence rates of smoking in OCD. To address these two phenomena, 113 individuals with OCD completed a battery of questionnaires assessing symptom severity, anhedonia, and smoking. 28.3% of the sample met criteria for clinically significant anhedonia, which correlated with Y-BOCS scores ( $r=0.44$ ), even when controlling for depressive symptoms. 13.3% of the sample endorsed current smoking, a lower rate than seen in psychiatric disorders (40–90%) and the general adult population (19%). Results highlight high rates of anhedonia and yet reduced prevalence of smoking in OCD. In contrast to the known positive association between anhedonia and smoking, a negative association emerged. Future research is needed to address the unique interface between anhedonia and reward responsiveness in OCD. Potential clinical implications are discussed.

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## 1. Introduction

Anhedonia, the inability to experience pleasure, is a quantifiable, valid construct that may be more heritable than depression (Bogdan and Pizzagalli, 2009). Researchers commonly use three categories of measures to assess anhedonia [i.e., self-report questionnaires, computerized tasks probing distinct components of reward processing, and imaging studies targeting brain activity in specific regions, predominantly the ventral striatum (VS)/nucleus accumbens (NAc)]. An abundance of psychiatric investigations, primarily in patients with schizophrenia, depression, substance dependence, and Parkinson's disease, but also in patients with bipolar disorder and PTSD, reports elevated scores on anhedonia scales (Leventhal et al., 2006; Franken et al., 2007; Assogna et al., 2011; Hatzigiakoumis et al., 2011; Di Nicola et al., 2012; Frewen et al., 2012). Although anhedonia is found in the majority of patients with major depressive disorder, it may be a distinct entity from depression as demonstrated by a plethora of studies reporting weak to moderate correlations between the two constructs

(Leventhal et al., 2006; Franken et al., 2007; Nakonezny et al., 2010) among non-psychiatric controls as well as in depression and other disorders. In the context of anhedonia, imaging studies repeatedly demonstrate reduced activation in the VS (and other regions associated with reward circuitry) in response to a variety of rewarding stimuli especially in schizophrenia and depression (Berridge and Kringelbach, 2008; Pizzagalli et al., 2009; Dichter et al., 2012).

To our knowledge, anhedonia has never been researched in Obsessive-Compulsive Disorder (OCD) or in OCD spectrum disorders. However, our clinical experience suggests that a significant percentage of OCD patients may be characterized by anhedonia. Two lines of evidence support this hypothesis. First, three imaging studies recently reported aberrant VS and insula activation in OCD patients during a monetary incentive task (Figue et al., 2011; Jung et al., 2011; Choi et al., 2012). One study found reduced VS activation in OCD patients in both the anticipatory and consummatory conditions (Figue et al., 2011), another only in the anticipatory condition (Jung et al., 2011), and another found aberrant activation in the insula but not in the VS (Choi et al., 2012). The second line of evidence stems from Deep Brain Stimulation (DBS) procedures, which have demonstrated benefits for patients with refractory OCD (McLaughlin and Greenberg, 2011), especially when targeting the VS/NAc (de Koning et al., 2011). In fact,

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anecdotal evidence suggests that DBS targeting the NAc alleviates anhedonic symptoms in treatment-refractory depression (Schlaepfer et al., 2008; Bewernick et al., 2010).

Of note, there are significant differences between the nature of brain pathophysiology in schizophrenia and depression compared to OCD. Whereas schizophrenia and depression are characterized by frontostriatal hypometabolism (Glahn et al., 2005; Price and Drevets, 2012), OCD is characterized by hypermetabolism with more pronounced differences in the prefrontal cortex (Baxter et al., 1990; Harrison et al., 2009). Additionally, in contrast to schizophrenia and depression, negative reward may be central to OCD given the rewarding properties of compulsions and mental rituals in reducing anxiety (Figeo et al., 2011). Consequently, this may cause an attenuation of otherwise naturally positively rewarding stimuli (Volkow et al., 2004; Figeo et al., 2011). Consistent with this assumption, OCD patients have exhibited impairment in adjusting their behavior following positive reward (monetary incentives; Nielen et al., 2009). In light of these findings, we hypothesized that OCD may be associated with anhedonia. We further hypothesized that other 'natural' prominent stimuli may be less rewarding in OCD. We examined patterns of cigarette smoking to further explore this possibility.

The brain's reward systems activate with nicotine administration, and nicotine enhances reward reactivity predominantly by increasing dopamine release from mesolimbic neurons to the ventral striatum (Grenhoff et al., 1986). Additionally, nicotine potentiates activity in the NAc (Pontieri et al., 1996). Thus, tobacco smoking is a relevant phenomenon for anhedonia research. Notably, psychiatric conditions characterized by reward deficits are associated with high rates of tobacco smoking. Compared to smoking prevalence rates in the general adult population (US, 19%; Centers for Disease Control and Prevention (CDC), 2012), smoking rates are significantly higher in schizophrenia (62–90%), bipolar disorder (69%), depression (60%), and Attention Deficit/Hyperactivity Disorder (ADHD; 42%) (Ziedonis et al., 2008; Dome et al., 2010; Aubin et al., 2012). Notably, these disorders are associated with prefrontal hypoactivation, and together with nicotine's property of increasing activation in mesolimbic circuits, a self-medication hypothesis has been proposed to account for the associated increased rates of smoking in these disorders (Winterer, 2010; Sousa et al., 2011). Moreover, insight into the rewarding value of smoking may be gained from research suggesting that anhedonia predicts smoking onset and escalation (Audrain-McGovern et al., 2012). However, OCD, which appears to be associated with significantly lower smoking rates, is an intriguing exception to the high prevalence of smoking in psychiatric disorders. Indeed, a small number of studies suggest that rates of cigarette smoking in OCD ranges between 5.5–14.5%, i.e., substantially lower than smoking rates in the general population (Bejerot and Humble, 1999; Baker-Morissette et al., 2004; McCabe et al., 2004).

In light of these epidemiological data, we speculate that nicotine might exert deleterious interactions with specific pathophysiological underpinnings of OCD (e.g., prefrontal cortex hyperactivation and basal ganglia dysfunction), giving rise to the low smoking rates in OCD. In addition, negative reinforcement cycles and the need to exert control over thoughts, behaviors, emotions, and situations characteristic of OCD might attenuate the reinforcing properties of naturally rewarding stimuli, raising the possibility that, in OCD, anhedonia might negatively correlate with the number of cigarettes consumed. The overarching goal of the present study was to test these hypotheses. Specifically we investigated anhedonia in the context of OCD and its association with disorder-specific symptom severity and smoking. We hypothesized that the prevalence and severity of clinically significant anhedonia would be significantly higher in OCD than in the general population. In addition, given the hypothesized uniqueness of anhedonia in OCD, we expected to find a significant association between anhedonia and OCD symptom

severity over and above depressive severity. In light of the hypothesized unique association between reward mechanisms and anhedonia in OCD, we further hypothesized that smoking rates in OCD would be lower than rates of smoking in patients with major psychiatric disorders and the general population. Finally, unlike typical findings in schizophrenia and depression, we expected to find a negative association between cigarette smoking and anhedonia in OCD.

## 2. Methods

### 2.1. Recruitment

To strengthen diagnostic validity, recruitment entailed four consecutive waves. The first wave included 25 participants with a verified diagnosis of OCD. These patients had participated in research studies or received treatment at the Massachusetts General Hospital OCD Clinic and were contacted directly by email or phone. Verified diagnoses for OCD patients included in the first wave were established using the Structured Clinical Interview for DSM-IV (SCID; First et al., 2002). The second wave included 22 individuals that contacted our program seeking treatment and/or participation in research studies and had consented to be contacted directly. The third wave ( $n=42$ ) was comprised entirely of members who responded to an advertisement posted on the group message board of the largest members-only online support group for OCD. The final wave ( $n=24$ ) included participants who responded to advertisements posted on the hospital, program, and International Obsessive Compulsive Foundation (IOCDF) web page, as well as flyers posted in specialty clinics.

### 2.2. Web-based screening and assessment tool

We used REDCap, a secure, web-based platform for building and managing online surveys (Harris et al., 2009). Participants first received information regarding this study and signed an online consent form. At this stage, participants learned of the compensation offered upon completion of the survey (a \$10 gift card). After providing informed consent, participants attested that they were at least 18 years of age and responded to a question regarding their English proficiency. Participants then completed a DSM-IV-based diagnostic questionnaire for OCD, preceded by the statement: "The next short section will assess your eligibility to participate in this study and complete this survey." All items in this DSM-IV based questionnaire included language that was based on DSM criteria, including the use of major keywords. For example, the first criterion reads: "Have you ever experienced recurrent and persistent thoughts, impulses, or images that were experienced as intrusive, unwanted or inappropriate and that caused anxiety or distress? (For example, fear of hurting others, fear of being contaminated or contaminate others with germs, a feeling you are responsible for things that are wrong etc.). Please note, check 'No' if these thoughts are limited to real life problems (for example, in case all recurrent thoughts are limited ONLY to worries regarding a relative that was recently hospitalized)." This part of the survey was designed to notify ineligible participants (i.e., those not meeting criteria for OCD) that they could not participate in the study (in which case participants were not able to go back and change their responses). After entering their initials, eligible participants were redirected to complete the survey, which took 20–40 min. This range accounts for different measures administered to participants that currently smoke, smoked in the past, or never smoked.

### 2.3. Data integrity and validity

Research suggests that data from web-based studies are more reliable than previously thought and are not significantly affected by 'non-serious' or repeat responders (Gosling et al., 2004). However, in light of the concerns and corresponding control measures suggested in the literature (Nosek et al., 2002), we used the following methods to increase diagnostic validity and data integrity:

1. OCD diagnosis was a prerequisite. Prior to participation, individuals attested their diagnosis by a licensed mental health professional (psychologist or psychiatrist).
2. Compensation was not mentioned in the flyers or online ads for the fourth recruitment wave.
3. The survey included several 'control' questions that appeared twice.
4. The survey included several open-ended questions requiring detailed report (e.g., medication dosages), which were reviewed for inconsistencies.
5. Participants were required to provide their email address in order to receive reimbursement. Participants providing their email address were also asked to respond to the question: "Please indicate if you agree to be contacted in the future by our research lab in order to participate in future studies. I agree to be

contacted by the MGH OCD and Related Disorders Programs research staff using the email I provided in order to be offered to participate in research studies." Of the 113 eligible participants, 106 responded positively to this item.

6. Participants were asked to describe how they learned of the survey. Given our phased recruitment strategies, we were able to identify potential imposters (e.g., one participant wrote that she heard about the study from a 'flyer in a hospital' prior to the stage in which we used flyers. This participant was excluded.)
7. We carefully examined the data for irregularities such as multiple entries, inconsistent or contrasting responses across similar items, and responses with zero variability across one or more measures.
8. High internal consistency coefficients were found across measures, with no significant differences between recruiting waves (see the Section 3).

#### 2.4. Participants

All procedures and measures were approved by the Institutional Review Board. The survey registered 156 complete entries. We identified 16 suspicious entries, which were subsequently excluded, resulting in 140 individual participants completing the survey. Participants were included if they were age 18 or older, proficient in English, and diagnosed with primary OCD. In order to inquire about possible comorbid disorders, participants were presented with a brief description of major DSM-IV disorders and were subsequently asked four multiple-response questions (i.e., "Based on these descriptions, please check any of the disorders you are likely to have"; "have you ever in your lifetime been diagnosed with one of the following disorders by a psychologist or psychiatrist?"). Two additional questions required participants to select the disorder that causes the most distress and significant interference with daily functioning. These items included the option to select "other" and specify in an open-ended text box. An additional item asked participants whether they considered OCD to be the most severe disorder they currently suffer from. Using the information provided from items concerning psychiatric diagnoses we excluded participants ( $n=27$ ) with a self-reported diagnosis of ADHD, bipolar disorder, autism, tic disorders, PTSD, anorexia, and bulimia nervosa. Notably, we considered OCD to be primary in cases where participants reported OCD as their most severe disorder and the primary cause for functional impairment and distress. In the resulting final sample ( $n=113$ ), 85% met our criteria for primary OCD, and the remaining 15% declared that OCD was either the primary cause for impairment or distress.

#### 2.5. Measures

The *Yale–Brown Obsessive Compulsive Scale – Self-Report* (Y-BOCS-SR; Baer, 1992) was used to assess obsessive-compulsive symptom severity. The Y-BOCS-SR is an analogous version of the clinician administered Y-BOCS (Goodman et al., 1989a; Goodman et al., 1989b), a gold standard measure of OCD symptom severity. The Y-BOCS-SR demonstrates good psychometric properties in clinical and non-clinical samples and is strongly correlated with the clinician administered Y-BOCS (Steketee et al., 1996). In addition, the Y-BOCS-SR has good internal consistency when administered online (Cronbach's  $\alpha=0.81$ ; Moritz et al., 2012). In the present study, internal consistency was very good for the YBOCS-SR Obsession, Compulsion, and Total scores (Cronbach's  $\alpha=0.86$ , 0.89 and 0.91, respectively).

The *Snaith–Hamilton Pleasure Scale* (SHAPS; Snaith et al., 1995) is a 14-item self-report instrument designed to assess hedonic tone. Participants are presented with 14 statements of enjoyable activities (e.g., "I would be able to enjoy my favorite meal," "I would enjoy a warm bath or a refreshing shower.") and must choose one of four optional responses: *Definitely Agree*, *Agree*, *Disagree*, and *Strongly Disagree*. Each of the *Agree* responses receives a score of 0 and each of the *Disagree* responses receives a score of 1. The SHAPS total score ranges from 0–14 with higher scores reflecting greater anhedonia severity. According to criteria established by Snaith et al. (1995), a cutoff score of more than two negative responses determines the presence of clinically significant anhedonia. The SHAPS has been shown to have good psychometric properties in terms of balancing sensitivity and specificity, as well as excellent internal consistency in clinical ( $\alpha=0.91$ –0.95) and non-clinical ( $\alpha=0.91$ ) populations (Franken et al., 2007; Nakonezny et al., 2010). The SHAPS demonstrated very good internal consistency in the present study (Cronbach's  $\alpha=0.91$ ).

The 21-item *Depression Anxiety Stress Scales* (DASS-21; Lovibond and Lovibond, 1995) is a short self-report measure designed to assess severity of depression, anxiety, and stress. Participants rate seven items of each subscale on a four-point Likert scale ranging from 0 (*never*) to 3 (*most of the time*). The DASS-21 has very good psychometric properties with excellent internal consistency in online administration (Cronbach's  $\alpha$  coefficients for the depression, anxiety, and stress subscales were 0.95, 0.93, and 0.94, respectively) (Zlomke, 2009). In the present study, Cronbach's  $\alpha$  coefficients for the depression, anxiety, and stress subscales were 0.93, 0.81, and 0.88, respectively.

Current smokers completed the *Fagerström Test for Nicotine Dependence* (FTND; Heatherton et al., 1991). The most widely used instrument to establish and quantify nicotine dependence, the FTND has six items that provide a total score

ranging between 0 and 10. The FTND has very good psychometric properties in non-psychiatric smokers as well as in clinical populations (Buckley et al., 2005). Good internal consistency has been reported for online administration of the FTND ( $\alpha=0.79$ ; Rueger et al., 2012). In the present study, internal consistency for the sample was 0.75.

#### 2.6. Statistical control procedures

Web-based research has been subject to scrutiny with regards to participants' intentional and unintentional indiscretions with regard to research participation. However, studies comparing web-based and face-to-face studies suggest that with appropriate control measures, web based research is reliable (Birnbaum, 2004; Gosling et al., 2004). As detailed above, we have taken several recommended precautions (Nosek et al., 2002; Reips, 2002) to increase data integrity and diagnostic validity before and during recruitment, and in the preliminary analysis phase. Given recent suggestions that comparison of within-group correlations as well as good and comparable reliability coefficients between samples may serve as a proxy for OCD diagnosis validity (Moritz et al., 2012), we compared Cronbach's alphas as well as within-group correlations between recruitment waves. We individually compared the Y-BOCS Cronbach's  $\alpha$  for the first recruitment wave (i.e., clinician-verified OCD) with the alphas obtained from each of the three sub-samples using the formula suggested by Feldt et al. (1987). No significant differences were found between the sub-samples (alphas ranging from 0.81 to 0.94). We used Fisher's  $Z$  transformation to compare within-group correlations between the Y-BOCS and the SHAPS and the Y-BOCS and the DASS depression subscale. These analyses yielded no significant differences between recruitment waves. Finally, for all outcome measures, we employed a standard imputation procedure for missing data on the basis of average subscale score.

### 3. Results

The demographic profile of the sample is presented in Table 1.

Table 2 presents clinical information. The samples' Y-BOCS scores represent a mild degree of severity, and all three DASS-21 subscale scores represent mild to moderate severity. Using the threshold determined by Snaith et al. (1995), 28.3% of our sample was characterized by clinically significant anhedonia, a prevalence rate significantly higher than that found in a normative sample (4.9%; Snaith et al., 1995) and among non-psychiatric controls (2%; Franken et al., 2007). Anhedonia severity ( $M=1.92$ ,  $S.D.=2.77$ ), as measured by the sum of negative responses on the SHAPS, was significantly higher than severity scores reported in Snaith et al.'s (1995) normative sample ( $(M=0.44$ ,  $S.D.=0.97$ ,  $N=82$ ),  $F(1,193)=21.471$ ,  $P<0.001$ ). Notably, others reported similarly low severity scores on the SHAPS in healthy controls (Nagayama et al., 2012; Santangelo et al., 2009).

Spearman zero order correlation analyses revealed significant positive correlations between the SHAPS and the DASS-21 depression<sup>1</sup> ( $r=0.544$ ,  $P<0.001$ ) and anxiety ( $r=0.417$ ,  $P<0.001$ ) subscales as well as with the Y-BOCS total score ( $r=0.442$ ,  $P<0.001$ ; Fig. 1), obsessions ( $r=0.433$ ,  $P<0.001$ ), and compulsions ( $r=0.386$ ,  $P<0.001$ ) subscales. To examine whether anhedonia was associated with OCD symptom severity above and beyond the severity of depressive and anxiety symptoms, three univariate ANCOVAs were conducted comparing the anhedonic-OCD group ( $n=32$ ) with the non-anhedonic OCD group ( $n=81$ ) on the three Y-BOCS scores while controlling for depressive and anxiety symptoms. Results of these analyses (Table 3) confirmed significantly higher Y-BOCS scores among the anhedonic-OCD patients compared to non-anhedonic OCD patients when adjusting for depressive and anxious symptoms. Moreover, Spearman partial correlations revealed a significant association between the SHAPS score and the Y-BOCS total ( $r=0.283$ ,  $P=0.003$ ), obsessions

<sup>1</sup> Our findings of approximately 30% shared variance between severity of anhedonia and severity of depression is in line with other reports, suggesting that anhedonia (as measured by the SHAPS) is a related, albeit distinct, construct from depression. In addition, our results reveal that only 39.7% (27/68) of OCD patients with a comorbid diagnosis of any depressive disorder meet the definition for clinically significant anhedonia.

**Table 1**  
Sample characteristics.

Characteristics	OCD sample
Gender, % (N)	
Male	37.2% (42)
Female	62.8% (71)
Age	
Mean (S.D.)	35.75 (12.55)
Range	18–61
Marital Status, % (N)	
Single/never married	52.2% (59)
Married	43.4% (49)
Divorced/separated	4.4% (5)
Race, % (N)	
Caucasian	90.3% (102)
Asian	9.7% (11)
Ethnicity, % (N)	
Hispanic	4.4% (5)
Non-Hispanic	95.6% (108)
Education, % (N)	
Less than high school diploma	0.9% (1)
High school diploma or GED	8.8% (10)
Some college or a 2-year degree	19.5% (22)
Bachelor degree or higher	70.8% (80)
Cigarette smoking, % (N)	
Current smokers	13.3% (15)
Employment status, % (N)	
Full time	50.9% (56)
Part time	13.6% (15)
Student	11.8% (13)
Unemployed	14.5% (16)
Retired	2.7% (3)
On disability	6.4% (7)
Annual household income, % (N)	
Less than \$35,000/Year	20.7% (23)
\$35,000–\$50,000/Year	13.5% (15)
\$50,000–\$75,000/Year	27% (30)
\$75,000–\$100,000/Year	13.5% (15)
Over \$100,000/Year	25.2% (28)

Note. OCD=Obsessive Compulsive Disorder.

**Table 2**  
Sample clinical characteristics.

Outcome measure	OCD sample
Age first diagnosed <i>M(S.D.)</i>	24.72 (9.92)
Y-BOCS Total score <i>M(S.D.)</i>	19.04 (7.52)
Y-BOCS Obsessions <i>M(S.D.)</i>	10.06 (3.82)
Y-BOCS Compulsions <i>M(S.D.)</i>	8.98 (4.32)
DASS-21 Depression <sup>a</sup> <i>M(S.D.)</i>	13.48 (11.35)
DASS-21 Anxiety <sup>a</sup> <i>M(S.D.)</i>	9.7 (8.32)
DASS-21 Stress <sup>a</sup> <i>M(S.D.)</i>	18.29 (9.64)
Presence of anhedonia % (N) <sup>b</sup>	28.3% (43)
SHAPS total <i>M(SD)</i>	1.92 (0.38)
Comorbid any depressive disorder % (N)	60.2% (68)
Comorbid social anxiety disorder % (N)	10.6% (12)
Comorbid body dysmorphic disorder % (N)	4.4% (5)
Comorbid panic disorder % (N)	15% (17)

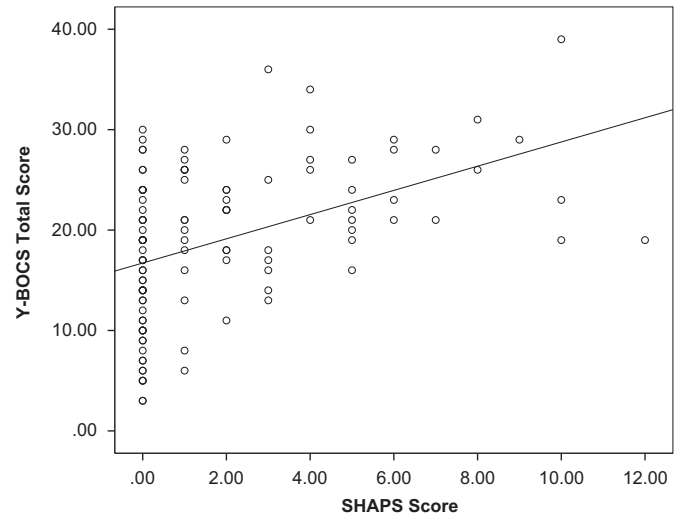
Note: Y-BOCS=Yale–Brown Obsessive Compulsive Scale; DASS-21=Depression Anxiety Stress Scale – 21 items; SHAPS=Snaith–Hamilton Pleasure Scale.

<sup>a</sup> In line with the scoring instructions, DASS-21 sum of subscales are multiplied by two.

<sup>b</sup> According to the criteria of Snaith et al. (1995).

( $r=0.261$ ,  $P=0.005$ ), and compulsions ( $r=0.255$ ,  $P=0.007$ ) subscales above and beyond the DASS-21 depression subscale scores.

Of the entire OCD sample, 13.3% ( $n=15$ ) were current smokers. This prevalence rate is lower than the prevalence found in a



**Fig. 1.** Correlation between severity of anhedonia (SHAPS score) and OCD symptom severity (Y-BOCS total score). Linear regression line is presented. No significant curvilinear distribution was found for this correlation.

US representative sample ( $N=84,700$ , 24% smokers; CDC, 2013) of adults without mental illness ( $\chi^2(1)=7.57$ ,  $P=0.006$ ), and did not differ significantly from a 2011 representative sample ( $N=33,014$ , 19% smokers; CDC, 2012) of the US adult population ( $\chi^2(1)=2.402$ ,  $P=0.12$ ). In addition, rates of smoking in our sample were lower in comparison to psychiatric disorders prominently characterized by anhedonia: schizophrenia (70–90%; Aubin et al., 2012), depressive disorders (37–69%; Ziedonis et al., 2008), and drug addiction (45–97%; Kalman et al., 2010; Pajusco et al., 2012). In light of evidence indicating that education and household income affects smoking rates (CDC, 2012), additional analyses were performed. We found no significant differences in education level and household income between OCD smokers vs. OCD non-smokers ( $P>0.05$ ). In order to rule out that our findings of reduced smoking prevalence in OCD may be due to OCD-specific reasons such as contamination concerns, we analyzed participants' open-ended responses concerning their primary and secondary reasons for smoking cessation attempts. The analysis yielded commonly-cited reasons such as peer and family pressure and health-related reasons but no indication of reasons associated with OCD-specific tendencies. With regards to dependence, the average FTND score represented a low level of dependence within the smoking subset.

Finally, in order to examine the association between anhedonia and smoking status, we compared the prevalence of clinically significant anhedonia and anhedonia severity between smokers and non-smokers in our sample. The OCD smokers and OCD non-smokers did not significantly differ in age, gender, or scores on the Y-BOCS, DASS-21, and SHAP anhedonia severity scale (all  $P>0.05$ ). However, a negative correlation, albeit non-significant, was found between the anhedonia severity score and number of cigarettes smoked per day ( $r=-0.485$ ,  $P=0.093$ ). Anhedonia severity score was also found to significantly negatively correlate with the FTND nicotine dependence score ( $r=-0.649$ ,  $P=0.012$ ). Notably, a strong positive correlation was found between the FTND dependence score and the number of cigarettes smoked per day ( $r=0.768$ ,  $P=0.002$ ).

#### 4. Discussion

To our knowledge, this is the first study to examine the clinical and phenomenological aspects of anhedonia in OCD. In accordance with our primary hypothesis, we found anhedonia to be prevalent in OCD (28.3%). This finding corresponds to recent imaging studies suggesting

**Table 3**  
Comparing Y-BOCS scores in OCD sub-samples, controlling for depressive and anxious symptoms.

	OCD+anhedonia (n=32)	OCD–anhedonia (n=81)	F(1,108)	P-value	Cohen's <i>d</i> effect size
Y-BOCS total score	23.81 (6.30)	17.16 (7.14)	6.585	0.012	0.97
Y-BOCS obsessions	12.44 (3.58)	9.12 (3.42)	5.052	0.027	0.76
Y-BOCS compulsions	11.38 (3.78)	8.04 (4.17)	5.380	0.022	0.83

Note. Y-BOCS=Yale–Brown Obsessive Compulsive Scale; OCD=Obsessive Compulsive Disorder.

deficient reward reactivity in OCD (Figeo et al., 2011; Jung et al., 2011; Choi et al., 2012). In addition, we found anhedonia severity to be higher in our sample than in normative samples (Franken et al., 2007). In line with our secondary hypothesis, we found anhedonia to be associated with OCD symptom severity over and above depressive symptoms. This finding suggests that greater OCD severity would entail more severe anhedonia. More importantly, this finding suggests that negative affect and depressive severity do not sufficiently account for the presence of anhedonia in OCD. One speculative account for the residual association between anhedonia and OCD is the need for explicit control of actions and thoughts in OCD, which in turn may hinder the ability to enjoy 'unstructured' positively rewarding activities. Providing support for this assumption, Vulink et al. (2006) recently found reduced capacity to experience pleasure from sexual activities in OCD participants as compared to controls. Although this effect was partially associated with disgust of bodily secretion (Aksaray et al., 2001), it is plausible that the unstructured nature of sexual activity together with the intolerance of uncertainty observed in OCD may impact the ability of these patients to enjoy sex.

These findings may be important on two accounts. First, little is known about reward mechanisms and incentive motivation (motivational learning) in OCD, both of which may play an important role in the psychopathology of the disorder. Our literature review did not yield any empirical studies on anhedonia in OCD, which is surprising given the known association and high comorbidity rates between depression and OCD (Ruscio et al., 2010). This may be due to the putative association between anhedonia and depression. For example, in the tripartite model of anxiety and depression, anhedonia and low positive affect were posited to represent the component of affect specific to depression (Clark and Watson, 1991) and were found to successfully differentiate between depression and anxiety (Watson et al., 1995). Second, current assessment procedures and CBT/ERP protocols for OCD treatment do not target anhedonia, which may improve significantly when treated with behavioral activation therapy (Dichter et al., 2012).

Unlike depression, OCD may be a disorder of negative reinforcement, along with the unique pattern of brain activation observed in OCD and evidence regarding unusually low prevalence of cigarette smoking, motivated us to examine the association between smoking and anhedonia in OCD. We found that 13.3% of our sample were daily smokers; this is lower than the reported prevalence rate in the general adult population in the US (19; CDC, 2012) and may be among the lowest rates in psychiatric disorders (Dome et al., 2010). This finding is in line with the notion of OCD as a protective factor against smoking and studies reporting low prevalence of smoking in OCD (5.5–14.5%; Bejerot and Humble, 1999; Baker–Morissette et al., 2004; McCabe et al., 2004). With regards to the association between smoking and anhedonia, we set forth an exploratory a-priori hypothesis that smoking will be negatively associated with anhedonia in OCD. Although we did not find significant differences in the presence or severity of anhedonia between smokers and non-smokers with OCD, we found that the number of cigarettes smoked per day was inversely correlated with the severity of anhedonia. The latter was also inversely correlated the level of nicotine dependence in smokers with OCD.

These results stand in direct contrast to the known positive association between anhedonia and smoking status (McLeish et al.,

2006; Gregor et al., 2007; Ameringer and Leventhal, 2010; Audrain-McGovern et al., 2012) as well as with the positive association between nicotine dependence and anhedonia (Cook et al., 2007; Leventhal et al., 2009). These findings, though seemingly counter-intuitive, are in line with the neurobiological and phenomenological similarities observed between OCD and drug addictions (Denys et al., 2004; Fontenelle et al., 2011). OCD may be viewed as a disorder of negative reinforcement in that OCD-related behaviors or mental rituals target anxiety/stress reduction (Fontenelle et al., 2011). Moreover, as proposed by Fontenelle et al. (2011) "Severe lifelong OCD may take precedence over normal hedonic behaviours, 'resetting' the reward system and imparting compulsive actions a hedonic quality because patients don't have time to obtain pleasure from anything other than OCD" (page 833). With regards to smoking behavior under the conceptual framework of "behavioral addiction," (Holden, 2001; Fontenelle et al., 2011) the vicious cycle of negative reinforcement results in incentive salience of the negatively reinforced stimuli over natural reinforcers (i.e., nicotine/smoking; Koob and Volkow, 2010). Notably, anxiety disorders such as generalized anxiety disorder, panic disorder and specific phobia, are associated with increased risk for smoking (e.g., Goodwin et al., 2012). However, as opposed to OCD, the governing mechanism in anxiety disorders is *avoidance* from the anxiety-provoking stimuli. Indeed, the prominent negative reinforcement cycle seen in OCD does not characterize other anxiety disorders, which are now distinct from obsessive compulsive and related disorders in the DSM-5 (American Psychiatric Association, 2013).

This study is not without limitations. First, our sample was primarily comprised of Caucasians and women, which may limit generalizability. In addition, a response bias inherent to online studies may be present. Moreover, while we employed cutting edge control measures to maximize validity and integrity of our data, including comparison to a subgroup of verified OCD participants, employing self-reported measures for diagnosis and assessment for comorbidities has some inherent limitations. Notably, it is important to keep in mind that multiple web-based psychology studies have yielded reliable results when appropriate control measures were employed (Birnbaum, 2004; Gosling et al., 2004). The lack of an active control group in this study may further limit findings. However, the very low prevalence of anhedonia in healthy controls reported across studies (2–5%; Snaith et al., 1995; Franken et al., 2007), support our conclusion that the prevalence of anhedonia found in our OCD sample may be significant. In addition, results of the analyses pertaining to our OCD-smokers subsample should be interpreted with caution due to the small sample size. Finally, several statistical tests were performed in the present study without alpha corrections for multiple comparisons. However, given the exploratory nature of the present investigation, such corrections were not implemented in order to reduce the probability of type II error.

## 5. Conclusion

The results of this study, exemplifying the presence of clinically significant anhedonia in OCD, may hold important theoretical and clinical implications. Hitherto overlooked by clinicians and

researchers, anhedonia may be an important symptom in OCD over and above its association with depressive severity. In addition, we suggest a unique mechanism underlying anhedonia in OCD that demands further exploration into the role of anhedonia in psychopathological mechanisms in OCD. Though further research is needed, we suggest incorporating anhedonia in the assessment and treatment processes of OCD, including the incorporation of behavioral activation protocols. Future research should attempt to incorporate anhedonia in psychopathological and neurobiological models of OCD. Notably, further empirical examination of correlates of anhedonia with different OCD dimensions (e.g., washing, checking, etc.) is warranted given the heterogeneous nature of OCD. Finally, the reduced prevalence of tobacco smoking in OCD and its negative association with anhedonia may contribute to our understanding of the important role of reward mechanisms in OCD, inclusive of its clinical, neurobiological and cognitive aspects.

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