

**- SAMPLE DOCUMENT ONLY -**

**Visit BetterEdit for details - <http://www.BetterEdit.com>**

Running Head: OBSESSIVE-COMPULSIVE DISORDER

Obsessive-Compulsive Disorder: A Review of a Biological and Behavioural Model

Ben Jardine

Obsessive-compulsive disorder (OCD) is a devastating culmination of anxiety disorders (Barlow & Durand, 2002), which can be both disabling and distressing for the affected person and also for friends and relatives (Rachman & Shafran, 1998). OCD is highly comorbid with other syndromes, particularly mood disorders, social phobia, substance abuse, depression and eating disorders (Craske, 1999). It tends to be chronic and was previously highly resistant to treatments (Minichiello, Baer & Jenike, 1988). However, presently there are a number of effective ways to treat OCD, which will be explored in this essay.

OCD usually involves having both obsessions and compulsions. Obsessions and compulsions are strongly associated with anxiety and even driven by anxiety (for these reasons, OCD is classified as an anxiety disorder in the Diagnostic and Statistical Manual of Mental Disorders–IV). Obsession refers to unwanted and distressing, recurrent thoughts or images which intrude into the individual's awareness, and that are resisted by the patient (Rachman and Shafran, 1998). Compulsions are continuous, purposeful and stereotypical behaviours or rituals that the patient performs to counteract the anxiety and distress triggered by obsessive thoughts (Kampman, Keijsers, Hoogduin & Verbraak, 2002); such as washing, checking, touching, counting, hoarding, arranging and praying (Rachman & Shafran, 1998). The individual feels driven to perform according to the specific rules so as to reduce the likelihood of a feared disastrous consequence occurring (Riggs & Foa, 1993). OCD patients are often aware that their compulsive behaviour is excessive or irrational, but nonetheless feel driven to perform it (Bram & Bjorgvinsson, 2004).

Biological, behavioural and cognitive paradigms are the typical models used to explain OCD (Jakes, 1996). In this essay, OCD will be explained from the behavioural and biological perspectives. A specific intervention will be described from each model: the serotonin reuptake inhibitor (SRI) treatment will be discussed in terms of the influence of the biological model, while the exposure response prevention (ERP) therapy will be discussed in terms of the effect of the behavioural paradigm in influencing the treatment. This will be followed by a discussion of the relative efficacy of each intervention and the outcome when integrating the two treatments.

#### *A biological explanation of OCD*

It has been proposed that some symptoms associated with OCD may be prescribed to structural or neurochemical disruptions (Salkovskis, 1998). Gross and colleagues (1998) posited that the underlying pathophysiology of OCD was due to hypersensitivity in the postsynaptic 5-hydroxytryptamine (5-HT) receptors and the abnormalities in the serotonin system. The involvement of the serotonin system is thought to revolve around altered sensitivity of the neurotransmitter receptor (Gross et al., 1998). In addition, the abnormal serotonergic system, developing from a neurochemical imbalance, could have also led to the biological hypothesis. Support for this hypothesis has come mainly from studies of the efficacy of serotonergic drugs, in which OCD patients were found to respond positively to serotonergic medication relative to other anxiety disorders and placebos (Riggs & Foa, 1993).

Grados and Riddle (2001) suggested that OCD involves problems in the communication between the front part of the brain (orbital cortex) and the deep structures

(basal ganglia). The dysfunction of the basal ganglia is likely to be implicated in OCD as it is connected to the repeated production of movement and thus could have led to the compulsive rituals (Grados & Riddle, 2001). Jakes (1996) stated that clusters of serotonin and serotonin receptors have also been found in the basal ganglia to further suggest the involvement of the region in OCD. Rachman (2004) stated that perhaps the reduced confidence in memory demonstrated in compulsive checking was due to insufficient levels of serotonin or abnormal basal ganglia.

There have been many studies that implicate the neurotransmitter serotonin in playing a central role in the development of OCD (Riggs & Foa, 1993; Lopez-Ibor & Lopez-Ibor, 2003). Therefore, the biological model explains the prevalence of OCD as emerging from a biochemical imbalance; specifically, the abnormal serotonergic system and the insufficient levels of serotonin.