
MOOD IN RELATION TO SUBCLINICAL OBSESSIVE-COMPULSIVE SYMPTOMS

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Obsessive-compulsive disorder (OCD) is considered an anxiety disorder, but shows comorbidity with other disorders in the affective and impulsive-compulsive spectra, including anxiety disorders, major depression, and drug addictions. Subclinical OCD symptoms are relatively common in nonclinical populations and share common neurobiological substrates with clinical OCD. In this non-clinical community sample, the relationship between the severity of obsessions and compulsions, as measured by the Yale-Brown Obsessive Compulsive Scale, related to the intensity of negative emotions (anger, depression, tension, confusion, and fatigue) but not positive emotion (vigor), as measured by the Profile of Mood States. These relationships were independent of demographic influences and psychoactive drug use frequency (alcohol, cannabis, opioid, major stimulants, MDMA, and hallucinogens). These likely reflect common neurobiological substrates for emotional and behavioral regulation in prefrontal-subcortical/limbic circuits, which show normal variations in the general population.

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Obsessive-compulsive disorder (OCD) is considered an anxiety disorder based on the prominent role anxiety plays in its core symptoms. Obsessions are defined as recurrent and intrusive thoughts, impulses, or images that create significant anxiety or distress, and compulsions are behaviors that alleviate the distress (American Psychiatric Association, 2000). However the emotional disturbances in this disorder are not limited to anxiety.

Several conditions are frequently comorbid with OCD, and it has been conceptualized as a spectrum disorder because it shares with several disorders features of phenomenology, clinical course, treatment response, genetic predisposition, and probable common pathophysiology. These include impulse-control disorders (e.g., trichotillomania, pathological gambling, compulsive spending), somatoform disorders (e.g., body dysmorphic disorder and hypochondriasis), eating disorders (e.g., anorexia and binge eating), compulsive sexual disorders, and Tourette's syndrome (Hollander, 1993; Rasmussen, 1994; McElroy, Phillips, & Keck, 1994). Hollander conceptualizes these on a continuum with risk avoidance on the compulsive end and risk seeking at the impulsive end (Hollander, 1993). OCD, in particular, is more closely associated with spectrum disorders than other anxiety disorders, such as social phobia and panic disorder (Richter et al., 2003).

It has also been proposed that these impulsive-compulsive spectrum disorders belong to a larger family of affective spectrum disorders (McElroy et al., 1994). These are proposed to include mood-related disorders (e.g., major depressive disorder, dysthymic disorder, premenstrual dysphoric disorder), anxiety disorders (e.g., panic disorder, posttraumatic stress disorder, social phobia, generalized anxiety disorder), attention-deficit/hyperactivity disorder, and others not typically thought of as primary psychiatric illnesses (e.g., cataplexy, fibromyalgia, irritable bowel syndrome, and migraine). Hudson and colleagues (1990, 2003) suggest this grouping of disorders based on response to serotonergic medications and strong aggregation in families.

In support of its designation as a spectrum disorder, several studies demonstrated a variety of emotional dysfunction in OCD. Rates of the comorbidity of OCD and major depression have been reported between 35% and 75% (Sciuto et al., 1995). Overbeek and colleagues (2002) reported depression in one third of a clinical sample of individuals with OCD, who also had a diminished response to treatment. Psychopathology may vary with subtypes of OCD: hoarding has been associated with greater elevated depression, anxiety, social dysfunction, and symptoms of dependent and schizotypal personality disorder compared to nonhoarding OCD (Frost et al., 2000). OCD has also been associated with anxiety, anger, and aggression (Stein et al., 1991).

Elevated scores of anger, depression, and anxiety were reported in women with OCD, who scored similarly to those with bulimia nervosa and greater than controls (Rubenstein et al., 1995)

A great deal of progress has been made toward understanding the neurobiology of obsessive-compulsive disorder. Functional and structural neuroimaging studies convergently point to dysfunction in prefrontal-subcortical circuits (Saxena et al., 2001; Tekin & Cummings, 2002). Structural neuroimaging shows abnormalities of orbitofrontal cortex and the striatum, whereas magnetic resonance spectroscopy shows decreased striatal and cingulate N-acetyl-aspartate, implying neuronal loss (Brambilla et al., 2002). Functional neuroimaging studies show hyperactivity in orbitofrontal cortex and the caudate nucleus. Hyperactivity in the anterior cingulate gyrus has also been reported, and cingulotomy is a surgical technique used in cases of intractable OCD (Kim et al., 2003). A variety of neuropathology in these structures, including seizure discharges, tumors, toxic exposure, trauma, and ischemic lesions, can cause OCD (Tekin & Cummings, 2002). OCD symptoms are associated with activation in the same regions in both OCD patients and healthy controls: dorsal and ventral prefrontal cortex and limbic structures (Mataix-Cols et al., 2003). There is evidence for serotonergic dysfunction in prefrontal-subcortical circuits in OCD. Genetic polymorphisms of the serotonin transporter (5-HTT) are associated with OCD, and serotonin reuptake inhibitors ameliorate symptoms in 40 to 60 percent of cases (Camarena et al., 2001; Kaplan & Hollander, 2003).

Other disorders of the impulsive-compulsive and affective spectra show aberrant activity in prefrontal-subcortical circuits (Davidson et al., 1999; Sheline, 2003). Other emotional states are associated activity in prefrontal-subcortical circuits. Anger, for example, is associated with increased activity in left orbitofrontal cortex, right anterior cingulate cortex, and bilateral anterior temporal poles (Dougherty et al., 1999). Affective and anxiety disorders may also commonly share abnormalities of neurotransmitter metabolism in prefrontal-subcortical circuits (Blier & Abbott, 2001; Krakowski, 2003).

Obsessions and compulsions are relatively common in nonclinical populations (Rachman & de Silva, 1978). In one epidemiologic survey, between 22% and 26% of 2200 respondents reported having obsessions or compulsions (Stein et al., 1997). However, only 0.6% received a DSM-IV diagnosis of OCD after clinical reappraisal. Many such individuals have been designated as "subclinical OCD" because they share the same characteristics but may not warrant a clinical diagnosis (Morris et al., 2000). Nonetheless, subclinical OCD has many features similar to clinical OCD (Frost et al., 1994;

Gershunny & Sher, 1995). For example, they show a similar pattern of deficits on neuropsychological tests sensitive to prefrontal-subcortical function, including design fluency and the Tower of Hanoi (Tallis, 1997; Mataix-Cols et al. 1999a, 1999b). Further, these deficits correlate with ratings of obsessionality or compulsions.

Substance abuse has been conceptualized as a compulsive behavior (Goodman, 1998; Hollander & Rosen, 2000). In support of this, obsessive and compulsive phenomena in alcoholism were evident in a modified version of the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) (Modell et al., 1992). In female subjects of a large sample, OCD was associated with dependence on nicotine, alcohol, and sedatives (Grabe et al., 2001). Compulsive phenomena in OCD and drug addictions may have some overlapping neurobiological basis in prefrontal-subcortical-limbic circuits (Robbins & Everitt, 2002; Volkow et al., 2002).

OCD and many disorders of emotion share common phenomenology and neurobiological underpinnings. Given the prevalence of subclinical OCD in the general population, this study sought to examine the relationships between emotional states and the severity of OCD symptoms in a nonclinical sample. Further, it sought to evaluate the contribution of psychoactive drug use to these relationships.

METHODS

Participants

Participants were a convenience sample ($N = 168$; 106 female, 6 male, 2 did not indicate sex) of adults. The age of participants ranged from 18 to 67 years ($M = 28.4$, $SD = 12.2$), and they had completed between 10 and 19 years of education ($M = 14.2$, $SD = 1.8$). They were recruited by research assistants who were asked to find community dwelling, noninstitutionalized adults. Participants were recruited via word of mouth from the local community (e.g., the college campus places of work).

An institutional review board approved the study and all participants agreed to an implied consent form in accordance with the Declaration of Helsinki and the ethical principles of the American Psychological Association. Participants did not receive any financial compensation for participating. In order to protect anonymity and confidentiality and encourage honest responding, participants were not asked any identifying information other

than basic demographics (age, sex, and education). They were also asked to fill out the questionnaires without being observed and to seal them in an envelope before returning them to the research assistant.

Measures

Yale-Brown Obsessive Compulsive Scale (YBOCS). The YBOCS is a commonly used, valid and reliable scale for rating obsessive and compulsive symptoms (Goodman et al., 1989a, 1989b). Five items pertain to obsessions: 1. Time Spent on Obsessions, 2. Interference from Obsessions (in everyday activities), 3. Distress from Obsessions, 4. Resistance to Obsessions, and 5. Control Over Obsessions. Items 6–10 are analogous for compulsions. The items are rated on a 5-point Likert scale (0–4), ranging from least to greatest severity. Separate scores were obtained for obsessions (YBOCS_o), compulsions (YBOCS_c), and the total score (YBOCS_t).

Profile of Mood States (POMS). A 35-item version of the POMS was utilized in this study (Grove & Prapavessis, 1992). This form has six subscales, identical to those used by the full POMS (Anger, Confusion, Depression, Fatigue, Tense, and Vigor). Total mood disturbance (TMD) was calculated as a sum of the negative emotional scales minus Vigor. Items were rated along a Likert scale (0 = Not at all, 1 = A little, 2 = Moderately, 3 = Quite a bit, 4 = Extremely). Both the long form and short form of the POMS have shown good reliability and validity (e.g., Reddon et al., 1985; Grove & Prapavessis, 1992).

Drug Use Frequency Questionnaire (DUF). The DUF is a questionnaire with established validity that inquires about the current use of psychoactive drugs (O'Farrell et al., 2003). Items are rated on a Likert scale, ranging from least to most frequent use: 0 = Never, 1 = Several Times, 2 = Once/month, 3 = Several times/month, 4 = 1–2 days/week, 5 = 3–4 days/week, 6 = 5–6 days/week, 7 = Every day. The drug use inquired in this study included tobacco, caffeine, alcohol, cannabis, opioids, major stimulants (e.g., cocaine, methamphetamine), methylenedioxymethamphetamine (MDMA), and hallucinogens. Colloquial names were provided for each class of drug to optimize the accuracy of reporting (e.g., “cocaine, amphetamine, speed, meth, and crank” for major stimulants). There were a total of 156 caffeine users, 77 tobacco users, 150 alcohol users, 49 cannabis users, 9 opioid users, 14 major stimulant users, 12 MDMA users, and 14 users of hallucinogens.

RESULTS

YBOCS total scores (Figure 1) ranged from 0 to 25 ($M = 9.8$, $SD = 6.4$). In terms of severity of OCD symptoms, these scores would classify 73 as sub-clinical, 72 as mild symptoms, 23 as moderate severity, and none as severe. These scores are clearly lower than those reported by Stein and colleagues (1997) for individuals with clinical OCD ($M = 19.0$, $SD = 4.6$). POMS total mood disturbance ranged from -18 to 109 ($M = 24.2$, $SD = 23.9$).

Pearson correlations were obtained for subscales of the YBOCS and POMS (Table 1). Positive correlations emerged between YBOCS_o and YBOCS_c scales and all of the negative affect scales (Anger, Confusion, Depression, Fatigue, and Tension) of the POMS. However, Vigor bore no relationship to either YBOCS_o or YBOCS_c scales. These relationships remained significant after partial correlations were performed to control for the influences of demographic variables (age, sex, and educational level), and the frequency of psychoactive drug use (caffeine users, tobacco users, alcohol users, cannabis users, opioid users, major stimulant users, MDMA users, and hallucinogens). These also remained significant after applying a conservative Bonferroni correction to the alpha level for the number of correlations performed ($p = .001$ for 42 correlations). The correlations obtained explained between 14% and

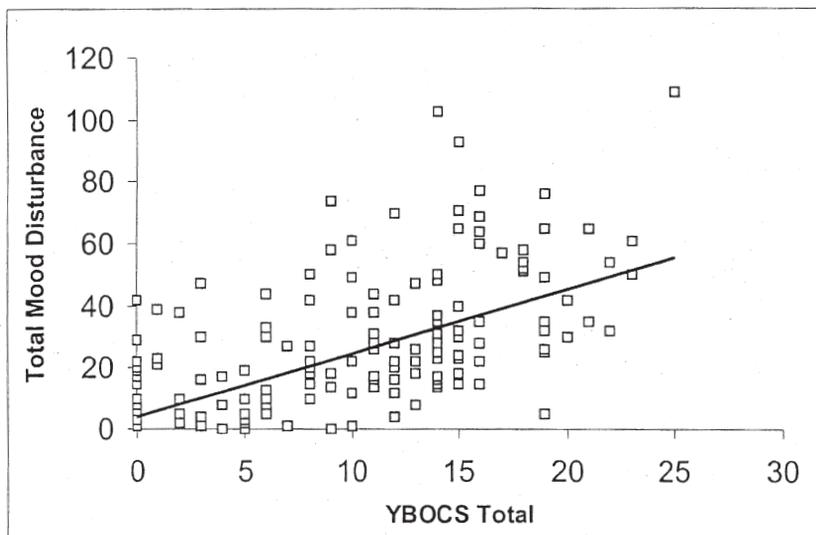


Figure 1. Scatterplot of the Yale-Brown Obsessive Compulsive Scale (YBOCS) total score and Profile of Mood States—Total Mood Disturbance (TMD).

Table 1. Correlations between the Yale Brown Obsession (YBOCS_O), Compulsion (YBOCS_C), and the Profile of Mood States subscales

	YBOCS		YBOCS		YBOCS	
	O ^a	C ^a	O ^b	C ^b	O ^c	C ^c
Anger	.53*	.40*	.50*	.38*	.52*	.38*
Confusion	.48*	.42*	.45*	.40*	.46*	.41*
Depression	.53*	.40*	.50*	.37*	.50*	.36*
Fatigue	.37*	.40*	.37*	.38*	.40*	.37*
Tense	.53*	.40*	.52*	.38*	.55*	.38*
Vigor	.01	.04	-.04	.02	-.07	-.05
TMD	.55*	.44*	.53*	.42*	.56*	.43*

N = 168, ^abivariate correlations and ^bpartial correlations controlling for age, sex, and education [*df* = 160]; ^cpartial correlations controlling for age, sex, education, and drug use [*df* = 151]; *p* < .001.

30% of the variance. YBOCS_O showed a slightly greater correlation with TMD than YBOCS_C ($r = .55, p < .001$ and $r = .44, p < .001$, respectively.)

DISCUSSION

This study demonstrates positive relationships between the severity of OCD symptoms and several negative emotional states in individuals with subclinical levels of OCD. Although OCD is defined, in part, by the presence of anxiety-producing obsessions, the distress in subclinical OCD is not limited to anxiety, and instead includes a variety of negative emotions. These findings were anticipated based on studies of clinical severity OCD populations who show greater anxiety, depression, anger, fatigue, and cognitive dysfunction compared to healthy controls. However, these relationships are not limited to clinical severity of OCD, but extend into subclinical OCD as well and appear to be continuously related. This relationship is logical because greater emotional distress would provide a stronger drive for compulsive behaviors.

The relationships observed here were not due to demographic influences. Further, although psychoactive drug use (including both legal and illegal substances) is common in the general population, and many forms of drug use do fit compulsive patterns of use, the correlations here were not notably diminished when the frequency of drug use was partialled out.

Common putative neurobiological mechanisms for OCD symptomatology and negative emotions provide a possible framework for interpreting the relationships observed here. OCD symptoms are associated with aberrant activity in prefrontal-striatal/limbic circuits. Anger, anxiety, fatigue, and depression

also have similarly been associated with dysfunction in prefrontal-subcortical/limbic circuits (e.g., Dougherty et al., 1999; Davidson et al., 2000; Chaudhuri & Behan, 2000). All of these states have also been associated with altered serotonergic functioning in the brain (Davis et al., 2000; Cleare & Bond, 1995; Blier & Abbott, 2001; Krakowski, 2003). Thus, it is feasible to hypothesize that underlying differences in prefrontal-subcortical/limbic circuits, including their neurochemical modulation, can lead to constellations of changes in emotional and behavioral states, which is the underlying argument behind spectrum disorders (McElroy et al., 1994). The continuous relationships observed here, which range from mild to moderate in severity, likely represent subtle variations in parameters of neurochemical and neuroanatomical functioning. For example, there are normal variations in the morphological development of neuroanatomical structures across individuals and across the life span that could underlie normal variations in behavior (Pfefferbaum et al., 1994).

This study further demonstrates some of the known similarities between clinical and subclinical OCD. Common features of emotion, of personality, cognition, and behavior have been established, and extend across a range of negative emotion. Future work in this area may further investigate other behaviors associated with subclinical OCD, particularly because people who manifest it may make up such a sizable proportion of the general population.

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