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The contribution of perceived control of stressful life events and thought suppression to the symptoms of obsessive–compulsive disorder in both non-clinical and clinical samples

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Abstract

The two studies presented in this paper investigated the impact of controllable versus uncontrollable stressful life events (SLE) and low versus high thought suppression upon symptoms of obsessive–compulsive disorder (OCD) in both a non-clinical sample (Study 1) and a clinical sample (Study 2). The sample for Study 1 consisted of 269 undergraduate university students and the sample for Study 2 consisted of 91 participants obtained from the Obsessive Compulsive and Anxiety Disorders Foundation of Victoria, Australia. Participants in both studies were given identical questionnaires measuring anxiety, depression, thought suppression, OCD, and, the control, magnitude and number of SLEs suffered during the previous 18 months. In both studies, analysis of covariance (ANCOVA) followed by post hoc tests indicated that high OCD scores were associated with high thought suppression and low perceived control over high magnitude stressful life events relative to controls. The results suggest that high thought suppression coupled with low control over stressful life events may interact with other predisposing factors, such as genetic vulnerability to produce OCD symptoms.

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1. Introduction

Obsessive–compulsive disorder (OCD) is one of the most debilitating and prevalent anxiety disorders, with a cross-cultural lifetime prevalence of about 2.5% (Karno, Golding, Sorenson, & Burnam, 1988; Nelson & Rice, 1997; Stein, Forde, Anderson, & Walker, 1997; Trivedi, 1996; Weissman et al., 1994). Sufferers typically spend large amounts of time obsessing about intrusive phenomena and feel compelled to carry out extensive rituals that are both distressing and time consuming (Abramowitz, 1997).

A number of laboratory studies have demonstrated that subjects who display a strong tendency to suppress their thoughts experience an increased frequency of the suppressed thoughts in what has become known as the “rebound effect” (Clark, Ball, & Pape, 1991; Lavy & van den Hout, 1990; Wenzlaff, Wegner, & Klein, 1991). In addition, some researchers have found that people with OCD tend to suppress their thoughts more than non-OCD subjects (Muris, Merckelbach, & Horselenberg, 1996; Salkovskis, 1985). In line with this, the “rebound” phenomenon has been postulated to be an important factor in the maintenance of OCD symptomatology, particularly the obsessional component of the disorder (Salkovskis & Campbell, 1994). However, the relationship between thought suppression and psychopathology remains unclear, since a number of researchers have failed to identify thought suppression as a predictor variable for increased intrusive phenomena (Kelly & Kahn, 1994; Roemer & Borkovec, 1994; Salkovskis & Campbell, 1994).

While considerable evidence is now available indicating that there is a major genetic contribution to the emergence of the disorder (e.g., Pauls & Alsobrook, 1999; Pollock & Carter, 1999) and to its co-morbidity with Tourette’s syndrome (Pauls et al., 1987), less interest has focused upon the environmental factors that might govern the initial onset of OCD.

There are good reasons for connecting onset of OCD with certain types of SLE. In a comprehensive review of the literature, Paykel and Dowlatshahi (1988) found that SLE were implicated in a number of psychological disorders including anxiety and depression. Similarly, Angst and Vollrath (1991) found that anxiety disorders are likely to be triggered by SLE. Other studies have found that SLE often precipitate depressive episodes (Brown & Bifulco, 1985; Overholser, Norman, & Miller, 1990).

One study which attempted to differentiate between the types of events involved in particular patterns of symptomatology found that events involving loss were more likely to lead to depressive symptoms, whereas events involving danger were more likely to lead to anxiety symptoms (Finally-Jones & Brown, 1981). On the other hand, other studies have found that recent SLE often play an important role in mixed forms of depression–anxiety, whereas they appear to only play a minor role in “pure” forms of depression and anxiety, where childhood and genetic factors are noted to be of greater importance (Alnaes & Torgersen, 1988; Torgersen, 1985). Although a number of theorists (see Warren & Zgourides, 1991)

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