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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

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