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## Letter to the Editor

**Improbability of response inhibition as a causal etiological factor of obsessive-compulsive disorder**


To the Editors:

In an article appearing in this journal, Harsányi et al. (2014) observed what they refer to as “severe executive impairment” in a sample of individuals with obsessive-compulsive disorder (OCD) and concluded that such impairments constitute a causal factor underlying compulsive rituals. They further suggested that with regards to obsessions and compulsions, “executive dysfunction is assumed to underlie both symptoms.” Harsányi et al. also hypothesized that failures in “behavioral-executive inhibitory processes” underlie the inability to stop compulsive rituals, once started, and deficient cognitive inhibition underlies the inability to stop obsessions. In addition, Harsányi et al. reported that the informant version of the self-report Dysexecutive Questionnaire (DEX) showed a significant positive correlation with the Compulsions, but not the Obsessions, subscale of the Yale-Brown Obsessive-Compulsive Scale (Y-BOCS). This discrepancy is explained by appealing to Wegner's (1994) model of thought suppression and arguing that suppression of obsessive intrusive thoughts in OCD is an automatic and effortless process that does not have a cognitive “cost.”

We note several problems with the conclusions that Harsányi et al. draw from their cross-sectional (correlational) data. First, causal inferences such as response-inhibition deficits underlying compulsive rituals cannot logically be drawn from correlational data. Such data are ambiguous as to whether underperformance on neuropsychological tests is a cause of, a consequence of, or in any way fundamental to, OCD symptoms. Harsányi et al. merely show that some relationship exists. Indeed, an equally plausible explanation for the findings of Harsányi et al. is that the anxiety and distress associated with obsessions and compulsions negatively influenced neuropsychological performance (Abramovitch et al., 2012). Alternatively, both OCD symptoms and performance could be influenced by a third variable not accounted for in the study. Experimental research involving manipulations of variables is necessary to determine whether one process is fundamental to the other. Moreover, models suggesting that neuropsychological impairments underlie OCD also suffer from specificity problems. First, these models fail to account for the heterogeneity of OCD symptoms. What determines whether a particular person with a response-inhibition deficit experiences checking, washing, or ordering symptoms? Second, a large number of psychiatric disorders, characterized by very different clinical pictures and putative pathophysiology, have been found to be associated with similar underperformance in executive functioning, including (but not limited to) schizophrenia and bipolar disorder

(Martínez-Arán et al., 2002), attention deficit/hyperactivity disorder (ADHD; Marije Boonstra et al., 2005), and depressive and anxiety disorders (Castaneda et al., 2008). In other words, what determines whether the response-inhibition deficits lead to OCD versus these other conditions?

A second set of problems that undermines the conclusions drawn by Harsányi et al. pertain to how executive functioning was assessed, as well as their interpretation of the extent of these impairments. Specifically, the DEX is not an objective neuropsychological measure, nor does it show strong correlations with objective neuropsychological tests of executive functioning (Chaytor et al., 2006). In addition, although Harsányi et al. compared their participants' DEX scores with the instrument's published norms (and determined that their OCD group suffered from severe executive function impairments), their interpretation relies on DEX scores that correspond to a range between the 31st and 50th percentile [or 0–0.5 standard deviations (S.D.)] from the normative sample's mean). The rule of the thumb for determining neuropsychological impairments, however, is at least a two S.D. difference (Lezak et al., 2012, p. 167). Thus, the reported percentile range does not meet the definition of a neuropsychological impairment (or even a trend toward such an impairment as defined by Lezak et al., 2012), let alone a “severe” degree.

Finally, the authors presuppose that compulsions are theoretically associated with response inhibition because these are “... rituals that they [individuals with OCD] are unable to stop.” In the realm of neuropsychology, performance on response-inhibition tasks (especially commission errors) is the gold standard indicator for behavioral impulsivity, which is prevalent in disorders associated with impulsiveness, such as ADHD (McAuley et al., 2014). However, studies show that individuals with OCD are no more behaviorally impulsive than are healthy controls (e.g., Shoval et al., 2006). Moreover, compulsions in OCD are not accidental impulsive acts that result from an inability to inhibit one's responses. Rather, they are carefully planned and executed, and are usually carefully timed in response to obsessions (American Psychiatric Association, 2013). In clinical work with patients, it is easy to observe that individuals with OCD are able to postpone or stop their rituals in certain circumstances (e.g., to avoid embarrassment, as part of behavior therapy), which indicates intact ability to inhibit these behaviors.

In sum, Harsányi et al.'s data do not support the conclusion of “severe” executive function impairments in OCD, nor do they support a causal role of executive functioning deficits in the etiology of OCD. Rather, the only inference that may be drawn on the basis of these data is that informant's ratings of behaviors associated with reduced executive functioning are in some way modestly associated with the severity of compulsions, but not obsessions.

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Received 23 December 2013; accepted 28 January 2014

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