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Nature vs. Nurture: In What Ways Do Genetic Factors and Environmental Factors Play a Role in the Causation of Obsessive-Compulsive Disorder?

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Abstract: This paper reviews the studies and other literature on genetic and environmental factors that contribute to the causation of Obsessive-Compulsive Disorder (OCD). The CTSC and belief and appraisal model are two popular explanations for OCD. This research found that OCD is a familial disorder, genetics affect childhood-onset OCD more than adults, and genetic factors play a more significant role in causing obsessive symptoms. Environmental factors are explored based on five learning experiences concluded as the origin of inflated responsibility in patients, parental rearing styles, and stressful or traumatic events likely to correlate with OCD. The gene-environmental interactions are complicated, and future research is needed.

Keywords: Obsessive-Compulsive Disorder (OCD), causation, genetic factors, learning experiences, inflated responsibility

1. Introduction

What factors are tied to the causation of obsessive-compulsive disorder (OCD) has long been a debatable topic. Models of OCD have been proposed from both a neurobiological perspective and a cognitive-behavioral perspective, and the factors causing OCD are concluded based on these models. There are controversial views about whether OCD is genetically heritable or nurtured by the environment. However, little research is aimed at finding a combination of both factors. Based on this context, this literature review will introduce the two prevailing models of OCD, one from a neurobiological perspective and one from a cognitive behavioral perspective, evaluate the studies on both genetic and environmental factors and finally discuss why is it complicated to determine the gene-environmental interactions in OCD and determine areas for future research [1-3]. This paper aims to achieve a more holistic view of the causation of OCD and suggest directions for future research.

2. Models of OCD

2.1. Introduction to OCD

A psychological condition known as obsessive-compulsive disorder (OCD) concerns either the presence of obsessions, compulsions, or both [4]. Habits are defined as the patient's conscious,

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invasive, and unwanted thoughts, intentions, and visuals that can be distressing or anxious. The patient tries to ignore such intrusion and usually aims to neutralize thoughts via the performance of a compulsion. Compulsions are recurring behavior or mental acts that the patients tend to carry out, but these behaviors are not connected to their aim of reducing anxiety and distress realistically. By performing the compulsions, the individual experiences temporary relief, but this reinforces the act of compulsion. This diagram explains why obsession leads to compulsive behavior and shows that the behavior will be reinforced [1].

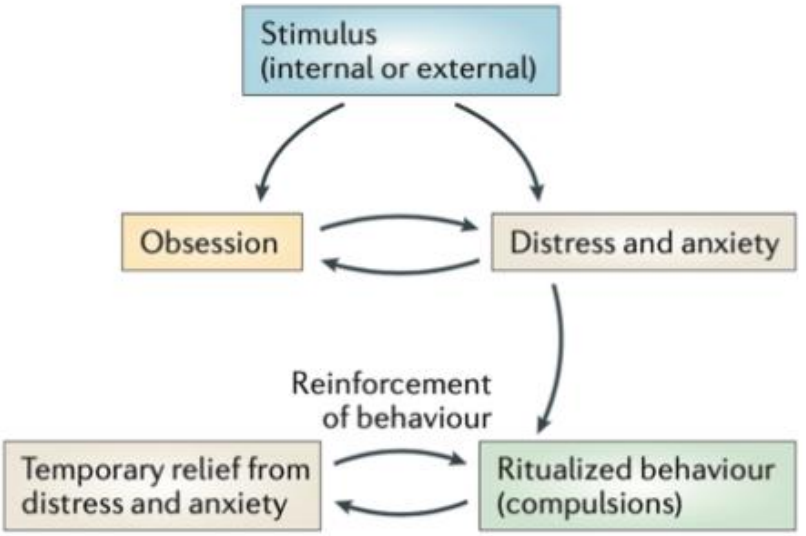

Figure 1: How OCD patients' behavior work.

2.2. The Cortico-Striato-Thalamo-Corticol (CTSC) Model

Through functional neuroimaging studies, Saxena proposed the CTSC model in 2000 to explain the differences in neurocircuit in OCD patients and healthy individuals. The orbitofrontal cortex (OFC), which the study indicates is a region of the brain, localizes the function of moderating heightened anxieties towards danger, hygiene, or threat, which OCD patients often worry about. The OFC is part of the orbitofrontal-subcortical pathway that fires the direct path, which has an excitatory function in the front striatal circuitry. In healthy individuals, the indirect way, which is the GABAergic pathway with an inhibitory role, will modulate the part of the direct path, so the influence of the OFC can be limited. However, in OCD patients, as Saxena stated, there is an imbalance between the two pathways; there is usually a lower activity in the indirect pathway, which leads to excessive working in the direct path. This, in turn, will lead to the hyperactivation of the orbitofrontal-subcortical pathway. The function of OFC cannot be inhibited enough. Thus, excessive worries about danger, cleanliness, or threat may result in obsessions and lead to compulsive rituals to neutralize the threat [5].

This model successfully identifies one difference between OCD patients and healthy individuals from a neurobiological perspective, paving the way for future research to identify genes that might be related to this circuitry to determine the causation of OCD from a biological point of view. Despite this work being done from a neurobiological perspective, one should note here that it doesn't mean that OCD is caused purely by biological factors, the environment can also alter the

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brain, so the causation of OCD remains open to both genetic and environmental factors. One limitation of Saxena's work is that the researcher focused on this one circuitry through neuroimaging studies. However, the interactions of the front striatal circuitry with other circuits remain unknown; more studies need to be done to determine how the brains of OCD patients work.

2.3. The Belief and Appraisal Model

This model is formulated from a cognitive-behavioral perspective and is not mutually exclusive with the CTSC model. It is two prevailing explanations of OCD from different perspectives.

The belief and appraisal model have formulated upon Beck's cognitive hypothesis that different psychopathology arose from varied categories of dysfunctional beliefs in 1976, so this model mainly explains the dysfunctional beliefs of OCD patients [2,6]. The main contributor to this model is Salkovskis, who claimed the cognitive-behavioral theory of OCD. He proposes that OCD patients pay more attention to the unwanted, intrusive thoughts similar to compulsive thoughts; they will wish to appraise those intrusions as dangerous or allocate individual responsibility for damage [3]. He proposed the concept of "inflated responsibility," which is how one individual misinterprets their responsibility to believe that they have power over something which they don't and that the individual will indicate responsibility to oneself for preventing or causing something from happening [2, 5, 7]. His model successfully explains why OCD patients are likely to appraise intrusions in terms of responsibility and identifies learning experiences that can lead to such beliefs and appraisals, which we will discuss later. Still, his theory is limited to responsibility, and he is unaware of other misconceptions about OCD patients. Based on Salkovskis, Rachman suggested that obsessions are broader, and misinterpretation doesn't only include responsibility; it can be any misinterpretation of the importance of an individual's ideas, images, or instincts [8]. There is more contemporary work done by the Obsessive-Compulsive Cognitions Working Groups, which recognized more specific beliefs of OCD patients that they conceptualized as giving rise to corresponding appraisals. They also developed smaller-scaled models to explain more particular types of OCD, which is helpful because OCD consists of many subtypes. If future researchers can identify the specific types, the causation of OCD can be investigated explicitly for each subtype.

The belief and appraisal model are supported by cognitive-behavioral theory and a range of empirical studies beginning in the 1980s; most evidence aligns with each other, so one could say that this model does have sufficient practical support [2]. The OCCWG and other modern researchers are working to improve the gaps and cover a broader range of beliefs and appraisals to fill in the areas of uncertainty about specific subtypes and their differences in their dysfunctional beliefs.

3. Genetic Factors

It has been the focus of numerous family investigations, twin studies, and genome-wide association studies to identify particular genetic contributions to the development of OCD and, more precisely, to determine the symptoms most likely to be influenced by genetic variables.

3.1. Family Studies

Family studies showed a significant association between OCD in the probands and their first-degree relatives and that childhood-onset or early-onset OCD is more attributed to genetic factors [9].

One comprehensive family study was done by Nestadt et al. in 2000, which used standardized diagnostic instruments and features and professional examiners for an interview. Two expert psychiatrists later reviewed all the diagnostic materials, including the assessment by a clinical examiner, in-depth discussion, audiotapes, and a clinal case summary. The probands and relatives

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were assessed to be separated into “definite,” “probable,” “not present,” and “unknown” according to DSM-IV criteria. Notably, the researchers noted symptoms of obsessions or compulsions even if the standard of OCD is not met, which provided a detailed insight into what role genetic factors play in the causation of OCD. The findings of Nestadt et al., consistent with Hettema’s review and meta-analysis of previous family research, show that first-degree relatives of probands have a noticeably higher probability of receiving an OCD diagnosis and have an almost 5-fold higher lifelong prevalence of the disorder [10]. The study also found that obsessions have a higher heritability than compulsions, which we could infer that genetics play a more significant role in causing obsessive symptoms. However, more study needs to be done to assert this result. Suppose this result is accorded with future studies. In that case, future research can focus on trying to identify genes that are correlated with obsessive behaviors and analysis for common gene variants that occur in both OCD patients and patients of other disorders which also have the component of compulsive symptoms; we see the possibility that this can be a new area of focus in searching for genetic causation of OCD. Another conclusion of the study is that onset at an early age of the symptoms is more familial than in older generations [9]. This aligns with Arumugham et al. study in 2014, and it is largely agreed that children are more affected by genetics, and its effect reduces as individuals grow older [11].

However, even though studies can conclude that OCD is a familial disorder, the aspect of genetic causation can only be inferred. This is because it is possible to argue that such a familial inheritance of OCD is due to the familiar environment in a family with an OCD patient and has little to do with genetics. It is also feasible that other family members can acquire the patient’s obsessive and compulsive behaviors.

3.2. Twin Studies

While family studies suggested the heritability of OCD, researchers had done twin studies to identify whether and how much genetics contribute to the causation of OCD.

The concordance rate in monozygotic twin pairs looked to be more than twice as high as the concordance rate in dizygotic twin pairs, according to studies comparing OCD onset in monozygotic twin pairs with dizygotic twin pairs [12]. This result is confirmed by a more contemporary analysis by Iervolino et al. in 2011, who also concluded that the concordance rate is higher in monozygotic twins [13]. Since monozygotic share nearly all their genes and dizygotic share half of their genes, the studies reveal that genetic factors contribute to the causation of OCD.

From another angle, in a review by van Grootheest et al. in 2005, the researchers assessed past twin studies done on children and adults. They concluded that the genetic liability for children is from 45% to 65%, while for an adult is lower, ranging between 27% and 47%; this accords with Nestadt et al. and other researcher’s conclusion in previous family studies of how the genetics in OCD play a more significant role in childhood-onset OCD than onset at an older age. Meanwhile, van Grootheest admitted that studies done on a larger scale need to be conducted to confirm the validity of his conclusion [11, 14].

3.3. Searching for Specific Genes

So far, no single nucleotide polymorphisms (SNP) have achieved genome-wide significance in case-controlled GWAS [1]. This suggests that the genetic factor in OCD might not be significant; at least, no single gene can determine the onset of this disease.

Candidate genes are also studied based on prior knowledge of OCD , and this links back to the neurobiological model that is being proposed, such as the CTSC model as introduced before [15]. In the CTSC model, the orbital-frontal-striatal pathway generates concerns associated with obsessions,

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and studies have suggested the role of protein in this pathway. Still, no other association is concluded [16]. Overall, the studies for specific gene variants have not found any particular gene that is aligned with other researchers, and more data is needed to conclude whether specific genes play a part in the causation of OCD; one should note that gene expression can also be affected by experiences which are environmental factors, and this is a bidirectional ambiguity that can hardly be avoided in the study for specific genes.

4. Environmental Factors

Environmental elements also contribute to the onset of OCD, with contradictory evidence for the correlated factors from multiple studies. Thus, no definite and sole environmental factor or experience is determined to be causing the disorder. Five types of learning experiences were proposed by Salkovskis et al. in 1999, which is based on his cognitive-behavioral model of OCD, and it accords with the existing learning theories [17]. Rooting from the types of learning experiences in general, family-rearing style, and traumatic or stressful events are two specific factors widely studied by later researchers and concluded a correlation with the cause of OCD [10]. Other factors such as socioeconomic status, infections, substance abuse, and perinatal complications are also studied, but less significance is concluded [12].

4.1. Five Types of Learning Experiences

As mentioned in the previous section of the cognitive-behavioral model of OCD, Salkovskis introduced the notion of "an inflated sense of responsibility" in OCD patients, and he proposed five learning experiences that can contribute to the origin of this concept [17]. The first experience is the promotion of an enormous sense of responsibility at a young age; children may learn that they have the power to change things that are out of control which may lead to misinterpretations of responsibility. The second experience strict rules of conduct and duty, which planted the idea of how things must be in the minds of individuals, then the individuals are likely to have more concerns if items are not aligned with their beliefs even if the detail did not matter in the specific context. A third experience is when individuals have not faced the content of responsibility in their childhood; this can be linked to having overprotective parents, which will be discussed as part of family-rearing styles. Thus, the child may be susceptible to ideas of responsibility and is more likely to misinterpret them. The fourth and fifth experiences are incidents of an individual's "action or inaction," their thoughts, or the combination of those two that accidentally lead to severe consequences that are usually negative. Through this experience, an association can be. Formed between the result and personal action or thought when they are not linked pragmatically or less connected than the individual assumes them to be.

4.2. Family-rearing Styles

Studies concluded that overprotective parents and paternal rejection are likely to be correlated with the causation of OCD [12], but there is conflicting empirical evidence for each.

As reviewed by Brander, eight out of thirteen studies [12] discovered a significant correlation between having children with an onset of OCD and having overprotective parents. In contrast, four studies came to the opposite conclusion [11]. The findings from studies on paternal rejection are more diverse. According to four research, rejection from the father is associated with OCD, while other studies also found that a disclaimer from both parents may be necessary. Further analyses found no correlation [12].

However, all the studies are not based on samples from a vast population; the studies used a small sample size from clinics, which reduces the generalizability of the studies. Another major

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methodological limitation is that the studies utilized the form of self-report. Patients likely link their disease to past experiences in the family; falsified memory should also be considered.

4.3. Stressful and Traumatic Events

Brown et al. conducted a study that reported that lifetime PTSD and OCD in individuals reached statistical significance [18]. Some patients were diagnosed with PTSD in the previous year of the onset of OCD. Grisham et al. reviewed the study and suggested that this study indicated how the beginning of PTSD can be a causal factor for OCD [10]. In Grisham's review, she noted in children with OCD, the patients have more real-life events, and they consider the adverse life events as more impactful. In Cromer et al.'s study, it is reported that traumatic life events increase OCD symptoms and their severity [10].

One standard limitation of the studies on the correlation between life events and OCD occurrence is that they are inconclusive enough to determine whether stressful and traumatic events can cause OCD; the studies lack a standardized criterion for characterizing the life events. Most studies rely on self-report, and the patients are likely to dramatize or exaggerate their adverse life events, so the results are hardly significant.

5. Conclusion

This examination of the literature leads us to conclude that genetic and environmental variables influence OCD. Regarding obsessive symptoms and the early start of OCD, hereditary factors are more critical, but ecological variables are more important in explaining the cause of exaggerated responsibility. It is difficult to determine the gene-environmental relationship for OCD because most study isolates the two elements. It is believed that multiple subtypes of OCD, which have not yet been precisely characterized, have unique causes, and each subtype's relationship with genetics and the environment can vary. Researchers also generally concur that OCD shares symptoms with other diseases and that these interactions make studying more challenging.

The existing studies paved the way for future research into the causal factors for OCD; other than improving methodological designs based on previous studies, this literature review suggests three future directions: more meta-analysis to solve the problem of small sample size, consider both genetic and environmental factors for future literature review and empirical study, and identify specific subtypes of OCD to study the particular causative factors in each subtype.

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