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Anorexia Nervosa: Neural Correlates of Obsessive-Compulsive Symptoms During
Food Reward Processing

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Table of Contents

Introduction	3
Epidemiology	3
Diagnostic Criteria and Symptomology of Anorexia Nervosa	4
Diagnostic Criteria and Symptomology of Obsessive-Compulsive Disorder and Diagnostic Boundary to Anorexia Nervosa	4
Obsessive-Compulsive traits and tendencies in Anorexia Nervosa	5
Neuroimaging findings in Anorexia Nervosa	7
Neural correlates of obsessive-compulsive symptoms	9
The Reward System and its Neural Mechanisms	10
The Reward System and Food Reward Processing in Anorexia Nervosa	10
Aims of This Study	13
Hypotheses	14
Methods	15
Study Design	15
Variables	16
Data Analysis	19
Results	20
Discussion	30
Conclusion	37
Sources	39
Appendix	56
Abstract (DE)	62
Abstract (EN)	63
List of Tables and Figures	56
List of Abbreviations	57
Supplementary Figures	59

Introduction

Anorexia nervosa (AN) is considered one of the most lethal psychiatric disorders (Meczekalski et al., 2013; Mehler et al., 2022; Van Eeden et al., 2021) with a frequently observed comorbid disorders (Himmerich et al., 2019) and a high chronification rate (Dobrescu et al., 2020; Fichter et al., 2017; Marcolini et al., 2024). Malnourishment and its immediate or longer term consequences (such as organ failure and pulmonary diseases), as well as suicide lead to its mortality rate estimated at around 5% - 5.6% (Søeby et al., 2024) or a standardized mortality ration (SMR) of 5.21 (Krug et al., 2025) and 5.86 (Arcelus et al., 2011) for AN. The heightened risks are especially relevant in the first 10 years after diagnosis (Søeby et al., 2024). Affected people experience a huge compromise in their life quality and wellbeing (Startup et al., 2012), cognitive abilities (Fielding et al., 2023). It immensely affects their long-term health, regarding the reproductive, cardiovascular, gastrointestinal, neuroendocrine and skeletal systems (Adamo & Pisano, 2023; Cass et al., 2020; Meczekalski et al., 2013; Springall et al., 2023). There is a high risk for medical complications, some of which are irreversible, even after remission (Falkai et al., 2018). The relevance of research on this disorder cannot be overestimated.

Epidemiology

The lifetime prevalence rates of AN are estimated to be up to 4% in females and 0.3% in males (Cass et al., 2020; Herpertz-Dahlmann & Altdorf, 2023; Van Eeden et al., 2021). It's most diagnosed in young females (Martinez-Gonzalez et al., 2020), with a female to male ratio regarding an anorexia nervosa diagnosis of 10:1 (Herpertz-Dahlmann & Altdorf, 2023). The number of undiagnosed males might be underestimated or present differently, for example through muscularity-oriented disordered eating (Nagata et al., 2020). The standardized mortality ratio (SMR) for males with AN was found to be higher than for females (Van Eeden et al., 2021).

For Austria, Zeiler et al. (2015) identified 30.9% of girls and 14.6% of boys to be at risk for eating disorders (ED) from a sample of 3610 adolescents. An epidemiological study based on a school sample (N = 3477) found a lifetime prevalence of 2.61% for eating disorders and a 0.53% lifetime prevalence for Anorexia nervosa, however for the clinical sample (N = 137) based on participants contacted through child- and adolescent psychiatries there was a 30.11% lifetime prevalence of AN. The combined school and clinical sample divided for girls and boys shows a 5.47% lifetime

prevalence of eating disorders for the combined school and clinical sample (Wagner et al., 2017).

Diagnostic Criteria and Symptomology of Anorexia Nervosa

The DSM-5, codes F50.01 and F50.02 (American Psychiatric Association, 2018) and the ICD-11 code 6B80 (World Health Organization, 2022) define the following diagnostic criteria for anorexia nervosa:

A significantly low body weight (in relation to age, height and gender), that is not attributable to any other health conditions or lack of food availability. For adults, the Body-Mass-Index (BMI) is used as a metric, with a BMI lower than 18.5kg/m² (ICD-11) or 17kg/m² (DSM-5) indicating underweight status. For children, the threshold is defined as a BMI below the 5th percentile of age-normative BMI and is often indicated not by weight loss but weight stagnation.

Persistent behavior that aims for a decreased energy intake (e.g. fasting) or increased energy requirement (e.g. exercise), leading to and resulting in a lower body weight. Additionally, the wide range of behaviors is backed by an intense fear of gaining weight.

An inaccurate perception of own bodyweight and shape, with one's self-worth being intensely tied to said distorted perception. Furthermore, ill individuals are unaware and/or disagree about the severity of health conditions and seriousness of their low bodyweight (Arbel et al., 2014). Weight loss is viewed as an accomplishment.

The ICD-11 and DSM-5 distinguish between two types of anorexia nervosa. The restrictive type (ICD-11: 6B80.x0, DSM: F50.01) induces weight loss or stagnation of weight by dieting, fasting and/or exercising, however is not engaging in binge-eating or purging during the last three months. The binge-purging type was engaged in bingeing and purging in the last three months.

Diagnostic Criteria and Symptomology of Obsessive-Compulsive Disorder and Diagnostic Boundary to Anorexia Nervosa

OCD, as defined by the DSM-5 (American Psychological Association, 2018) and the ICD-11 (World Health Organization, 2022), describe the presence of obsessions, compulsions, or both. The experience of recurrent and persistent thoughts is disturbing and unwanted and met with attempts to ignore and suppress them (e.g. by giving in to the compulsion). Individuals with OCD feel forced to perform obsessive

repetitive behaviors or mental acts. Said behaviors are aimed at reducing stress, however they are clearly excessive and not connected to any stress sources in a realistic way. Additionally, they are time consuming and impairing important areas of life. A symptom-dimensions perspective has been proposed as an alternative to categorical descriptions and two motivational dimensions underlying obsessive-compulsive symptoms have been found: harm avoidance and incompleteness (Summerfeldt et al., 2014).

An important distinction for the OCD diagnosis is the lack of better explanation of the symptoms by any other mental disorder (e.g. ruminations by major depressive disorder) (World Health Organization, 2022). The distinction between AN and OCD, however, was a highly debated topic (Serpell et al., 2002), with a relationship between both disorders suggested as early as 1939 by Palmer and Jones. The current consensus in research is that AN is distinct from OCD (Di Luzio et al., 2024). The reasons include the AN specific time of onset during puberty, denial of illness, lack of insight, body-related preoccupations being regarded as sensible and the obsessions and compulsions being perceived as ego syntonic, therefore aligning with one's values and self-image (Phillips & Kaye, 2014). In OCD, symptoms are ego-dystonic, with affected individuals typically perceiving their obsessions and compulsions as unwanted, but feeling incapable to stop them (Kaye et al., 1992).

Obsessive-Compulsive traits and tendencies in Anorexia Nervosa

Most patients with anorexia nervosa experience a lifetime mental disorder, most commonly mood disorders, anxiety disorders, obsessive-compulsive disorders and substance use disorders (Fernandez-Aranda et al., 2007; Herpetz-Dahlmann & Dahmen, 2019; Marucci et al., 2018; O'Brien & Vincent, 2003). Obsessive-compulsive disorder occurs in 10-62% (Riquin et al., 2021), with most AN diagnosed individuals experiencing obsessions or compulsions throughout their life. A comorbid disorder relates to higher clinical severity (Himmerich et al., 2019; Riquin et al., 2021) and higher treatment resistance (Nagy et al., 2023). The overlaps regarding the compulsive nature of behaviors are striking. OCD and AN consistently show systematic co-occurrence (Herpetz-Dahlmann & Dahmen, 2019).

Research has identified shared personality traits, such as perfectionism and neuroticism (Holden, 1990; Ortiz et al., 2022; Williams et al., 2022; Young et al., 2013). Even though the correlation between both AN and OCD cannot be fully

explained by personality traits alone (Pollack & Forbush, 2013; Yilmaz et al., 2022), trait contributions to symptomatology remain clinically relevant. There is a lot of evidence regarding certain traits, such as perfectionism, concern over mistakes, intolerance of uncertainty, and obsessive beliefs to be associated with illness course and outcome in AN and OCD patients, as well as comorbid patients (Cordeiro et al., 2015; Crane et al., 2007; Levinson et al., 2019a; Levinson et al., 2019b; Touzeau et al., 2025). Not only do those traits occur before onset of an eating disorder, but they often persist after recovery (Holtkamp et al., 2005; Wagner et al., 2006).

Beyond trait-related correlates, the acute underweight state appears to substantially shape clinical presentation. The acute underweight state has been associated with individually highest scores of depression, anxiety, and obsessionality, with symptom levels decreasing during weight restoration (Pollice et al., 1998). Consistently, obsessive-compulsive symptoms appear to be highest in acutely ill inpatients, with weight restored AN patients having significantly lower symptoms scores (Mattar et al., 2012; Meule & Voderholzer, 2022; Roberts et al., 2011). It remains unclear whether the persistence of obsessive-compulsive symptoms reflects a stable trait or a state-dependent consequence of the illness, and it is just as uncertain which symptoms features are associated with malnutrition, and which were present before the onset of AN or were independent from it. An example for that is the often trait-like cognitive deficit of poor set-shifting and lack of cognitive flexibility, with high illness duration and severity contributing to said weaknesses (Sternheim et al., 2022). Contrarily, Rao et al. (2008) mentions certain cognitive alterations, such as set-shifting abilities, to be state-independent and found them to be present in recovered patients. Emotion regulation remains a long-term challenge for individuals recovered from AN, even with improvement of alexithymia, anxiety and depression (Castro et al., 2021).

On a biological level, there is an overlap in genetics (Di Luzio et al., 2024; Mas et al., 2013) and structural abnormalities in the brain. The latter have been found in the right insula, superior temporal gyrus, rolandic operculum and inferior frontal gyrus, which may relate to the similarity in neuropsychological features (Wan et al., 2025). Genetic and neurobiological factors are to be differentiated for obsessive-compulsive manifestations regarding severity and content across development of the person since childhood (Leckman et al., 2001; Leckman et al., 2009).

OCD often precedes eating disorder symptoms (Rao & Wadford, 2025). Childhood OCD is considered as a risk factor for eating disorders by Micali et al. (2011). Individuals with both diagnoses tend to develop the ED at a younger age and experience a more chronic disorder progression (Lo Sauro et al., 2012). Interestingly, obsessive-compulsive symptoms are more prevalent in eating disorders with bingeing and purging, than in restrictive EDs (Di Luzio et al., 2024).

At a symptom level, obsessions seem to be the common dimension of both disorders and need particular attention in assessment and treatment of AN with and without a comorbid OCD diagnosis. Subthreshold presence of obsessive-compulsive symptoms in the clinical context is highly prevalent and to be considered. Obsessions are related to most AN symptoms (Levinson et al., 2019), even obsessions that are not directly associated with eating (Di Luzio et al., 2024). A systematic review of 51 studies with more than 15,000 participants further confirmed the association between obsessions, compulsions and eating disorders - specifically cognitive aspects may play an important role in the comorbidity between OCD and ED (Acar et al., 2026).

Taken together, the evidence supports an interaction model in which trait vulnerabilities (such as perfectionism and intolerance of uncertainty) present a risk and fuel persistence, whereas state effects of malnutrition amplify rigidity and obsessional symptom expression, with relative contributions varying across illness stages.

Neuroimaging findings in Anorexia Nervosa

Food restriction in Anorexia not only affects the brain function but also changes the structure of the brain (Alfano et al., 2020). Adolescents are affected by gray matter (GM) loss even more than adults (Seitz et al., 2016). GM volume (Boto et al., 2017), as well as cortical thickness, significantly decreases and white matter (WM) structure significantly changes, in various regions in persons with AN with severe disease severity regarding weight loss (Bär et al., 2015). The regions that are affected the most are the prefrontal, parietal and temporal cortex, hippocampus, amygdala, insula, thalamus and cerebellum, and WM tracts, e.g. corona radiata and superior longitudinal fasciculus (Kappou et al., 2021). That is clinically relevant, because GM volume is correlated with cognitive deficits (Seitz et al., 2016). In populations with AN, adults as well as adolescents show brain volume reductions in regions associated with reward processing (Dahlberg et al., 2017). Comorbid personality disorders are

also associated with GM alterations in the thalamus, cingulate and parieto-occipital regions, which persist after weight restoration (Lenhart et al., 2025). While the pattern of reduction persists in short-term weight recovery, long-term recovered patients show no significant deficits anymore (Seitz et al., 2016). Clinicians should prioritize fast weight recovery, especially for adolescents, to reduce the time spent in starvation, to preserve brain volume and the normal development of neural plasticity, and therefore protect cognitive flexibility, learning and the effectiveness of psychotherapeutic treatment (Keeler et al., 2023; Seitz et al., 2016).

Functional neuroscience findings in AN are typically taken from task-based functional magnetic resonance imaging (fMRI), resting-state fMRI and positron emission tomography (PET). Central AN features, such as cognitive inflexibility and deficits in emotional processing and executive control, are related to functional abnormalities across several areas and networks. High cognitive inflexibility is a predictor of poor illness outcome, even after controlling for body weight and duration of illness (Favaro, 2018).

For people with AN, differences in intrinsic functional activity, a decreased resting-state functional activity in the bilateral anterior and median cingulate cortex, and an increased resting-state functional activity in the right parahippocampal gyrus have been found by Su et al. (2021). A lower resting-state functional connectivity was demonstrated between the nucleus accumbens (NAcc) and superior frontal gyrus, correlating with greater Eating Disorder Examination (EDE) scores (Haynos et al., 2018). Studies on functional imaging found alterations in large-scale brain networks, such as default mode network, executive control network and salience network (Kappou et al., 2021) and sensorimotor network (Zhang et al., 2018). Resting-state fMRI studies have found brain alterations in networks and areas involved with cognitive control, inflexibility and altered processing and integration of body signals (Gaudio et al., 2016).

The anterior cingulate cortex (ACC) is considered especially relevant (Gaudio et al., 2015; Olsavsky et al., 2019). It is part of the emotion-related limbic system and cognition-related prefrontal cortex and plays a role in both domains as a “top-down” player providing cognitive abilities, such as volitional attention (White et al., 2018) and the preferential processing of stimuli and responses (Crottaz-Herbette & Menon,

2006). The ACC is therefore an important part of the executive control network (Stevens et al., 2011).

Neural correlates of obsessive-compulsive symptoms

Since there is little research regarding the neural basis of obsessive-compulsive symptoms in AN, findings from OCD research will be used as a basis to bring out potential models.

Across studies, abnormalities have been reported for various aspects. Key regions associated with obsessive-compulsive behavior are the orbitofrontal cortex (OFC), ACC, amygdala and cortico-striatal regions (Abe et al., 2015; Maia et al., 2008; Milad & Rauch, 2012). OCD is also often conceptualized as a cortico-striatal-thalamo-cortical (CSTC) dysfunction (Alturaiqi et al., 2025). Reduced connectivity within the limbic CSTC loop has been associated with greater symptom severity in OCD patients (Hou et al., 2013; Posner et al., 2014). Said dysfunction leads to hyperactivations in the OFC, ACC and basal ganglia (Casale et al., 2011; Kwon et al., 2014; Leckman et al., 2022). Hyperactivation of the OFC is associated with intrusive thoughts, while hyperactivation of the ACC is associated with non-specific anxiety (Del Casale et al., 2011). Consistent with that, there are findings for activations in the OFC and ACC during symptom provocation, which are hypothesized to be related to the patients' effort to resist (Rotge et al., 2008). Furthermore, activation in the limbic CSTC circuit and insula were associated with heightened emotional processing of visceral and offensive stimuli, such as disgust, which may demonstrate a dysfunctional process appraisal of objects and events in OCD (Husted et al., 2006). Temporo-limbic regions, typically associated with emotion processing, were revealed as important for neurobiological alterations in OCD (Reess et al., 2016).

Functional neuroimaging studies have observed changes in several brain regions following various therapeutic intervention for OCD, most notably decreased metabolism and perfusion of the caudate, ACC, thalamus, regions of the prefrontal cortex (PFC), including the OFC, among others (Bijanki et al., 2021; Norman et al., 2021). Some studies find the activation pattern changing to a normal level after successful treatment (Nabeyama et al., 2007).

There are no studies directly investigating the neural overlap between OCD and anorexia nervosa, specifically in the context of consummatory reward processing. The mechanisms that reinforce the OCD habit formation overlap functionally with

neural systems of reward processing. Examining obsessive-compulsive symptoms in the context of anorexia nervosa may help discover potential transdiagnostic mechanisms that may contribute to understand reward-related alterations in brain activation.

The Reward System and its Neural Mechanisms

First, general reward processing needs to be explained. A reward is experienced when the brain associates any stimuli, such as activities, events or substances, with a desirable outcome (Schultz, 2006). The associations result in a behavioral shift towards the (re)obtainance of stimulus with following positively experienced effect (Lewis et al., 2022) in the form of motivation (Berridge & Robinson, 2003). Reward processing is not a simple construct, it describes multiple components, including motivation, cost-benefit decision making and action-outcome association, among others (Treadway & Zald, 2013), but research mostly focuses on incentive salience (“wanting”) and hedonic responses (“liking”) (Berridge, 2009). The two pillars of reward, wanting and liking, are interconnected and typically work together (Robinson et al., 2015). However, they are two distinct, separable systems (Berridge, 1996).

Incentive salience wanting is a not necessarily conscious process (Berridge, 2009). Wanting is found to work within a larger dopamine-related mesolimbic circuit (Nguyen et al., 2021). Early research was done mostly on animal models and effort-based tasks, proposing the mesolimbic dopamine system, that innervates the ventral striatum, to be responsible for processing reward-relevant information and provide motivation for behavioral effort (Berridge & Kringelbach, 2015). Liking reactions are located in small so-called “hedonic hotspots”, which include the orbitofrontal cortex, insula, nucleus accumbens and ventral palladium (Nguyen et al., 2021). The NAcc is important and functions as a part of the neural circuit that responds to reward-associated cues and regulates cost-based decision making. It is crucial for accepting high costs and overcoming high efforts for obtaining rewards (Day et al., 2011) and assessing risks associated with the reward (Sugam et al., 2014).

The Reward System and Food Reward Processing in Anorexia Nervosa

Alterations in reward processing and its subcomponents have been associated with various psychiatric disorders (Clements et al., 2023; Treadway & Zald, 2013). For example, in depression rewards appear to lose incentive, while irrelevant stimuli are overvalued in schizophrenia, and in addiction salient and reward-predictive cues are

overvalued and can trigger compulsive behavior (Ryan & Skandali, 2016). Building on this, reward dysregulation can be distinguished between two profiles. Reward hyposensitivity, which relates to anhedonia, is seen in motivational deficits in depression. Hypersensitivity is the opposite and is present in symptoms of excessive motivation, as often seen in bipolar disorder (Nusslock & Alloy, 2017).

For anorexia nervosa, attempts to explain abnormalities in reward processing typically focus on anhedonia, which is observed in AN to a greater degree than in healthy controls – a finding that persists even after weight restoration (Dolan et al., 2022; O'Hara et al., 2015). However, the finding is not uniform across studies, as Pines et al. (2024) reported no reduced experience of pleasure in adolescents with AN when controlling for depression scores.

While reduced enjoyment is a useful clinical signal, it is not diagnostically specific. Treadway & Zald (2013) compare headache, which is a symptom rather than a cause, to lower reported pleasure, which is by itself also not indicating whether the dysfunction lies in motivational drive, hedonic capacity or downstream processes, such as valuation and learning.

A common way to assess hedonic components is through taste, including in individuals with AN. Beyond nourishment, food serves as a primary reward, which is represented in a food-specialised brain structure additionally to the core reward system (Alonso-Alonso, 2015; Sescousse et al., 2013). Food liking and wanting are two separable constructs (Jiang et al., 2015) and incentive-salience wanting for food relates to appetite (Berridge, 1996). Internal states modulate these processes: hunger typically enhances reward-sensitivity, while satiety reduces reward system sensitivity (Cassidy & Tong, 2017). High-caloric food is preferentially consumed in both animal and human studies, a preference that is not driven by subjective emotions. Instead, it seems like mesolimbic dopamine contributes to the promotion of conditioned behavior – which then usually results in the most rewarding and caloric intake (Saleem, 2016).

Further neuroimaging studies illustrate how metabolic state interacts with the energy content of food. The OFC is considered especially important, as regions in the OFC are associated with the processing of food related information and are central to understanding human eating behavior (Kringelbach et al., 2012). In healthy individuals, food deprivation increases activity in the OFC when a person is

presented with high calorie foods (Siep et al., 2009). By contrast, for AN individuals there is a lower connectivity between the OFC and the hypothalamus, which is central to feeding regulation (Frank et al., 2016). In the AN population, hunger fails to upregulate reward valuation circuitry, and instead elevated responses in cognitive control remain regardless of feeding state (Wierenga et al., 2015).

There are inconsistencies about the direction of the dysregulation of neural circuits related to reward processing across studies (Haynos et al., 2020). One proposal is that aspects of rewards may be “contaminated” by punishment in AN due to overlapping processing of reward and punishment (Keating, 2010). AN patient’s altered response in taste-reward tasks may rather reflect a fear of weight gain associated with the calories they’re supplied with in the study paradigm (Keating et al., 2012). Behaviorally, both acute and weight-restored AN participants have shown less implicit “wanting” for high-calorie foods – a pattern that is inverted from healthy controls (Cowdrey et al., 2013).

Illness stage and development may further shape reward circuitry in AN. A study using monetary rewards on recovered patients showed elevated dorsolateral prefrontal cortex (dlPFC) activity in the anticipation phase for a reward, and a stronger functional connectivity between the dlPFC and medial orbitofrontal cortex (mOFC) compared to controls, suggesting remitted AN patients to have a stronger engagement in self-regulatory processes (Ehrlich et al., 2015). For food-related tasks, Mendez-Torrijos et al. (2024) showed a weakened maturation of resting-state connectivity from young to adult AN patients, in comparison with HCs. While the resting-state network reactivity to high-caloric food, present in younger (<21 years) patients with AN, it decreases with age and is no longer present in adult patients with AN. The reason for that might be structural atrophy or functional changes that take place throughout the illness and do not normalize with remission (Boto et al., 2017; Scaife et al., 2017). Interestingly, women remitted from AN also do not adequately increase activation of reward valuation circuitry when hungry and instead show elevated activation in cognitive control (Wierenga et al., 2015).

Neuroimaging data relates a dysfunction in the fronto-striatal neural circuit, including the ACC, to altered reward processing in AN (Keating, 2010). Feeding-state independent findings of a greater activation in the food-cue paradigm (exposure to high-calorie food images) show up for following regions: dorsal anterior cingulate

cortex (dACC), dlPFC, hippocampus, caudate and putamen for the AN and atypical AN group vs HC (Eddy et al., 2023). Relatedly, elevated expected value signals in the ACC in response to food stimuli are hypothesized to be due to increased conflict-detection during reward processing contributing to food restriction in acute AN (Olsavsky et al., 2019).

A meta-analysis on task-related fMRI studies in patients with AN reported increased activity in the left superior temporal gyrus and bilateral ACC during reward-related tasks. They also found increased activity in the cerebellum, midcingulate cortex and right fusiform gyrus, but decreased activity in the left ACC in food-related tasks (Zhong et al., 2023). In more complex reward processing tasks, a fronto-striatal neural circuit dysfunction was found to be related to altered reward processing in AN (Keating et al., 2012). Increased activations in fronto-striatal circuits and striatal regions possibly support the overriding of hypothalamic inputs and are associated with the persistent restrictive eating habits in AN patients (Frank et al., 2016; Steward et al., 2018). Overall, these findings support a model in which patients with AN have an increased top-down control and executive abilities to inhibit motivational drives when it comes to motivationally salient cues (Ehrlich et al., 2015; Kaye et al., 2013).

Aims of This Study

While AN is a disorder with a homogenous phenotype, the course of the illness varies (Treasure et al., 2015). Obsessive-compulsive features frequently occur in eating disorders, and in AN their presence has been associated with a more severe and chronic illness (Cumella et al., 2007). This highlights the potential value of assessing and targeting OCD symptoms in addition to ED-focused treatment, as it seems to be beneficial for overall treatment outcomes (Bee et al., 2020).

In this context, the aim of the master's thesis was to clarify how obsessive-compulsive symptomatology and reward-related neural processing relate to one another across the trajectory of AN.

First, I wanted to characterize obsessive-compulsive symptom severity across illness stages. This has previously been researched by Wiedenmann (2025) for a smaller subset ($n = 121$) of the current sample ($n = 164$). Considering the relevance for the subsequent hypotheses and the completeness of the data this thesis has access to, posing an identical question is justified – on one hand in the context of this thesis, on the other hand as a replication check for Wiedenmann's research.

Second, I wanted to test whether neural activation during the consummatory phase of food reward processing differs between individuals with AN and healthy controls, and whether there is a difference between acutely ill individuals compared to partially or fully remitted ones. Lastly, I wanted to examine whether obsessive-compulsive symptom severity explains variability in activation during reward consumption, and whether the strength of a possible effect is different across participant groups.

Based on previous research and my research questions, I formulated a set of confirmatory hypotheses.

Hypotheses

OCI-R scores in AN level groups and HC

H1a: Obsessive-compulsive symptoms are higher in acutely ill AN patients than in patients in partial remission, full remission, and healthy controls.

H1b: Obsessive-compulsive symptoms are higher in AN patients in partial remission than in patients in full remission and healthy controls.

H1c: Obsessive-compulsive symptoms are higher in AN patients in full remission than in healthy controls.

Brain activity during food reward consumption

H2a: AN patients of all levels show greater alteration in brain activation during food reward consumption compared to HC.

H2b: Acute AN patients show a greater alteration in brain activation during food reward consumption compared to AN patients partially or fully in remission.

Role of obsessive-compulsive symptoms

H3a: OCI-R scores across all participants are associated with altered brain activation during food reward consumption.

H3b: The association between OCI-R scores and altered brain activation is different across all levels.

Methods

Study Design

The data used in this master's thesis is part of the "AN-BEL" project. The research project, involving the University of Vienna and the Medical University of Vienna, uses a translational psychiatric approach. The focus lies on social and non-social reward processing in Anorexia Nervosa. Various psychological assessments, facial electromyographic activity (fEMG), functional and structural MRI scans were conducted. Data was collected between February 2022 and November 2025. For my master thesis, only clinical data and functional MRI data was analyzed.

Considering that this thesis is part of a bigger project that has been going for four years, some of the hypotheses coincide with questions that have already been researched. However, I now had access to the full sample and posing the same questions was therefore justified to corroborate previous findings. Furthermore, this is the first thesis that focuses on obsessive-compulsive symptoms in the context of consummatory food reward processing. Hypotheses that are similar or identical to research that has already been done with a subset of this data have been marked as such and differences in the findings will be mentioned.

The present study used a cross-sectional design to compare obsessive-compulsive symptoms across different stages of anorexia nervosa and to examine brain activation during consummatory reward processing across AN of different illness stages and healthy controls.

Recruitment

Participants were recruited through the Department of Child and Adolescent Psychiatry at the Vienna General Hospital and through the distributions of flyers and information on the project on social media platforms and research participant platforms, as well as in-person at educational institutions.

Inclusion and Exclusion Criteria

Inclusion criteria were female sex, age 12-30 years, sufficient German language proficiency, written informed consent by the participant or, for minors (<18 years) by a legal guardian. Eligibility for the AN group required current or past AN diagnosis according to DSM-5 criteria. Patients were then assigned to subgroups according to illness status: acutely ill, partially remitted or fully remitted.

Partial remission was defined by a BMI > 18.5, no more restricted food intake or engagement in binge eating episodes or vomiting; but still had a fear of weight gain or body image disturbances. The total EDE score had to fall within two standard deviations of normative values. These criteria had to be maintained for at least three months.

Full remission required a BMI >19 or having gained 85% of ideal body weight, with no restrictive eating, binge eating and vomiting. Additionally, there needs to be an absence of fear of weight gain and body image disturbance. The total EDE score had to be within 1.5 standard deviations of normative values, and remission criteria had to be maintained for at least 12 months.

Healthy controls were required to have no current or lifetime psychiatric or neurological disorder. General exclusion criteria applied to all participants were intellectual disabilities, psychotic or bipolar disorders, organic psychosyndrome, cardiovascular diseases, lung disorders, metabolic disorders and pregnancy. MRI-related exclusions were claustrophobia and metallic implants, such as pacemakers.

179 participants were enrolled in the study. For analysis of clinical data, 8 participants were excluded due to dropping out or having an excluding psychiatric diagnosis. Additionally, 7 participants were excluded due to missing data on the OCI-R questionnaire. For fMRI analysis of hypotheses 2a and 2b, from 150 participants that took part in the scan, 10 participants were excluded due to missing scan data on the food trial parts of the experiment, technical issues or participants not completing the scanning procedure. Final sample sizes for each analysis are reported in the respective results sections. For fMRI analysis of hypotheses 3a and 3b, additional 3 participants were excluded due to missing OCI-R scores.

Variables

Groups

For the analyses a categorical group variable was created. The four groups included were AN patients with full syndrome (AI-AN), partial remission (PR-AN), full remission (FR-AN) and healthy controls (HC).

Eating Disorder Symptomatology

For the assessment of the current state of illness for AN-subgroup allocation through eating disorder symptoms the German version of the Eating Disorder Examination

(EDE; Hilbert & Tuschen-Caffier, 2016) was used. The EDE is a semi-structured clinical interview for assessing the specific ED psychopathology in children and adults. Four subscales are evaluated: Restraint, Eating Concern, Weight Concern, and Shape Concern. The Restraint subscale measures attempt to restrict food intake. The Eating Concern subscale assesses anxiety, guilt and preoccupation related to eating. The Weight Concern subscale reflects concerns and dissatisfaction regarding own body weight. The Shape Concern subscale measures worries and discomfort associated with body shape.

The EDE evaluates the frequency and severity of behaviors specific to eating disorders over the past 28 days. It uses a seven-point rating scale, from 0 (absence of ED-specific behavior) to 6 (daily occurrence or extreme severity). The interview provides subscale scores and a total EDE score, with higher values indicating greater psychopathology. The total score demonstrates good reliability, with a Cronbach's alpha of .93.

OCD Score

The Obsessive-Compulsive Inventory Revised (OCI-R) is a questionnaire used to assess the severity and nature of obsessive-compulsive symptoms through self-report (Foa et al., 2002). The symptoms are assessed on 6 subscales: washing, checking, ordering, obsessing, hoarding and neutralizing. It contains 18 items that are rated on a 5-point Likert scale from 0 (not at all) to 4 (extremely). The items each describe an obsessive-compulsive symptom and are rated according to the level of impairment and distress they caused for the past month. The total score ranges from 0 to 72, the subscale scores range from 0 to 12. A higher score sum represents higher obsessive-compulsive symptomatology. The OCI-R achieved good to excellent internal consistency. Cronbach's alpha ranged from .76 to .95 for the total score and most subscales, with only the Neutralizing subscale showing satisfactory reliability. The German version was used for the study. The factor structure of the original English version was confirmed in the German sample, as well as convergent and divergent validity, approving it as a reliable and valid measure of obsessive-compulsive symptoms (Gönner et al., 2007).

Neuroimaging Data

The neuroimaging data was preprocessed and optimized for subsequent analysis and a customizable script for further analysis of the fMRI data was provided by the

thesis supervisors. The first level analysis was then conducted on an individual basis. A contrast image was created for each subject, contrasting the blood-oxygenation-level-dependent (BOLD) response during the consumption of a reward with the BOLD response during the presentation of a fixation cross. Second-level analyses used said contrast images.

Experimental Design

This study is based on a recently developed and validated experimental paradigm by Chiappini et al. (2022). It was designed to assess anticipatory wanting and consummatory liking responses through two reward modalities: food rewards, delivered orally, and social rewards, delivered through touch. Both kinds of rewards vary systematically in intensity – there are three reward levels. For the food stimuli, the participants are administered with three types of milk: cocoa milk, sugared milk and a blend consisting of 25% cocoa milk and 75% sugared milk. Participants were previously informed that milk stimuli contained identical calories to ensure a focus on taste preference. In between the food deliveries participants get water to rinse their mouths and neutralize the most recent taste.

The clinical examination was the first phase of the experiment for the participants. Clinical psychologists assessed the participants with various clinical questionnaires, including the EDE interview and OCI-R self-report questionnaire.

For the second part of the study, participants received an introduction to the scanning process. Participants' maximum voluntary contraction was measured with a dynamometer, establishing a baseline for subsequent measurements. Participants were then exposed to all stimulus types and asked to rank them using a button box. Participants were asked to rank the stimuli by their personal preference. The ranking was then used to tailor the conditions to the participant during testing. Following the visual announcement of each reward, subjective ratings of wanting and liking were acquired to capture explicit reward responses. Implicit wanting was captured in the form of the force measured on a hand dynamometer and implicit liking through functional electromyography (fEMG). Functional magnetic resonance imaging (fMRI) was used to neural correlates of reward processing.

The current thesis specifically focuses on the actual consummatory (not-anticipatory) reward processing and relevant clinical data. Data related to social touch rewards

and anticipatory linking and wanting assessments will not be described or analyzed further.

fMRI Data Acquisition

MRI examinations were performed at the Center of Excellence High-field MRI of the Medical University of Vienna. A 3 Tesla Siemens Prisma MRI scanner (Siemens Medical, Erlangen, Germany) with a 64-channel head-neck coil was used. The participant's head was supported with No Motion Correction pads within the head coil. BOLD functional imaging was performed using a T2 weighted single-band echo planar imaging (EPI) sequence with the following parameters: Echo time = 36 ms, repetition time = 2.5 s, flip angle = 66°, 44 axial slices coplanar to the line connecting the anterior and posterior commissure, field-of-view = 140 x 140 x 132 mm, matrix size 96 x 96, voxel size = 1.45 x 1.45 x 3 mm, interslice gap = 3 mm.

Data Analysis

Statistical analysis of descriptive and clinical data was performed using R (R Core Team, 2025). Neuroimaging data processing was conducted using Statistical Parametric Mapping (SPM25; Tierney et al., 2025), which is a toolbox operated in MATLAB (MathWorks, 2024). Second-level analysis output coordinates were anatomically located using the SPM built-in Neuromorphometrics atlas. Coordinates are reported in Montreal Neurological Institute (MNI) space. ChatGPT (OpenAI, 2025) was used to assist with statistical analyses.

Demographics

Before hypothesis testing, analyses were done to explore demographic characteristics and clinical variables. Assumptions of normality and homogeneity of variances were assessed. Levene's test indicated heterogeneity of variances, $F(3, 160) = 3.70$, $p = .013$. Shapiro-Wilk tests showed a deviation from normality for three groups, HC ($W = 0.93$, $p = .002$), PR-AN ($W = 0.91$, $p = .006$) and FR-AN ($W = 0.89$, $p = .004$).

Statistical Analysis of Hypotheses 1a, 1b, and 1c

For H1a-c, group differences in obsessive-compulsive symptoms (OCI-R score) were assessed. A one-way analysis of variance (ANOVA) with the four clinical levels (HC, AI-AN, PR-AN, FR-AN) evaluated differences in OCI-R scores. Given the heterogeneity of variances, Welch's ANOVA was conducted. Planned Holm-adjusted

contrasts tested the a priori specified group differences in H1a-H1c. Post-hoc pairwise comparisons (Tukey HSD) and non-parametric Kruskal-Wallis test were then run. Effect sizes (Cohen's d) were reported for each pairwise comparison.

Statistical Analysis of Hypotheses 2a and 2b

Group differences in neural activation were tested with two-sided t-tests. Age was entered as a covariate. For H2a, all AN participants were compared against healthy controls. For H2b, a two-sided t-test compared acute AN against (all) remitted AN. Whole-brain analysis was performed in SPM25. The statistical threshold was set at $p < .05$, corrected for family-wise error (FWE) at the voxel level. Exploratory analyses at threshold $p > .001$ and $k > 5$ were conducted.

Statistical Analysis of Hypotheses 3a and 3b

For 3a, OCI-R total scores were entered as a predictor in a multiple regression model across all participants to test for positive and negative associations between obsessive-compulsive symptomatology and neural activation during food reward consumption. Again, age was entered as a covariate, statistical threshold was set at $p < .05$, corrected for FWE at the voxel level. Exploratory analyses at threshold $p > .001$ and $k > 5$ were conducted. For 3b, group differences in the same association were tested through a full factorial model with the four group levels, controlling for age. OCI-R scores were entered as a separate, group-specific covariate, and an OCI-activation slope was estimated within each group. An omnibus F-contrast was used to test whether these slopes differed across groups. T-contrasts were conducted to explore between-group slope differences, at threshold $p > .001$ and $k > 5$.

Results

Sample Characteristics

The number of participants for each analysis varied due to missing data across different measures.

For the analysis of clinical data for H1, the final sample included 164 participants – 33 AI-AN, 38 PR-AN, 31 FR-AN, and 62 HC. For the fMRI analyses of H2a and 2b, the final sample included 140 participants – 30 AI-AN, 36 PR-AN, 25 FR-AN, and 49 HC. For the fMRI analyses of H3a and H3b, the final sample included 137 participants, 27 AI-AN, 36 PR-AN, 25 FR-AN and 49 HC.

Exploratively, participants' demographics for age and BMI were examined. The values reported in Table 1 are based on the sample included in the analysis of H1.

Table 1

Means, Standard Deviations, and One-Way Analyses of Variance in Descriptive and Clinical Characteristics

Variable	HC (A) (<i>n</i> = 62)		FR-AN (B) (<i>n</i> = 31)		PR-AN (C) (<i>n</i> = 38)		AI-AN (D) (<i>n</i> = 33)		<i>F</i> (3, 160)
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Age (years)	19.6	(3.7)	21.1	(3.7)	20.6	(3.6)	17.3	(3.4)	6.97***
BMI (kg/m ²)	21.4	(2.5)	21.6	(2.9)	20.8	(2.4)	16.4	(2.0)	33.92***
OCI-R, total score	13.0	(8.5)	10.5	(8.8)	17.4	(11.3)	28.5	(14.5)	19.57***

Note. HC: healthy controls, FR-AN: fully remitted anorexia nervosa, PR-AN: partially remitted anorexia nervosa, AI-AN: acutely ill anorexia nervosa, BMI: Body Mass Index, OCI-R: Obsessive-Compulsive Inventory-Revised.

*** $p < .001$.

H1: Group Differences in Obsessive-Compulsive Symptoms

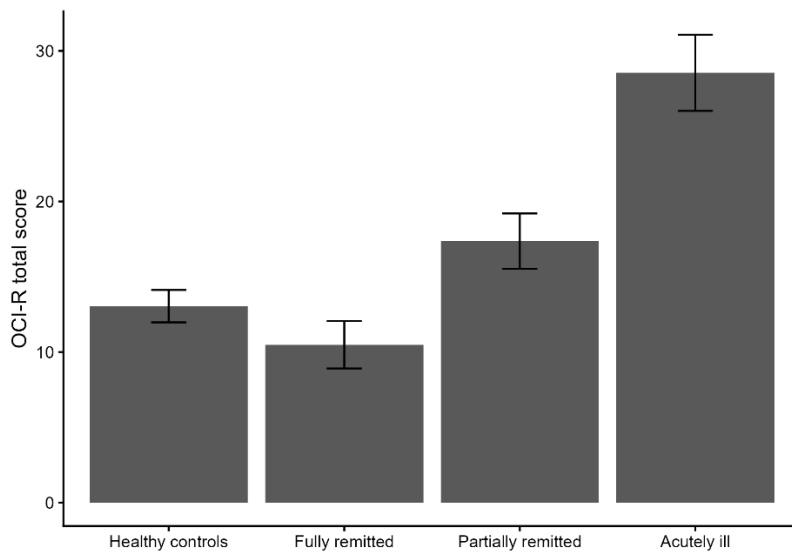
A one-way ANOVA indicated significant group differences in OCI-R total scores, $F(3, 160) = 19.57$, $p < .001$, with a large effect ($\eta^2 = .27$). Given unequal variances, a Welch's ANOVA was conducted as a robustness check and confirmed the omnibus group effect, $F(3, 73.60) = 13.62$, $p < .001$. A Kruskal–Wallis test further supported the presence of group differences, $\chi^2(3) = 36.04$, $p < .001$. Planned Holm-adjusted contrasts supported H1a: acutely ill AN patients had higher OCI-R scores than partially remitted AN ($p < .001$, $d = 1.05$), fully remitted AN ($p < .001$, $d = 1.69$), and healthy controls ($p < .001$, $d = 1.45$). H1b was partially supported: partially remitted AN exceeded fully remitted AN ($p = .025$, $d = 0.65$) for OCI-R scores, but not healthy controls ($p = .102$, $d = 0.41$). H1c was not supported, as fully remitted AN did not differ significantly from healthy controls ($p = .276$, $d = -0.24$).

Group differences are illustrated in Figure 1. Tukey's HSD showed that the acutely ill AN group scored higher than the partially remitted AN group ($p < .001$), fully remitted

AN group ($p < .001$), and healthy controls ($p < .001$). Partially remitted AN did not differ significantly from healthy controls ($p = .205$). Fully remitted AN did not differ from healthy controls ($p = .694$). OCI-R scores were highest in the acutely ill AN group ($M = 28.55$, $SD = 14.53$), followed by partially remitted AN ($M = 17.37$, $SD = 11.32$), healthy controls ($M = 13.05$, $SD = 8.47$), and the fully remitted AN group ($M = 10.48$, $SD = 8.77$).

Figure 1

Group Differences in OCI-R Scores Between Participant Groups



Note. Differences in obsessive-compulsive symptoms, measured by the Obsessive-Compulsive Inventory-Revised (OCI-R), between healthy controls, fully remitted, partial remitted, and acutely ill anorexia nervosa patients. Error bars represent 95% confidence intervals.

H2: Group Differences in Whole-Brain Activation

H2a: Brain activation differences between AN Subgroups and HC

Whole-brain second-level two-sample t-tests with voxel-wise family-wise error (FWE) $p < .05$ were conducted. No suprathreshold clusters were detected for either contrast direction (AN > HC; HC > AN), indicating that there are no significant whole-brain group differences in activation during consumption between the combined AN group and healthy controls in the present sample.

An exploratory analysis with uncorrected $p < .001$ revealed activations in the contrast HC > AN for three clusters ($k = 34$ and $k = 9$ and $k = 5$) and its peaks in the cerebral white matter, and activations in the contrast AN > HC for one clusters ($k = 9$) and its peak which could not be labelled, see Table 2.

H2b: Brain activation differences between AN Subgroups

Whole-brain second-level two-sample t-tests with voxel-wise FWE $p < .05$ were conducted. No suprathreshold clusters were detected for either contrast direction (AI-AN > FR-AN & PR-AN; FR-AN & PR-AN > AI-AN), indicating that there are no significant whole-brain group differences in activation during consumption between the combined remitted AN group and acute AN group in the present sample. An exploratory analysis with uncorrected $p < .001$ with an extent threshold of $k > 5$ revealed significant activations in both directions. In the contrast AI-AN > PR-AN & FR-AN it found two clusters ($k = 15$, $k = 8$) and its peaks, which could not be labelled by Neuromorphometrics. In the contrast PR-AN & FR-AN > AI-AN it found one cluster ($k = 16$) in the right cerebral white matter.

Table 2

Group Differences Between HC and All AN During Food Reward Consumption

Contrast	Brain region	k	$T(Z)^a$	x	y	z
HC > AN	No label	34	3.84 (3.73)	-50	-56	-52
	Left Cerebral White Matter	9	3.30 (3.23)	-18	-8	36
	Right Cerebral White Matter	5	3.23 (3.17)	30	-44	24
AN > HC	No label	9	3.54 (3.46)	6	54	-30

Note. Voxel-wise threshold $p < .001$ (uncorrected). No clusters survived FWE correction. Coordinates are reported in MNI space. k = cluster size.

^a of peak-level t-score (z-score)

Table 3

Group Differences Between Acute AN and Remitted AN During Food Reward Consumption

Contrast	Brain region	<i>k</i>	<i>T</i> (<i>Z</i>) ^a	<i>x</i>	<i>y</i>	<i>z</i>
AI-AN > PR-AN & FR-AN	No label	15	3.55 (3.46)	-56	-60	-36
	No label	8	3.37 (3.29)	-18	-22	-28
PR-AN & FR-AN > AI-AN	Right Cerebral White Matter	16	3.25 (3.18)	42	-44	18

Note. Voxel-wise threshold $p < .001$ (uncorrected). $k > 5$ is reported. No clusters survived FWE correction. Coordinates are reported in MNI space. k = cluster size.
^a of peak-level t-score (z-score)

A visual representation of found activations is presented in Appendix Figure A for contrasts HC > AN and AN > HC, Figure B for contrasts AI-AN > PR-AN & FR-AN and PR-AN & FR-AN > AI-AN.

H3: OCI-R Scores and Whole-Brain Activation

H3a: Association of OCI-R scores and brain activation across all participants

A whole-brain analysis testing for associations between OCI-R scores and brain activation revealed no suprathreshold clusters when applying a voxel-wise FWE correction threshold of $p < .05$. For a positive association (meaning higher OCI-R scores revealing greater activation) two small clusters showed up at an uncorrected threshold of $p > .001$, as seen in Table 4 and as a visual representation in Figure C in the Appendix.

Table 4

Positive Association between OCI-R scores and Brain Activation Across All Participants

Brain region	Cluster p_{FWE}	k	$T(Z)^a$	Peak p_{FWE}	x	y	z
Right Cerebral White Matter	.893	21	3.44 (3.36)	.836	2	4	-8
No label	.937	9	3.41 (3.33)	.860	18	-20	-40

Note. Voxel-wise threshold $p < .001$ (uncorrected). No clusters survived FWE correction. Coordinates are reported in MNI space. k = cluster size.

^a of peak-level t-score (z-score)

H3b: Association of OCI-R scores and brain activation across participant groups

The F-contrast, voxel-wise FWE-corrected at $p < .05$, revealed no significant interaction between participant group and obsessive-compulsive symptom severity. At an uncorrected threshold of $p > .001$, 12 clusters with $k > 5$ appeared, as reported in Table 5 and Figure 2. The biggest clusters appeared in the left middle occipital gyrus and the cerebral white matter.

T-contrasts at a threshold of $p < .001$ for between-group OCI-R slope differences revealed ten clusters in condition HC > AI-AN, see Table 6 and Figure 3. The biggest clusters showed up in the cerebral white matter, cerebellum white matter, and the right calcarine cortex.

T-contrasts at a threshold of $p < .001$ for between-group OCI-R slope differences showed nine clusters in condition FR-AN > PR-AN, see Table 7 and Figure 4. The biggest clusters appeared in the right cerebellum white matter and right cerebral white matter.

Table 5

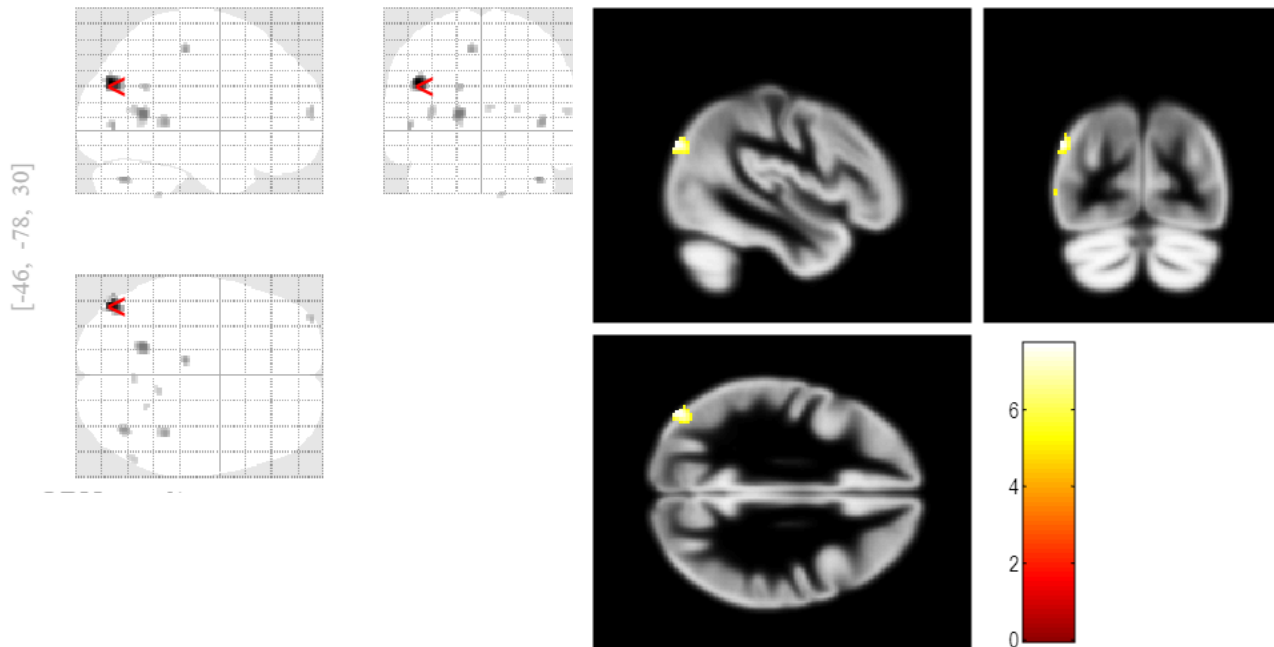
Interaction Between Participant Group and Obsessive-Compulsive Symptom Severity During Food Reward Consumption

Brain region	Cluster p_{FWE}	k	$F(4, 128)$	Peak p_{FWE}	x	y	z
Left middle occipital gyrus	.569	96	7.70	.176	-46	-78	30
Left Cerebral White Matter	.792	55	6.16	.748	-18	-56	10
Right Cerebellum White Matter	.965	17	5.75	.900	38	-68	-34
Left Cerebral White Matter	.968	16	5.73	.906	-8	-26	52
Right Cerebral White Matter	.909	32	5.70	.915	40	-40	4
Left inferior occipital gyrus	.988	7	5.59	.939	-52	-76	2
No label	.973	14	5.42	.969	-36	62	12
Left Cerebral White Matter	.984	9	5.39	.973	-16	-54	28
Brain Stem	.991	5	5.20	.989	12	-44	-44
Right Cuneus	.986	8	5.17	.991	4	-62	14
Right middle temporal gyrus	.980	11	5.14	.992	58	-62	12
Right Cerebral White Matter	.991	5	5.09	.994	22	-52	12

Note. Voxel-wise threshold $p < .001$. No clusters survived FWE correction. $k > 5$ is reported. Coordinates are reported in MNI space. k = cluster size.

Figure 2

Interaction Between Participant Group and Obsessive-Compulsive Symptom Severity During Food Reward Consumption



Note. Whole-brain results of the interaction between participant group and obsessive-compulsive symptom severity (OCI-R scores) on neural activation during consummatory food reward processing. The activation map displays regions in which the association between OCI-R scores and neural activation differed between groups. Voxel-wise threshold $p < .001$. Coordinates are reported in MNI space.

Table 6

Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (HC > AI-AN)

Brain region	Cluster p_{FWE}	k	$T(Z)^a$	Peak p_{FWE}	x	y	z
Left Cerebral White Matter	.482	134	4.04 (3.91)	.307	-6	-26	52
No label	.805	47	3.96 (3.84)	.372	-50	-64	-46
Right calcarine cortex	.692	75	3.81 (3.70)	.520	16	-62	4
No label	.841	38	3.80 (3.69)	.532	40	12	62

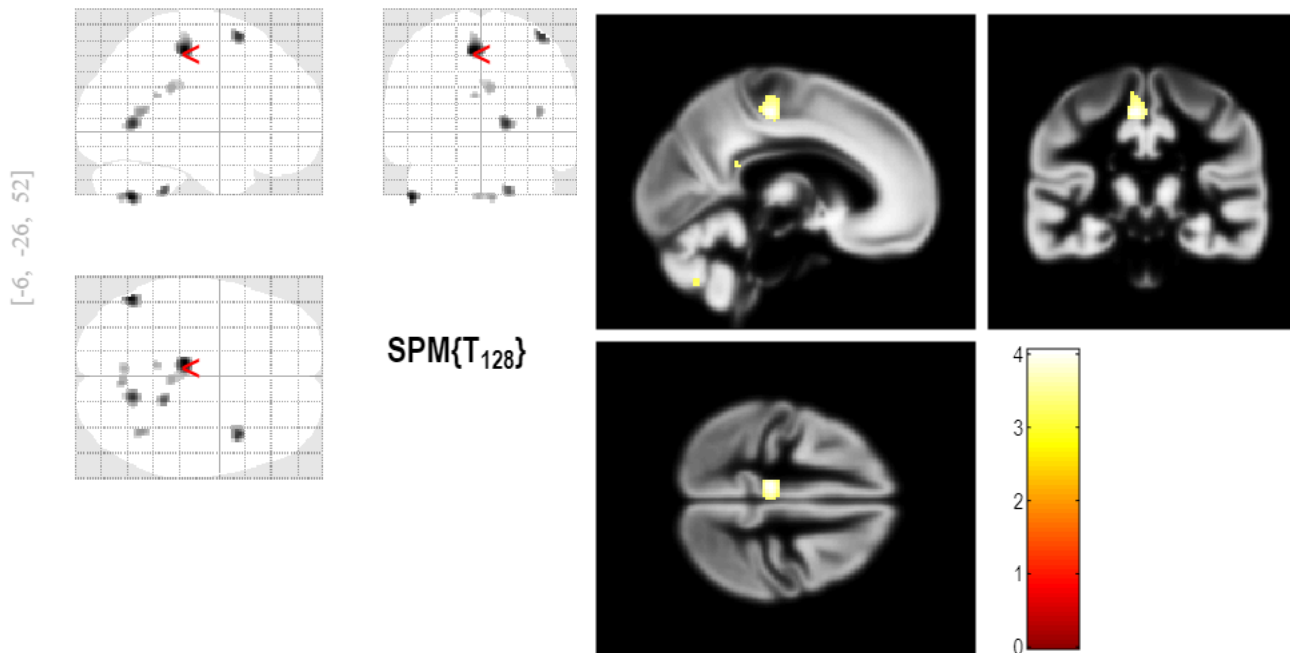
Brain region	Cluster p_{FWE}	k	$T(Z)^a$	Peak p_{FWE}	x	y	z
Right Cerebellum White Matter	.873	30	3.68 (3.58)	.655	18	-40	-42
Right Cerebral White Matter	.877	29	3.45 (3.37)	.860	38	-56	12
Right Cerebellum Exterior	.869	31	3.41 (3.33)	.888	6	-70	-46
Cerebellar Vermal Lobules VIII-X			3.35 (3.28)	.918	-4	-68	-46
Right posterior cingulate gyrus	.833	40	3.37 (3.29)	.909	4	-36	28
Left Cerebral White Matter	.959	6	3.32 (3.25)	.932	-6	-46	22

Note. Voxel-wise threshold $p < .001$. $k > 5$ is reported. Coordinates are reported in MNI space. k = cluster size.

^a of peak-level t-score (z-score)

Figure 3

Between-Group Differences in OCI-R- Activation Slopes During Food Reward Consumption (HC > AI-AN)



Note. Whole-brain results of the slope contrast comparing the association between obsessive-compulsive symptom severity (OCI-R scores) and neural activation during consummatory food reward processing, showing a stronger positive association between OCI-R scores and neural activation for HC than for AI-AN. Voxel-wise threshold $p < .001$. Coordinates are reported in MNI space.

Table 7

Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (FR-AN > PR-AN)

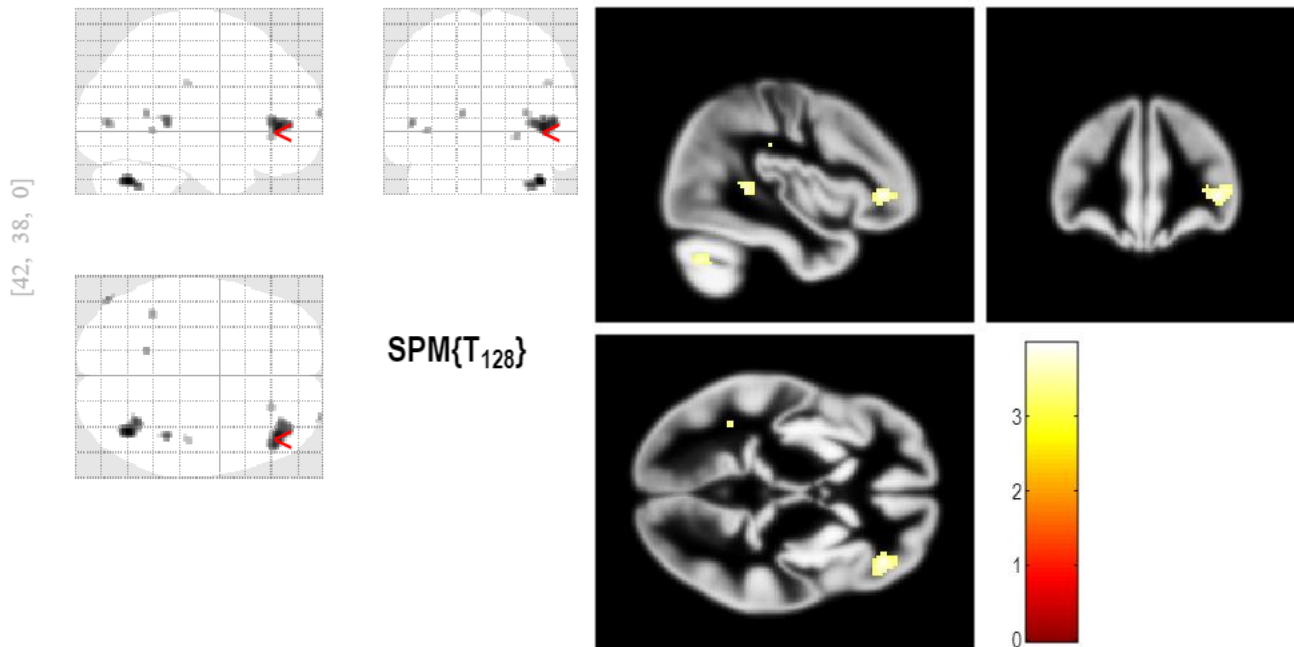
Brain region	Cluster p_{FWE}	k	$T(Z)^a$	Peak p_{FWE}	x	y	z
Right Cerebellum White Matter	.582	104	3.96 (3.84)	.369	38	-66	-34
			3.69 (3.59)	.642	34	-60	-38
Right Cerebral White Matter	.338	189	3.87 (3.75)	.520	42	38	0
Right Cerebral White Matter	.865	32	3.62 (3.53)	.532	42	-38	6
Left inferior occipital gyrus	.939	12	3.49 (3.40)	.655	-50	-80	4
Left Cerebral White Matter	.946	10	3.39 (3.31)	.860	-16	-52	10
Left Cerebral White Matter	.939	12	3.38 (3.31)	.888	-40	-48	-2
No label	.959	6	3.38 (3.30)	.918	30	68	10
Right Cerebral White Matter	.946	10	3.35 (3.27)	.909	22	34	-6
Right Cerebral White Matter	.949	9	3.28 (3.21)	.932	46	-22	30

Note. Voxel-wise threshold $p < .001$. $k > 5$ is reported. Coordinates are reported in MNI space. k = cluster size.

^a of peak-level t-score (z-score)

Figure 4

Between-Group Differences in OCI-R- Activation Slopes During Food Reward Consumption (FR-AN > PR-AN)



Note. Whole-brain results of the slope contrast comparing the association between obsessive-compulsive symptom severity (OCI-R scores) and neural activation during consummatory food reward processing, showing a stronger positive association between OCI-R scores and neural activation for FR-AN than for PR-AN. Voxel-wise threshold $p < .001$. Coordinates are reported in MNI space.

Discussion

The aim of this study was to explore neural correlates of consummatory food reward processing across different stages of anorexia nervosa and in healthy controls and how they relate to obsessive-compulsive symptoms. The study examined group differences in obsessive-compulsive symptom severity, group differences in neural activation during food reward consumption, and associations between obsessive-compulsive symptoms and neural activation, as well as its differences across illness stages.

Obsessive-Compulsive Symptoms Across Illness Stages

Consistent with H1a, acutely ill AN patients reported higher obsessive-compulsive symptoms, as measured by OCI-R, than partially remitted AN, fully remitted AN and healthy controls. Effect sizes for these differences were large, indicating that

obsessive-compulsive symptoms are elevated in the acute phase of the disorder. My finding aligns with previous studies that show heightened levels of obsession and compulsivity in acutely ill AN patients and a reduction of these symptoms with weight restoration and therapy targeting the eating disorder (Mattar et al. 2012; Meule & Voderholzer, 2022).

H1b was partially supported. Partially remitted AN patients reported higher obsessive-compulsive symptoms than fully remitted AN but did not differ from healthy controls. This is different from Wiedenmann's (2025) findings, which did not find significant differences in OCI-R scores between partially remitted and fully remitted AN groups.

The HC group even had slightly higher obsessive-compulsive symptom scores than the fully remitted AN group, however, the difference was not significant and H1c was not supported. These findings suggest that obsessive-compulsive symptoms are heightened during the acute phase of AN. Due to the cross-sectional design of my thesis, one cannot draw conclusions about a gradient in the severity of obsessive-compulsive symptoms throughout illness stages.

These results with the full dataset therefore mostly corroborate Wiedenmann's (2025) findings, except for the difference in the contrast of PR-AN and HC - a finding that was not present yet in the smaller subset of participants. The results are furthermore partially in accordance with previous research. In weight-restored AN, obsessive-compulsive symptoms, as well as other comorbid depressive and anxious symptoms, appear much improved (Mattar et al., 2012; Meule & Voderholzer, 2022). They have been found to be lower in long-term recovered AN patients than in those that are acutely ill, while still significantly higher than in healthy women (Holtkamp et al., 2005), or at the same level as in healthy women (Roberts et al., 2011). To my knowledge, there are no longitudinal studies that follow the same AN cohort from acute illness to long-term full remission with repeated assessments of obsessive-compulsive symptoms. Existing findings suggest a combination of state- and trait-like components, with obsessive-compulsive symptoms diminishing with weight restoration, yet often not fully reaching normal levels. In contrast, my study did not find significant differences between partially remitted AN and healthy controls, which supports the idea that obsessive-compulsive symptoms in AN are strongly influenced

by acute illness-related factors and suggesting a primarily state-dependent expression of obsessive-compulsive symptoms.

While both disorders are highly comorbid, the order of their appearance could be explained by distinct pathways in the course of the disorder. For example, individuals with OCD and anxiety may seek psychiatric help earlier, possibly reducing the likelihood of transitioning to AN. On the other hand, a unique connection between high birth weight and increased risk of subsequent OCD in AN population has been reported (Zhu et al., 2025). Longitudinal studies are rare, but one population-based study from Sweden showed that individuals first diagnosed with OCD had an increased risk (risk ratio, $RR = 3.6$) for a later diagnosis of AN, whereas individuals first diagnosed with AN had an even greater risk ($RR = 9.6$) of a later diagnosis of OCD, with a moderately high genetic correlation (Cederlöf et al., 2015).

Neural Activation During Consummatory Food Reward

Contrary to H2a and H2b, no significant whole-brain group differences in activation during food reward consumption were observed between the combined AN group and healthy controls, or between acutely ill AN and combined remitted AN, when applying voxel-wise FWE correction. Exploratory analysis at an uncorrected threshold ($p < .001$) indicated reduced activation in the left and right cerebral white matter in AN compared to healthy controls; and reduced activation in the right cerebral white matter in acute AN compared to both remitted groups combined; however, as these findings did not survive correction for multiple comparisons they must be interpreted with caution.

The absence of group differences seems inconsistent with most prior literature, which repeatedly reports altered neural responses to food and reward cues in AN, especially hyperactivation of fronto-striatal and cingulate regions and cognitive control networks (Ehrlich et al., 2015; Keating et al., 2012; Steward et al., 2018; Wierenga et al., 2015). The mentioned studies have focused on anticipatory or cue-related phases of reward processing, for example viewing food images or anticipating taste, and provide evidence that AN is characterized by alterations in anticipation and cognitive control around food cues (Jiang et al., 2019; Oberndorfer et al., 2013; Sanders et al., 2015; Wierenga et al., 2015). It is therefore plausible that group differences are stronger during anticipatory phases and decision-making, and less so during the consumption of controlled food stimuli inside the MRI scanner, as seen in

the present study, which only analyses the consummatory phase, therefore the actual receipt of the food reward.

Obsessive-Compulsive Symptoms and Neural Activation

H3a predicted an association between obsessive-compulsive symptom severity and neural activation during consumption of food rewards across all participants. This was not supported by the present data. Even though exploratory analyses at an uncorrected threshold revealed two small clusters, the effect did not survive correction for multiple comparisons. This suggests that, for the consummatory phase of food reward processing, obsessive-compulsive symptom severity is not related to any neural activation patterns across the sample.

Specifically in the eating-disorder context, differences in activation during anticipation are a very prominent finding (Yu et al., 2022). A meta-analysis of monetary reward tasks presented that anticipation robustly shows activation in the ventral striatum, while receipt shows a different pattern as a comparably weaker activation of the ventral striatum, and some activation in cortical regions that are not clearly defined as reward-related (Jauhar et al., 2021). Contrarily, Oldham et al. (2018) observed increased likelihood of OFC and ventromedial prefrontal cortex (vmPFC) and posterior cingulate cortex (PCC) activity for reward outcome but not anticipation. A meta-analysis by Gürsel et al. (2018) emphasized heterogeneity and a dysfunction at network-level for OCD patients, which could implicate a reduced likelihood of detecting a relationship between symptom level and neural activation in a mixed sample and a single task phase. Consistent with that, Uniacke et al. (2018) suggest that there may be a difference in obsessive-compulsive symptoms and OCD regarding their neural underpinnings, as they found no association between limbic CSTC connectivity and obsessive-compulsive symptoms in an AN population. There is a considerable possibility that symptom-related effects are more likely to emerge during other phases within a reward task paradigm, as other studies show that OCD-related reward abnormalities are evident during reward learning, prediction error signaling and decision making (Figeet et al., 2011; Hauser et al., 2017).

H3b predicted a difference in association between OCI-R scores and neural activation across groups, however the data did not support this. A full factorial analysis revealed no significant group-dependent OCI-R interaction in neural activation. Only when exploring the data at an uncorrected threshold of $<.001$ do we

see activation, with the biggest clusters located in the left middle occipital gyrus, left and right cerebral white matter and right cerebellum white matter.

Exploratory follow-up slope contrast (HC > AI-AN) at the uncorrected threshold of $p < .001$ showed differences between healthy controls and acute AN. In healthy controls, higher OCI-R scores were associated with increased activation in clusters in the left exterior cerebellum. In the contrast of partially remitted AN and fully remitted AN (FR-AN > PR-AN), higher activation slopes could be observed in fully remitted AN in the right cerebellum white matter and right cerebral white matter. Again, since these findings did not survive correction for multiple comparisons, they cannot be interpreted as robust effects.

Functionally, the core function of the left middle occipital gyrus is visual processing (Palejwala et al., 2020; Su et al., 2021). Cerebral and cerebellum white matter relates to global cognitive function (Baarsen et al., 2016; De Groot et al., 2001). Cerebellar structural and functional abnormalities have been linked to OCD (Yang et al., 2025) and functional connectivity between cerebellar and visual regions has been reported in OCD populations (Kashyap et al., 2021).

One possible interpretation of heightened relation of OCI-R scores and activation in acute and partially remitted AN patients is a greater heterogeneity in neural responses, which may obstruct a consistent relationship between OCI-R scores and neural activation, resulting in a pattern that differs from the one observed in healthy controls and fully remitted AN patients, respectively. Overall, my findings suggest that we do not see a uniform association between obsessive-compulsive symptoms and neural activation across the whole sample, and that we also cannot clearly state that the relationship between symptoms and neural activation may vary by illness stage. Given the limited statistical power, the uncorrected findings must be interpreted with caution and considered as preliminary evidence for future studies that should better assess the shift of neural correlates of obsessive-compulsive symptoms across illness stages.

Clinical and Theoretical Implications

A recent review of 14 studies on non-pharmacological treatment efficacy for comorbid AN and OCD showed no improvement of OCD symptoms, most of those treatments were tailored around AN (Steinhoff et al., 2025). For therapy forms targeting bulimia nervosa, Reilly et al. (2022) found improvement in ED related obsessive-compulsive

symptoms but not general obsessive-compulsive symptoms. At the same time, obsessive-compulsive symptomatology has been linked to poorer treatment outcome and a more chronic illness course (Cumella et al., 2007). OCD symptoms can be targeted just as effectively for individuals with a comorbid eating disorder, as in patients that do not have an eating disorder (Van Passel et al., 2025). Targeting obsessive-compulsive symptoms directly may be important when dealing with AN patients with comorbid OCD or subclinical presence of obsessive-compulsive symptomatology (Meule & Voderholzer, 2022; Simpson et al., 2013).

Clinically similar or identical symptoms can have different underlying conditions, therefore relying on diagnostics of symptoms alone is not always the optimal and only criteria basis. Brossollet et al. (2023) discuss how machine learning could be used to improve diagnostics by using biomarkers, such as through MRI, to support clinicians in diagnostics and treatment selection and to predict treatment response. Similarly, collective effort for bigger datasets and a research focus on assessment of reward processing circuitry and underlying mechanisms could bring forward understanding and therefore assessment and treatment of patients with anorexia nervosa and identify at-risk individuals for a very influential comorbidity in the form of OCD.

Limitations

There are several strengths and limitations to consider for interpreting the present findings.

Regarding sample demographics, only female participants were included. While it reflects the higher prevalence of AN in females, it limits generalizability to the male and gender-diverse population. The selection of the clinical sample mostly through a psychiatric population may not be fully representative of the whole AN population. It was not possible to consider different subtypes of AN for this study, such as the binge-purge subtype. Research suggests higher levels of impulsivity, obsessions and compulsions and a more severe course in binge-purging eating disorders compared to purely restrictive presentation (Di Luzio et al., 2024). Additionally, greater affected cognitive control systems in other eating disorders compared to AN have been found (Wonderlich et al., 2021). It would be of interest to consider different forms of AN and other EDs in further research.

The age range of participants included 12- to 30-year-olds, which is a wide developmental period from early adolescence to adulthood. Importantly, the acutely ill AN group was significantly younger than the fully remitted group, reflecting the requirement for fully remitted participants to be symptom-free and at normal weight for a minimum of 12 months, but also younger than the other groups taken together. Neurodevelopmental research shows that reward and cognitive control related systems mature at different rates across adolescence and young adulthood (Casey et al., 2008; Konrad et al., 2013). Ideally future research should have age-matched groups as to not limit statistical power by controlling for age.

The overall sample size for the fMRI analyses ($N = 137$ and $N = 140$) is relatively large compared to most task-based neuroimaging studies. For fMRI analyses, the sample size in each subgroup ranged from 25 to 49 participants. Even though the median neuroimaging study sample size is about 25, it may be too little to show robust effects, with reproducible findings requiring thousands of individuals (Marek et al., 2022; Turner et al., 2018). The analyses may have been too underpowered to detect small associations. The use of voxel-wise FWE correction provides control for false positives but also increases the risk of false negatives and making it difficult to detect subtle effects. This conservative approach may explain null findings for H2 and H3. Additionally, group sizes were unequal, with a much larger group of healthy controls and partially remitted AN patients, which may have further affected statistical power.

Another limitation is that no region-of-interest (ROI) analysis was conducted. While fronto-striatal and other reward-related regions were considered theoretically relevant, the interaction between reward processing in AN and obsessive-compulsive symptoms was less obvious. The decision for a whole-brain approach was based on providing broader coverage. Future studies could employ ROI analyses to increase sensitivity.

A task-related limitation is that the present thesis focused exclusively on the consummatory phase. The anticipatory phase, which is strongly linked to motivational “wanting” processes (Berridge, 2009), was not analyzed. Further research should examine both phases for a better understanding of reward processing alterations in AN and their association with obsessive-compulsive symptomatology.

Considerable strengths of the study are the inclusion of multiple illness stages (acutely ill, partially remitted, fully remitted, and healthy controls), which allows for broader view across illness stages and its specific presentation, rather than a simple patient and control contrast. Due to the cross-sectional design of the study, interpretations regarding causality cannot be made. However, the study might provide a basis for generating ideas about illness progression across different stages of anorexia nervosa.

Conclusion

The goal of my thesis was to shed light on the processes happening during food reward consumption for different illness stages in AN and how obsessive-compulsive symptoms relate to them.

The first finding was that OCI-R scores do differ between groups, being most elevated in the acute stage, followed by the partially remitted group, healthy controls and then fully remitted AN patients. This indicates that state-effects of the acute phase of illness play a considerable role in obsessive-compulsive symptomatology. From a clinical perspective this offers a basis for further integrating assessment and treatment of obsessive-compulsive symptoms in AN as part of a sustained and more holistic recovery.

Next, a whole-brain analysis of activation during consummatory food rewards was conducted, with no conclusive results. That may be due to the chosen time frame of consummatory reward consumption in the experiment for the analysis, as previous research offers consistent evidence for altered brain activation related to reward cues and reward anticipation, rather than consumption.

Lastly, I investigated the association of OCI-R scores and brain activation during food reward consumption across all participants, and how it may present differently for each group. Null findings for OCI-R specific brain activation across all participants can be interpreted as obsessive-compulsive symptom severity not being related to any neural activation for consummatory food reward processing. Participant group-dependent OCI-R activation was not found either. Therefore, based on our sample, one cannot conclude that the relationship between obsessive-compulsive symptoms and neural activation during consummatory food reward processing differs by illness stage. There is some remarkable activation seen under an uncorrected threshold,

however, those findings should be interpreted with caution. Future research should focus on obsessive-compulsive symptoms and their role in a possible shift in neural correlates across the course of illness regarding consummatory food reward processing, as well as further assessing the mechanisms for anticipatory food reward processing and reward processing of other stimuli.

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Appendix

List of Tables and Figures

Figure 1	Group Differences in OCI-R Scores Between Groups	p. 22
Figure 2	Interaction Between Participant Group and Obsessive-Compulsive Symptom Severity During Food Reward Consumption	p. 27
Figure 3	Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (HC > AI-AN)	p. 28
Figure 4	Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (FR-AN > PR-AN)	p. 30
Figure A	Higher Brain Activation for HC vs. All AN	p. 59
Figure B	Higher Brain Activation for AI-AN Group vs. PR-AN and FR-AN	p. 60
Figure C	Positive Association of OCI-R Score and Brain Activation Across All Participants	p. 61
Table 1	Descriptive and Clinical Characteristics	p. 21
Table 2	Group Differences Between HC and All AN During Food Reward Consumption	p. 23
Table 3	Group Differences Between Acute AN and Remitted AN During Food Reward Consumption	p. 24
Table 4	Positive Association Between OCI-R scores and Brain Activation Across All Participants	p. 25

Table 5	Interaction Between Participant Group and Obsessive-Compulsive Symptom Severity Association During Food Reward Consumption	p. 26
Table 6	Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (HC > AI-AN)	p. 27-28
Table 7	Between-Group Differences in OCI-R–Activation Slopes During Food Reward Consumption (FR-AN > PR-AN)	p. 29

List of Abbreviations

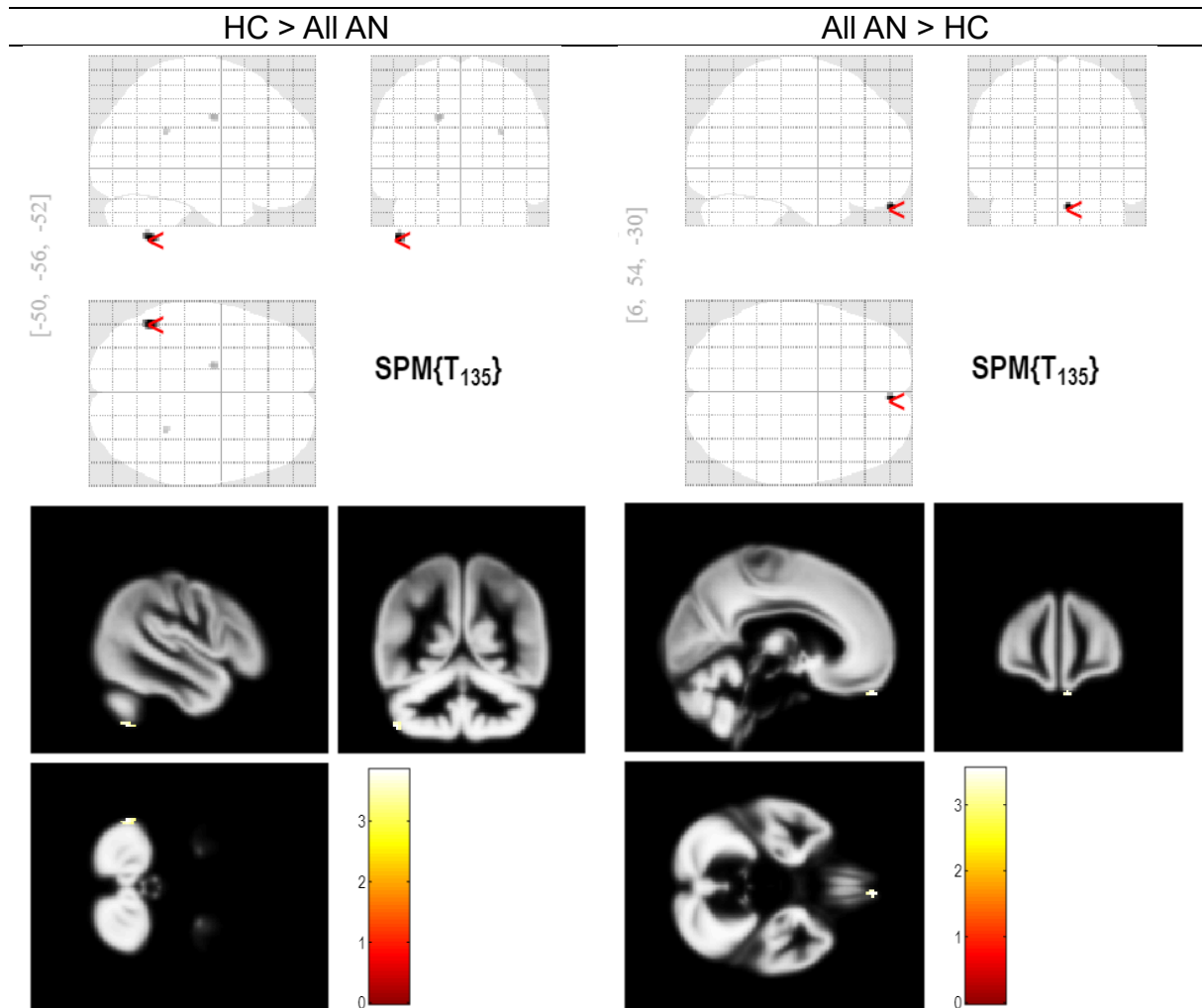
ACC	Anterior Cingulate Cortex
AI-AN	Acutely Ill Anorexia Nervosa
AN	Anorexia Nervosa
ANOVA	Analysis of Variance
BMI	Body Mass Index
BOLD	Blood-Oxygenation-Level-Dependent
CSTC	Cortico-Striatal-Thalamo-Cortical
dIPFC	Dorsolateral Prefrontal Cortex
ED	Eating Disorder
EDE	Eating Disorder Examination
fMRI	Functional Magnetic Resonance Imaging
FR-AN	Fully Remitted Anorexia Nervosa
FWE	Family-Wise Error Rate
GM	Gray Matter
HC	Healthy Control
HSD	Honestly Significant Difference
MNI	Montreal Neurological Institute
mOFC	Medial Orbitofrontal Cortex
MRI	Magnetic Resonance Imaging
NAcc	Nucleus Accumbens

OCD	Obsessive-Compulsive Disorder
OCI-R	Obsessive-Compulsive Inventory-Revised
OFC	Orbitofrontal Cortex
PFC	Prefrontal Cortex
PR-AN	Partially Remitted Anorexia Nervosa
ROI	Region Of Interest
RR	Risk Ratio
SPM	Statistical Parametric Mapping
vmPFC	Ventromedial Prefrontal Cortex
WM	White Matter

Supplementary Figures

Figure A

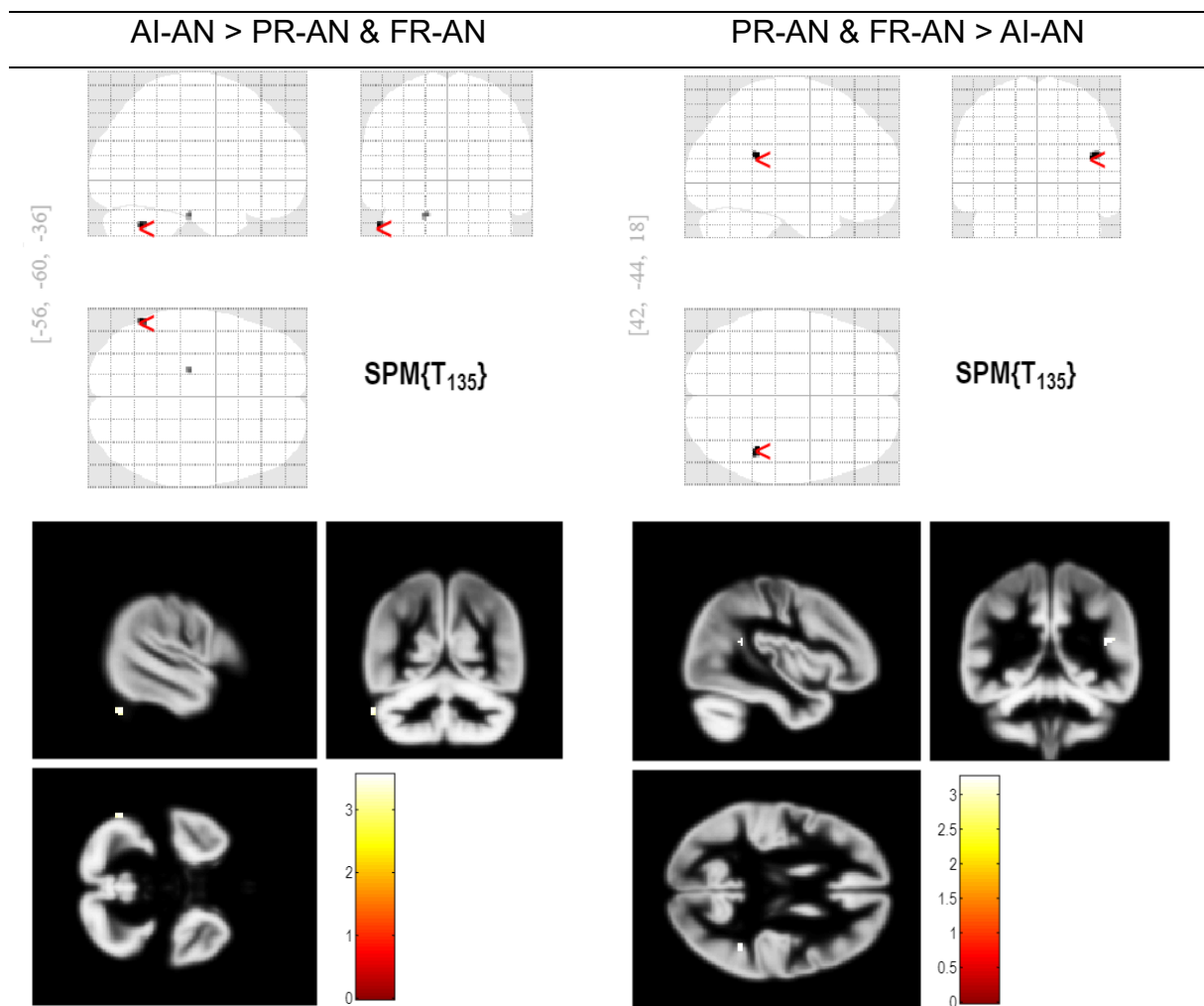
Higher Brain Activation for HC vs. All AN



Note. Whole-brain results for the contrast *HC > all AN* (left) and *all AN > HC* (right). Statistical parametric map thresholded at $p < .001$ (*uncorrected*) with a cluster extent threshold of $k \geq 5$ voxels. Results are displayed in MNI space.

Figure B

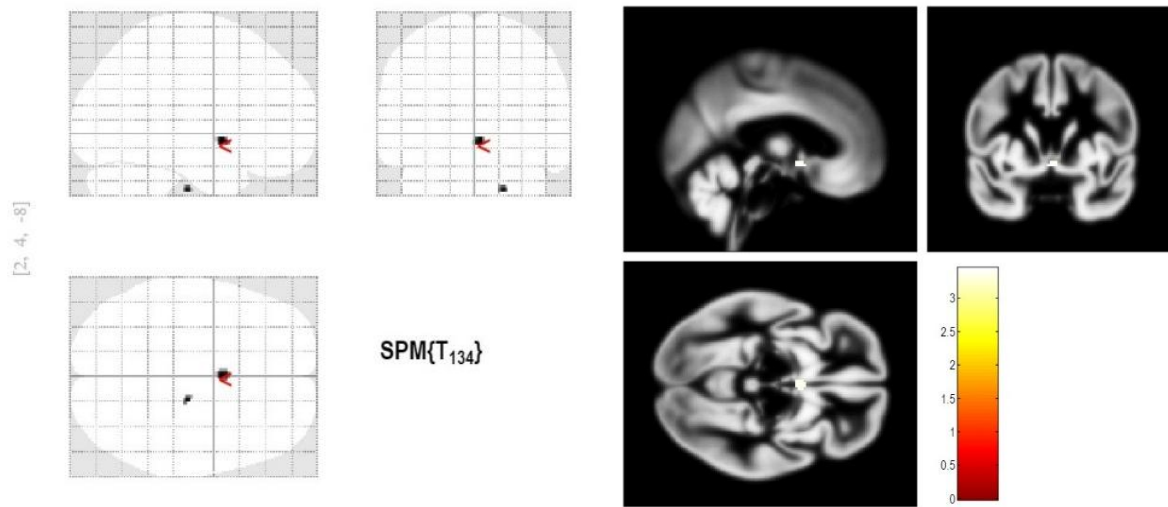
Higher Brain Activation for AI-AN Group vs. PR-AN and FR-AN



Note. Whole-brain results for the contrast *AI-AN > PR-AN and FR-AN* (left) and *PR-AN and FR-AN > AI-AN* (right). Statistical parametric map thresholded at $p < .001$ (uncorrected) with a cluster extent threshold of $k \geq 5$ voxels. Results are displayed in MNI space.

Figure C

Positive Association of OCI-R Score and Brain Activation Across All Participants



Note. Brain regions showing a positive association between OCI-R scores and neural activation during consummatory food reward processing across all participants. Statistical parametric map thresholded at $p < .001$ (*uncorrected*). Results are displayed in MNI space.

Abstract (DE)

Anorexia nervosa (AN) ist eine schwerwiegende psychische Störung, mit einer hohen Komorbidität mit Zwangsstörungen. Veränderungen in der Belohnungsverarbeitung wurden sowohl im Verlauf der Erkrankung als auch in der Remission gefunden. Diese Arbeit untersuchte, ob der Ausprägungsgrad zwanghafter Symptome Unterschiede in der Belohnungsverarbeitung zwischen AN-Patientinnen und gesunden Kontrollpersonen erklärt. Innerhalb eines validierten experimentellen Paradigmas wurden neuronale Belohnungsreaktionen auf Nahrungsreize mithilfe von funktioneller Magnetresonanztomographie (fMRT) erfasst. Die neuronale Aktivierung des gesamten Gehirns während der Konsumphase von Nahrungsbelohnungen wurde gemessen, aufbereitet und analysiert. Anschließend wurden Hypothesen zur moderierenden Rolle zwanghafter Symptome für die Belohnungsverarbeitung überprüft, wobei die Symptome anhand des Obsessive-Compulsive Inventory Revised (OCI-R) erhoben wurden.

Die Ergebnisse zeigen, dass sich die OCI-R Werte zwischen verschiedenen Krankheitsstadien der AN und gesunden Kontrollpersonen unterscheiden. Die höchsten Werte wurden bei akut erkrankten Patientinnen beobachtet, gefolgt von Patientinnen in partieller Remission, gesunden Kontrollpersonen und schließlich Patientinnen in voller Remission. Es konnte kein Zusammenhang zwischen OCI-R Werten und der neuronalen Verarbeitung beim Konsum von Nahrungsbelohnung gezeigt werden. Auch zwischen den Versuchspersonengruppen gab es keinen signifikanten Unterschied bezüglich des Einflusses der OCI-R Werte auf die neuronale Verarbeitung von Nahrungsbelohnung.

Im klinischen Setting ist das Erfassen und Behandeln von Zwangssymptomatik im Rahmen der Therapie von Anorexia Nervosa zu empfehlen. Durch weitere Untersuchung des Zusammenhangs zwischen zwanghaften Symptomen und Veränderungen in der Belohnungserwartung und -verarbeitung könnte ein vertieftes Verständnis über Verlauf und Persistenz von AN gewonnen werden.

Abstract (EN)

Anorexia nervosa (AN) is one of the most quality of life affecting and deadly psychiatric disorders, with a frequent comorbidity with obsessive-compulsive disorder (OCD). Dysfunctional reward processing has been discovered across illness stages. This thesis investigated whether obsessive-compulsive symptom severity explains differences in reward processing among female AN patients and healthy controls. Within a validated experimental paradigm neural reward responses to food stimuli were assessed using functional magnetic resonance imaging (fMRI). Whole-brain neural activation was measured, processed and analyzed for food reward consumption. Hypotheses regarding the moderating role of obsessive-compulsive symptoms, which was assessed using the Obsessive-Compulsive Inventory Revised (OCI-R), for reward processing were tested.

The main result was that OCI-R scores do differ between illness stages of AN and healthy controls, with highest scores in the acute stage, followed by partially remitted AN, healthy controls and then fully remitted AN patients. However, the connection between OCI-R scores and consummatory food reward processing in all participants could not be confirmed. Furthermore, there was no difference between participant groups regarding the impact of obsessive-compulsive symptoms on neural activation during consummatory food reward processing.

Clinicians are advised to consider assessing and treating obsessive-compulsive symptomatology in anorexia nervosa treatment. By further evaluating the connection between obsessive-compulsive symptoms and reward anticipation and reward processing alterations, understanding of the course and duration of AN might advance.