Introduction to the Special Section: Cognitive-Behavioral and Neuroscientific Approaches to Obsessive-Compulsive and Related Phenomena: Why the Need for an Interface?

Michael Kyrios
Swinburne University of Technology, Melbourne, Australia

Cognitive and behavioral theories of Obsessive-Compulsive Disorder (OCD) and related disorders (e.g., Body Dysmorphic Disorder [BDD] and tic disorders) have led to the development of effective treatments, although clinically significant outcomes are not consistent and both dropout and relapse rates remain high. Researchers have turned to alternative theoretical frameworks to advance our understanding of these disorders and to provide novel insights into either new interventions or the tailoring of existing interventions. Neuroscientific evidence from neuropsychological, functional and structural imaging, and electrophysiological studies has implicated the role of frontal-striatal dysfunction in OCD and related disorders. However, to date, neuroscientists and cognitive-behaviorists have tended to publish in separate journals, with few attempts at integrating their respective evidence bases to advance understanding of psychopathological processes, or in progressing knowledge of treatment-related processes. This special edition presents four articles on OCD and related disorders which present results from studies integrating neuroscientific and cognitive-behavioral approaches, methodologies, and measures. This introduction to the special edition outlines some of the background of this initiative, and contextualizes the articles.

The experience of unwanted intrusive thoughts, images, urges, or doubts that cause distress, and the compulsive and/or ritualistic responses to such intrusions are characteristic of Obsessive-Compulsive Disorder (OCD) and related disorders such as Body Dysmorphic Disorder (BDD) and Tourette’s Syndrome. The way in which individuals respond to unwanted intrusive experiences, as well as the mechanisms underpinning
compulsive responses and their maintenance, plays an important role in cognitive-behavioral models of these disorders (Meyer, Levy, & Schnurer, 1974; Rachman, 1997, 1998, 2002; Salkovskis, 1985; Veale, 2004). In particular, cognitive theorists consider that the anticipation of negative outcomes associated with intrusive experiences in OCD is premised on beliefs such as inflated personal responsibility, perfectionism, intolerance for uncertainty, overestimation of threat, and the need to control intrusions because of their perceived importance (OCCWG, 1997). Behavioral models propose that negative reinforcement of compulsive responses or neutralization results from decreases in distress or discomfort immediately following their engagement (Meyer et al., 1974). However, such responses to intrusions also increase their frequency and salience, setting in motion a compounding cycle that reinforces erroneous beliefs about the meaning attributed to these intrusions.

Cognitive-behavioral models of OCD and related disorders have been instrumental in the development of effective interventions, such as exposure with response prevention (ERP) and cognitive-behavior therapy (CBT), for these disabling disorders (Abramowitz, 2006; Buhlmann, Reese, Renaud, & Wilhelm, 2008; Chang, Piacentini, & Walkup, 2007; Cook & Blacher, 2007; Fisher & Wells, 2005; Rosa-Alcáz, Sánchez-Meca, Gómez-Conesa, & Marín-Martínez, 2008; Williams, Hadjistavropoulos, & Sharpe, 2006). Behavioral treatments such as ERP target the compulsive responses to unwanted intrusions. Cognitive therapy, either alone or combined with behavioral treatment, utilizes techniques such as cognitive restructuring and behavioral experiments to correct erroneous beliefs and appraisals of intrusions (Abramowitz, 2006), and has also been shown to be effective in OCD (Freeston, Rheume, & Ladouceur, 1996; Wilhelm, Steketee, Reilly-Harrington, Dekersbach, Buhlmann, & Baer, 2005), BDD (Buhlmann et al., 2008; Williams et al., 2006), and Tourette's Syndrome (Chang et al., 2007; Cook & Blacher, 2007). Despite their relative effectiveness, cognitive and behavioral approaches to the treatment of obsessive-compulsive and related disorders demonstrate limitations such as high drop-out and relapse rates and poor treatment adherence (Buhlmann et al., 2008; Kyrios, 2003; Rosa-Alcáz et al., 2008; Vogel, Stiles, & Gotestam, 2004). Fisher and Wells (2005) reviewed clinically reliable change following treatment for OCD, and reported that only 50-60% of patients responded to ERP, with even lower recovery rates (25%) for both ERP and CBT when using asymptomatic criteria. Such findings suggest that we need to augment existing interventions or develop alternative treatments and, perhaps, theoretical models to guide such interventions.

In an attempt to broaden our understanding of factors that may require attention in treatment, a number of alternative conceptual frameworks have emerged in recent years for OCD and related disorders. For instance, variables such as reasoning styles, attachment patterns, self-construals, and sensitivity to one's sense of moral self-worth have been associated with obsessive-compulsive symptoms and disorder (Bhar & Kyrios, 2007; Doron, Moulding, Kyrios, & Nedeljkovic, 2008; Doron, Moulding, Kyrios, Nedeljkovic, & Mikulincer, 2009; Ferrier & Brewin, 2005; O'Connor, Aardema, & Pélissier, 2005; Purdon, Cripps, Faull, Joseph, & Rowa, 2007; Szechtman & Woody, 2004). Furthermore, a broad range of cognitive factors linked to vulnerability and maintenance have been identified in BDD patients, including endorsement of delusional beliefs, high fantasy proneness, lower self-esteem, and higher self-ambivalence (Labuschagne, Castle, Dunai, Kyrios, & Rossell, 2010). A recent study found some specificity in the relationships between various self constructs and BDD versus social anxiety symptoms in nonclinical participants (Phillips, Moulding, Kyrios, Nedeljkovic,
& Mancuso, in press). It was concluded that a cognitive-behavioral model of BDD that incorporates ambivalent self-perceptions as a cognitive component, provides further explanatory power in the development and etiology of BDD. Interventions involving self-construals, self-defining memories, and other narratives have already made an impact in mood and anxiety disorders (Singer & Blaghov, 2004), although they have yet to impact significantly on the management of obsessive-compulsive spectrum disorders.

In addition to these psychological theories, neuroscientific models of OCD and related disorders have also emerged, reflecting a more general trend to investigate neurocognitive factors in psychological disorders (for example, see Atchley & Hardi, 2007 for a discussion on the potential of cognitive neuroscience to advance our understanding of depression). Neuroscientific models of disorders such as schizophrenia and autism spectrum disorder have already led to cognitive rehabilitation treatments, which have had some success (Kayser, Sarfati, Besche, & Hardy-Bayle, 2006; Wolf, Tanaka, Klaiman et al., 2008).

Neuroscientific models of OCD are based on evidence from functional, and structural imaging, neuropsychological, and electrophysiological studies that support the presence of dysfunction in frontal-striatal brain areas (Greisberg & McKay, 2003; Rotge, Guelh, Dilharreguy, et al., 2009; Saxena, Brody, Schwartz, & Baxter, 1998; Zald & Kim, 1996). Our own research in OCD has supported neuropsychological, functional and some possible structural differences in OCD patients relative to clinical and non-clinical controls. For instance, in a neuropsychological study, we found impaired planning, and strategy, nonverbal memory deficits (especially spatial working memory), and slowed response speed in OCD relative to normal controls, and patient groups with major depression and panic disorder (Purcell, Maruff, Kyrios, & Pantelis, 1998a, 1998b). Using univariate and multivariate approaches to analysis of functional imaging in order to generate a more robust profile of brain activity in OCD patients versus healthy controls undertaking an inhibitory control task, we found that both methods supported abnormal corticostriatal activity in OCD patients (Harrison, Yücel, Shaw, et al., 2006). Utilizing MRI, we also examined morphological and volumetric properties in three key brain areas associated with OCD, namely the orbitofrontal, anterior cingulate, and caudate nucleus (Riffkin, Yücel, Maruff, et al., 2005). Using an automated voxel-based analysis on patients with OCD and gender-, age-, and education-matched healthy controls, we found no significant structural differences in these key brain areas. Finally, using an inhibitory control paradigm designed specifically to probe the medial frontal cortex, especially the dorsolateral anterior cingulate and the supplementary motor area, we found significant deactivation in the rostral anterior cingulate in OCD participants relative to normal controls (Yücel, Harrison, Wood, et al., 2007). In the same study, biochemical markers of neuronal integrity and function were significantly reduced in the dorsal anterior cingulate of OCD patients.

Similarities have been reported between OCD and related disorders, inclusive of Tourette’s, trichotillomania and BDD, in their neurobiological substrates (Chamberlain, Fineberg, Blackwell, Clark, Robbins, & Sahakian, 2007; Feusner, Yaryura-Tobía, & Saxena, 2008; Harris & Singer, 2006; Hollander, Braun, & Simeon, 2008). Our own research with BDD patients found neurocognitive deficits such as between search errors on a spatial working memory task, but not on spatial span, pattern recognition, or motor speed tasks (Dunai, Labuschagne, Castle, Kyrios, & Rossell, 2010). Importantly, the severity of BDD, depressive, or anxiety symptoms were not correlated with performance on any of the neurocognitive tasks. Overall, our findings were indica-
tive of a disorder with varying degrees of delusional thinking with executive function deficits involving on-line manipulation, planning, and organization of information. Evidence from lesion and neuroimaging studies suggests frontal lobe dysfunction may be associated with BDD. However, the association of cognitive and neurocognitive factors in BDD remains unknown.

The link between neuroscientific and cognitive-behavioral correlates, as well as the concerns of cognitive-behavioral researchers and therapists about the heuristic value of neuroscientific models, has yet to be examined systematically. There has been very little literature integrating neuroscientific and cognitive-behavioral findings in the psychopathology literature. In the OCD area, notable exceptions include Otto (1992), who proposed a neuropsychological perspective of normal and abnormal information processing in OCD. Furthermore, O'Connor (2002) presented an integrated cognitive-behavioral and psychophysiological model of tic disorders. More recently, Nedeljkovic and Kyrios (2007) examined metamemory beliefs in OCD, proposing a relationship between lowered confidence in cognitive abilities and compromised neuropsychological performance in OCD.

Neuroscientists and cognitive-behaviorists publish in separate journals and often lack knowledge of each other's methodologies and theoretical bases, thus limiting the opportunities for cross-pollination, which could lead to theoretical developments, advances in our understanding of psychopathological processes, and increased heuristic value of each approach. The cognitive neuroscience and imaging area has been criticized on various philosophical and pragmatic grounds, including its less than optimal consideration of the phenomenology of psychological disorders, on which cognitive models of psychopathology place particular importance (Fuchs, 2002; Fusar-Poli & Broome, 2006). However, it has been argued that cooperation linking phenomenology, psychopathology, and cognitive science is necessary for further advancement of our understanding of disorders (Fuchs, 2002).

Focusing discussion on the utility of neuroscientific methodologies for cognitive theory and therapy, a number of potential applications can be identified. First, brain processes and their proximal or functional markers (e.g., brain biochemistry, neuropsychological deficits, EEG patterns, functional or structural properties of key brain areas) could provide markers of vulnerability to the development of disorders or to psychological processes associated with disorder. For instance, the neuroscientific study of moral reasoning has identified specific brain processes which have been associated with antisocial personality disorder (Braun, Léveillé, & Guimond, 2008) and which could have important implications for our understanding of the moral-based factors in OCD (cf. Doron et al., 2008). Neuroscientific studies have also identified brain-related processes associated with attachment patterns in animals and humans (Bora, Yücel, & Allen, 2009; Gunnar & Fisher, 2006). As already indicated, such attachment patterns have already been linked to OCD (Doron et al., 2009), although such links are tentative at this stage. Furthermore, constructs such as “cognitive inflexibility” have been identified by neuroscientists as an important etiological and phenomenological component of OCD (Chamberlain, Fineberg, Blackwell, Robbins, & Sahakian, 2006; Gu, Park, Kang, et al., 2008) and, despite some limited theoretical discourse in the constructivist literature (e.g., Guidano & Liotti, 1983) and an abundance of similar or related concepts in the cognitive and psychological literature (e.g., openness, “just right” experiences, over-valued ideation, inference-based reasoning deficits), there has been little cross-fertilization between neuroscientists and cognitive-behaviorists in developing a more complete definition of this potentially useful construct.
Second, brain patterns and neurocognitive measures could predict treatment outcomes or progress our knowledge of treatment-related processes. For instance, a recent review of neuroimaging studies following psychological treatment for anxiety and mood disorders concluded that decreased OCD symptoms following ERP was associated with functional changes in the caudate-orbital-thalamic brain circuit, reflecting enhanced self-regulatory control in CBT-treated individuals with OCD (Frewen, Doozois, & Lanius, 2008). However, the number of existing studies is very small, power is often compromised, control conditions are often nonexistent, and there is a paucity of studies with related disorders such as BDD and Tourette’s. Furthermore, new imaging technologies are being developed (e.g., magneto-encephalography [MEG]) that have yet to be utilized in such studies. Finally, recent advances in biological treatments for severe infractory OCD [e.g., Deep Brain Stimulation (see Mian, Campos, Sheth, & Eskandar, 2010)] have found some support but still require integration with CBT in order to maintain gains. However, no controlled studies have been undertaken, reflecting a general lack of communication between relevant disciplines.

This special section on the utility of neuroscience to cognitive-behavioral theories and management of OCD and related disorders presents four articles, which in different ways attempt to fill gaps in the existing literature. The first paper by Nedeljkovic and colleagues examines the relationship between OCD symptom improvement following manualised CBT and changes in neuropsychological performance on a spatial working memory task, compared to a test-retest control cohort. Two articles on OCD-related disorders follow, further reflecting the potential utility of neuroscientific factors. Labuschagne and colleagues examine a case series of patients with BDD, describing their clinical features and outlining the results of detailed cognitive assessments, including tests of executive function, facial affect perception, and general social cognition, with implications for treatment and for cognitive theories of BDD. Also examining the utility of neuroscientific factors in understanding treatment processes, Lavoie and colleagues explore the hypothesis that symptomatic amelioration associated with CBT for Tourette’s Syndrome is related to changes in cerebral activity as assessed via electroencephalograms (EEGs) obtaining event-related potentials (ERP) during motor tasks. Finally, the fourth article by Fitzgerald and colleagues draws on neuropsychological and cognitive-behavioral approaches to OCD, using an experimental design to examine the association between memory performance, cognitive confidence, and OCD phenomena in a nonclinical cohort.

REFERENCES


