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Reconceptualizing and Renaming Eating Disorders as Anankastic Eating Disorders

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Abstract

Eating disorders is fundamentally an obsessive-compulsive disorder with eating-related symptoms, focusing on feeding, body shape, size and weight. Obsessive-compulsive personality disorder (OCDP) traits are commonly associated with eating disorders (EDs), with evidence indicated that these traits predispose and exacerbate the ED illness course. In any case, 'Anorexia' is not the central pathogenic feature in the so-named 'anorexia nervosa disorder'. These patients do not loss appetite; in fact, they restrict feeding. It is obvious that these patients suffer from a specific and severe form of an 'anankastic personality', having the aberration of restrict feeding, which indicate an 'anankastic eating disorder'. We need to rethink about ED from an obsessive-compulsive disorder (OCD) perspective, and reconceptualizing and renaming ED as 'Anankastic Eating Disorders'. Placing eating-related symptoms in an OCD/OCDP framework could help focusing more on reducing ED-associated compulsions and avoidance behaviors, as the primary treatment targets. This aspect will suggest, among others, on medication selection and dosage, such as high-dose and long period SSRIs, for targeting eating-related obsessive/compulsive symptoms. This will also encourage relevant research, e.g. within RDoC, having the potential to inform the development of a unified, dimensional, and biobehaviorally-grounded psychiatric nosology.

Keywords

Anorexia nervosa, anankastia, anankastic personality, eating disorder, Obsessive-compulsive personality disorder

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Let's start with anorexia nervosa

The case of 'anorexia nervosa' is a typical paradigm of a worldwide scientific misconception, having serious diagnostic and therapeutic consequences. For many decades 'anorexia nervosa', as indicate the label, is perceived as a disorder related to 'lack of orexis' (from the Greek 'orexis' for 'appetite' and 'anorexia' for the 'loss of appetite'), having until now serious problems with therapy.

Truth is that 'anorectic' individuals do not loss appetite. In fact, they just restrict feeding, by stirring food, separating the food to be eaten, counting bites, cutting food into geometric shapes, cutting food into small pieces, chewing for a long time, taking long breaks between bites, and eating extremely slowly. Patients have an intense 'sense of mastery / sense of control over world', leading to an 'over-control' over her body shape and weight. In this frame, patients develop a controlling behavior on eating, resulting in weight loss and body shape preoccupations **[1]**.

Most individuals with anorexia nervosa and bulimia nervosa will achieve long-term recovery. Rates of recovery from bulimia nervosa peak in the first decade of follow-up, while rates of recovery from anorexia nervosa continue to increase over 2 decades of follow-up **[2]**. DSM 5 also suggests to Specified for: "Full remission: After full criteria met, none of the criteria met for sustained period of time".

Having in mind the above findings, we may ask: Is anorexia nervosa a self-healed disease, starting at about 13 years old, and ending 10 or 20 years later? And what is happening in these patients after this period? Are they healthy? Do they suffer from any disorder? Don't they need any monitoring, supporting or treating during this "Full remission" period?

Truth is that these patients will continue to live in the same obsessive/compulsive realm. That's why we believe the diagnosis 'anorexia nervosa' is a virtual diagnosis. We all know these patients are not 'anorexic', but we still design criteria and therapies for anorexics. This explains our good diagnostic reliability, but the very bad content validity, in our clinical practice. And that puts a burden on patients.

We need to resolve the whole circle of misconceptions between patients, psychiatrists, other specialties, scientific institutions and journals, pharmaceutical companies, legal agencies, and media. We suggest a specific type of a life-long 'anankastic (eating) disorder', as the basic construction of the so-named 'anorexia nervosa', as analyzed below.

Obsessive-compulsive personality disorder and eating disorders

Traits characteristic of Obsessive-compulsive personality disorder (OCPD) such as perfectionism, inflexibility, rule-driven behaviours, and a need for order and symmetry, are prevalent within eating disorders (ED) samples (American Psychiatric Association, 2013). On the other hand, individuals with anorexia nervosa (AN) and bulimia nervosa (BN) display behaviours that are highly concordant with OCPD, such as perfectionism, rigidity, restricted affectivity, and a propensity to derive and adhere to rule-bound behaviours **[3]**.

OCPD is characterized by stable neurocognitive traits such as perfectionism, rigidity and a focus on detail, with associated behavioral tendencies such as behaviors aimed at achieving intra or inter-personal control, and miserliness that adversely affect psychosocial function and impair quality of life. The nosological status of OCPD remains uncertain, as it is noted to share diagnostic traits and clinical overlap with certain Obsessive Compulsive and Related Disorders, including OCD, body dysmorphic disorder and hoarding disorder, and with neurodevelopmental disorders such as Autism Spectrum Disorders.

New editions of the International Classification of Diseases – 11th Revision (ICD-11) are moving towards a dimensional model of PD asessment, which involves both an evaluation of PD severity (mild, moderate or severe) and of the presence of five stylistic trait domains: negative affectivity, detachment, disinhibition, dissociality and anankastia (WHO 2018). Notably, the DSM-5 definition of OCPD considerably overlaps with the ICD-11 proposed trait of "anankastia", which is described as rigid perfectionism, and emotional and behavioral constraints to ensure conformity to standards (WHO 2018).

OCDP traits are commonly associated with EDs, with evidence demonstrating that these traits predispose and exacerbate the ED illness course. Interpersonal distrust emerged as a possible bridging pathway connecting childhood OCPD traits to eating disorder symptoms **[4]**. While OCPD traits have been shown to confer risk for the development of EDs, OCPD traits and EDs have a complex interrelated relationship. OCPD traits, such as cognitive inflexibility and rigidity, may become intensified, which may serve to further maintain the ED. Therefore, OCPD traits, while evident prior to the onset of the ED, may become exacerbated during the acute illness phase of AN and BN, further maintaining the ED **[3, 5].**

Finally, different neurocognitive structures, as well physiological processes, may contribute to the development of OCD/OCPD behavior. Research showed that individuals with AN-restricting-type tended to have higher levels of ghrelin, a hormone responsible for signaling hunger. These increased ghrelin levels could lead to binging episodes, which could, in turn, be followed by a compensatory purging, eventually developing into a regular cycle and thus starting the transition to the binge/purge subtype **[6]**. Neuroimaging studies in OCPD indicate the involvement of specific neurocircuitry, including the precuneus and amygdala. However, according to Marincowitz et al (2022) **[7]**, although knowledge in this area has advanced, there are insufficient data on which to base a comprehensive model of the neurobiology of OCPD.

Treatment trials

Less than 10% of OCD patients are currently receiving evidence-based therapy. Cognitive Behavioral Therapy (CBT) with exposure and response prevention (E/RP) is one of the first-line evidence-based treatments for OCD. Indeed, several meta-analyses have found a significant reduction of OCD

symptoms after a psychotherapy including E/RP, with 40–50% of patients achieving symptom remission **[8]**. A review indicated a number needed to treat (NNT) of three for CBT and five for SSRIs, with the additional benefit of fewer side effects and relapses **[9]**. However, those results should be interpreted considering potential biases, such as the exclusion from the CBT trials of patients with comorbidities or the most severe cases of OCD. SSRI monotherapy is suggested as an option for patients with insufficient compliance for psychotherapy. Some meta-analyses suggest even longer waiting periods, showing a progressive improvement up to 30 weeks after the initiation of SSRI therapy **[10].**

Drugs are widely prescribed for anorexia nervosa (AN) in the nutritional, somatic, and psychiatric fields. Meta-analysis of Blanchet et al (2019) **[11],** described some small sized (12-44 subjects) and short duration (12-54 weeks) trials with selective serotonin reuptake inhibitors (SSRIs) treatment, having controversial results. Most of them improved depressive symptoms, perceptions of ineffectiveness, lack of interoceptive awareness, and perfectionism, but not weight gain. The inconsistent evidence on the effectiveness of antidepressants in treating AN patients, necessitates the need for additional studies with a larger sample size and longer follow-up duration.

AN is a complex disease caused by multiple factors and evaluating its long-term prognosis is crucial. Chiu et al (2023) **[12]** assessed the BMI course trend between different medications and timepoints, in order to improve AN treatment in clinical practice. Their study provided insights and highlights three key findings: 1) medication adherence is crucial in treating AN, 2) frequent switching of medications may not promote weight gain and may also require a re-establishment of rap- port with patients with AN, and 3) pharmacotherapy, especially antidepressants, is more effective than no treatment.

Recently, Gradl-Dietsch et al (2023) **[13]** reported that an off-label treatment of a 15-year-old female patient with anorexia nervosa (AN) with human recombinant leptin (metreleptin) for nine days was associated with self-reported increments of appetite and hunger, resulting in rapid weight gain and substantial improvement of eating disorder cognitions and of depression. According to DSM, intense fear of gaining weight in AN patients, may indicate BMI is a key measure of treatment outcome of AN.

However, is this conceptualization in the right direction? Can really weight gain improve AN patients? Or, can weight gain reduce patient's obsessive/compulsive eating behavior, giving an overall improvement? This way of thinking is the result of a scientific misconception, believing that 'anorexia' or 'weight gain' are the central features of eating disorders. This surely result in the design and production of incorrect psychotherapeutic techniques or drugs. And perhaps this is one of the reasons, if not the most important, that patients with eating disorders have such poor therapeutic results.

The clinical utility of the ICD-11 PD model with regards to anankastia

According to DSM-5, AN is characterized by a restriction of energy intake, intense fear of gaining weight, and a disturbance in the way in which one's body weight or shape is experienced. The two subtypes of AN are *restricting type* and *binge-eating/purging type*. The restricting subtype is associated with an earlier age of onset, a better prognosis, and a greater possibility of crossover to the other subtype. The binge-eating/purging subtype exhibits higher levels of core eating disorder (ED) psychopathology such as dietary restraint, eating concern, and shape/weight concerns.

On the other hand, according to the DSM-5, OCD is characterized by the presence of obsessions and/or compulsions. Obsessions are recurrent and persistent thoughts, urges, or images that are experienced as intrusive and unwanted, whereas compulsions are repetitive behaviors or mental acts that an individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.

OCPD is a common mental health problem that is still relatively under-recognized and lacks empirical investigations. The review of Gecaite-Stonciene et al (2021) **[14]** suggests that the ICD-11 PD model is a diagnostically valid and clinically useful approach to OCPD. Specifically, the ICD-11 *anankastia* domain overlaps with DSM5 OCPD traits, with factor analyses of the ICD-11 PD model further supporting the diagnostic validity of this domain. There is some support for the clinical utility of the ICD-11 PD model with regards to *anankastia*. Empirical findings generally support the conceptual considerations that the ICD-11 model more comprehensively covered the area of personality pathology than the DSM-5 model, with Anankastia revealed as a more specific domain of personality disorders, as well as more cohesively located within the overall personality structure, in comparison to Psychoticism **[15]**.

It has been proposed eating disorders are a variant of Obsessive-Compulsive Disorder (OCD) **[16]**. Both BN and AN-binge purge (AN-BP) subtype demonstrating deficits in inhibitory control. Moreover, obsessive/compulsive symptomatology and/or obsessive-compulsive personality traits contribute greater complexity to individual cases and may make these patients more resistant to treatment **[5]**. Genetic studies found no shared causative factor for OCD and eating disorders within families, but OCPD traits are specific familial risk factor for anorexia nervosa **[17]**.

Thinking about Eating Disorders from an Anankastic perspective

Individuals with OCD recorded higher *desire for control* and lower *sense of control*, relative to healthy individuals, and a higher desire for control than the anxiety group, suggesting some specificity to OCD **[18]**. Across eating disorders, *loss of control eating* includes both subjective and objective binge episodes and is associated with psychopathology. Forney et al

(2014) **[19]** suggested that loss of control eating is a clinically significant feature in Purging Disorder (PD). Loss of control eating is associated with disinhibition, hunger, depressive symptoms, negative urgency, distress and impairment above and beyond the effects of age, BMI, and purging frequency in PD. Given these associations, loss of control eating within PD appears to be a marker of severity. Thus, assessing frequency of loss of control eating in PD would provide highly relevant information in clinical settings and may be considered for inclusion in defining PD in diagnostic systems.

We propose renaming of Anorexia Nervosa (AN) in *Over-controlled Eating Disorder* or *Restricting type Eating Disorder*. Here, the OCD/OCPD traits of the need to control things, to self-control, to control body weight, and the compulsions to sustain a just-right appearance of particular body parts, reflect the ED symptomatology of restriction of energy intake relative to requirements, leading to a significantly low body weight. Patients severely limit their diet or food intake, though sometimes they use other methods to limit the impact of food on their bodies, including laxative and enema misuse and excessive exercise. This leads to malnutrition and a host of related health problems. In parallel, patients are controlled to hide their strong emotions, claiming they are not angry, living in a chronic emptiness. (Table 1).

On the other hand, we propose the renaming of Binge-eating Disorder in *Loss of control Eating Disorder*, or *Disinhibited Eating Disorder* or *Cyclical Eating Disorder*. Here, the OCD/OCPD traits of compulsive behavior to minimize situational distress reflects the lack of control over eating, resulting in eating much more rapidly than normal, until feeling uncomfortably full, eating large amounts of food when not feeling physically hungry, or eating alone. Some patients move from restrictive anorexia nervosa to the binge/purge type, by binge eating, characterized by various purging methods, such as self-induced vomiting, as compensatory behavior. We also consider Bulimia Nervosa as a Loss of control Eating Disorder, since the OCD/OCPD traits of disgust, distress, and fear of weight gain, in combination with the compulsive/impulsive behavior to minimize distress, leads to an ED symptomatology, characterized by a lack of control over eating during the episode. This is accompanied with recurrent inappropriate compensatory behaviors, in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, or other medications, fasting, or excessive exercise.

Table 1. Eating Disorders (ED) vs Anankastic Eating Disorders (AED). Domains, characteristics and main derivations.

Eating Disorders (ED) (DSM 5 / ICD 11)	Anorexia Nervosa	Binge-eating Disorder, or Binge-eating/ purging type
ED derivations	Intense fear of gaining weight, Restriction of energy intake relative to requirements, leading to a significantly low body weight	Periods of binge eating, followed by the use of various purging methods, such as self-induced vomiting, as compensatory behavior
ED taxonomy	Anorexia Nervosa (AN)	• Bulimia Nervosa (BN) • Binge Eating Disorder (BED) • Other Specified Feeding and Eating Disorder (OSFED) • Avoidant/Restrictive Food Intake Disorder (ARFID) • Unspecified Feeding or Eating Disorder (UFED)
Anankastic Eating Disorders (AED) (renaming proposals)	**Over-controlled Eating Disorder, or Restricting type Eating Disorder**	**Loss of control Eating Disorder, or Disinhibited Eating Disorder or Cyclical Eating Disorder**
OCD derivations	A need to control things, A need to self-control, A need to control her body, A need to control her weight	Breaking down the internal tendency to control things and oneself
OCPD derivations	Compulsions to maintain weight gain and sustain a just-right appearance of particular body parts	Compulsive behavior to minimize situational distress
Treatment	SSRIs, Physiological and Diet monitoring, Psychological support	SSRIs, Physiological and Diet monitoring, Psychological support

Epilogue

We need to think about ED from an OCD/OCPD perspective. Whether ED is ever reclassified as OCD or a related disorder in the DSM/ICD awaits future research, but the evidence is hard to ignore. Placing eating-related symptoms in an OCD/OCDP framework could serve to focus more on reducing ED-associated rituals, compulsions and avoidance behaviors, as the primary treatment targets. This model will help patient's tolerance of distress and uncertainty, instead of trying to explain or stop

directly obsessions. This aspect will also inform on medication selection and dosage for targeting eating-related obsessive and compulsive symptoms, e.g., high-dose and long period SSRIs, and encourage relevant research **[20]**.

With the accumulation of so much neuroscientific knowledge we rather need new models of understanding mental illness. Grounded in clinical neuroscience and guided by brain modeling, psychiatry can improve both assessment and treatment strategies **[21,22]**. When even more scientific evidence will be gathering, we could be in a position to study 'Anankastic Eating Disorders' in a dimensional framework, according to the Research Domain Criteria (RDoC) or/and the Hierarchical Taxonomy of Psychopathology (HiTOP), which have the potential to inform the development of a unified, dimensional, and biobehaviorally-grounded psychiatric nosology **[23]**.

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