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تتقدم إليكم جامعة بدر بالقاهرة بالشكر على ما تبذلونه من جهد مادي ومعنوي لإصدار المجلة، فتميزكم المشهود خير قدوة، ممتنين لعملكم الدؤوب وتفوقكم الباهر، ونتمنى لكم المزيد من النجاحات المستقبلية.

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Neural Mechanisms of Executive Function Tasks in Obsessive-Compulsive Disorder and Anorexia Nervosa: A Comparative Systematic Review

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Abstract: Executive functions are a set of cognitive tools that helps in navigating through a complex and unpredictable world. Prominent research has extensively evaluated the neural basis of executive functions in psychiatric disorders. Particularly, inferences on whether obsessive- compulsive disorder and anorexia nervosa present with similar brain activity whilst performing an executive function task were raised. This is due to the high comorbidity and shared symptomatology between the two disorders. Therefore, a systematic review was conducted to synthesize research that applied executive function tasks during functional magnetic resonance imaging in obsessive-compulsive disorder and anorexia nervosa. Following the PRISMA guidelines, 43 studies comparing the executive performance of patients of each disorder and healthy participants were chosen. This review revealed that although both disorders may activate similar brain areas, they may be engaging in diverse neural pathways for each executive function task. Conclusively, common notions on increased self-referential processing and discrepancy between internal and external models were given by most of the included studies. The evidence for comparing neural activation in both disorders was conflicting and incongruous. More research using consistent experimental paradigms and assessing both disorders concurrently is required.

Keywords: executive function, obsessive-compulsive disorder, anorexia nervosa, functional magnetic resonance imaging

INTRODUCTION

Adaptability is born out of the ability to produce non-automatic responses in face of a world of unpredictability and complexity (Barkley, 2001; Hawkins & Blakeslee, 2007). Fundamentally, executive functions are perceived to embody adaptation and learning. Although commonly referred to as cognitive tools, executive functions could be explained as the inherent drive to harmonize internal models with external realities (Pezzulo, 2012). Through planning, anticipating, and reflecting, one can attain perseverance and a sense of integrity and direction. Recent research has commenced towards explaining executive functions through new lenses. Traditionalistic views asserted that the brain merely reacts to upcoming stimuli; however, prevailing views contend that the brain is in a dynamic cycle where it plans, anticipates and predicts upcoming stimuli and actively updates any predictive errors to enable behaviour with intention and purpose (Kopp, 2012; Parr et al., 2023; Pezzulo, 2012).

There is no doubt that altered executive functioning could lead to disrupted self-governance (Barkley, 2001). Research in psychology and psychiatry have emphasized the importance of studying changes in executive function and cognitive control in psychiatric disorders. Prominently, results have shown compromised abilities in accurate decision making (Cáceda et al., 2014; Scholl & Klein-Flügge, 2017); a dissonance between desired goals and actions taken (Gillan et al., 2015; Gillan et al., 2016; Griffiths et al., 2014; Voon et al., 2017); and inability to produce accurate predictions and update internal models (Doll et al., 2012; Gradin et al., 2011; Stephan & Mathys, 2014).

Research postulates that obsessive-compulsive disorder (OCD) and anorexia nervosa (AN) might share similar cognitive dysfunctions and neural mechanisms. With results showing high comorbidity between these disorders (Drakes et al., 2021; Mandelli et al., 2020; Pollack & Forbush, 2013); shared symptomatology (Altman & Shankman, 2009; Latif & Moulding, 2024; Williams et al., 2022); and genetic relatedness (Yilmaz et al., 2018), it is intuitive to see whether they exhibit similar executive functions and neural mechanisms. OCD is popularly characterised through the well-known 'OCD Loop', in which unwanted and intrusive obsessions give rise to anxious feelings that could only be relieved by performing ritual habits or compulsions, and the cycle perpetuates (Laposa et al., 2018; Robbins et al., 2011; Salkovskis, 1985; Taylor et al., 2007). Similarly, anorexia nervosa has shown to exhibit a similar cycle. Obsessions and unwanted negative thoughts about weight, appearance and food rises anxiety levels, which inevitably leads to compulsive actions such as body checking, purging and restricting (Matsunaga et al., 1999; Shafran, 2002; Sherman et al., 2006). Likewise, these compulsions only lead to short-lived relief. Here, one can observe a profound struggle with the very essence of executive control. A shift from conscious deliberation of aligning goals with actions to a manifestation of routines and automated responses is seen.

Changes in executive control in patients with AN or OCD is observed throughout the literature. For instance, a large sample of patients with AN were assessed using the Wisconsin Card Sorting Task and were compared to healthy participants (Tchanturia et al., 2012). Results revealed that patients faced difficulty and performed worse than healthy controls. High preservative errors in both patients with active illness and recovered patients attests to the notion that eating disorders have impaired executive functioning, specifically cognitive flexibility and

inhibition of prepotent responses (Tchanturia et al., 2012). Similar conclusions were found when comparing the performance of OCD patients and healthy controls in a variety of executive functioning tasks such as go/no-go task, stop-signal task, Wisconsin Card Sorting task (WCST), and n-back task (Martínez-Esparza et al., 2021; Youssef et al., 2020). Sternheim et al.'s (2022) study has highlighted the importance of self-reported measures alongside executive function tasks, explaining that both AN and OCD patients self-reported more inflexibility did not link to their actual performance.

Despite such evidence, opposing results were identified. Although both patient groups present with executive deficits, neuroimaging outcomes differed. To explicate, Thomas et al.'s (2022) review has concluded that a divergence between behavioural performance and neural activity between AN and OCD patients. In other words, even if both patient groups present with similar executive deficits in cognitive-behavioural tasks, the underlying neural mechanism functions differently. This was further supported by Li et al. (2023), in which poor performance in inhibitory tasks was not linked to similar brain activity in eating disorders and OCD. Evidence from previous reviews has mainly centred on domain-based analysis such as inhibition, cognitive flexibility, and working memory. However, much of the research have used different executive function tasks to assess the same domain. This could lead to confusions due to (1) different operationalizations of the same domain, (2) tasks may engage in selective and specific cognitive processes, and (3) the same task can measure many domains, making it difficult to isolate one domain to a specific task. The argument of whether to measure executive function as domain-general or domain-specific processes or to assess it through domain-based or task-based analysis has been discussed in past research (for a deeper read, see Assem et al., 2024; Chan et al., 2007; Jurado & Rosselli, 2007; Löffler et al., 2024). Therefore, the following review will aim to synthesize research that assessed executive function in OCD and AN using a task-based approach.

METHODS

Literature Search and Study Selection

This systematic review was carried out using the PRISMA 2020 Guidelines (Page et al., 2021). Both Scopus and PubMed were used pinpoint functional magnetic resonance imaging studies examining executive function in OCD and AN until 17th November 2024. The search strategy consisted of five items: population (OCD or AN); fMRI imaging; executive function domains; and executive functioning tasks. The full detailed search strategy can be found in the Appendix. After duplicates were removed, M.S and A.A both independently screened the titles and abstracts of the remaining studies and assessed against the eligibility criteria. Studies that coincided with the eligibility criteria were taken for full-text examination. Any disagreements were resolved through discussions and reaching consensus.

Eligibility Criteria

Inclusion criteria consisted of the following:

1. Articles to be published in peer-reviewed journals, in English, with full-text access.
2. Articles to be published from 2000 to 2024.
3. Authors report that participants are diagnosed with OCD or AN through a standardized diagnostic criterion (e.g. DSM-5)

4. All participants (both healthy controls and patient group) must perform executive function tasks (e.g. go/no-go; n-back; WCST) during fMRI scanning.

Exclusion criteria included the following:

1. Authors report using the same subject datasets.
2. Participants undergo an executive function task that includes affective or food stimuli.
3. Participants performed executive function task during other forms of neuroimaging techniques.
4. Participants performed executive function tasks outside the fMRI scanner.
5. Participants undergo intervention or cognitive training programs prior to the fMRI scan (unless fMRI results were reported prior to the intervention/training programs).

Data Extraction

All chosen studies were divided into two folders and were uploaded on Zotero (a reference manager software). Data extraction was performed independently by each reviewer; however, all information was uploaded on a shared Excel spreadsheet for cross-validation. Important information that was looked for were: study characteristics (e.g. authors and year of publication); sample characteristics (e.g. age); fMRI analysis and executive function paradigm used; and case-control contrasts. Full study details can be found in Tables 1 and 2 (in the Appendix).

Quality Assessment

Quality assessment was conducted for the included articles using the Standard Quality Assessment Criteria (SQAC; Kmet et al., 2004; Wollesen et al., 2019). Quality assessment was based on the following criteria: (1) question and objective of study is sufficiently described, (2) study design is identified and appropriate to study's research question, (3) information on sample selection, input variables, and inclusion/exclusion criteria are described and appropriate, (4) sufficient description of sample demographic characteristics are given, (5) outcome and exposure measures are precisely described and defined, (6) study's methodological analyses are described and appropriate, (7) reports of estimates of variance in results are provided, (8) results are reported sufficiently in detail, (9) all conclusions are supported by the data. Points for each criterion were as follows: 2 points if criteria are fulfilled, 1 point if criteria are partially fulfilled, 0 points if criteria are not fulfilled. Total quality score is out of 18, and a minimum score of 14 (75-77%) is required for a study to be considered a high-quality study.

RESULTS

Study Selection

A total of 332 studies were obtained in the literature search: 86 studies from PubMed and 238 studies from Scopus. With regards to research that included OCD patients, a total of 230 studies were obtained (n = 64 PubMed; n = 166 Scopus). After excluding 105 studies due to inappositeness and 54 studies were removed due to duplication, 71 studies were chosen for full-text examination. Out of the 71, 43 studies were excluded for reasons such as: EF tasks were done outside the fMRI scanner; fMRI results were reported post-treatment; EF tasks included an emotional stimulus; same sample datasets were used. Moreover, 3 studies were found from previous literature reviews (Gruner & Pittenger, 2016; Nakao et al., 2014; Saxena et al., 2009).

Therefore, a total of 31 fMRI studies that were concerned with executive function tasks in OCD patients were selected for this review.

With regards to research that included AN patients, a total of 94 studies were obtained (n = 22 PubMed; n = 72 Scopus). A total of 22 studies were chosen for full-text examination after 62 incompatible studies and 10 duplicates were excluded. After full-text examination, 13 studies were excluded due to the following reasons: participants were stress-induced; participants had to perform physical activity; participants were either hungry or satiated during fMRI scanning; food and affective stimuli was included; EF tasks were taken outside fMRI. Backward literature search extracted 4 additional studies (Fuglset et al., 2016; Kappou et al., 2021). This resulted in a total of 13 fMRI studies that analysed executive function tasks in AN patients included in this review.

High quality scores (threshold of >=14, 75-77%) were given to 37 studies. The remaining 6 studies received a good quality score, with scores ranging from 12-13 (66-72%), and thus were not excluded from the study. Overview of the scores can be found in the Appendix.

Study Characteristics

The final 43 chosen studies consisted of 2,068 participants, with sample sizes varying from 15 to 100. Thirty-one of OCD-focused studies and seven of AN-focused studies included adult samples (ages ranging from 21 to 51), with three OCD-focused studies and six AN-focused studies included adolescent samples (ages ranging from 13 to 18).

Executive Function Tasks

The studies addressing OCD examined the following executive functioning tasks: Go/No-Go (n = 7), N-back (n = 6), Task-Switch (n = 4), Stroop (n = 4), Tower of London (n = 3). Other tasks included Stop-Signal (n = 3), Simon Task (n = 1), Flanker Task (n = 2), Wisconsin Card Sorting Task (WCST, n = 2), Item-Recognition Working Memory Task (n = 1), and Imagination, Suppression and Erase Task (n = 1).

In terms of AN-targeted studies, most common executive functioning tasks used were the Go/No-Go Task (n = 3) and the WCST (n = 3). Other tasks included Set-Shift (n = 2), Stop-Signal (n = 1), Target-Detection Task (n = 1), N-back (n = 1), Implicit Sequence Learning Task (n = 1), Embedded Figures Task (n = 1).

fMRI data analysis

While almost all studies have applied general linear modeling (GLM), psychophysiological interactions (PPI; n = 7) and functional connectivity (FC; n = 4) have been used. Whole-brain analysis was conducted in 89% of studies (n = 27 OCD-focused studies; n = 13 AN-focused studies), whilst 69% of studies used region of interest analysis (ROI; n = 23 OCD-focused studies; n = 8 AN-focused studies).

Brain Activation in OCD and AN

Brain regions that were reported in at least 50% of the studies were taken into consideration. In the following results, numbers in brackets refer to subregions. Subregions were counted by each instance they were reported, acknowledging that the same subregion has been reported by more than one study. Twenty out of 32 OCD-specific studies have reported more activation in healthy controls compared to OCD in frontal (n = 26), cingulate (n = 20), temporal

(n = 20), sensorimotor (n = 15), subcortical and limbic regions (n = 21) (Cocchi et al., 2011; De Wit et al., 2012; Fitzgerald et al., 2005; Gu et al., 2007; Han et al., 2011; Heinzel et al., 2017; Kang et al., 2012; Koch et al., 2012; Koçak et al., 2011; Masharipov et al., 2023; Morein-Zamir et al., 2015; Page et al., 2009; Remijnse et al., 2013; Roth et al., 2007; Schlösser et al., 2010; Thorsen et al., 2020; Tolin et al., 2013; Vaghi et al., 2017; Van Den Heuvel et al., 2005).

Twenty-three out of 32 OCD-focused studies have reported more activation in OCD compared to HC in frontal (n = 29), temporal (n = 13), parietal (n = 17), and sensorimotor regions (n = 19) (De Wit et al., 2012; Fitzgerald et al., 2005; Fuglset et al., 2016; Gruner & Pittenger, 2016; Gu et al., 2007; Han et al., 2011; Heinzel et al., 2017; Henseler et al., 2008; Hough et al., 2016; Kang et al., 2012; Kappou et al., 2021; Kim et al., 2021; Koçak et al., 2011; Masharipov et al., 2023; Morein-Zamir et al., 2015; Nakao et al., 2008; Page et al., 2009; Remijnse et al., 2013; Roth et al., 2007; Schlösser et al., 2010; Thorsen et al., 2020; Tolin et al., 2013; Vaghi et al., 2017; Vanderwee et al., 2003).

Seven out of 13 AN-focused studies reported more activation in HC compared to AN in frontal (n = 6), cingulate (n = 4), sensorimotor (n = 5), occipital (n = 7), subcortical (n = 5), insula and opercular regions (n = 4; Castro-Fornieles et al., 2019; Sato et al., 2013; Suttkus et al., 2021; Wierenga et al., 2014; Zastrow et al., 2009).

Seven out of 13 AN-focused studies reported more activation in AN compared to HC in prefrontal (n = 5), parietal (n = 4), and occipital regions (n = 9) (Castro-Fornieles et al., 2019; Lao-Kaim et al., 2015; Lock et al., 2010; Noda et al., 2021; Van Autreve et al., 2016; Wierenga et al., 2014).

Figures 1 and 2 show common brain area activation patterns reported by most studies (refer to the Appendix). In OCD, hyperactivation was seen in the following brain areas: middle frontal gyrus (MFG); inferior frontal gyrus (IFG); precentral gyrus (PRG); postcentral gyrus (PoCG); inferior parietal lobule (IPL); supramarginal gyrus (SMG); and pre-supplementary motor area (pre-SMA; Cocchi et al., 2011; De Vries et al., 2013; De Wit et al., 2012; Heinzel et al., 2017; Hough et al., 2016; Kim et al., 2021; Meram et al., 2020; Morein-Zamir et al., 2015; Remijnse et al., 2013; Schlösser et al., 2010). Hypoactivation was shown in the posterior cingulate cortex (PCC) and premotor cortex (PM; Gu et al., 2007; Koçak et al., 2011; Page et al., 2009; Van Den Heuvel et al., 2005).

Conflicting results were shown in the dorsolateral prefrontal cortex (DLPFC), superior frontal gyrus (SFG), superior temporal gyrus (STG), middle temporal gyrus (MTG), anterior cingulate cortex (ACC), supplementary motor area (SMA), precuneus (PRCU), and cerebellum (Cocchi et al., 2011; De Vries et al., 2013; Fitzgerald et al., 2005; Gu et al., 2007; Han et al., 2011; Heinzel et al., 2017; Hough et al., 2016; Kang et al., 2012; Kim et al., 2021; Koch et al., 2012; Koçak et al., 2011; Liu et al., 2023; Maltby et al., 2004; Masharipov et al., 2023; Meram et al., 2020; Morein-Zamir et al., 2015; Nakao et al., 2008; Page et al., 2009; Remijnse et al., 2013; Roth et al., 2007; Schlösser et al., 2010; Thorsen et al., 2020; Tolin et al., 2013; Van Den Heuvel et al., 2005).

In AN, increased brain activation was reported in the PRCU (Lao-Kaim et al., 2015; Van Autreve et al., 2016; Noda et al., 2021), and cuneus (Castro-Fornieles et al., 2019; Noda et al., 2021). Decreased brain activation was shown in the IFG, MFG, PRG, cerebellum, and lingual gyrus (LG; Castro-Fornieles et al., 2019; Sato et al., 2013; Wierenga et al., 2014; Zastrow et al.,

2009). Conflicting results were seen in the ACC (Lock et al., 2010; Wierenga et al., 2014; Zastrow et al., 2009). Both OCD and AN have presented increased overall activation of the insula during executive functioning tasks (Heinzel et al., 2017; Kim et al., 2021; Nakao et al., 2008; Roth et al., 2007; Tolin et al., 2013; Van Autreve et al., 2016).

Brain Activation and Executive Function Tasks

Brain Activation during Go/No-Go Task

Seven studies have used the go/no-go task to measure executive functioning in OCD. Out of these studies, the most reported brain regions were the ACC (n = 5), thalamus (n = 4), caudate (n = 4). Other brain areas included the posterior cingulate (n = 3), precuneus (n = 3), insula (n = 2), and middle temporal gyrus (n = 2). Three studies have reported more activation in OCD patients compared to healthy controls in the ACC (Maltby et al., 2004; Morein-Zamir et al., 2015; Tolin et al., 2013); whereas two studies have reported less activation (Masharipov et al., 2023; Page et al., 2009). This was also shown in the caudate nucleus (CN), in which two studies have reported hyperactivity in OCD (Masharipov et al., 2023; Roth et al., 2007) and two other studies have reported hypoactivity in OCD (Masharipov et al., 2023; Page et al., 2009). Of the studies that reported activation in the thalamus, two have reported more activation in healthy controls compared to OCD patients (Masharipov et al., 2023; Page et al., 2009), and only one study has reported variable activity levels (Morein-Zamir et al., 2015).

In terms of research pertaining to AN patients, three studies have applied the go/no-go task and have reported distinct brain activations. Two studies have identified heightened activity compared to healthy controls and one study has reported the contrary. For instance, Lock et al. (2010) have reported elevated activity in AN (binge-eating type) compared to HC in the following brain areas: PRG, ACC, MTG, STG, hypothalamus, and DLPFC. On the other hand, heightened activity in the cuneus and precuneus were reported by Noda et al. (2021). With regards to low-level activation compared to HC, the amygdala and hippocampus were reported (Suttkus et al., 2021).

Taking a closer look at between-group comparisons, similar brain regions but with distinct neural activity is shown. These include the following: MTG, ACC, PRCU, PoCG, CN, LG (Lock et al., 2010; Maltby et al., 2004; Masharipov et al., 2023; Morein-Zamir et al., 2015; Noda et al., 2021; Page et al., 2009; Roth et al., 2007; Tolin et al., 2013).

Brain Activation during Stroop Task

Overall, only two studies addressing OCD have assessed executive functioning through the Stroop task. There was no consensus on brain regions reported. Through ROI analysis, Schlösser et al. (2010) reported elevated activity in OCD in the dACC, DLPFC, PRG, SFG, MFG, SPL, and IPL, and reduced activity levels in the occipital lobe. In contrast, Page et al. (2009) reported higher activity in posterior cingulate gyrus (PCG) and cerebellum, and reduced activity in the precuneus, MTG, STG, STJ, IPJ, and SPL.

Brain Activation during Stop-Signal Task

Three studies (one used ROI analysis and the other whole-brain analysis) presented distinct neural activity in OCD patients during stop-signal tasks. Essentially, de Wit et al.'s (2012) findings suggest that OCD patients exhibit higher brain activity in the pre-SMA compared to controls, and

lower brain activity in the IFG and inferior parietal cortex (IPC). Through whole-brain analysis, brain regions that exhibit greater activation in OCD include the parahippocampal gyrus (PHG), SPC, and cerebellum (Kang et al., 2012). Moreover, healthy controls showed greater neural activations compared to patients in the middle temporal cortex (MTC), middle occipital cortex (MOC), PRG, superior temporal cortex (STC), ACC, CN, putamen, fusiform face area (FFC), calcarine (CALC), mid-cingulate cortex (MCC), and cerebellum (Kang et al., 2012). During successful inhibition, HC>OCD results were found in the IFG, hippocampus, MCC, and precuneus (Thorsen et al., 2020). During failed inhibition, HC>OCD results were found in the dmPFC, IFG, operculum and white matter (Thorsen et al., 2020). Their study also examined group differences in right and left amygdala connectivity during successful inhibition. OCD>HC results were found in right amygdala connectivity with pre-SMA, IFG, MePFC, MTG, MOG, CN, and putamen. Left amygdala connectivity was observed in MePFC, IFG, calcarine sulcus and white matter (Thorsen et al., 2020). HC>OCD were only found in right amygdala connectivity with hippocampus (tail), precuneus, and white matter (Thorsen et al., 2020).

Comparatively, Wierenga et al. (2014) aimed to analyse executive functioning in anorexia nervosa during the stop-signal task. Neuroimaging results demonstrate reduced neural activation in AN adolescents compared to healthy adolescents in many brain regions including the ACC, MFG, and PCC, with patients not presenting post-error slowing (Wierenga et al., 2014).

Brain Activation during Wisconsin Card Sorting Task

Only two studies (out of 32 included studies) were found to apply the WCST to measure executive functioning in OCD patients. Analyses reveal that OCD patients exhibit hyperactivation in various brain regions compared to controls. Many activations were reported, spanning across the frontal, parietal and occipital regions known for sensorimotor and cognitive processing (Kim et al., 2021). Comparatively, the PRCU, ANG, IFG have been implicated in both OCD and AN patients. Both PRCU and ANG have been shown to be hyperactivated in both patient groups when compared to controls (Kim et al., 2021; Lao-Kim et al., 2015). The IFG revealed inverse activation patterns, with OCD showing hyperactivation and AN showing hypoactivation when compared to controls (Kim et al., 2021; Sato et al., 2013). Interestingly, a recent study has reported no significant between-group differences amongst AN, OCD and HC adolescents in brain activation during WCST (Bohon et al., 2019).

Brain Activation during N-Back Task

Findings reveal common hyperactivation of the PFC, STG, PRCU in OCD compared to healthy controls (De Vries et al., 2013; Heinzel et al., 2017; Meram et al., 2020; Nakao et al., 2008; Vanderwee et al., 2003). Moreover, Nakao et al.'s (2008) study revealed hyperactivation of the DLPFC, insula, and cuneus. With regards to heightened activity in healthy controls compared to OCD patients, only two studies (out of six) have reported significant results (Heinzel et al., 2017; Koch et al., 2012). Such results were observed in regions related to visual, motor and emotional processing such as SMA, IOL, PoCG, CN, and amygdala (Heinzel et al., 2017). Interestingly, no significant between-group differences were found between AN and HC (Lao-Kaim et al., 2013).

Brain Activation during Task-Switch and Set-Shift Tasks

Distinct brain activations have been reported during task-switching in OCD. The PoCG was shown to be hyperactive in OCD compared to HC, according to Remijnse et al. (2013) and

Liu et al. (2023). Remijnse et al. (2013) have reported more activation in the ACC and putamen and less activation in the anterior prefrontal cortex (APFC). Although Liu et al. (2023) found no hypoactivation in OCD, they did report hyperactivation in the precuneus, middle occipital cortex (MOC), middle cingulate gyrus (MCG), and superior parietal cortex (SPC). Moreover, more brain activity in HC compared to OCD was shown in the following areas: middle temporal gyrus (MTG), STG, occipital lobe (OL), ACC (rostral and dorsal), PCC, MeFC, VLPFC, PMC, CN, OFC, parietal lobe (PL), uncus, thalamus and hippocampus (Gu et al., 2007; Han et al., 2011). With regards to OCD, patients have shown increased activation in the ventromedial prefrontal cortex and the orbitofrontal cortex during task-switch tasks (Gu et al., 2007). Hyperactivation was reported in the SPL and insula, which was associated with slower reaction times (Liu et al., 2023). This suggests the need for more brain activity to participate in the task.

During set-shifting, frontal, parietal, and occipital subregions have mainly been reported. For instance, AN patients have exhibited more activation in parietal subregions such as the precuneus, IPL, SPL, SMG (Van Autreve et al., 2016); occipital subregions such as the cuneus and calcarine cortex (De Vries et al., 2013); and in other areas like the insula and dorsal-premotor cortex (PMD; Van Autreve et al., 2016). With regards to less brain activation in AN compared to HC, occipital subregions such as inferior occipital (IO), middle occipital (MO), calcarine, lateral fusiform and lingual gyrus; frontal subregions such as the MFG, IFG, and PCG; and other regions such as the cerebellum have been reported (Castro-Fornieles et al., 2019).

Brain Activation during Tower of London Task

Through the Tower of London task (TOL), van den Heuvel et al. (2005) have reported greater brain activation in OCD in the following areas: VLPFC, anterior temporal cortex (ATC), cingulate cortex, PHG, and brainstem. Oppositely, hypoactivation was shown in DLPFC, MFC, precuneus, PMC, IPC, CN, and putamen (Vaghi et al., 2017; Van Den Heuvel et al., 2005). Also, Kim et al. (2020) have reported reduced functional connectivity between DAN and DMN and LFPN networks in OCD.

Brain Activation during other Executive Function Tasks

The following studies have used other executive function tasks to report brain activity in OCD. Differently, Koçak et al. (2011) have applied an Imagination, Suppressing, and Earsing task to measure executive function-related brain activity in OCD. Overall, healthy controls have exhibited more brain activation in the PCC, SFG, and IPL compared to OCD (Koçak et al., 2011). No hyperactivation in OCD was reported.

Conversely, the opposite effect was found during an item-recognition task, with OCD presenting increased brain activation over HC. This increased brain activation was specific to the precentral sulcus, IPC, IFG, and IFS (Henseler et al., 2008).

During the Flanker task, differential activation patterns in neighbouring subregions were reported. For instance, the rostral and subgenual part of the ACC was more active in OCD relative to HC, in contrast to the dorsal part of the ACC, where HC showed more active relative to OCD (Fitzgerald et al., 2005; Grützmann et al., 2014). Other brain areas that displayed OCD>HC include the MeTG, anterior insula (AIC), SFG, IFG, SMA and amygdala (Fitzgerald et al., 2005; Grützmann et al., 2014). Brain areas that exhibited HC>OCD include the pre-SMA, SMA, and STG (Fitzgerald et al., 2005).

In Cocchi et al.'s (2011) study, they have applied the Flanker task, along with the Stroop and Simon tasks and have reported brain regions that were commonly activated during these tasks. Essentially, they reported greater brain activation in OCD relative to HC in brain regions involved in the paralimbic and sensorimotor networks. These regions include the anterior insular, pre-SMA, SMA, sensorimotor cortex, and primary motor cortex (Cocchi et al., 2011). Mainly brain regions included in the salience network and default mode network were reported to be more activated in HC relative to OCD. These regions include the MeFC, dPFC, dACC, and MCC (Cocchi et al., 2011).

With regards to AN-focused studies, studies that have applied the target-detection task and the embedded figures task have not reported any increased brain activity in AN (Leslie et al., 2021; Zastrow et al., 2009). During target-detection, the following brain areas were reported for HC>ED: MeFG, MFG, ACC, PoCG, PCG, thalamus, subthalamic nucleus, putamen, globus pallidum, and cerebellum (Zastrow et al., 2009). During an implicit sequence learning task, only between-group differences were found in the thalamus, with more activation shown in HC than AN (Firk et al., 2015).

DISCUSSION

The aim of this systematic review was to examine executive function-related brain activity in anorexia nervosa and obsessive-compulsive disorder. Throughout research, both disorders have shown executive dysfunction, which has been used to explain the manifest clinical characteristics (Del Casale et al., 2015; Diaz-Marsa et al., 2022; Kuelz et al., 2003; Martínez-Esparza et al., 2021). There are many notions that there is a possibility of shared neural mechanisms due to shared symptomatology between the two (Altman & Shankman, 2009; Gershkovich et al., 2017; Hoffman et al., 2012). The results of this review reveal distinctive neural mechanisms for each disorder. This is consistent with Thomas et al.'s (2022) review, in which AN and OCD may be utilizing different neural pathways for each executive function domain. They highlight that despite both presenting similar behavioural impairments, the way their brain adapts or compensates for executive dysfunction is different. Li et al.'s (2023) meta-analysis has also shown that both disorders present disorder-specific brain activation during inhibition.

The current review has found distinct brain activity when comparing the research based on tasks. Contrasting brain activation was observed when looking at within-group comparisons for the same tasks. For instance, research that used the go/no-go task to measure executive functioning in OCD have reported obverse results in areas such as the ACC, CN, and thalamus. Between-group comparisons for tasks were almost implausible due to research utilizing diverse tasks for each disorder.

When looking at neural activity during the n-back task, most studies have shown agreeable results regarding the correlation between task difficulty or working memory load and brain activation. For instance, during easier trials of the n-back task, OCD patients have shown hyperactivation of many brain areas such as the dACC, SMA, and IPL (De Vries et al., 2013; Koch et al., 2012; Vanderwee et al., 2003). However, this hyperactivation reduces during harder levels, revealing notions that these brain areas do not adjust well to changes in task difficulty (De Vries et al., 2013; Heinzel et al., 2017; Koch et al., 2012).

Many studies have suggested the notion of a compensatory mechanism during prolonged hyperactivation of frontoparietal areas (De Vries et al., 2013). For example, van der Wee et al.

(2003) have reported hyperactivation in the ACC during all n-back task levels, suggesting a possibility of compensation due to disturbed executive functioning other than working memory. Similar suggestions have been proposed by De Vries et al., (2013) in the DLPFC, pre-SMA, and precuneus. Interestingly, Koch et al. (2012) have reported a non-linear association between task difficulty and activation in the ACC (particularly in the dorsal area). This contrast with van der Wee et al.'s (2003) study could be due to differences in the nature of the n-back task (i.e., spatial or verbal).

Studies assessing task-switch or set shifting have noted a disintegration or conflict between internal and external models. In anorexia nervosa, higher activation in the precuneus, cuneus, lingual gyrus and insula during switching tasks is explained by higher self-referential processing and internal sensations, and less attention on external stimuli (Castro-Fornieles et al., 2019; Van Autreve et al., 2016). Similarly, reduced anticorrelation between the FPN and DMN in OCD is explained as patients' inability to disengage from internal thoughts during a task (Liu et al., 2023).

Through the Tower of London task (TOL), studies have suggested the notion of shifting from goal-directed planning to habitual responses. Vaghi et al. (2017) have reported reduced activation of the DLPFC, with weaker connections with putamen during the Tower of London task. This could suggest failure of the top-down control and could potentially explain the change from goal-directed planning to habitual responses. Furthermore, van den Heuvel et al.'s (2005) study has shown increased brain activity in the VLPFC, ACC, PHC, ATC, and brainstem, particularly as the task difficulty increases.

Moreover, studies have reported a disconnection between initial learning and cognitive flexibility. Particularly, AN patients have shown hyperactivation in the precuneus during the first trial of a switching task, suggesting that patients are able to learn new associations quickly (Lao-Kim et al., 2015). However, this pattern reduces in the second trial. Lao-Kaim et al. (2015) reported hypoactivation in the caudate nucleus after learning a new rule, suggesting a preservation error. This is further expanded through Sato et al.'s (2013) study, in which patients showed hypoactivation in the parahippocampal cortex and ventrolateral prefrontal cortex. This may suggest reduced ability to plan future events.

Additionally, studies report that OCD may present an over-active error-monitoring system and generate a greater affective response to errors or conflicting stimuli. Of particular interest, hyperactivation of the ACC has been reported by three studies. Schlösser et al. (2010) particularly noticed increased activation in the dorsal part of the ACC. Fitzgerald et al. (2005) and Maltby et al. (2004) have reported higher activation in the rostral part of the ACC, suggesting that OCD patients posit high sensitivity to errors and generate greater affective responses. Also, Maltby et al. (2004) have noted that higher activation in the posterior cingulate could offer two explanations: OCD patients perceive conflict or error monitoring tasks to be highly emotional or that there is a discrepancy between expected and predicted outcomes, resulting in higher prediction errors.

Contrastingly, Wierenga et al. (2014) and Suttkus et al. (2021) have reported a hypoactive error-processing control and emotional system during go/no-go and stop-signal tasks in anorexia nervosa. Particularly, hypoactivation was shown in the MFG and PCC, suggesting that patients do not monitor their mistakes (Wierenga et al., 2014). Also, patients have shown less activation in the amygdala and hippocampus compared to healthy controls, suggesting that patients are less emotionally sensitive to errors (Suttkus et al., 2021). Reduced error-monitoring in AN could be explained by an overall diminished activity of the cognitive control system. Research has proposed

that altered activation of ACC during inhibition and altered error-monitoring could explain that patients with AN may engage in self-control processes that perpetually subside receptivity to errors (Geisler et al., 2017).

Most reported brain region among all studies was the ACC. Previous research has proposed that the ACC, specifically the dorsal area, plays a core role in inhibitory cognitive control (Salehinejad et al., 2021). Propositions have also suggested that activation of the ACC is required when elevated level of control and conflict information processing is needed (Braver et al., 2001). That is, the ACC signals the conflict information processing to other regions involved in the cognitive control network; thereafter, these brain regions activate task-relevant information and inhibit task-irrelevant information (Botvinick et al., 2001; Braver et al., 2001). Moreover, it is postulated that the dACC is involved in the consolidation of reinforcement history, goal-driven efficiency and aversive anticipation (Andrzejewski et al., 2019; Botvinick et al., 2001; Liu et al., 2024; Shenhav et al., 2013; Shenhav et al., 2016; Yu & Desrivières, 2023). Cingulate abnormalities in OCD suggests that patients have excessive or disproportionate amount of error or conflicting monitoring (Zhao et al., 2023). Zhao et al., (2023) explain OCD as misattribution of pertinence to neutral or harmless stimuli due to disruptions in the salience network (particularly the dACC and anterior insula). When looking at cingulate-subcortical circuits, it has been suggested that OCD is a result of overreliance of the direct pathway. In other words, the direct pathway is known for supporting and enacting certain behaviours; whereas the indirect pathway is known for inhibiting the direct pathway during moments when changing or switching a behaviour is needed (Salehinejad et al., 2021). Hence, accounts denote OCD to be a disorder of arbitration (Lee et al., 2014; Robbins, 2024).

In respect to preliminary research analysing ACC activation in AN, inferences posit an aberrant self-control and self-referential system (Lee et al., 2013; Northoff & Bermpohl, 2004; Northoff et al., 2006). For instance, synchronous activity between the dACC and the retrosplenial cortex in AN signifies heightened introspective cognition (Lee et al., 2013). Such results propose a crucial role of the dACC in the cognitive control of appetite and body image. Suggestions that AN can be characterized as a disorder of excessive self-control and suppression of instinctive processes were given (Lee et al., 2013). This is further expanded through the connectivity between the ventral striatum, which is associated with value assignment and prediction error; the vmPFC, which is associated with the conciliation of subjective value and learning; and the dACC, which is associated with adaptive goal-directed behaviour and erroneous actions (Lee et al., 2013; Northoff & Bermpohl, 2004). Accordingly, hypoactivation in the ACC during inhibition could suggest diminished ability towards adaptive control, detecting erroneous or conflicting cues, and weighing reinforcement history and value assignment functionally (Lee et al., 2013).

The evidence gathered from the current review has several limitations. The first limitation is the lack of consistent experimental paradigm in assessing executive functioning. Such limitation has been mentioned in prior reviews (Thomas et al., 2022). Therefore, it is not a surprise that the studies would report different brain area activations. Moreover, the inclusion of studies published in English only and with most studies using WEIRD populations could potentially lead to missed insights and overgeneralization of results. Also, only one study has assessed the difference between AN and OCD patients concurrently, with the rest of the studies have focused on only one population (Bohon et al., 2019). Interestingly, this study has reported no significant neural difference between AN, OCD and HC groups (Bohon et al., 2019). This could either be interpreted that there truly is no difference between these populations or that it is due to the task used in the

study. Therefore, more research is required to assess all groups concurrently with the same task; rather than trying to compare results based on studies who have adopted different experimental methodologies.

The current review has highlighted the need for an exhaustive delineation of the neural mechanisms underlying executive functioning tasks in AN and OCD. Although between-group comparisons on neural activity were difficult, the current review reveals a common profile of reduced integration/consensus between internal and external world models and increased self-referential processing. Such results push forward research towards assessing executive function, its relation to reinforcement learning and whether AN and OCD differ in this context. Perhaps applying methods of predictive coding or active inference (Parr et al., 2022; Redish & Gordon, 2022) could offer new insights on how executive function is truly represented in the brain for both groups.

List of abbreviations

ACC: anterior cingulate cortex

AIC: anterior insula cortex

ANG: angular gyrus

AN: Anorexia Nervosa

APFC: anterior prefrontal cortex

ATC: anterior temporal cortex

CALC: calcarine fissure/cortex

CN: caudate nucleus

DAN: dorsal attention network

DMN: default mode network

DLPFC: dorsolateral prefrontal cortex

dPFC: dorsal prefrontal cortex

dACC: dorsal anterior cingulate cortex

fMRI: Functional Magnetic Resonance Imaging

FFC: fusiform face area

IFG: inferior frontal gyrus

IFS: inferior frontal sulcus

IPL: inferior parietal lobule

IPC: inferior parietal cortex

IPJ: inferior parietal junction

IOL: inferior occipital lobule

LG: lingual gyrus

LFPN: left frontoparietal network

MCG/C: mid-cingulate gyrus/cortex

MFG/C: middle frontal gyrus/cortex

MeFC: medial frontal cortex

MePFC: medial prefrontal cortex

MeTG: medial temporal gyrus

MOG/C: middle occipital gyrus/cortex

MO: middle occipital

MTG/C: middle temporal gyrus/cortex

PMC: premotor cortex

PMD: dorsal premotor cortex

PM: premotor area

PCG/C: posterior cingulate gyrus/cortex

PHG: parahippocampal gyrus

PoCG: postcentral gyrus

PRCU: precuneus

PRG: precentral gyrus

Pre-SMA: pre-supplementary motor area

PFC: prefrontal cortex

SFG: superior frontal gyrus

SMG/A: supplementary motor gyrus/area

SPC: superior parietal cortex

SPL: superior parietal lobule

STG/C: superior temporal gyrus/cortex

STJ: superior temporal junction

VLPFC: ventrolateral prefrontal cortex

WCST: Wisconsin Card Sorting Task

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Consent for Publication

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Both M.S and A.A screened and evaluated all included studies used in the review and M.S was a major contributor in writing the manuscript. All authors read and approved the final manuscript.

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Appendix A

Figure 1. *PRISMA Flow Diagram for fMRI studies that examined executive function tasks in AN patients*

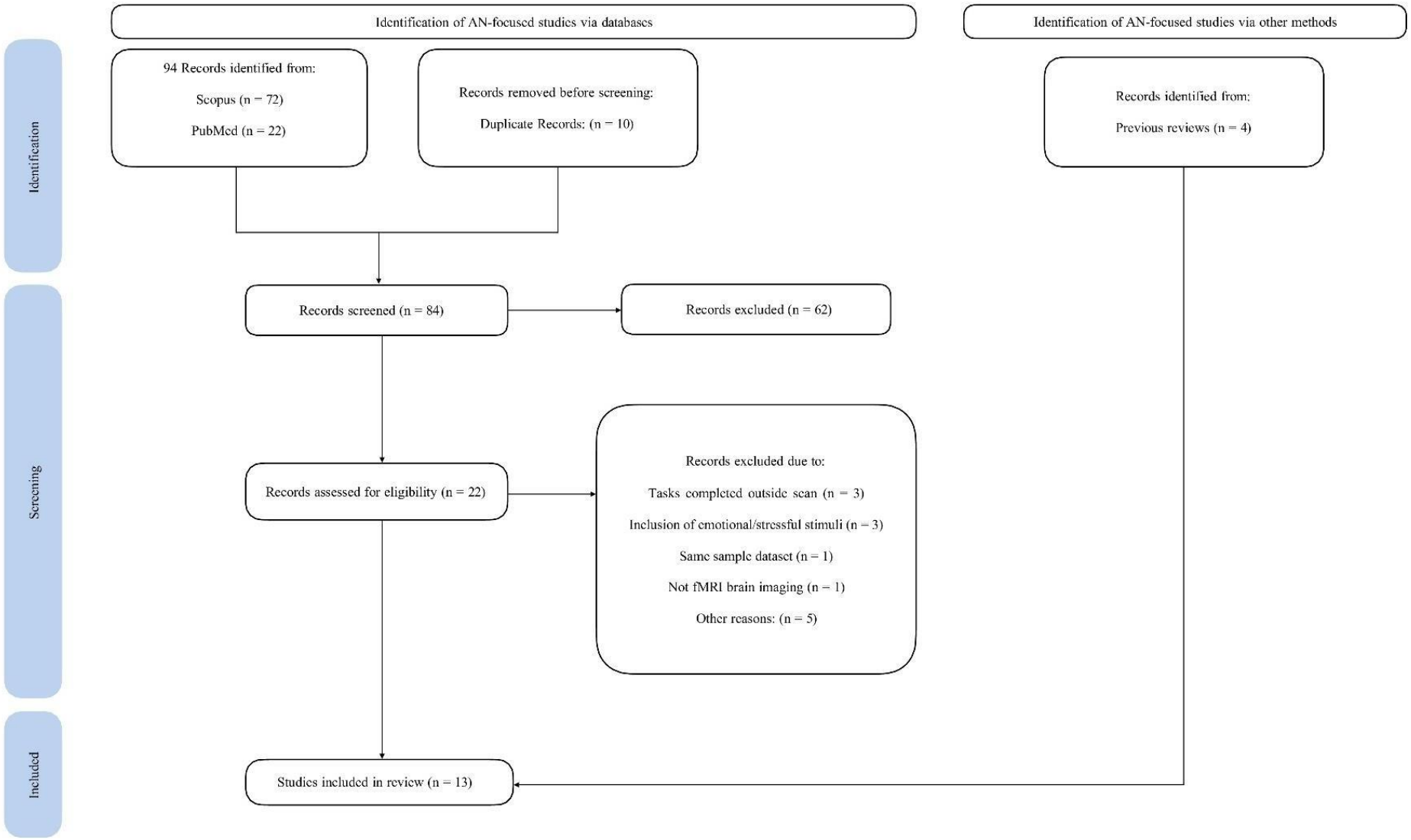
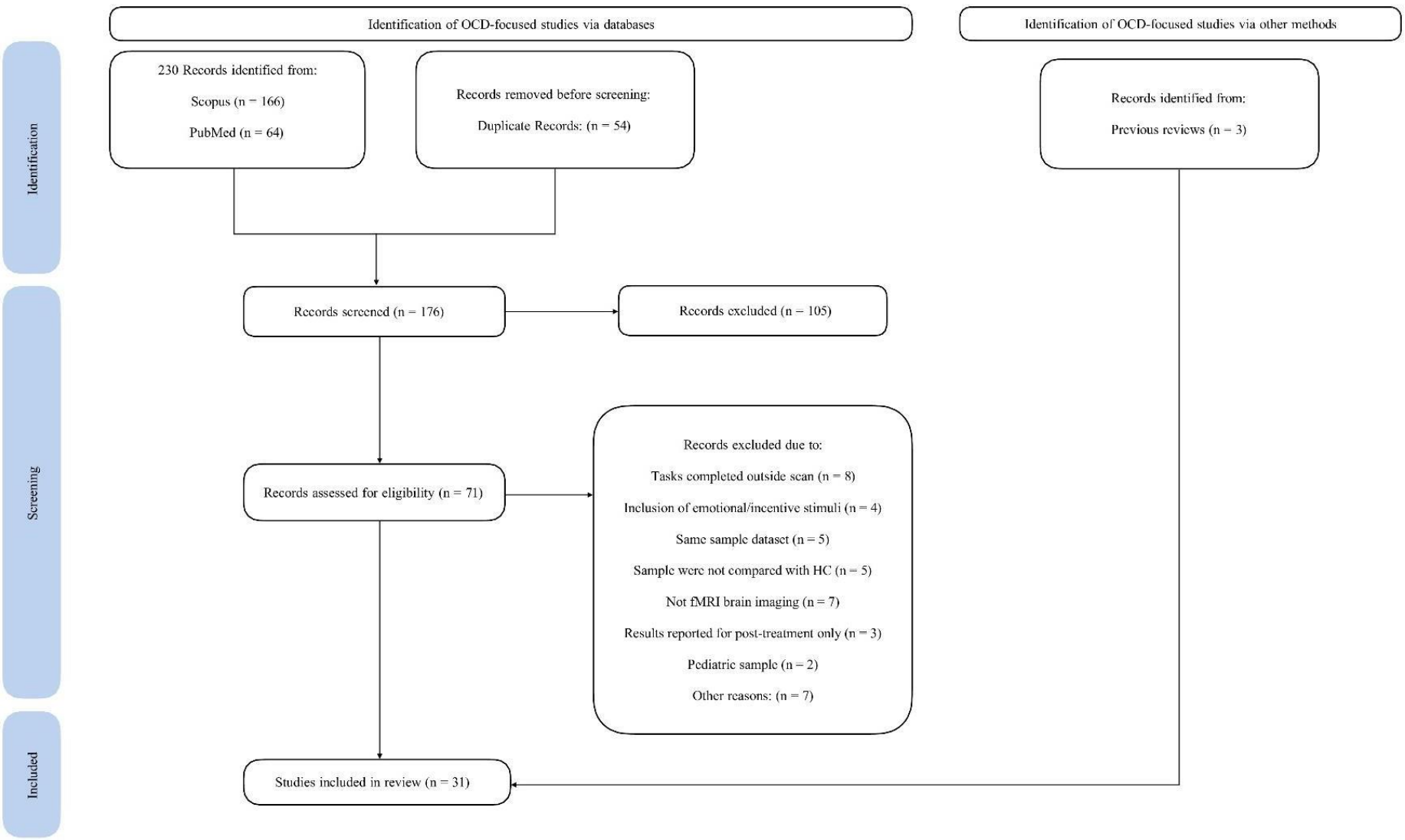


Figure 2. *PRISMA Flow Diagram for fMRI studies that examined executive function tasks in OCD patients*



Appendix B

Table 1. *fMRI Studies examining executive function tasks in OCD patients that were included in the current review*

	Study	Sample				fMRI	Fo	
	Authors	N	M/F	Age	Task	Analysis	OCD>HC	HC>OCD
1	Kim et al., 2021	OCD 30 HC 30	27/3 29/1	25.0 ± 5.2 22.8 ± 2.1	WCST	ROI; Whole-Brain Analysis	31	-
2	Gu et al., 2007	OCD 21 HC 21	18/3 18/3	23.6 ± 4.5 24.8 ± 3.7	Task-Switch	ROI; Whole-Brain Analysis	-	21
3	Vanderwee et al., 2003	OCD 11 HC 11	0/11 0/11	34.1 ± 9.6 34.8 ± 9.7	N-Back	ROI; Whole-Brain Analysis	2	-
4	Nakao et al., 2008	OCD 40 HC 25	16/24 10/15	33.3 ± 8.9 30.9 ± 7.1	N-Back	Whole-Brain Analysis	4	-
5	de Wit et al., 2012	OCD 41 HC 37	21/20 18/19	38.6 ± 9.8 39.7 ± 11.6	Stop-Signal	ROI	1	2
6	Tolin et al., 2013	OCD 24 24	18/6 4/20	33.54 ± 13.03 51.29 ± 9.88	Go/No-Go	ROI; Whole-Brain Analysis	1	-

			HC						
8	Koçak et al., 2011	12	OCD	6/6	27 ± 5.80	Imagination, Suppression, and Erasing	ROI; Whole-Brain Analysis	-	6
		12	HC	6/6	25.08 ± 3.32	Cards			
	Remijnse et al., 2013	18	OCD	4/14	33 (10-54)	Task-Switch	Whole-Brain Analysis	3	1
9			HC						

		29	9/20	33 (22-53) OCD				
10	Page et al., 2009	10	10/0	39.1 ± 10.2 HC	Go/No-Go; Stroop	Whole-Brain Analysis	7	9
		11	11/0	34.1 ± 10.1				
11	Hough et al., 2016	17	8/9	36.1 ± 10.4 OCD	Go/No-Go; Stroop	Whole-Brain Analysis	2	-
		17	8/9	36.1 ± 10.4 HC				
12	Vaghi et al., 2017	25	12/13	44.8 ± 16.2 OCD	Tower of London	ROI; Whole-Brain Analysis	-	2
		21	3/18	37.90 ± 14.31 HC				
		21	18/3	36.45 ± 8.54				
13	Kang et al., 2012	18	12/6	24.9 ± 5.9 OCD	Stop-Signal	ROI; Whole-Brain Analysis	5	13
		18	12/6	24.7 ± 2.7 HC				
14	Henseler et al., 2008	11	n/a	32.64 ± 7.17 OCD	Item-Recognition	ROI; Whole-Brain Analysis	10	-
11			n/a	33.73 ± 15.29 HC				
15	Maltby et al., 2004	14	5/9	39.36 ± 13.66 OCD	Go/No-Go	ROI	7	-
14			5/9	36.55 ± 11.36 HC				
16	Morein-Zamir et al., 2015	19	5/14	37.79 ± 10.10 OCD	Go/No-Go	ROI; Whole-Brain Analysis	3	13
19			5/14	36.16 ± 11.26 HC				

17	Roth et al., 2007	OCD 12	5/7	37.8 ± 13.2	Go/No-Go	Whole-Brain Analysis	4	7
		HC						
14			6/8	34.9 ± 13.2				
			OCD					
18	Schlösser et al., 2010	21	5/16	31.3 ± 10.2	Stroop	ROI	18	1
			HC					
21			5/16	28.8 ± 8.3				
			OCD					

19	Koch et al., 2012	21	5/16	29.4 ± 9.4	N-Back	ROI	2	1
HC		21	5/16	27.5 ± 6.1				
20	de Vries et al., 2013	OCD 43	22/21	38.1 ± 9.7	N-Back	ROI; Whole-Brain Analysis	3	-
		HC 37	17/20	39.2 ± 11.5				
21	Cocchi et al., 2011	OCD 17	8/9	32.8 ± 10.8	Stroop; Simon; Flanker	ROI; Whole-Brain Analysis	6	5
		HC 19	10/9	30.6 ± 7.2				
22	van den Heuvel et al., 2005	OCD 22	7/15	21-49	Tower of London	ROI; Whole-Brain Analysis	10	9
		HC 22	11/11	23-51				
23	Fitzgerald et al., 2005	OCD 8	6/2	27.4 ± 8.5	Flanker	ROI; Whole-Brain Analysis	11	7
		HC 7	5/2	30.0 ± 8.6				
24	Masharipov et al., 2023	OCD 14	9/5	30.0 ± 10.6	Go/No-Go	ROI; Whole-Brain Analysis	-	9
		HC 34	10/24	25.9 ± 5.2				
25	Liu et al., 2023	OCD 42	25/17	21.86 ± 4.85	HC			

		48	21/27	20.65 ± 2.10		Task-Switch	ROI; Whole-Brain Analysis	7		-
		OCD								
26	Meram et al., 2020	28	10/18	16.53 ± 3.07		N-Back	ROI; Whole-Brain Analysis	26		-
27			HC 10/17	16.32 ± 2.70						
		OCD								
27	Han et al., 2011	10	9/1	23.2 ± 4.5		Task-Switch	ROI; Whole-Brain Analysis	-		22
20			HC 18/2	24.3 ± 2.9						
		OCD								
28	Thorsen et al., 2020	31	12/19	30.19 ± 9.21		Stop-Signal	ROI; Whole-Brain Analysis	17		12
		HC								

		26	8/18	31 ± 10.73						
29	Bohon et al., 2019	OCD 11	0/11	15.64 ± 2.01		WSCT	Whole-Brain Analysis	-	-	
		HC 24	0/24	15.29 ± 1.65						
30	Kim et al., 2020	OCD 17	12/5	26.4 ± 6.0		Tower of London	Whole-Brain Analysis	4	-	
		HC 21	11/10	26.0 ± 5.3						
31	Grützmann et al., 2014	OCD 84	39/45	31.6 ± 9.5		Flanker	Whole-Brain Analysis	2	-	
		HC 99	41/58	31.4 ± 9.7						

Table 2. *fMRI Studies examining executive function tasks in AN patients that were included in the current review*

Study		Sample			fMRI		Foci	
	Authors	N	M/F	Age	Task	Analysis	AN>HC	HC>AN
1	Wierenga et al., 2014	11	0/11	AN 16.0 ± 2.0	Stop-Signal	ROI; Whole-Brain Analysis	5	11
12			0/11	HC 14.9 ± 1.8				
2	Lao-Kaim et al., 2015	32	0/32	AN 18-41	WCST	Whole-Brain Analysis	2	-
32			0/32	HC 22-46				
3	van Autreve et al., 2016	29	0/29	AN 22 ± 6 AN-R; 24 ± 4 AN-BP	Set-Shift	ROI; Whole-Brain Analysis	12	-
15			0/15	HC 22 ± 4				
4	Castro-Fornieles et al., 2019	30	0/30	AN 14.9 ± 1.3	Set-Shift	ROI; Whole-Brain Analysis	3	15
16			0/16	HC 15.3 ± 1.4				
5	Sato et al., 2013	15	0/15	AN 23+/- 7 AN; 21± 9 AN-R; 26 ± 9 AN-BP;	WCST	ROI; Whole-Brain Analysis	-	3
15			0/15	HC 22 ± 3				
6	Zastrow et al., 2009	15	0/15	AN 24.2 ± 2.3	Target-Detection	Whole-Brain Analysis	-	29
15			0/1	HC 23.1 ± 3.6				

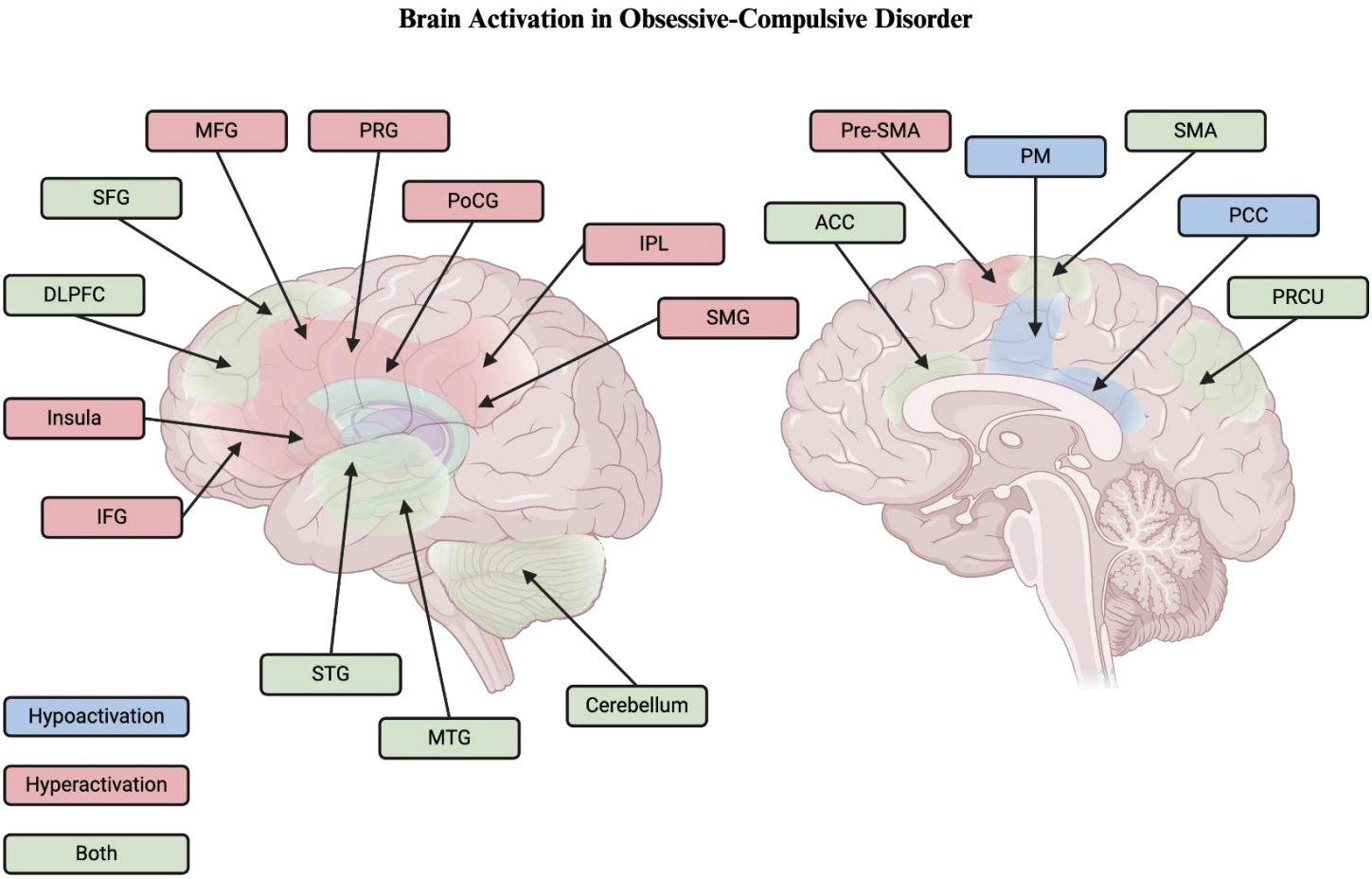
			5						
7	Lock et al., 2010	27	0/27	AN 15.02 ± 1.74 AN-R; 17.26 ± 1.23 AN-BP/BN	Go/No-Go	ROI; Whole-Brain Analysis	6	-	
13			0/13	HC 15.93 ± 1.39					
8	Noda et al., 2021	23	0/23	AN 37.04 ± 9.88	Go/No-Go	Whole-Brain Analysis	4	-	
17			0/17	HC 36.24 ± 10.59					
9	Suttkus et al., 2021	16	2/14	AN 24.88 ± 7.85	Go/No-Go	Whole-Brain Analysis	-	2	
21			2/19	HC 26.29 ± 6.9					
10	Bohon et al., 2019	14	0/14	AN 15.79 ± 1.93	WCST	Whole-Brain Analysis	-	-	
24			0/24	HC 15.29 ± 1.65					

AN

HC	11	Lao-Kaim et al., 2013	31	n/a		18-46	N-Back	ROI; Whole-Brain Analysis	-	-
AN			31	n/a		22-34				
	12	Firk et al., 2015	19	0/19	HC	15.9 ± 1.5	Implicit Sequence Learning	ROI; Whole-Brain Analysis	-	1
AN			20	0/20		15.9 ± 1.9				
	13	Leslie et al., 2021	19	0/19	HC	16-25	Embedded Figures Task	ROI; Whole-Brain Analysis	-	-
			20	0/20		16-25				

Appendix C

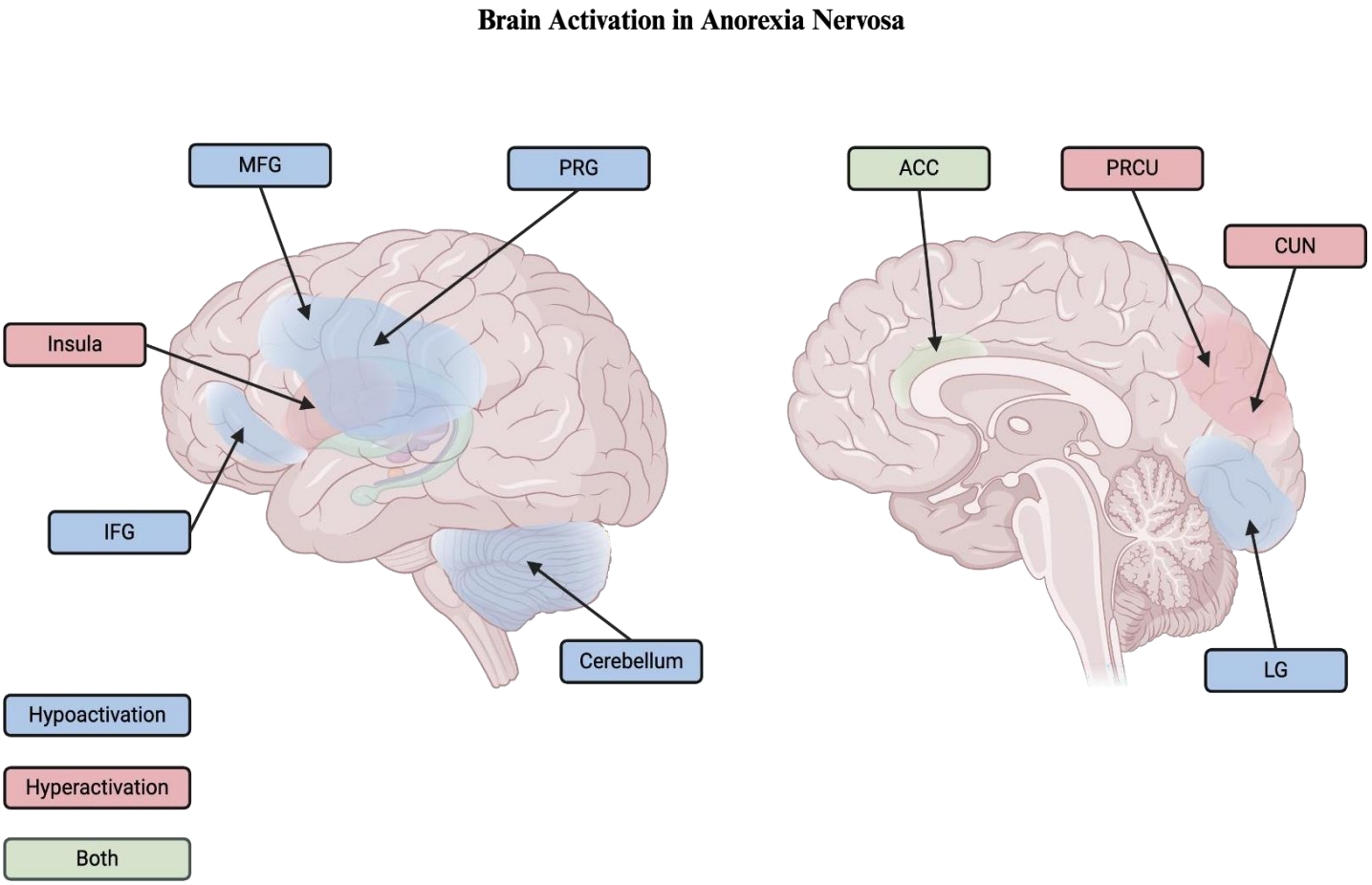
Figure 1. *Common Brain Area Activation Patterns in Obsessive-Compulsive Disorder.*



Note. The figure shows brain area activations that were commonly reported by the included studies. Areas shaded in blue represent hypoactivation (OCD<HC) and areas shaded in red represent hyperactivation (OCD>HC). Brain areas that were commonly reported, but with inconsistent brain activation patterns (i.e., some studies reported hypoactivation, while others reported hyperactivation) were shaded in green.

PM: pre-motor area, PCC: posterior cingulate cortex, IFG: inferior frontal gyrus, MFG: middle frontal gyrus, PRG: precentral gyrus, PoCG: postcentral gyrus, IPL: inferior parietal lobule, SMG: supramarginal gyrus, pre-SMA: pre-supplementary motor area, MTG: middle temporal gyrus, STG: superior temporal gyrus, DLPFC: dorsolateral prefrontal cortex, SFG: superior frontal gyrus, ACC: anterior cingulate cortex, SMA: supplementary motor area, PRCU: precuneus. Created in BioRender. Sayed, M. (2025) https://BioRender.com/z92i759

Figure 2. *Common Brain Area Activation Patterns in Anorexia Nervosa.*



Note. The figure shows brain area activations that were commonly reported by the included studies. Areas shaded in blue represent hypoactivation (AN<HC) and areas shaded in red represent hyperactivation (AN>HC). Brain areas that were commonly reported, but with inconsistent brain activation patterns (i.e., some studies reported hypoactivation, while others reported hyperactivation) were shaded in green.

IFG: inferior frontal gyrus, MFG: middle frontal gyrus, PRG: precentral gyrus, LG: lingual gyrus, PRCU: precuneus, CUN: cuneus, ACC: anterior cingulate cortex. Created in BioRender. Sayed, M. (2025) https://BioRender.com/z92i759

Appendix D

Table 3. *Search Strategy for fMRI studies that examine executive function tasks in OCD or AN patients*

Database		Search Query
PubMed	#1	"functional magnetic resonance" [Title/Abstract] OR "fmri" [Title/Abstract] OR "neuroimaging" [Title/Abstract]
	#2	"stroop" [Title/Abstract] OR "wisconsin card sorting" [Title/Abstract] OR "trail making" [Title/Abstract] OR "go no go" [Title/Abstract] OR "n back" [Title/Abstract] OR "flanker" [Title/Abstract] OR "rey osterrieth complex figure" [Title/Abstract] OR "tower of london" [Title/Abstract] OR "task switch" [Title/Abstract] OR "stop signal" [Title/Abstract]
	#3	"executive function" [Title/Abstract] OR "working memory" [Title/Abstract] OR "plan*" [Title/Abstract] OR "cogntive flexibility" [Title/Abstract] OR "inhibit*" [Title/Abstract] OR "inhibitory control" [Title/Abstract] OR "set shift*" [Title/Abstract] OR "attentional control" [Title/Abstract] OR "problem solving" [Title/Abstract] OR "error monitor*" [Title/Abstract]
	#4	"ocd" [Title/Abstract] OR "obsess*" [Title/Abstract] OR "compuls*" [Title/Abstract] OR "obsessive compulsive disorder" [Title/Abstract]
	#5	"anorexi*" [Title/Abstract] OR "anorexia nervosa" [Title/Abstract] OR "eating disorder" [Title/Abstract]
	#6	#1 AND #2 AND #3 AND #4
	#7	#1 AND #2 AND #3 AND #5
	#1	(TITLE-ABS-KEY(functional magnetic resonance) OR TITLE-ABS-KEY(fmri) OR TITLE-ABS-KEY(neuroimaging))
	#2	(TITLE-ABS-KEY(stroop) OR TITLE-ABS-KEY(wisconsin card sorting) OR TITLE-ABS-KEY(trail making) OR TITLE-ABS-KEY(go no go) OR TITLE-ABS-KEY(simon effect) OR TITLE-ABS-KEY(simon task) OR TITLE-ABS-KEY(stop signal) OR TITLE-ABS-KEY(n back) OR TITLE-ABS-KEY(flanker task) OR TITLE-ABS-KEY(rey osterrieth complex figure) OR TITLE-ABS-KEY(tower of london) OR TITLE-ABS-KEY(task switch))

Scopus	#3	(TITLE-ABS-KEY(executive function*) OR TITLE-ABS-KEY(working memory) OR TITLE-ABS-KEY(plan*) OR TITLE-ABS-KEY(cognitive flexibility) OR TITLE-ABS-KEY(inhibit*) OR TITLE-ABS-KEY(inhibitory control) OR TITLE-ABS-KEY(set shift*) OR TITLE-ABS-KEY(attentional control) OR TITLE-ABS-KEY(problem solving) OR TITLE-ABS-KEY(error monitor*))
	#4	(TITLE-ABS-KEY(ocd) OR TITLE-ABS-KEY(obsess*) OR TITLE-ABS-KEY(obsessive compulsive disorder) OR TITLE-ABS-KEY(ocd obsessive compulsive disorder) OR TITLE-ABS-KEY(compuls*))
	#5	(TITLE-ABS-KEY(anorexia) OR TITLE-ABS-KEY(anorexia nervosa) OR TITLE-ABS-KEY(anorexi*) OR TITLE-ABS-KEY(eating disorder))
	#6	#1 AND #2 AND #3 AND #4
	#7	#1 AND #2 AND #3 AND #5

Appendix E

Table 4. *Quality Assessment for fMRI studies examining executive function tasks in OCD and AN patients*

Author	Year	1	2	3	4	5	6	7	8	9	Quality Score
Kim et al.	2021	2	2	1	1	1	1	1	1	2	12
Gu et al.	2007	2	2	2	2	2	2	2	2	2	18
Vanderwee et al.	2003	1	2	2	2	2	2	2	2	2	17
Nakao et al.	2008	1	2	1	2	1	1	2	2	2	14
de Wit et al.	2012	2	2	2	2	1	2	2	2	2	17
Tolin et al.	2013	2	2	1	2	1	2	2	1	1	14
Heinzel et al.	2017	2	2	1	1	2	1	2	2	2	15
Koçak et al.	2011	2	2	0	1	2	2	2	2	2	15
Remijnse et al.	2013	1	2	1	2	2	2	1	1	2	14
Page et al.	2009	1	2	1	2	2	1	1	1	2	13
Hough et al.	2016	2	2	2	2	2	2	1	1	2	16
Vaghi et al.	2017	1	2	2	2	2	2	2	2	2	17
Kang et al.	2012	1	2	2	2	2	2	2	2	2	17
Henseler et al.	2008	2	2	2	2	2	2	1	2	2	17
Maltby et al.	2004	2	2	0	1	2	2	2	2	2	15
Morein-Zamir et al.	2015	1	2	2	2	2	2	2	2	2	17
Roth et al.	2007	1	2	2	2	2	2	2	2	2	17
Schlösser et al.	2010	2	2	2	2	2	2	2	2	2	18
Koch et al.	2012	2	2	2	2	2	2	2	2	2	18
de Vries et al.	2013	2	2	2	2	2	2	2	2	2	18
Cocchi et al.	2011	2	1	2	2	2	2	2	2	2	17
van den Heuval.	2005	2	2	1	2	2	2	1	2	2	16
Fitzgerald et al.	2005	1	2	0	2	2	2	2	2	2	15
Masharipov et al.	2023	2	2	0	2	2	2	1	2	2	15
Liu et al.	2023	2	2	2	2	2	2	2	2	2	18
Meram et al.	2020	1	2	0	2	1	2	1	2	2	13
Han et al.	2011	2	2	0	2	2	2	2	2	2	16
Thorsen et al.	2020	2	2	2	2	2	2	2	2	2	18
Bohon et al.	2019	1	2	2	2	2	1	1	1	2	14
Kim et al.	2020	2	2	0	2	2	2	2	2	2	16

Grützmann et al.	2014	1	2	0	2	2	1	2	1	2	13
Wierenga et al.	2014	2	2	1	2	2	2	2	2	2	17
Lao-Kaim et al.	2015	2	2	1	2	2	2	2	2	2	17
Van Autreve et al.	2016	2	2	0	2	2	2	2	1	2	15
Castro-Fornieles et al.	2019	2	2	1	2	2	2	2	2	2	17
Sato et al.	2013	1	2	2	2	2	2	1	2	2	16
Zastrow et al.	2009	1	2	1	1	2	2	1	1	1	12
Lock et al.	2010	2	1	2	2	1	2	1	1	2	14
Noda et al.	2021	1	1	1	2	2	2	2	1	1	13
Suttkus et al.	2021	1	1	1	2	2	2	1	2	2	14
Lao-Kim et al.	2013	1	1	1	2	2	2	2	2	1	14
Firk et al.	2015	2	2	2	2	2	1	1	1	1	14
Leslie et al.	2021	1	1	1	2	2	2	2	2	2	15