

# [The biological basis of obsessive-compulsive disorder](https://assignbuster.com/the-biological-basis-of-obsessive-compulsive-disorder/)

Obsessive-Compulsive Disorder (OCD) is a debilitating anxiety disorder that initiates feelings of distress to those suffering from it. Such obsessive-compulsive disorders include hoarding disorder, body dismorphic disorder, trichotillomania (hair-pulling disorder), and excoriation (skin-picking disorder) (American Psychiatric Association, 2013). According to the Diagnostic and Statistical Manual of Mental Disorders (DSM-V), OCD is divided into two categories: obsessions and compulsions. Obsessions are disturbing, persistent thoughts that provoke unwanted feelings of anxiety and nervousness. Compulsions influence individuals to perform certain behaviors in an effort to reduce these anxious feelings. Such examples include hand washing, organizing, counting, repeating words, constant fear of germs, and et al. Consequently, obsessions and compulsions are unwarranted and excessive, as individuals constantly feel obligated to perform certain rituals in an effort to experience temporary relief (Rachman, 2017).

OCD is portrayed as one of the top ten leading causes of disability around the world (Davis, 2008). Patients suffering from OCD display a variety of cognitive and behavioral characteristics, including perfectionism and an overwhelming need for control. Such individuals tend to create negative scenarios, in addition to anticipating the worst. Foreshadowing negative situations only increases the likelihood of such situations from occurring, which increases obsessive-compulsive behavior. Negative thoughts do not make the situation less intimidating, but rather makes the situation worse. Thus, it is important to understand that OCD is an unbearable impairment.

Given that OCD demands a multitude of timely explanations, it is essential to recognize the plethora of factors that contribute to obsessive-compulsive behavior. Such factors include psychological, social, environmental, and cultural influences. However, recent studies have explored the possible biological basis of OCD, in addition to identifying the possible pathophysiological and neuroanatomy of this disorder. Likewise, helpful medications and treatment methods have been designed in order to alleviate the uncomfortable symptoms of OCD. Thus, the biological basis of OCD is influenced by the interactions between nature and nurture. By identifying the biological basis of OCD, in addition to determining several treatment approaches, individuals can remove the stigma that is often associated with obsessive-compulsive disorder.

Environmental Factors

With respect to nurture, OCD can arise from various environmental influences including cultural factors, familial backgrounds, traumatic life events, and social relationships. In particular, stressful life events (SLEs) are considered one of the major contributors to obsessive-compulsive behaviors. Research studies involving the relationship between SLEs and OCD initiated significant results. Rosso et al. (2012) was interested in diagnosing individuals who suffered from OCD with and without SLEs preceding obsessive-compulsive behavior. The researchers were further concerned with identifying the specific types of SLEs that may trigger obsessive-compulsive behavior among the participants. The results demonstrated that 200 participants experienced OCD symptoms when a stressful life event was presented initially. Additional data signified that females were more likely to experience obsessions and compulsions, as opposed to their male counterparts. The researchers insinuated the notion that (1) “ hospitalization of a family member,” (2) “ major personal physical illness,” and (3) “ loss of a personally valuable object” were significant SLEs that contributed to the onset of obsessive-compulsive behaviors. Therefore, the researchers hypothesize that minimizing a connection to stressful life events may reduce OCD, given that SLEs are detrimental to one’s psychological and physiological well-being.

Biological Basis, Pathophysiology, and Neuroanatomy

With regards to nature, twin and family studies have demonstrated possible genetic factors underlying OCD. Such studies revealed that obsessive-compulsive behaviors were significantly higher among individuals who were related to one another. Family members connected to adults with OCD tended to experience the symptoms of OCD twice as often as the control population. Likewise, family members connected to children and adolescents with OCD symptoms were ten times more likely to exhibit such obsessions and compulsions (Grootheest et al., 2012). Nevertheless, it is obvious that future research studies should be conducted in order to clarify these results. Additionally, the significance of family genetics as a possible contributor to OCD should not be overlooked.

Research concerning the possible genetic influences of OCD continued with Mattheisen et al. (2015). Mattheisen and his team of researchers conducted a genome-wide study with respect to obsessive-compulsive disorder. They stressed the significance of identifying a particular genetic component of OCD in an effort to create effective treatment methods to alleviate such symptoms. The researchers rigorously analyzed 5, 601 human genomes in total, which included: (1) 1, 406 individuals suffering from OCD, (2) 1, 000 family members with OCD, and (3) 2, 655 individuals from the general population. The results indicated a significant relationship between patients with OCD and a protein tyrosine phosphokinase gene (PTPRD). PTPRD branches from the protein tyrosine phosphatase (PTP) family; PTP is designed to maintain cell growth and axon guidance. The researchers accentuated the fact that this association is groundbreaking for a variety of reasons. Primarily, they found that PTPRD might be linked to attention deficit hyperactivity disorder (ADHD). This is important because ADHD shares similar etiological influences comorbid with OCD. Likewise, they established that PTPRD is linked to knowledge and memory retention in animals. Such areas involving learning and memory are associated with OCD in humans as well. In addition, the researchers discovered that PTPRD works in conjunction with the SLITRK1 family gene, which regulates synaptic transmission. SLITRK1 binds with PTP, initiating the early release of excitatory messages and allowing over-excited signals to travel through neural pathways. SLITRK is further associated with OCD in animals, highlighting the notion that animals can be used to model OCD in humans. Consequently, the researchers stressed the importance of exploring the possible genetic variables of OCD found in animals. Such discoveries will create an understanding for recognizing the neural pathways of this disorder in humans.

Along with the possible genetic influences, the pathophysiology and neuroanatomy of OCD has yet to be determined. Nevertheless, research studies have declared that abnormalities in the brain may contribute to the biological basis of OCD. Functional neuroimaging of patients suffering from OCD have reported high activity levels in the frontal lobe, which is responsible for motor control, problem solving, and reasoning. In particular, the orbitofrontal cortex (OFC), the anterior cingulate cortex (ACC), and the caudate nucleus are areas located within the frontal lobe that are associated with obsessive-compulsive symptoms (Maia, Cooney, & Peterson, 2008). Respectively, the OFC is involved with cognitive functioning and decision-making, the ACC entails emotion and cognition, and the caudate nucleus comprises the basal ganglia. The basal ganglia initiates wanted behaviors, while inhibiting those behaviors that are unwanted. The reality is that damage to these neuroanatomical zones may possibly initiate the onset of obsessive-compulsive behaviors.

The cortico-basalganglia-thalamo-cortical loop contains the OFC and ACC; two regions that are connected to the basal ganglia (Alexander, DeLong, & Strick, 1986). Research studies propose that the disconnection involving passages through the basal ganglia may contribute to obsessive-compulsive symptoms. The direct pathway begins at the cortex, moves to the thalamus (sensory relay station), and returns to the cortex. This direct pathway is excitatory, which indicates that neurons are constantly firing. Thus, it has been suggested that over-excited signals along the passage route result in a “ positive feedback loop” that may trap the signals of obsessive-compulsive thoughts. If this occurs, the thalamus begins to communicate positive signals to the OFC, which will initiate compulsive responses (Beucke, 2013).

Treatment Methods

These significant findings helped to initiate treatment methods designed to alleviate obsessive-compulsive symptoms. Therapeutic methods revolve around cognitive-behavioral therapies. Such methods allow patients to alter their ritualistic behaviors, in addition to minimizing feelings of anxiety and nervousness. With respect to pharmacotherapy, medications center on the biological aspect of OCD. Such medications help to boost serotonin levels in the brain, which ultimately reduces the outcome of obsessions and compulsions. Therefore, patients suffering from OCD will be less hyperactive following the implementation of cognitive-based therapy and/or successful pharmacotherapy.

Cognitive-behavioral therapy (CBT) exposes patients to a series of approximations with regards to the anxiety-producing stimulus under relaxed conditions. In time, anxious feelings are extinguished. A major component of CBT is systematic desensitization, which allows the therapist to teach the patient specific relaxation techniques. This serves as a form of exposure therapy given that the patient learns to overcome fears by exposure to the fear-producing conditions (Foa, 2010). Essentially, systematic desensitization exposes the patient to the obsessive stimulus, while slowly increasing the strength of the exposure. This works to minimize feelings of anxiety. Upon successful completion of systematic desensitization, the patient’s obsessive-compulsive behavior may subside.

With regards to the biological component of OCD, psychopharmacological treatment methods have been utilized in an effort to diminish obsessive-compulsive behaviors. Such medications serve as agonists, given that their function is to facilitate an increase in serotonin levels. Tricyclic antidepressants (TCA), such as Clomipramine, work to effectively treat OCD. Clomipramine is sold under the brand name Anafranil, the first drug used to demonstrate symptom relief. As a TCA, clomipramine works to boost norepinephrine and serotonin levels in the brain, in addition to reducing reuptake back to the axon terminals. Consequently, Anafranil decreases the intensity of unwanted thoughts and behaviors (Kellner, 2010). Likewise, Kellner signified that selective serotonin reuptake inhibitors (SSRIs) successfully increase serotonin levels in the brain by preventing reuptake in the synapse. He further implied that higher doses of SSRIs are more effective in combating obsessive-compulsive behaviors, as opposed to reduced dosages. Such SSRIs include Prozac, Zoloft, Paxil, and Lexapro. In addition, Figueroa et al. (1998) proposed that clomipramine and SSRIs in combination are successful alleviators of obsessive-compulsive symptoms. However, Fineberg et al. (2012) strongly argues that SSRIs should be utilized, as opposed to clomipramine, given that clomipramine has a dangerously high toxicity level, which may possibly result in death. SSRIs, on the other hand, rarely results in death with overdose.

Given these facts, including supporting data, it is apparent that obsessive-compulsive behavior may have a biological component that must be considered when discussing a successful treatment methodology. The truth of the matter is that OCD hinders daily activities and relationships, negatively impacting one’s lifestyle. It is for this reason that obsessive-compulsive behaviors must be viewed as potentially debilitating, and therefore, require a multi-faceted treatment plan.

## References

* Alexander, G. E., DeLong, M. R., & Strick, P. L. (1986). Parallel organization of functionally segregated circuits linking basal ganglia and cortex. Annual Review of Neuroscience, 9 , 357-381. doi: 10. 1146/annurev. ne. 09. 030186. 002041
* American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders (5th ed.). Arlington, VA: American Psychiatric Publishing.
* Beucke, J. C., Sepulcre, J., Talukdar, T., Linnman, C., Zschenderlein, K., Endrass, T., … Kathmann, N. (2013). Abnormally high degree connectivity of the orbitofrontal cortex in obsessive-compulsive disorder. JAMA Psychiatry, 70 (6), 619-629. doi: 10. 1001/jamapsychiatry. 2013. 173
* David, L. J. (2008). Obsession: A history. Chicago, Illinois: University of Chicago Press.
* Figueroa, Y., Rosenberg, D. R., Birmaher, B., & Keshavan, M. S. (1998). Combination treatment with clomipramine and selective serotonin reuptake inhibitors for obsessive-compulsive disorder in children and adolescents. Journal of Child and Adolescent Psychopharmacology, 8 (1), 61-67. doi: 10. 1089/cap. 1998. 8. 61
* Fineberg, N. A., Brown, A., Reghunandanan, S., & Pampaloni, I. (2012). Evidence-based pharmacotherapy of obsessive-compulsive disorder. International Journal of Neuropsychopharmacology, 15 (8), 1173-1191. doi: 10. 1017/S1461145711001829
* Foa, E. B. (2010). Cognitive behavioral therapy of obsessive-compulsive disorder. Dialogues in Clinical Neuroscience, 12 (2), 199-207. Retrieved from https://www. ncbi. nlm. nih. gov/pmc/articles/PMC3181959/
* Grootheest, D. S., Cath, D. C., Beekman, A. T., & Boomsma, D. I. (2012). Twin studies on obsessive-compulsive disorder: A review. Twin Research and Human Genetics, 8 (5), 450-458. doi: 10. 1375/183242705774310060
* Kellner, M. (2010). Drug treatment of obsessive-compulsive disorder. Dialogues in Clinical Neuroscience, 12 (2), 187-197. Retrieved from https://www. ncbi. nlm. nih. gov/pmc/articles/PMC3181958/
* Maia, T. V., Cooney, R. E., & Peterson, B. S. (2008). The neural basis of obsessive-compulsive disorder in children and adults. Development and Psychopathology, 20 (4), 1251-1283. doi: 10. 1017/S0954579408000606
* Mattheisen, M., Samuels, J. F., Wang, Y., Greenberg, B. D., Fyer, A. J., McCracken, J. T., … Nestadt, G. (2015). Genome-wide association study in obsessive-compulsive disorder: Results from the OCGAS. Molecular Psychiatry, 20 (3), 337-344. doi: 10. 1038/mp. 2014. 43
* Rachman, S. (2007). Unwanted intrusive images in obsessive-compulsive disorders. Journal of Behavior Therapy and Experimental Psychiatry, 38 (4), 402-410. doi: 10. 1016/j. jbtep. 2007. 10. 008
* Rosso, G., Albert, U., Asinari, G. F., Bogetto, F., & Maina, G. (2012). Stressful life events and obsessive-compulsive disorder: Clinical features and symptom dimensions. Psychiatry Research, 197 (3), 259-264. doi: 10. 1016/j. psychres. 2011. 10. 005