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The role of the orbitofrontal cortex in normally developing compulsive-like behaviors and obsessive–compulsive disorder

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Abstract

Mounting evidence concerning obsessive–compulsive disorders points to abnormal functioning of the orbitofrontal cortices. First, patients with obsessive–compulsive disorder (OCD) perform poorly on tasks that rely on response suppression/motor inhibition functions mediated by the orbitofrontal cortex relative to both normal and clinical controls. Second, patients with OCD exhibit functional hyperactivity in lateral orbitofrontal and related structures corresponding with symptom severity. In this article, we compare these neurocognitive correlates of OCD with the executive and neural underpinnings of "compulsive-like" behaviors that are common in normal childhood. We discuss the phenomenology and natural history of normative compulsive-like behaviors as well as the behavioral, emotional, and cognitive continuities between typical and pathological obsessive–compulsive behaviors. We then examine associations between children's executive performance deficits and their observed compulsive-like characteristics. We relate these patterns to executive deficits shown by adults with OCD. Finally, we speculate on the developmental neurobiology of children's compulsive-like behaviors, with particular attention to orbitofrontal functions including behavioral and emotional regulation, and we suggest similarities and differences with the neurobiology of OCD. In making these comparisons, we hope to open a dialogue between researchers who study underlying brain pathologies associated with OCD and those who explore the neurocognitive bases of normal development.

1. Introduction

In this article, we discuss recent research and theory on the development of rituals, habits, and compulsive behaviors, and the possible role of the orbitofrontal cortices in the development and maintenance of these behavioral patterns. Our discussion begins with a review of the neurocognitive deficits associated with obsessive–compulsive disorder—inferred from tasks of executive function as well as neuroimaging studies. After relating these deficits to covariance between children's compulsive and executive activities, we consider how interactions between the executive and emotional functions of the orbitofrontal cortex play a role in both pathological and normative compulsive behavior patterns.

2. Obsessive–compulsive disorder

Obsessive–Compulsive Disorder (OCD) is an anxiety disorder characterized by intrusive, troubling thoughts that are perceived as the products of one's own mind (i.e., not thought insertion as in hallucinations associated with schizophrenia) or repetitive, compulsive behaviors (APA, 1997). The obsessions or compulsions are time-consuming, and lead to impairment in functioning. The thoughts and behaviors associated with OCD are viewed as senseless, and egodystonic—that is, they are counter to the individual's motives, goals, identity, and self-perception and thus create significant subjective distress. The clinical presentation of OCD is highly variable, but some obsessions may include thoughts of death or harm befalling a loved one, doubting (that one *may* have hurt someone), thoughts/fears of contamination, blasphemous thoughts or impulses, counting, and preoccupations with symmetry or neatness.

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Compulsions are repetitive behaviors or rituals that are carried out in a rigid, ritualized, and circumscribed manner. Like obsessions, compulsions are egodystonic, such that the individual does not experience gratification from carrying out the compulsion; rather, the compulsion is viewed as senseless and troubling, but he or she feels compelled to perform it nonetheless. Traditionally, compulsions are thought to neutralize, prevent, or reduce stress or anxiety. Common compulsions may include excessive handwashing, elaborate rituals such as a specific dressing or eating routine, touching or tapping, lining up objects in straight lines or symmetrical patterns, retracing steps, and checking. Certain compulsions may be concomitant with particular obsessions. Fear of contamination, for example, may accompany excessive handwashing. Doubting may accompany checking behaviors. Thus, in some instances, while clearly excessive, there is some "logical," causal connection between obsessions and compulsions, whereas in other instances the connection between them seems more remote and may invoke magical thinking in order to tie together the thoughts and behaviors (Evans, Milanak, Medeiros, & Ross, 2002).

Though both obsessions and compulsions are troubling to the individual with OCD, carrying out a particular compulsion may temporarily relieve the anxiety brought on by the obsession. Handwashing, as just noted, may reduce the anxiety produced by thoughts of contamination. The excessive nature of the compulsion, however, creates its own distress and it appears that the individual may be caught up in a kind of negative reinforcement loop. Some patients with OCD report that they must engage in a compulsion a certain number of times, whereas others will repeat the behavior until it satisfies some sensory-perceptual criterion as feeling "just right." Perfectionism and scrupulosity are also features of the disorder that may involve having to say or do things in a certain order, or a certain number of times; but more often these behaviors are associated with Obsessive–Compulsive Personality Disorder (OCPD).

OCD is among the most common psychiatric disorders, with lifetime prevalence estimates as high as 2.5% (Karno, Golding, Sorensen, & Burnam, 1988). Modal age of onset is 6–15 years of age for males and 20–29 years of age for females. Familial predisposition points to a genetic role in OCD. Concordance rates are higher monozygotic than dizygotic twins, and children of first-degree relatives with OCD are at a greater risk for developing the disorder than is the general population (Lenane et al., 1990; Pauls, Alsobrook, Goodman, Rasmussen, & Leckman, 1995; Pauls, Raymond, & Robertson, 1991). Family history of Tourette syndrome (TS) is also associated with increased risk of developing OCD, and TS is a common comorbidity of OCD, occurring in 35–50% of OCD cases (Evans, King, & Leckman, 1995). Some OCD–TS comorbidity estimates are even higher, but some questions remain as to whether these reflect actual co-morbidity or difficulty in distinguishing between certain compulsions and complex motor tics (Evans et al., 1995).

Some motor tics are relatively easily distinguished from compulsions, in that tics are rapid, jerking movements whereas compulsions are not. Some tics, though, involve complex muscle groups and are relatively orchestrated, making them appear more compulsive-like. Similarly, some compulsions may involve touching or tapping that is virtually indistinguishable from certain motor tics. Stereotypic motor movements common to pervasive developmental disorders are more rhythmic than compulsions or tics. And self-injurious behavior, though repetitive and seemingly "compulsive," is considered a stereotypic movement and not a compulsion. Perseverative behaviors are a broad class of behaviors, referring to previously acquired motor responses or cognitions that, although appropriate in some contexts, are inappropriate in others yet cannot be inhibited. Thus, many tics, compulsions, and stereotypic behaviors may be considered perseverative in the sense that they are motor responses with adaptive origins that are inappropriate to a given situation and difficult, if not impossible, to inhibit. It is important to note that while significant efforts have been made to differentiate these various kinds of motor movements, their similarities warrant further scrutiny as possibly reflecting similar underlying etiologies.

In childhood onset OCD, compulsions tend to emerge significantly earlier than obsessions. Whether the differential onset of obsessions and compulsions actually reflects the natural history of childhood OCD is unclear, given that younger children may be unable or unwilling to express the content of their obsessive thoughts. The presentation of child and adult forms of OCD are quite similar, though children may have poorer insight. Given that symptom expression is generally similar for children and adults with OCD, it has been suggested that whatever neurological maturation is required for the presentation of OCD is intact relatively early in development (Bolton, 1996). This is clearly an important clue for relating the neurobiology of normal childhood compulsions to that of OCD.

The evolution of our understanding of OCD over the past century reflects paradigmatic changes in our conceptions of mental illness. Models of pathogenesis have included psychoanalytic interpretations as well as models of operant conditioning fueled largely by the success of exposure therapy (e.g., in panic and agoraphobia). More recently, cognitive and cognitive-behavioral accounts for OCD have come to the fore, and these too have been linked with some therapeutic success.

Still more recently—in the past two decades—advances in the neurosciences have shed light on possible

neurobiological and neuropsychological mechanisms of obsessive–compulsive disorder. Such advances promise to integrate previous models of pathogenesis in that they speak to the mechanisms that underlie anxiety and arousal, learning and memory, and a variety of "executive" functions that are thought to be related to intrusive thoughts and repetitive behavior patterns. In the following sections, we review briefly the neuropsychological and neurobiological literature on obsessive–compulsive behaviors. Next, we present recent empirical and theoretical efforts suggesting similarities as well as differences in the development of normative compulsive behaviors in children and the symptoms associated with OCD.

3. The neuropsychology of OCD[1]

The behaviors associated with (adult-onset) OCD have been linked to a wide variety of cognitive deficits (Tallis, 1997). These findings are relatively inconsistent, largely born of differences in methodological approaches, such as inclusion–exclusion criteria for subjects, medication status, matching approaches, comorbidities, as well as assessment measures (Schultz et al., 1999). Nonetheless, some patterns do emerge; suggesting that OC behaviors are associated with neuropsychological deficits.

Numerous studies report that patients with OCD evidence deficits in executive functions. Executive functions refer to a broad range of abilities including planning, goal-directed behaviors, self-regulation, maintenance of cognitive set and set-shifting ability, impulse control, motor inhibition, sustained attention, and working memory (Schultz et al., 1999). Working memory serves to hold goal-oriented representations "on line," so that problems can be resolved without reliance on previously learned associations. Deficits in nonverbal and procedural memory have also been explored, and some impairment in visual-motor integration and visuospatial functioning has also been reported in OCD (Schultz et al., 1999). We now review evidence for associations between OCD symptoms and three distinct executive functions, working memory, set-shifting, and response inhibition.

3.1. Working memory deficits

While much has been made of working memory deficits in OCD, the empirical findings are mixed. A comparison of Tower of London (ToL) performance (the ToL assesses primarily planning, but also involves working memory) between patients with OCD, other psychiatric patients as well as normal controls yielded no significant findings (Purcell, Maruff, Kyrios, & Pantelis, 1998). This finding corroborates previous null findings in the Trail-making test (Schmidtke, Schorb, Winkelmann, & Hohagen, 1998; Veale, Sahakian, Owen, & Marks, 1996). Patients with OCD did, however, perform worse than comparison groups on a task of spatial working memory involving visual memory for prior actions (Schmidtke et al., 1998). This makes sense when considering that certain OC behaviors involve doubting and checking—behaviors that one might associate with visual memory for actions, though these studies did not differentiate among subtypes or quality of OC symptom expression. Nevertheless, a link between OC behavior and working memory deficits has not been clearly demonstrated.

3.2. Set-shifting deficits

Data on set-shifting ability are somewhat more consistent. Several studies report that OC patients perform significantly worse than other psychiatric groups and normal controls in terms of their ability to shift cognitive set (Abbruzzese, Bellodi, Ferri, & Scarone, 1995; Head, Bolton, & Hymas, 1989; Hollander & Wong, 1996; Hymas, Lees, Bolton, Epps, & Head, 1991; Lucey et al., 1987). Perhaps the most common neuropsychological task of set-shifting abilities is the Wisconsin Card Sort Task (WCST). Lucey et al. (1987) reported that OCD subjects made significantly more perseverative errors on the WCST than normal controls. In addition to group differences, linear associations were found between the obsessive factor of the Yale-Brown Obsessive Compulsive Scale and WCST errors. The overall cognitive rigidity and inflexibility that comprises the clinical presentation of many OC patients corroborate associations between poor set-shifting and OCD.

Patients exhibiting obsessional slowness performed worse on cognitive set-shifting tasks than OC patients not exhibiting obsessional slowness, but no linear relation between slowness and set-shifting emerged (Gehring, Himle, & Nisenson, 2000). Moreover, some patients with OCD are shown to have longer latency in their responses on EF tasks (Gehring et al., 2000). Presumably, greater response latency indicates that subjects spend more time generating alternate responses. Checking or doubting, a common symptom of OCD, may translate into longer response latencies, resulting from heightened error detection. The longer latency of response for OC patients seems peculiar to EF tasks, however, as latency of response on control (non-EF) tasks was not associated with OC symptoms (Behar et al., 1984). Thus, individual elements of behavioral responses can be performed at normal speeds, but responses that involve linking and sequencing behaviors are slowed in patients with OCD (Sawle, Hymas, Lees, & Frackowiak, 1991; Schultz et al., 1999).

[1] Portions of this section were published in Schultz, Evans, and Wolf (1999).

Non-clinical OC behavior patterns may also be linked with poorer set-shifting abilities. Zohar, LaBuda, and Moschel-Ravid (1995) report that, within a normal population, scores on the Maudsley Obsessive–Compulsive Inventory were related to the WCST. As reported in studies on clinical samples, number of perseverative errors and reaction time were both significantly related to Maudsley scores on the checking factor, though the amount of shared variance was modest. In addition, normal subjects who scored one standard deviation above the mean on a self-report OC inventory performed poorly on the Tower of Hanoi (ToH; a measure of planning ability), relative to subjects scoring one standard deviation below the mean on the OC inventory (Matiax-Cols et al., 1999). Checking behaviors and total score were both linearly related to number of moves and time to complete the TOH. Both sets of findings argue for a continuum approach to OCD, whereby the same cognitive deficits associated with OCD may be found in subclinical obsessions and compulsions.

Not all studies find that set-shifting deficits are common to OCD. When matching OC patients to controls on demographic variables such as age, sex, and education level, no group differences emerged on set-shifting as assessed by the WCST (Abbruzzese, Ferri, & Sarcone, 1995). Others (e.g., Grau, 1991) report that IQ is a better predictor of WCST performance than OCD, and Cox (1997) reviews evidence that WCST deficits associated with OCD may be entirely explained by IQ differences. These and other researchers suggest that set-shifting deficits in OCD may have been overestimated (Schultz et al., 1999).

3.3. Response inhibition deficits

Perhaps the most consistent evidence for EF deficits in OCD come from tests of motor inhibition and response suppression. A study of treatment-naive, recent-onset OCD children and adolescents revealed deficits on tests of response suppression, with severity of OC symptoms positively correlated with response suppression errors (Rosenberg, Dick, O'Hearn, & Sweeney, 1997). Others report significant correlations between OC symptom severity and children's inability to suppress automatic responses (Cox, 1997). Rosenberg and colleagues (Rosenberg et al., 1997) found that, relative to matched controls, 12 patients with OCD demonstrated poor performance on an oculomotor suppression task. These findings are consistent with previous work (Tien, Pearlson, Machlin, Bylsma, & Hoehn-Saric, 1992) noting greater error rate on a goal-guided anti-saccade task that requires subjects to move their eyes away from visually presented targets (thus inhibiting a powerful reflexive tendency to orient toward novel stimuli; Schultz et al., 1999). Similarly, several studies report deficits in patients with OCD on tasks of object alternation, but not on set-shifting tasks. Object alternation tasks require that subjects give a certain response for one stimulus, but not for another, followed by reversal of the response rule (Cavedini, Ferri, Scarone, & Bellodi, 1998; Gross-Isseroff et al., 1996). Not only did these studies reveal group differences, but they also reported linear associations between symptom severity and perseverative errors of commission on response suppression/object alternation tasks (Gross-Isseroff et al., 1996).

3.4. Summary

Taken together, the various lines of evidence for EF deficits in OCD consistently show impairment of response suppression and motor inhibition abilities, with somewhat less consistent evidence for reduced set-shifting abilities, and patchy evidence for working memory deficits. Also notable is the finding that longer response latencies are common to tests of motor inhibition/response suppression as well as set-shifting abilities.

4. The neurobiology of OCD

As noted earlier, several regions of the prefrontal cortices presumably subserve the executive functions. Specifically, the dorsolateral prefrontal cortex (DLPFC) is associated with cognitive set-shifting, planning, and working memory (Schultz et al., 1999). Though some questions remain as to the specificity of the DLPFC in set-shifting ability, a recent meta-analytic study suggests that WCST perseverative errors are more common in patients with damage to the dorsolateral prefrontal cortices as opposed to more posterior brain regions and other regions of the frontal lobes (Demakis, 2003).

The orbitofrontal region, in contrast, is more typically associated with motor suppression and response inhibition, as gleaned from studies of patients with orbitofrontal lesions (Fuster, 1989; Malloy, Bihrle, Duffy, & Cimino, 1993). Various regions of the orbitofrontal cortices are especially activated during response suppression tasks such as the Continuous Performance Task and variations of this task (Casey et al., 1997), and cross-sectional data indicate that activation of the dorsal and lateral prefrontal cortices during this task decreases with development (Casey et al., 1997). Activity in the orbitofrontal and cingulate cortices is associated with performance (errors of commission) on a go-no-go task (Casey et al., 1997, 2001). Studies using event-related potentials indicate that increased orbitofrontal activity is associated with inhibition of responses in the no-go condition (Bokura, Yamaguchi, & Kobayashi, 2001). As noted above response suppression errors are commonly reported in patients with OCD as is hyperactivation of

the orbitofrontal cortices (Tamm, Menon, & Reiss, 2002).

The OFC is involved in many functions both related to and unrelated to inhibition, most of which reside at the intersection of cognitive, emotional, and behavioral regulation. These functions include evaluation of the motivational significance of stimuli, learning appropriate responses to rewarding and aversive stimuli, switching responses when it is advantageous to do so, and registering and regulating emotional states (e.g., Barbas, 1995; Bechara, Damasio, & Damasio, 2000; Davidson & Irwin, 1999; Rolls, 1999). The OFC has also been credited with its own kind of working memory, including representations of the significance of stimuli and of one's own emotional states (e.g., Schoenbaum & Setlow, 2001). The OFC includes several distinct regions, each unique in its anatomy, connectivity, and cell structure. A convenient way to classify these regions is to divide them into medial and lateral areas. The medial (and more posterior) region is thought to be involved in motivational evaluation, especially reward and incentive motivation, as well as stimulus-response learning, and it is more directly connected to paralimbic, limbic, and diencephalic structures (e.g., insular cortex, amygdala, and hypothalamus) subserving emotional responding and emotion regulation. The lateral (and more anterior) region is implicated in behavioral inhibition, response suppression, selection of one response over others (e.g., Bokura et al., 2001; Bradshaw & Sheppard, 2000), and the *reappraisal* (or cognitive regulation) of emotional significance (Ochsner, Bunge, Gross, & Gabrieli, 2002). This region is more advanced in its cell type, and it is more connected to higher neocortical systems, particularly the DLPFC. Importantly, both the medial and lateral OF systems are connected to the basal ganglia, including the striatum. The medial OFC is strongly connected to the VTA and nucleus accumbens, with which it participates in a circuit controlling basic incentive motivation (Depue & Collins, 1999). The lateral OFC is connected to the caudate nucleus, with which it participates in the coordination of motor activity. Both orbital–striatal streams project to the thalamus, from where they return to frontal and motor cortical systems, comprising feedback circuits that modulate motivation and action. Finally, regions of the OFC are connected to the anterior cingulate cortex (ACC), another frontal region subserving executive functions. The ACC is involved in overriding prepotent response patterns, self-monitoring and error detection, and selection among competing responses (Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999; Carter et al., 2000)—functions that clearly overlap with those of the lateral OFC, such as setting an appropriate behavioral course in motivationally ambiguous circumstances. In vivo fMRI work indicates involvement of the ACC, not only while subjects are committing response errors, but also during challenging tasks when errors are likely (Carter et al., 1998; Casey et al., 2001).

Although set-shifting and response inhibition are considered distinct cognitive functions reflecting different brain loci (DLPFC for set-shifting and OFC for response inhibition), it is important to point out that these may be overlapping functions. That is, whereas set-shifting requires the ability to adopt a new rule (such as a sorting principle) and attend to various stimulus dimensions, it also requires response suppression and response selection in order to inhibit responding to the previously learned rule. Therefore, while the DLPFC has generally been identified with set-shifting abilities, it is likely that the OFC and ACC are involved in set-shifting performance as well.

Evidence from brain-injured patients has long tied the prefrontal cortices to perseverative behaviors, inability or impaired ability to shift cognitive set, impairments in working memory, poor motor suppression, and reductions in inhibitory control. Therefore it should not be surprising that the neural basis of OCD is often assigned to the prefrontal cortices as well. Specifically, OCD patients show hyperactivity of the lateral OFC, caudate nucleus, and ACC, all three of which are thought to work together to inhibit or terminate inappropriate responses and to select and monitor preferred behavioral sequences. PET studies have found increased glucose metabolism in the OFC, caudate, ACC, and thalamus (Baxter, 1990; Baxter et al., 1987; Baxter et al., 1992; Rauch et al., 1994; Rauch, Savage, Alpert, et al.; see Saxena et al., 1999; Schwartz, Stoessel, Baxter, Martin, & Phelps, 1996; for a review). In dense-array ERP research, OCD patients demonstrate an exaggerated error-monitoring component localized to the ACC (Gehring et al., 2000). Moreover, the initiation of OCD symptoms increases blood flow to orbital and caudate regions (McGuire et al., 1994; Rauch et al., 1994). For example, McGuire et al. (1994) observed right lateral OFC activation corresponding with the provocation of OC symptoms. Finally, successful intervention seems to reduce these activation patterns. Baxter and colleagues (Baxter, 1990; Baxter et al., 1992; see also Schwartz et al., 1996) observed reductions in glucose metabolization in the OFC and caudate nucleus in OCD patients following a 10-week trial of both pharmacological and cognitive-behavioral therapy (CBT). Saxena et al. (1999) found symptom reduction induced by pharmacological therapy to correspond with reduced metabolism in right lateral-anterior OFC as well as right caudate nucleus. And fMRI work has noted increased activation of the cortico-striatal systems following symptom provocation in OCD patients (Breiter, Rauch, Kwong, & Baker, 1996). These data suggest not only that symptoms of OCD are associated with orbitostriatal hyperactivation, but that the causal relation between brain and behavior associated with OCD may be bi-directional.

Importantly, these brain–behavior relations are presumed to characterize children and adolescents as well as adults (Bradshaw & Sheppard, 2000). That compulsive behaviors tend to emerge earlier, ontogenetically speaking, than obsessions in the natural history of OCD further suggests that anxiety may follow from, rather than drive, compulsions.

One question brought to mind by these findings is: why hyperactivation? Because OCD is characterized by dysfunctions in inhibition, response suppression, set shifting, and response selection, one might expect brain regions subserving these functions to be damaged rather than hyperactivated. Indeed, lesions to the lateral OFC produce increased perseveration and failure to inhibit inappropriate responses in reversal learning tasks in non-human primates (Roberts & Wallis, 2000), paralleling deficits shown by OCD patients. Several authors report that damage to orbitofrontal and striatal regions in humans produce some of the same deficits (see review by Cox, 1997). Yet, it is hyperactivation that corresponds with the diagnosis and symptoms of OCD. A possible explanation is that orbitostriatal hyper-activation reflects a regulatory difficulty, in which conscious effort must be continually expended in order to inhibit behavioral patterns that are difficult to terminate. Bradshaw and Sheppard (2000) refer to an overactive orbital warning system, and Saxena, Bota, and Brody (2001) and others describe an imbalance between direct and indirect striato-thalamic pathways. Perhaps a poorly regulated orbitostriatal system is less efficient, less sensitive, or less available, when it comes to inhibiting prepotent responses in tests of executive function.

In fact, while a failure to inhibit can be said to characterize some OCD symptoms, an excess of activity can be said to characterize others. From this latter perspective, hyperfrontality makes good sense. The hyper-selectivity of particular behavioral repertoires reflects a heightened discrimination between normally minor aberrations in stimuli, resulting in exaggerated distinctions between behaviors and/or sensory perceptual phenomena. This may, in turn, result in marked distinctions between behavioral and sensory phenomena that are "just right" and those that are not. Still, neither dysregulation nor hyper-activation fully explains the well-known presence of anxiety in OCD.

Perhaps a clue linking orbitostriatal dysregulation, hyper-activation, and anxiety is an fMRI finding of robust activity in the lateral OFC corresponding to the "reappraisal" (or effortful regulation) of negative emotional stimuli (Ochsner et al., 2002). In this study, subjects were asked to reappraise unpleasant situations depicted by pictures, thereby reducing felt negative emotions. These emotions no doubt included anxiety, as the pictures showed scenes of threat and violence. Thus, in OCD, a continuous need to regulate anxiety might over-stimulate the lateral OFC, detracting from its capacity to execute a flexible control process. However, it is also possible that anxiety is a result, rather than a cause, of compulsivity associated with OCD.

Advances in the neurobiology and neuropsychology of OCD and other perseverative behavior disorders may shed light on the maturational changes that give rise to repetitive behaviors, intrusive thoughts, and circumscribed interest patterns that are common in the behavioral repertoire of typically developing young children. Indeed, such exploration may even help us understand the adaptive nature of some repetitive behaviors, such as their maintenance of strategies that are ontogenetically and phylogenetically advantageous. Conversely, the study of atypical repetitive behaviors may be served by a better understanding of repetitive behavior patterns that are typical in early development. In the next section, we review recent literature on the normal ontogenesis of repetitive, compulsive behavior in young children and discuss the possible adaptive and maladaptive correlates of children's normal routines and rituals. We also discuss emergent cognitive capacities as well as the role of phase-appropriate anxiety in the development of what we have previously called "compulsive-like" behaviors.

5. Normative childhood rituals and "compulsive-like" behaviors

Rituals, perseverative, and compulsive behaviors, and other repetitive behavior patterns (as well as circumscribed interest patterns) have been studied, almost exclusively, in the context of brain injury and psychopathology. But increasingly, researchers are gaining an appreciation for the remarkable similarities between the broad range of repetitive behaviors that define severe psychopathology and those behaviors that are ubiquitous among typically developing young children. Thus, in the debate as to how broadly to define the compulsive-spectrum disorders, one must also consider the habits, compulsions and idiosyncrasies of normative development, and their possible continuity with pathological manifestations of these phenomena.

Historically, many scholars of developmental psychology have recognized that most young children go through a compulsive-like phase. Freud (1919), Erikson (1968), Gesell (1928), Werner (1948), Piaget (1962), and Vygotsky (1962) all made some mention of children's repetitive, ritualistic, and compulsive behaviors, though this topic has almost completely eluded empirical scrutiny. Workers in the applied arenas such as pediatrics recognize too that children may go through periods of odd behaviors, such as compulsivity and fetishism, equivalent to those seen in even the most severe compulsive disorders. However, these are often dismissed by mental health experts as something that one is to either

"keep an eye on" or ignore altogether, as they represent "only a phase." Children's compulsive-like behaviors may include rigid routines, strong likes and dislikes, food neophobia, acute perceptions of minute flaws in toys or clothes, and perceptions of subtle changes in the environment. Even repetitive movements that are markers of severe disorders—stereotypic motor movements such as body rocking or head-banging, and tics—are seen in typically developing children. Yet we know virtually nothing of the normative variant of this spectrum of behaviors (Marks, 1987).

Only within the past five years have researchers begun to explore the phenomenology and natural history of children's compulsive-like behaviors. Yet the correlates of typical compulsive-like behaviors remain ambiguous. Are children's compulsive-like behaviors associated with anxiety, fears and phobias? Are children's compulsive-like behaviors a function of cognitive and neuropsychological development and brain maturation? Are early and more intense compulsive behaviors in typically developing children markers for later psychopathology? Are children's typical compulsions adaptive? Finally, what are the continuities and discontinuities between children's normative compulsive-like behaviors and those behaviors that define psychopathology?

6. The phenomenology and natural history of compulsive-like behaviors

As is the case with the symptom expression of OCD, the features of children's compulsive-like behaviors are varied and multifaceted. By around age two children begin to engage in repetitive behaviors and to establish routines, particularly surrounding mealtime and bedtime. With regard to the bedtime ritual, children may require that parents engage in the same behaviors each night, such as reading a certain book, or watching the same video over and over. Children may involve a treasured object, such as a teddy bear or toy, in their rituals. Consider James Sully's (1896, as cited in Werner, 1948) account of a young child's bedtime ritual:

> A mother writes to Sully about her 2:7-year-old boy: "After I have kissed him and given him my hand I must also kiss his doll. Then I have to shake the doll's hands, and do the same to the four hooves of a toy horse, which lies at the foot of his bed. When all this has been done he rises in bed and begs: 'Kiss me again and then say goodnight just once more.'"

Werner seems to suggest that the compulsive nature of 2- and 3-year-olds is common knowledge (see also, Evans, 2000):

> We all know that during infancy children want to eat and be dressed in some particular fashion. In agreement with this attitude are those ceremonial rules and ritualistic practices of the child... These rituals may be so set that any neglect or alteration is felt to be a symbol of disruption of a state of affairs in which "something is wrong." We are unable to state definitively just when the all or nothing reaction evolves into the formal ceremonial, that is, when it becomes real magical behavior.

Several noteworthy concepts are presented in these narratives of Sully and Werner. First, there is an element of exactness or attention to detail that characterizes many aspects of compulsivity. Second, this exactness involves both a sensory-perceptual awareness of minute details in the child's surroundings, and also the necessity of the sequentiality of events that comprise the ritual or routine. Third there is the *necessity* itself—the inflexibility of events or behaviors that precludes spontaneity or adaptation of the ritual.

Other aspects of children's compulsivity include what we call "just right" behaviors. This refers to the tendency of children to arrange or order objects or engage in behaviors until they satisfy some subjective criterion of being "just so." Young children are concerned with object symmetry, balance and wholeness (Gesell, Ames, & Ilg, 1974). So the compulsivity of typical children involves both carrying out certain behaviors in an invariant sequence as well as heightened sensory perceptions and attention to minute details, imperfections, and geometric properties of objects. Attachment to a favorite object, and perseveration on certain thoughts and objects—including body parts—are also among the compulsive behaviors that appear to be common to most, if not all, children in the preschool years of development.

Though this intriguing developmental phenomenon has attracted the interest of theorists for over a century, little is known about its emergence, natural history, or association to later obsessive–compulsive behavior (Marks, 1987). Therefore, our first cross-sectional study examined the natural history of compulsive-like behavior, using parents' reports on a 19-item inventory of normative compulsions (The Childhood Routines Inventory, or CRI; Evans et al., 1997). Factor analysis of the CRI indicated that two principal components represent the variance across these items. One component we refer to as "Just right" behaviors. Just right behaviors reflect the aforementioned sensory-perceptual aspects of compulsive behaviors. The second principle component—"Repetitive behaviors"—comprises those items relating to repeating rituals, habits, and daily routines. Our results (Evans et al., 1997) revealed that compulsive-like behaviors are highly prevalent by the age of two years—with 80% of the sample engaging in some compulsive-like behaviors. Compulsive-like behaviors remain prevalent through the fifth year of life, after which they appear to decrease significantly, though they do not disappear altogether. One longitudinal study (Evans and Gray, submitted) corroborates this developmental trajectory. Moreover, developmental changes appear to be dependent on mental age: children

with mental retardation (Down syndrome) engaged in a similar frequency and intensity of compulsive-like behaviors as did their mental age- but not their chronological age-matched counterparts (Evans & Gray, 2000). A recent cross-sectional study on children aged seven to fifteen years suggests that, while children across these age groups engage in less compulsive-like behavior than do preschoolers, such behaviors are still relatively common (Stayton, 2000; Stayton & Evans, in preparation).

Some scholars claim that it is easy to differentiate between pathological compulsions and those that characterize "normal" childhood. Rapoport and Inoff-Germain (2000), for example, dismiss the similarities between the rituals and compulsions of typical children and clinically-significant compulsions by simply writing that typical compulsions are relatively short-lived, more flexible, and not associated with subjective distress. But this is not true. We have found, for example, that although compulsive-like behaviors may be considered a phase, children's compulsive-like behaviors are highly correlated over a 2-year period (Evans & Klinepeter, 2002). Second, rigidity, not flexibility, defines even typical rituals and compulsions, and third, even typical compulsive-like behaviors in young children are associated with fears, phobias, and anxiety (Evans, Gray, & Leckman, 1999). The frequency and intensity of children's bedtime rituals are significantly correlated with their fears surrounding bedtime. In middle childhood, parents' reports of their children's compulsive-like behaviors are correlated with scores on the Speilberger trait anxiety scale (Stayton, 2000; Evans & Stayton, 2003). Moreover, even slight disruptions in children's rituals or routines can be met with extreme resistance, resulting in interruptions in sleep patterns and food refusal. During the enactment of the compulsive-like behaviors themselves, it may be evident that the child is not deriving pleasure from the ritual. Consider again one of Werner's (1948) anecdotes of a typically developing child's bedtime ritual: "One child always felt compelled to say her prayers standing up in bed as straight and stiff as a ramrod; although, because of the lack of heat in the room, this was at times an unpleasant procedure, nevertheless it had to be followed" (Werner, 1948, p. 361).

Thus, the compulsive-like behaviors of typically developing children may indeed be quite rigid and inflexible; they are associated with fears, phobias, and anxieties, and the acts themselves may be experienced as egodystonic. Because of this, the compulsive-like behaviors of typical children may have more in common with pathological compulsive behaviors than previously thought (Evans et al., 1999; see also Bolton, 1996). Many of the behaviors associated with normative childhood, such as perfectionism and rigid adherence to rules, may also resemble OC personality disorder (OCPD). OCPD tends to be less associated with anxiety than does OCD, and historic and contemporary accounts of normative compulsive-like behaviors do not highlight anxiety. But again, our own accounts suggest that anxiety and subjective distress may play a role in the normative manifestations of OC behaviors. However, links between normative compulsive behaviors and OCPD merit exploration. In general, we caution against any model that disregards the continuity between normative and pathological compulsive behaviors in favor of a rigid taxonomy.

The next question, then, is whether such behaviors also tap the same neuropsychological and neurobiological underpinnings.

7. Developing executive functions and their hypothesized neural substrates

As noted earlier, executive functions refer to a broad range of cognitive and behavior control capabilities, that appear to undergo a period of development from infancy through the preschool years and beyond. Most relevant to our discussion are those EFs that underpin response inhibition, set-shifting, and selecting among competing behavioral tendencies. These are the capabilities that allow children to control their impulses, behave flexibly, with attention to contextual cues, and formulate rewarding action plans (Robbins, 1998). They are also the capabilities that are compromised in obsessive–compulsive disorders in adulthood.

One of the earliest indices of executive function is successful performance on Piaget's A-not-B task (Diamond & Goldman-Rakic, 1989). Infants learn to ignore the hiding place where an object was previously placed in favor of its new location, from about the age of 9–12 months. This is essentially a task of response inhibition in which infants learn to inhibit a prepotent response in favor of a more rewarding one. The A-not-B error has been studied extensively by a number of scholars and is used as an index of early executive function (see Diamond, Prevor, Callender, & Druin, 1997, for a review, and their work with children with disabilities). Considerable evidence links the A-not-B task to the DLPFC in both human infants and rhesus monkeys (Diamond & Goldman-Rakic, 1989). Also, both the OFC and the ACC are thought to come on-line by the latter half of the first year. Posner and Rothbart (1998) as well as Harman and Fox (1997) emphasize the maturation of the ACC, and discuss its contribution to self-regulation and distress reduction in later infancy. Schore (1994, 1997) cites evidence for the development of (especially) the right OFC by the end of the first year, somewhat later than ACC development, and discusses its critical role in emotion regulation.

The onset of normal compulsive-like behaviors begins just before the age of two and may reach its zenith between two and five years. What aspects of executive

development correspond with this age range? Dunn (1988) describes the advent of rule-focused behavior beginning at about 18 months and continuing to age 3. Children of this age begin to learn rules, particularly concerning dirt and cleanliness and the suppression of certain impulsive behaviors. They can also contextualize rules, as evidenced by their frequent testing of which rules apply in which contexts. At about the same age, children begin to perform successfully on object reversal tasks, as when they learn to select a previously unrewarded stimulus that is now paired with a reward (Overman, Bachevalier, Schuhmann, & Ryan, 1996). Also at around age two children begin to learn ambiguous associations that can only be disambiguated via contextual cues (Clohessy, Posner, & Rothbart, 2001). All these capabilities seem to point to OFC maturation.

Overman, Bachevalier, Schuhmann, and McDonough-Ryan (1997) hypothesize that two- to three-year-old reversal learning *and* distress regulation depend on developing orbitofrontal function. This interpretation is based, in part, on evidence that boys develop OFC function earlier than girls do. Girls made significantly more errors on the object reversal task than boys, until the age of about 3 years when both were performing well (Overman et al., 1996). Also, about 15% of younger girls demonstrated extreme distress when failing to make the reversal, whereas no younger boys showed distress. Young female monkeys and adult monkeys with orbitofrontal damage both perform equivalently, and relatively poorly, on the same reversal task (Overman et al., 1997), hence the link with orbitofrontal capacity. Of particular relevance for OCD development, human studies show gender differences in callosal axons projecting from the OFC: men attain maximal callosal size in this region at age 20, well ahead of women who do not attain it until 40–50 years (Cowell, Allen, Zalantino, & Denenberg, 1992). The implications for OCD development will be discussed later. For now, what is important is that the onset of normative compulsive-like behavior patterns corresponds with the age of onset of rule learning, rule-testing, reversal learning, and contextual disambiguation, all of which appear to be linked with orbitofrontal maturation (see also Zelazo & Reznick, 1996; Zelazo, Reznick, & Spinazzola, 1998).

At the age of two to five, when normative compulsive-like behaviors are on the rise, children learn to inhibit their impulses, delay gratification, and suppress behaviors that are associated with punishment. Executive development across the same age span has been documented in conflict-resolution tasks. Posner and Fan (in press) cite research conducted in their lab by Gerardi-Caulton (2000) and Rothbart, Ellis, and Posner (in preparation), in which children are required to push a button on the opposite side of a screen from a presented cue, thus overriding their tendency to push the same-sided button. While 2-year-olds are incapable of this response inhibition, 3-year-olds both perform well and also more slowly, demonstrating what Posner and Rothbart (1998) have called "effortful control." Adults engaged in this task show ACC activation, implying that the development of this capability in toddlers may reflect ACC development (Posner & Fan, in press; Posner & Rothbart, 2000). However, because the lateral OFC is also known to mediate response inhibition, and because the left OFC shows a growth spurt at 2–3 years as well (Chiron et al., 1997), it is quite possible that maturation of the OFC and ACC work together in supporting the emergence of effortful self-control.

Interestingly, there is an apparent dissociation in early development, between preschool children's abilities to understand (or at least recite) a particular rule and their abilities to execute behaviors according to the rule (Zelazo & Jacques, 1996). For example, when asked to sort objects according to one dimension (such as color) and then switch to a new dimension (such as shape) children perseverate and continue to sort according to the first dimension—even though they respond correctly when verbally queried as to the new (second) sorting rule. Thus behaviorally, the children's established response patterns are more potent than their conscious understanding of the rules during such sorting tasks, and presumably this ability changes with maturation of the DLPFC (see Zelazo & Jacques, 1996 for a review on rule use).

A number of authors relate executive development at around age 3–4 to the onset of self-control and consideration for others in social situations. Inhibitory control contributes to the development of conscience in young school-aged children (Kochanska, Murray, & Coy, 1997), and children's self-control fosters awareness of responsibility for their own actions (Derryberry & Reed, 1996). Moore (this issue) provides evidence for the development of prudence, or delay of gratification, during the same period. In these and related studies, behavior regulation and affect regulation are considered extensions of a more fundamental capacity for effortful control which relies in large part on the ability to inhibit prepotent responses (Posner & Rothbart, 1998, 2000).

8. Associations between neuropsychological performance and normal compulsive-like behaviors

Our recent work has demonstrated that, in school-aged children, tasks of set-shifting and response inhibition/motor suppression are related to the frequency and intensity of typically developing children's compulsive-like behaviors. Children six to eleven years of age were administered a series of computer-generated tasks assessing set-shifting and response inhibition (Evans & Iobst, 2003a, 2003b).

Motor suppression/response inhibition tasks were as follows:

1. Color Discrimination/inhibition. On this task three colored squares (2 in. × 2 in.) appear on a screen and children are instructed to click a mouse when the square in the middle matches either of the outer two squares, and to inhibit clicking when the middle square matches neither. Sets of squares are presented every 3 s, and latency of response and errors of omission and commission are recorded.
2. On a related task a 12 in. × 6 in. border appears on the screen. Inside the screen a small square (.5 in. × .5 in.) appears intermittently (approximately every 2 s) and alternates between two (and later four) colors. Subjects are instructed to click a mouse when the color of the border and the color of the square match, and to inhibit clicking the mouse when they do not. Again, latency of responses and inhibition errors are recorded.

Set-shifting was assessed through two tasks:

1. In Symbolic Display Match, children are required to discover a rule for matching two objects, on color, shape, or color and shape, by clicking a mouse when two objects match. Children are not informed of the matching rule, but are given feedback from the computer as to whether their response was correct or incorrect (a beep or buzz). As in the WCST, the rule switches, and children are expected to adapt to the new matching rule.
2. Finally, a Conceptual Discrimination task presented children with a 3×2 matrix of six different shapes. Children are instructed to highlight and click on the object that does not match the others. Features that can differentiate objects include, shape, color, and size, and the rule for matching/not matching changes.

Six multiple regression analyses were performed. Each regression equation was used to predict a different factor from the Childhood Routines Inventory (CRI) discussed previously. Three factors reflected the *mean* frequency/intensity of children's compulsive-like behaviors: an overall mean CRI score (Mean CRI) and a mean score for each of the two factors (Mean Just Right and Mean Repetitive Behaviors). Three variables reflected the *total number* of compulsive-like behaviors in which children engaged: the total number of items endorsed (out of 19) for the whole CRI (Total CRI) and the total number of items endorsed for each factor (Total Just Right and Total Repetitive Behaviors). Because scores on the CRI tended to decrease with age, age was entered into the regression equation among the other predictor variables.

Results revealed that a combination of set-shifting and response inhibition scores predicted variance in children's compulsive-like behaviors. None of the tasks predicted significant variance in Mean CRI or Mean Just Right scores beyond the variance predicted by age (which was about 15–17%). However, mean Repetitive Behaviors was positively predicted by errors in Conceptual Discrimination (a set-shifting task), response latency on Color Discrimination (a response suppression task), and inhibition errors on Color Discrimination, for a total of 38% of the variance. Thus, children's observed degree of repetitive behavior was predicted by a combination of poor set-shifting and poor response-inhibition.

For the compulsive-like behavior variables representing the total number of items endorsed on the CRI, Conceptual Discrimination errors predicted 12% of the variance. Conceptual Discrimination errors also predicted variance in the number of "Just Right" items endorsed, as well as the total number of repetitive behaviors endorsed (13% and 29% of the variance, respectively).

A combination of tasks reflecting set-shifting and response suppression capabilities thus served as predictors of children's normative compulsive-like behaviors. For all results summarized above, increased compulsivity was associated with worse performance on cognitive/neuropsychological task performance. Such findings suggest that, with children's normative compulsive-like behaviors, just as with pathological OCD symptoms in adults, the same underlying executive functions appear to be compromised. As discussed previously, set-shifting tasks require response suppression as well as the higher-order cognitive functions of selecting and remembering the appropriate rule. Therefore, the results reported here may be explained by differences in the functioning of the OFC (and ACC) circuitry responsible for inhibiting prepotent responses.

9. A hypothesized developmental neurobiology of normative compulsive-like behaviors

So far we have reported that children's compulsive-like behaviors appear by about the age of two years, then increase in prevalence and remain highly characteristic of normal development until about the age of five, when they start to diminish in prominence. These behaviors include the repetition of invariant sequences, often based on attention to small changes in the environment, as well as a tendency toward excessive precision or symmetry, and they are related to anxiety both at the trait and state level. In particular, repetitive sequences of behavior appear to ward off anxiety in normal childhood, as evidenced by the common finding, both by parents and researchers, that anxiety-producing situations such as bedtime are most likely to elicit compulsive routines and the interruption of these routines can produce great distress. We have also reviewed evidence that capabilities including object reversal, response inhibition, contextual disambiguation, and con-

flict resolution come on line at about the same age as normative compulsive behaviors. These capacities have been associated with the maturation of the orbitofrontal cortices, as well as the ACC, and they have been hypothesized to reflect a developing capacity for "effortful control" (Posner & Rothbart, 1998). Finally, we demonstrated that individual differences in performance on executive tasks that rely on response inhibition/suppression, and which therefore suggest orbitofrontal involvement, correlate with observed differences in compulsive-like behaviors in early childhood, just as they do in pathological forms of OCD.

Clinical OCD has been consistently linked to hyperactivation of the lateral OFC, ACC, and caudate nucleus—structures that work together to regulate a coherent and flexible stream of behavior. In reviewing these findings, we noted that hyperactivation of these systems may imply dysregulation, resulting in inadequate and inflexible behavioral control capacities. We also noted that hyperfrontality is consistent with the amplified tendency toward effortful response selection and associated vigilance commonly observed in OCD. To apply these propositions to normative compulsive-like behaviors, and particularly their relation to age-appropriate anxieties, we return to Ochsner and colleagues' (2002) discovery that the lateral OFC is specifically activated by the effortful control of negative emotions.

Putting these pieces together, we hypothesize a critical interaction between the development of inhibitory self-control and the need to regulate age-specific anxieties across the age-span of 2–5 years. Socialization practices are known to correspond to the timetable of maturing regulatory capabilities (Gordon, 1989; Thompson, 1994). Impulse control is among the tasks associated with the epoch of development when the OFC and related structures mature. A lack of mastery of impulse control often stresses parent-child interactions resulting in threats and punishment by parents, which in turn, increases anxiety (Dunn, 1988). This may result in a child's preoccupation with being "good" rather than "bad" and learning to distinguish, obey and internalize the rules for appropriate conduct (Dunn, 1988). It would also explain a good deal of the anxiety young children experience as a result of their own impulsive behavior. As they mature, the lateral OFC/ACC functions that permit response inhibition and effortful control may also be recruited by the demands of anxiety reduction, particularly when anxiety is linked with the failure to inhibit impulsive behavior. This may dominate the lateral OFC with emotion-regulation demands, making it less flexible and less sensitive when it comes to the routine requirements of behavior control. Thus, lateral OFC activation dedicated to the regulation of anxiety may in some ways mimic the hyperactivation of the same system in pathological OCD. The result, in both cases, may be an over-attuned but rigid and stereotypic style of behavior regulation, in place of a more flexible and intermittent mechanism of behavior control. However, the continual maturation of the same neural systems (particularly in the left hemisphere—Chiron et al., 1997) may permit increasingly effective self-control capabilities over the next few years, such that, by the age of 5 or 6, anxiety is generally reduced, behavioral regulation is more automatic, and compulsive-like behavior rituals are no longer required.

If this hypothesis is even partially correct, then normative compulsive-like behavior may be similar to pathological OCD with respect to excessive demands on the response-inhibition and response-selection capabilities of the lateral OFC and perhaps the ACC as well. However, the involvement of the striatum, and particularly the caudate nucleus, may differentiate the two constellations. Perhaps adult OCD patterns begin with striatal dysregulation, as postulated by some theorists, and only then require an excessive degree of OFC activation, shifting the burden of behavioral control from unconscious to effortful mechanisms. Future research, and especially more detailed comparisons of pathological and normative compulsive patterns, will be necessary to resolve this puzzle. However, one piece of evidence suggests an even greater continuity between developmental compulsive behavior and adult pathology. As we noted earlier, males develop clinical OCD far earlier than females, about 10 years earlier on average. We have also reviewed evidence that OFC development is later in females than males, with peak callosal density roughly 20 years later for women than men. It is possible that these sexual dimorphisms are related, and that, even in pathological OCD, the developmental trajectory of OFC maturation is an important parameter. This speculation exemplifies one of the main themes of this article: that comparing the neurobiology and neuropsychology of pathological and normative compulsive patterns may enhance our understanding of each, based on our knowledge of the other.

We do not mean to suggest that children's early compulsive behaviors are necessarily precursors to later OCD. How, when, or whether, normal childhood rituals and compulsions develop into OCD, is not clear, but one indicator as to when normative behaviors become problematic is when the normative routines and compulsions become out of step with "the rest" of development; or whether the compulsive-like behaviors are sufficiently rigid and pervasive, chronic, or subjectively distressing so as to impede the mastery of phase-specific developmental tasks in the realms of cognition, emotion, socialization, etc. When asked to report the earliest signs or onset of their children's OCD behaviors, parents of adolescents with OCD are readily able to identify the early "symptoms" as the habits and routines that comprise normative development (Leonard, Goldberger,

Rapoport, Cheslow, & Swedo, 1990). This gives an illusion of continuity between the early normative compulsions and later OCD. Conversely, parents who report that their typically developing three-year-old engaged in elaborate bedtime routines and other compulsive-like behaviors will, when the child is seven years old, deny that such behaviors ever existed, unless of course the compulsions have persisted, worsened or evolved into OCD. Early and even extreme levels of compulsive behaviors do not necessarily indicate a vulnerability to OCD. We suggest that the routines, habits and compulsions that exist (and are even necessary) in all humans, may involve underlying mechanisms similar to those that represent pathology, and in this sense are continuous with them.

We conclude then, with the proposition that repetitive behavior patterns, rituals, and compulsions that are ubiquitous among typically developing young children are not only phenomenologically similar to the behaviors associated with OCD, but they may in fact share a common underlying neurobiology. As in OCD, the repetitive and compulsive-like behaviors associated with normative ontogenesis are related to the development of motor-suppression and response inhibition, and later, set-shifting ability. These cognitive tasks are, in turn, governed by certain regions of the orbitofrontal cortices that are implicated in the pathogenesis of OCD. In typically developing children response inhibition and motor suppression abilities (and the maturation of the orbitofrontal cortices) are critical to the development of self-regulation and the organization, execution and regulation of emotions and behaviors. Rather than view childhood habits, routines, and compulsions (as well as accompanying cognitive and emotional phenomena, e.g., anxieties, fears, focused interests), as qualitatively distinct from pathological compulsions and obsessions, we argue that the normality and psychopathology of compulsive behaviors are complementary domains of study that merit further empirical inquiry and theoretical consideration.

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