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Does cognitive self-consciousness link older adults' cognitive functioning to obsessive-compulsive symptoms?

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ABSTRACT

To elucidate how obsessional symptoms might develop or intensify in late-life, we tested a risk model. We posited that cognitive self-consciousness (CSC), a tendency to be aware of and monitor thinking, would increase reactivity to aging-related cognitive changes and mediate the relationship between cognitive functioning and obsessive-compulsive disorder (OCD) symptoms. Older adults (M_{age} = 76.7 years) completed the Dementia Rating Scale-2 (DRS-2), a CSC measure, and an OCD symptom measure up to four times over 18 months. A model that included DRS-2 age and education adjusted total score as the indicator of cognitive functioning fit the data well, and CSC score change mediated the relationship between initial cognitive functioning and changes in OCD symptoms. In tests of a model that included DRS-2 Initiation/Perseveration (I/P) and Conceptualization subscale scores, the model again fit the data well. Conceptualization scores, but not I/P scores, were related to later OCD symptoms, and change in CSC scores again mediated the relationship. Lower scores on initial cognitive functioning measures predicted increases in CSC scores over time, which in turn predicted increases in OCD symptoms over the 18 months of the study. Implications for understanding late-life obsessional problems are discussed.

Obsessive-Compulsive Disorder (OCD) is a commonly occurring psychiatric disorder characterized by recurrent intrusive thoughts, images, or impulses that increase distress (i.e., obsessions), and repetitive thoughts or actions intended to alleviate this distress (i.e., compulsions; American Psychiatric Association, 2013). Although research on OCD has been prolific, research on OCD in older adults has been neglected (e.g., Calamari, Wilkes, & Prouvost, in press; Carmin, Calamari, & Ownby, 2012). Little is known about late-life OCD, including how to best treat older people with the disorder, which is problematic because OCD is associated with serious disability and distress across the lifespan (e.g., Steketee et al., 2012). Although estimates of the prevalence of OCD in older adults are lower than in other age groups in some epidemiologic studies (e.g., lifetime prevalence, 0.7%; Kessler et al., 2005), these estimates have several limitations including the exclusion of older adults living in supported environments such as nursing homes, where estimates of OCD have been much higher (see Carmin et al., 2012; for a review).

There is growing recognition by gerontologists that the complex changes characteristic of late-life can influence adjustment broadly and affect the occurrence of late-life emotional disorders (e.g., Woods, 2008). Late-life adjustment is affected by significant stressors including important changes in older peoples' social support (e.g., the passing of friends or the loss of one's spouse) and by increasing health problems (e.g., Calamari et al., in press). Significant changes in cognitive functioning characterize even normal aging (e.g., Salthouse, 2010), and although there are multiple factors related to cognitive decline, there is an established association between anxiety (see Beaudreau & O'Hara, 2008; for a review) and depression (e.g., Bielak, Gerstorf, Anstey, & Luszcz, 2011), however, the causes of this relationship are not well understood (e.g., Beaudreau & O'Hara, 2008; Woods, 2008). Most often, the associations between mood, anxiety, and cognitive functioning have been evaluated from the perspective that chronic mood or anxiety symptoms or disorders result in later, more significant declines in cognitive functioning, while few investigators have longitudinally evaluated how cognitive functioning might affect the trajectory of anxiety, mood, or other disorders symptoms (e.g., Salthouse, 2012). In the present study, we evaluated how developmental stage related changes in cognitive functioning might affect the

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development of obsessional symptoms in late-life. Calamari, Janeck, and Deer (2002) hypothesized that older adults' concerns about changes in cognitive functioning and related vigilance about their cognitive abilities might influence reactivity to aspects of cognition including their reactions to common negative intrusive thoughts.

The etiology of OCD is not known, but neurobiological models posit that the neuropathology reliably associated with the condition plays a causative role. Condition-related neuropathology includes abnormal functioning of frontal–striatal circuits involving the orbitofrontal cortex, anterior cingulate cortex, thalamus, and caudate (Baxter et al., 1992; Breiter et al., 1996; Rauch et al., 1994; Saxena & Rauch, 2000). The dearth of research on older adults with OCD precludes any conclusions as to whether the neuropathology underlying late-life OCD is different, although the results of several investigations suggest that the same underlying abnormalities might be involved (Henin et al., 2001; Roth, Milovan, Baribeau, & O'Connor, 2005; Taylor, 2011).

Although neurobiological models of OCD place different emphases on the affected neurologic systems and the system related functional abnormalities, Rauch and Savage's (2000) model focused on resultant excessive effortful, conscious processing. Rauch and Savage posited that the frontal-striatal hyperactivity that characterizes OCD results in a non-conscious processing gating disturbance such that innocuous stimuli, which are typically processed non-consciously and which do not elicit focused attention, are often processed consciously and inefficiently by people with OCD. An overly frequent shift to effortful, conscious processing is understood to be importantly related to many of the symptoms seen in OCD.

Cognitive theories of OCD emphasize the role of certain dysfunctional beliefs (e.g., responsibility; importance of thoughts), which are understood to drive the problematic appraisal of common negative intrusive thoughts (e.g., Frost & Steketee, 2002; Salkovskis, 1985). Although cognitive theories place different emphasis on the types of problematic beliefs most important to OCD, some theorists have focused on metacognitive processes. Wells (2000; 2009) emphasized specific aspects of metacognition as etiologic in mood, anxiety, and obsessional problems. In an evaluation of a measure of multiple metacognitive constructs, cognitive self-consciousness (CSC), an excessive awareness of and attention to thought experiences, was the only measure that differentiated OCD patients from patients with Generalized Anxiety Disorder (Cartwright-Hatton & Wells, 1997).

In evaluations of the CSC construct that followed, individuals with structured clinical interview diagnosed OCD scored higher on a measure of CSC compared to both clinical and nonclinical controls (e.g., Goldman et al., 2008; Janeck, Calamari, Riemann, & Heffelfinger, 2003). CSC measure scores correlated significantly with multiple OCD symptom measure scores in mixed clinical–nonclinical samples ($r = 0.64$) (Marker, Calamari, Woodard, & Riemann, 2006), in OCD and clinical comparison groups ($r = 0.55$ - 0.46) (Goldman et al., 2008) and in a non-clinical group ($r = 0.45$) (Cohen & Calamari, 2004). Further, CSC measure scores remained associated with OCD symptom scores even after controlling for general negative affect (Jacobi, Calamari, & Woodard, 2007), intrusive thought appraisals (Cohen & Calamari, 2004), worry (de Bruin, Muris & Rassin, 2007), and OCD-related dysfunctional beliefs (Janeck et al., 2003), suggesting that CSC is a distinct OCD-related cognitive process. Additionally, Exner and colleagues demonstrated that CSC scores mediated the relationship between OCD and performance on object memory test (Exner et al., 2009) and selective attention evaluations (Koch & Exner, 2015), areas of cognitive functioning sometimes found impaired in OCD. As a result of the associations between CSC and OCD, Janeck et al. (2003) posited that elevated CSC might increase the detection of personally relevant negative intrusive thoughts, make their misappraisal more likely, and hinder the dismissability of these thoughts. Janeck et al. went on to theorize that CSC could be a cognitive risk factor for OCD, and noted that the construct shared similarities with the neuropathology-related dysfunction affecting cognitive processing in Rauch and Savage's (2000) model of OCD.

In several investigations, CSC has been directly linked to a behavioral marker of OCD neuropathology, implicit procedural learning, as measured by performance on the serial reaction time task (SRT; Nissen & Bullemer, 1987). Impaired performance on the SRT task was directly associated with the cortical-striatal dysfunction associated with OCD (e.g., Deckersbach et al., 2002). Further, Goldman et al. (2008) found that OCD patients' performance was impaired in comparison to an anxious control group on the SRT, and that higher scores on a measure of CSC were related to longer reaction times (impaired performance). As a result of the potential importance of the CSC construct to cognitive and neurobiological theories of OCD, and as a result of the association between CSC and OCD-related cognitive functioning differences, we tested whether CSC might play an important role in late-life OCD.

There has been only one prior evaluation of a risk model of late-life obsessional problems. Using a cross sectional design, Teachman (2007) employed structural equation modeling to evaluate whether the relationship between OCD-related dysfunctional beliefs and OCD symptoms was mediated by a measure of subjective concerns about cognitive decline. As predicted, dysfunctional beliefs (e.g., over importance of thoughts; perceived need to control thoughts) was related to OCD symptoms through the mediator, concerns about cognitive functioning. She found that this mediational relationship was the same in the older and younger adult samples included in her study.

In the present study, we undertook a longitudinal evaluation of a risk model that included CSC as a mediational process or as a moderator. We tested whether CSC mediated the relationship between older adults' cognitive functioning and the development of OCD symptoms (see Figs. 1 and 2). We posited that late-life cognitive functioning changes, even changes that are within normal limits, would lead to a greater focus on cognitive processes and increasing CSC. Further, we predicted that increasing CSC would result in greater reactivity to personally relevant, negative intrusive thoughts and more OCD symptoms. Reactivity to negative intrusive thoughts is importantly related to OCD symptoms (e.g., Salkovskis, Richards, & Forrester, 1995). Additionally, CSC has been posited to be a relatively stable individual difference characteristic that might not directly influence OCD symptoms, but could interact with other variables to increase obsessional problems (Janeck et al., 2003). Therefore, we also tested for the presence of an interaction between our cognitive functioning predictor variables and the CSC mediator and the relationship to OCD symptoms. We also tested this relationship because predictor -mediator interactions will affect the estimation of mediational relationships (e.g., MacKinnon, 2008).

We tested whether a measure of global cognitive functioning or performance on measures of specific cognitive functions most sensitive to OCD-related neuropathology would affect OCD symptom levels though the CSC mediator, or interact with CSC, to predict later OCD symptoms. We predicted that that lower Dementia Rating Scale-2 (DRS-2; Jurica, Leitten, & Mattis, 2001) total scores, and lower scores on the DRS-2 Initiation-Perseveration (I/P) and Conceptualization subscales, which largely require executive