Working Memory Deficits in Obsessive-Compulsive Disorder Patients

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Abstract: Obsessive-compulsive disorder (OCD) is a long-term mental illness characterized by constant unwanted thoughts and repeated actions that cause severe impact on daily life. This review explores OCD's working memory deficits and their connection to abnormal brain activation, particularly in the anterior cingulate cortex (ACC), orbitofrontal cortex (OFC), and prefrontal cortex (PFC). Neuroimaging and behavioral studies suggest that these deficits cause cognitive overload in OCD patients, impairing decision-making and problem-solving processes. Despite growing evidence of executive function impairments in OCD, research results remain inconsistent due to task design limitations and small, homogeneous samples. This review identifies limitations of current research, such as task design and sample selection, and suggests future improvements, including standardized, ecologically valid tasks and larger, more diverse samples. Some possible treatments will be mentioned, including cognitive remediation therapy (CRT) and cognitive behavioral therapy (CBT), which are feasible ways to address working memory deficits and improve living quality for OCD individuals.

Keywords: Obsessive-compulsive disorder (OCD), Working Memory Deficits, Neuroimaging Studies

1. Introduction

Obsessive-compulsive disorder (OCD) is a chronic disease often emerging during adolescence or early adulthood, affecting about 1-3% of the worldwide people [1,2]. With OCD, individuals experience persistent and unwanted thoughts, followed by repetitive behaviors to alleviate the distress [1,3-5]. Fear of dust, germs, or environmental toxins is a common compulsive thought [6,7]. OCD sufferers may continuously fear getting contaminated by touching objects, people, or surfaces [8,9]. Through rituals like excessive hand washing, showering, or cleaning the house, OCDs can reduce their concern about contamination. The interplay between obsession and compulsive behaviors creates a highly time-consuming negative cycle, making OCD patients suffer negative impacts on their personal, social, and career lives [10].

The quest to understand the underlying neuropsychological mechanisms of OCD and behavioral impairments has challenged researchers for decades and is still ongoing. While more evidence suggests executive function (EF) impairments in OCD, the results have been inconsistent and highly generalized [11-13]. An exacting area that has received growing attention relates to the role of working memory in the OCD core symptoms.

Working memory (WM) is an essential component of executive function, initially proposed by Baddeley and Hitch in 1974. Based on the early short-term memory (STM) model, they proposed a

new model in which working memory is not only a passive storage system (as in the early STM model) but an active system that enables effective decision-making, problem-solving, and goal-directed behaviors by retaining and manipulating information over short periods [14-18]. The standard capacity of working memory is about 7 ± 2 items [19], but recent research suggests that working memory may only hold about 3-5 chunks of information [20]. Meanwhile, its existence lasts only 10-15 seconds [21]. Therefore, working memory deficits may severely impair cognitive control, with OCD only remembering existing behaviors for a shorter time and being unable to filter unwanted thoughts, leading to the repetition of the time-consuming cycle in OCD [22]. Although the OCDs' general executive dysfunction has been mentioned in many studies, including impaired cognitive flexibility, inhibition, and stereotype switching, the specific working memory deficits and their link with possible brain abnormalities remain insufficiently explored [11,23-24].

Recent neuroimaging studies appear to find evidence for abnormal brain activation in OCD patients, particularly in the prefrontal cortex (PFC), orbitofrontal cortex (OFC), and anterior cingulate cortex (ACC), areas responsible for executive functions [22,25-27]. However, it has been unclear whether these abnormal activations, compared to normal individuals, present a compensatory mechanism for OCD's impaired working memory or whether they reflect a fundamental neural network dysfunction that controls cognitive processes [28]. This gap in the research emphasizes the need for further study on the correlation between working memory impairments and brain activities in the OCD population. Accordingly, this paper will try to fill this blank by exploring the following research question: How are OCD's working memory dysfunctions related to specific brain activities? The review hypothesizes that OCD individuals have bad performance and abnormal responses in critical brain regions, such as ACC, OFC, and PFC, during working memory tasks. These unusual activations may serve as compensatory functions to manage the overload cognitive demands associated with intrusive thoughts and compulsive behaviors.

2. Literature review

2.1. Neuroimaging research

A study by Van der Wee et al. in 2003 recruited 11 untreated female OCD patients and 11 controls to perform N-Back working memory tasks with different difficulty levels under fMRI to support their hypothesis that OCD patients might perform poorer on a high workload working memory task compared to controls. This bad performance would be associated with an unusual brain activation, particularly in the anterior cingulate cortex (ACC). The research's results showed a highly increased error rate in OCD subjects while doing the most challenging task (3-back). At the same time, the fMRI images indicated that all participants used similar brain regions: the anterior cingulate cortex (ACC), the dorsolateral prefrontal cortex (DLPFC), and the parietal cortex, but OCD patients had larger activation in the ACC. Whereas they concluded that the increased activity in the ACC was unrelated to clinical symptoms, which means the observed neurological differences may be compensatory [22].

Building on this work, Nakao et al. [26] used neuropsychological assessment and fMRI to explore working memory deficits in OCD patients and their relationship to symptom severity. In the neuropsychological assessment, 40 OCD patients gained lower points than 25 control subjects while doing the WMS-R test and R-OCFT recall test. During the N-Back task conducted under fMRI, OCD patients showed stronger right DLPFC and left STG activation in the harder 2-Back task. There was also a positive correlation between symptom severity (measured by Y-BOCS) and right orbitofrontal cortex (OFC) activation. Evidence from this research supports that OCD patients have specific working memory deficits, especially in spatial cognition and visual delayed recall, which result in abnormal brain activations. Besides, researchers suggest that overactivation may reflect

compensatory mechanisms, where the brain excessively uses certain regions to cope with cognitive demands [26].

A meta-analysis by Rotge et al. in 2009 further supported these findings by examining structural abnormalities related to the cortico-striatal-thalamo-cortical (CSTC) circuit in OCD patients, including decreased volumes of the anterior cingulate cortex (ACC) and orbital frontal cortex (OFC). At the same time, the increased thalamic volume was linked to the OCD symptom severity [29]. Combined with functional abnormalities in the anterior cingulate cortex (ACC) and orbital frontal cortex (OFC) found by [26], the evidence suggests that both structural and functional deficits interact to affect OCD patients' cognitive control and error monitoring.

2.2. Behavioral research

Expanding on these insights, Harkin and Kessler [30] proposed the hypothesis that three components of the EBL system (efficiency of executive functioning, binding complexity, and memory load) together cause memory deficits in OCD patients. By analyzing the literature around the 2000s, they found that OCD patients' working memory deficits do not perform consistently across tasks but rather vary according to the executive function demands, binding complexity, and memory load. For example, verbal working memory impairments were mainly found in tasks requiring complex organizational strategies, while visuospatial memory impairments appeared in high executive demands tasks [30].

Jaafari et al. [31] explored these theoretical frameworks by testing 32 OCD patients and 32 gender, age, and education-matched control participants with multiple psychological evaluations: whether impairments in verbal and visuospatial aspects of working memory directly led to obsessive-compulsive behaviors in OCD patients. During the working memory assessments, OCD participants had significantly lower than healthy individuals on reading and positional width tests, suggesting that verbal and visuospatial working memory are impaired. For the checking behavior test, they modified and applied previous study's [29] image comparison task to reduce participants' checking requests by displaying two images on the same screen. The results showed that OCD sufferers had more checking behaviors compared to controls, which supported the link between working memory impairments and compulsive behavior in OCD [31]. Though the findings contradict Harkin and Kessler's conclusion that specific task demands (especially those involving complex bindings and high executive load) could amplify working memory deficits, these behavioral studies [30,31] have shown the versatility of working memory dysfunctions in OCD patients.

More recently, Rosa-Alcázar et al. [27] compared working memory in Obsessive-Compulsive Disorder (OCD) with that of Generalized Anxiety Disorder (GAD) and Social Anxiety Disorder (SAD) patients while including the potential influence of comorbidity and medication. Assessing working memory through the Numerical Breadth (verbal) and Corsi Block (visuospatial) tests, researchers found that people with OCD performed poorly on verbal and visuospatial working memory tasks compared to the GAD and SAD groups and that medication affected visuospatial working memory in all participants. These findings indicated that working memory deficits are the core feature of OCD patients' cognitive impairments, leading to their repetitive, rigid thinking and behaviors [27].

Another study by Heinzel et al. in 2021 extended the subjects of their research to first-degree relatives of OCD individuals to determine if working memory deficits in OCD are also found in not yet symptomatic individuals, i.e., if working memory deficits are OCD's endophenotypes. Participants (OCD patients, OCD patients' first-degree relatives, and healthy controls) were asked to find hidden markers in boxes and avoid returning to previously selected boxes during a spatial working memory task. The researchers finally compared the three groups' total scores (between-search errors and strategy scores). They found that OCD patients performed poorest on the spatial

working memory tests, unaffected relatives performed moderately, and healthy controls did the best. However, no significant differences occurred between relatives and healthy controls or between OCD patients and relatives [28]. This evidence showed that OCD patients' working memory deficits could not become an endophenotype of OCD; on the contrary, it again supported the results of previous studies [22,26-27,29,31] that working memory deficits were directly correlated with the main symptoms of OCD (obsession and compulsion).

In conclusion, the above studies have proved the connection between working memory deficits, repetitive behavior symptoms, and abnormal brain activation in OCD patients by neuropsychological assessments, fMRI studies, and behavioral tasks. Neuroimaging studies mentioned that OCD's working memory deficits were linked to abnormal brain activation, especially in ACC, PFC, and OFC regions, suggesting possible compensatory mechanisms. The following study by Harkin and Kessler [30] explored the subtle differences in subtype working memory deficits across specific tasks, as detailed with these deficits varying with task demands, while other studies [27-28,31] confirmed the direct role of working memory deficits in OCD symptom presentation.

3. Discussion

This section will critically discuss these studies' methodology for testing OCD working memory deficits, highlighting why these approaches were used, their limitations, and potential improvements.

3.1. Task design

Most studies used the N-Back task to test OCD patients' working memory capacity, a highly accepted working memory measurement that includes multiple difficulties [22,26,32-33]. Its characteristics of manually setting task complexity and requiring sustained participants' attention and cognitive control allow researchers to assess performance and neural activation in OCD patients quickly. In addition, Rosa-Alcázar et al. [27] used other tasks, such as the Corsi Block Test and working memory paradigm, to separate verbal and visuospatial memory deficits, thus providing a full understanding of cognitive functioning.

Despite their advantages, these task designs have some limitations. Lack of standardization between studies complicates the comparison of results [30]. Also, variations in task difficulty, length, and specific cognitive demands may lead to inconsistent findings regarding degrees of working memory deficits in OCD [28]. Heavy reliance on artificial lab-based tasks like N-Back tasks might fail to capture the cognitive challenges OCD patients face in the real world, thus limiting ecological validity [22,26]. Moreover, in 2013, Jaafari et al. mentioned that tasks often concentrate on isolated cognitive functions and may ignore the potential interaction of emotional and motivational factors [31].

3.2. Sample selection

Sample selection usually impacts the findings' validity and generalizability. Most studies have matched OCD patients' age, gender, and education level with other groups and healthy controls [26], [27-31]. However, there were limitations in some studies' small sample, which increased the probability of Type II error, leading to potentially important findings being neglected and the results not being generalizable enough [22,26-27,31]. Differences in symptom severity, comorbidities (e.g., depression or anxiety), and medication status among OCD samples might confound the results [26,27].

To address these limitations, future research on OCD working memory deficits should try standardizing tasks closely based on realistic settings, such as real-life simulation tasks or adaptive working memory challenges. This could provide more accurate information about OCD patients' daily cognitive difficulties and the emotional and motivational effects. Furthermore, sample sizes

should be larger and more diverse, including OCD patients with different symptom levels and comorbidities. Besides, longitudinal designs might be tried to track cognitive changes over time within different populations (including untreated, treated, and medicated patients), thus providing a deeper understanding of how working memory deficits develop and how they respond to treatment.

4. Conclusion

This paper reviewed several neuroimaging and behavioral studies to find the links among OCD patients' working memory deficits, abnormal brain activation, and OCD symptoms [22,26-31]. Neuroimaging evidence consistently proves that OCD individuals have working memory deficits, especially in tasks requiring high cognitive load, which are often paired with excessive brain activation in ACC, OFC, and PFC. These hyperactivations may serve as compensatory mechanisms to manage the overload cognitive demands of intrusive thinking and compulsive behaviors [22,26-27]. Behavioral studies further support these results, finding working memory impairments directly related to OCD's repetitive, compulsive behaviors [27-28,31]. These deficits are also variable, increasing as cognitive demands rise [30].

However, the previous studies [22,26-31] had limitations regarding task design and sample selection, such as an overreliance on laboratory settings for manualized tasks, a lack of standardization, and unrepresentative populations. Future studies should improve these disadvantages by mimicking daily practices and expanding the sample size, thus increasing the validity and generalizability of the results. Potential interventions for OCD patients' working memory deficits include Cognitive Remediation Therapy (CRT). This trains patients to develop effective strategies and enhances cognitive process flexibility [34]. Another option is Cognitive Behavioral Therapy (CBT) for direct working memory training, which helps OCD patients better manage intrusive thoughts and reduce obsessive-compulsive behaviors [35-37].

Overall, this review believes that research should continue improving methods to better understand the complex nature of OCD patients' working memory deficits and provide more reliable clinical findings in the future. These will help us develop practical diagnostic tools and treatment strategies, finally improving OCD patients' cognitive functioning and living quality.

References

- [1] Stein, D. J., Costa, D. L. C., Lochner, C., Miguel, E. C., Reddy, Y. C. J., Shavitt, R. G., van den Heuvel, O. A., & Simpson, H. B. (2019). Obsessive-compulsive disorder. Nature Reviews Disease Primers, 5(1), 52. https://doi.org/10.1038/s41572-019-0102-3
- [2] Brock, H., Rizvi, A., & Hany, M. (2024). Obsessive-compulsive disorder. In StatPearls. StatPearls Publishing. https://www.ncbi.nlm.nih.gov/books/NBK553162/
- [3] Manarte, L., Andrade, A. R., do Rosário, L., & others. (2021). Executive functions and insight in OCD: A comparative study. BMC Psychiatry, 21, 216. https://doi.org/10.1186/s12888-021-03227-w
- [4] Singh, A., Anjankar, V. P., & Sapkale, B. (2023). Obsessive-compulsive disorder (OCD): A comprehensive review of diagnosis, comorbidities, and treatment approaches. Cureus, 15(11), e48960. https://doi.org/10.7759/cureus.48 960
- [5] Rizvi, A., & Nolte, C. (2024). Obsessive-compulsive disorder: Diagnosis, clinical presentations, and implications. In G. Bennett & E. Goodall (Eds.), The Palgrave encyclopedia of disability. Palgrave Macmillan. https://doi.org/10.1007/978-3-031-40858-8_150-1
- [6] Szechtman, H., Shivji, S., & Woody, E. Z. (2023). Pathophysiology of obsessive-compulsive disorder: Insights from normal function and neurotoxic effects of drugs, infection, and brain injury. In Handbook of neurotoxicity (pp. 2195-2216). Springer International Publishing. https://doi.org/10.1007/978-3-031-15080-7_118
- [7] Singla, N. (2024). Discussing methods of treatment for obsessive-compulsive disorder by using sustainable technology. In Manufacturing technologies and production systems (pp. 132-140). CRC Press.
- [8] Aardema, F. (2020). COVID-19, obsessive-compulsive disorder, and invisible life forms that threaten the self. Journal of Obsessive-Compulsive and Related Disorders, 26, 100558. https://doi.org/10.1016/j.jocrd.2020.100558

- [9] Sulaimani, M. F., & Bagadood, N. H. (2021). Implication of coronavirus pandemic on obsessive-compulsive disorder symptoms. Reviews on Environmental Health, 36(1), 1-8. https://doi.org/10.1515/reveh-2020-0054
- [10] Maddux, J. E., & Winstead, B. A. (2019). Psychopathology: Foundations for a contemporary understanding (5th ed.). Routledge.
- [11] Youssef, A. M., AbouHendy, W. I., Elshabrawy, A., & Amin, S. I. (2020). Executive function in obsessive-compulsive disorder at Zagazig University Hospitals: A case-control study. Middle East Current Psychiatry, 27, 1-10. https://doi.org/10.1186/s43045-020-00033-0
- [12] Martínez-Esparza, I. C., Olivares-Olivares, P. J., Rosa-Alcázar, Á., Rosa-Alcázar, A. I., & Storch, E. A. (2021). Executive functioning and clinical variables in patients with obsessive-compulsive disorder. Brain Sciences, 11(2), 267. https://doi.org/10.3390/brainsci11020267
- [13] Zainal, N. H., & Newman, M. G. (2022). Executive functioning constructs in anxiety, obsessive-compulsive, post-t raumatic stress, and related disorders. Current Psychiatry Reports, 24(12), 871-880. https://doi.org/10.1007/s119 20-022-01390-9
- [14] Baddeley, A. D., & Hitch, G. (1974). Working memory. In G. H. Bower (Ed.), Psychology of learning and motivation (Vol. 8, pp. 47-89). Academic Press.
- [15] Baddeley, A. D. (2000). The episodic buffer: A new component of working memory? Trends in Cognitive Sciences, 4(11), 417-422. https://doi.org/10.1016/S1364-6613(00)01538-2
- [16] Engle, R. W. (2010). Role of working-memory capacity in cognitive control. Current Anthropology, 51(S1), 17-26. https://doi.org/10.1086/650572
- [17] Radüntz, T. (2020). The effect of planning strategy learning and working memory capacity on mental workload. Scientific Reports, 10, Article 7096. https://doi.org/10.1038/s41598-020-63897-6
- [18] De Vita, F., Schmidt, S., Tinti, C., & Re, A. M. (2021). The role of working memory on writing processes. Frontiers in Psychology, 12, Article 738395. https://doi.org/10.3389/fpsyg.2021.738395
- [19] Miller, G. A. (1956). The magical number seven, plus or minus two: Some limits on our capacity for processing information. Psychological Review, 63(2), 81-97. https://doi.org/10.1037/h0043158
- [20] Cowan, N. (2010). The magical mystery four: How is working memory capacity limited, and why? Current Directions in Psychological Science, 19(1), 51-57. https://doi.org/10.1177/0963721409359277
- [21] Goldstein, E. B. (2010). Cognitive psychology: Connecting mind, research, and everyday experience (3rd ed.). Wadsworth Publishing.
- [22] Van der Wee, N. J. A., Ramsey, N. F., Jansma, J. M., Denys, D. A., Van Megen, H. J. G. M., Westenberg, H. M. G., & Kahn, R. S. (2003). Spatial working memory deficits in obsessive-compulsive disorder are associated with exce ssive engagement of the medial frontal cortex. NeuroImage, 20(3), 2271-2280. https://doi.org/10.1016/j.neuroima ge.2003.05.001
- [23] Christensen, K. J., Kim, S. W., Dysken, M. W., & Hoover, K. M. (1992). Neuropsychological performance in obsessive-compulsive disorder. Biological Psychiatry, 31(1), 4-18. https://doi.org/10.1016/0006-3223(92)90003-I
- [24] Abbruzzese, M., Ferri, S., & Scarone, S. (1995). Wisconsin Card Sorting Test performance in obsessive-compulsive disorder: No evidence for involvement of dorsolateral prefrontal cortex. Psychiatry Research, 58(1), 37-43. https://doi.org/10.1016/0165-1781(95)02670-r
- [25] Busatto, G. F., Buchpiguel, C. A., Zamignani, D. R., Garrido, G. E., Glabus, M. F., Rocha, E. T., & Castro, C. C. (2000). A voxel-based investigation of regional cerebral blood flow abnormalities in obsessive-compulsive disorder using single photon emission computed tomography (SPECT). Psychiatry Research: Neuroimaging, 99(1), 15-27. https://doi.org/10.1016/S0925-4927(00)00050-0
- [26] Nakao, T., Nakagawa, A., Nakatani, E., Nabeyama, M., Sanematsu, H., Yoshiura, T., ... & Kanba, S. (2009). Working memory dysfunction in obsessive-compulsive disorder: A neuropsychological and functional MRI study. Journal of Psychiatric Research, 43(6), 784-791. https://doi.org/10.1016/j.jpsychires.2008.10.013
- [27] Rosa-Alcázar, A. I., Rosa-Alcázar, Á., Martínez-Esparza, I. C., Storch, E. A., & Olivares-Olivares, P. J. (2021). Response inhibition, cognitive flexibility, and working memory in obsessive-compulsive disorder, generalized anxiety disorder, and social anxiety disorder. International Journal of Environmental Research and Public Health, 18(7), 3642. https://doi.org/10.3390/ijerph18073642
- [28] Heinzel, S., Bey, K., Grützmann, R., Klawohn, J., Kaufmann, C., Lennertz, L., Wagner, M., Kathmann, N., & Riesel, A. (2021). Spatial working memory performance in people with obsessive–compulsive disorder, their unaffected first-degree relatives, and healthy controls. BJPsych Open, 7(e208), 1-3. https://doi.org/10.1192/bjo.2021.1052
- [29] Rotge, J.-Y., Guehl, D., Dilharreguy, B., Tignol, J., Bioulac, B., Allard, M., Burbaud, P., & Aouizerate, B. (2009). Meta-analysis of brain volume changes in obsessive-compulsive disorder. Biological Psychiatry, 65(1), 75-83. https://doi.org/10.1016/j.biopsych.2008.06.019
- [30] Harkin, B., & Kessler, K. (2011). The role of working memory in compulsive checking and OCD: A systematic cla ssification of 58 experimental findings. Clinical Psychology Review, 31(6), 1004-1021. https://doi.org/10.1016/j.c pr.2011.06.004

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- [31] Jaafari, N., Frasca, M., Rigalleau, F., Rachid, F., Gil, R., Olié, J.-P., Guehl, D., Burbaud, P., Aouizerate, B., Rotg é, J.-Y., & Vibert, N. (2013). Forgetting what you have checked: A link between working memory impairment and checking behaviors in obsessive-compulsive disorder. European Psychiatry, 28(2), 87-93. https://doi.org/10.1016/ j.eurpsy.2011.07.001
- [32] Esmaeeli, S. (2021). The effectiveness of neurofeedback training on the working-memory of people with obsessivecompulsive disorder symptoms. European Journal of Molecular and Clinical Medicine, 8(1), 1795+.
- [33] Yue, J., Zhong, S., Luo, A., Lai, S., He, T., Luo, Y., & Jia, Y. (2021). Correlations between working memory impairment and neurometabolites of the prefrontal cortex in drug-naive obsessive-compulsive disorder. Neuropsychiatric Disease and Treatment, 17, 2647–2657. https://doi.org/10.2147/NDT.S296488
- [34] van Passel, B., Danner, U., Dingemans, A., & others. (2016). Cognitive remediation therapy (CRT) as a treatment enhancer of eating disorders and obsessive-compulsive disorders: Study protocol for a randomized controlled trial. BMC Psychiatry, 16, 393. https://doi.org/10.1186/s12888-016-1109-x
- [35] Jaeggi, S. M., Buschkuehl, M., Shah, P., & others. (2014). The role of individual differences in cognitive training and transfer. Memory & Cognition, 42(3), 464–480. https://doi.org/10.3758/s13421-013-0364-z
- [36] Spencer, S. D., Stiede, J. T., Wiese, A. D., Goodman, W. K., Guzick, A. G., & Storch, E. A. (2023). Cognitivebehavioral therapy for obsessive-compulsive disorder. Psychiatric Clinics of North America, 46(1), 167-180. https://doi.org/10.1016/j.psc.2022.10.004
- [37] Syros, I., & Anastassiou-Hadjicharalambous, X. (2024). Implementation of cognitive behavioral therapy in a child with obsessive-compulsive disorder: A case study. OBM Neurobiology, 8(3), 231. https://doi.org/10.21926/obm.n eurobiol.2403231