Orthorexia Nervosa: an Emerging Eating Disorder with Deep Psychological Origins

Abstract
Orthorexia nervosa (ON) refers to an obsession with the quantity and quality of food ingested. While altering one’s diet is a common treatment to improve health, individuals diagnosed with ON obsess over their diet. Where’s more, individuals plagued by orthorexia nervosa feel badly about themselves if they fail to adhere to their ideal nutritional guidelines. Although little research has been performed to properly diagnose people with ON, this manuscript aims to shed light on the developing epidemic and will be the first publication, to our knowledge, to compile a list of psychological signatures that may help future clinicians diagnose this emerging eating disorder.

Keywords: Orthorexia nervosa; Diets; Psychological disorder

Abbreviations: ON: Orthorexia Nervosa; OCD: Obsessive-Compulsive Disorder; OCPD: Obsessive-Compulsive Personality Disorder

Introduction
Orthorexia nervosa (ON) is a complex and serious motivated behavioral condition with high morbidity and mortality [1]. Characterized by self-starvation, excessive exercise, morbid eating restraint, and overvalued fear of unhealthy eating, ON has a driven nature and bears phenomenological similarity to the addictions [2]. Dysregulation in the neurological circuitry underlying anxiety and reward, as well as environmental induced disturbances within homoeostatic epicenters, are implicated in the etiology and maintenance of ON [3-7]. Most of these physiological changes are state-related and reversible; however, growing evidence suggests some feed forward, contributing to entrainment and maintenance of excessive food restriction and exercise behaviors. Interesting most cases of ON occur in females [8-10]. Although little is known, we speculate that the relatively narrow peripubertal window of onset [7] and marked female preponderance suggest a role for estrogen in facilitating development of the disorder. The mechanism for this relationship may pertain to estrogen’s regulation of gene transcription within neurotransmitter systems relevant to food reward, stress vulnerability or anxiety and is likely to differ in new onset and chronic cases. Trait anxiety has been linked to risk for eating disorders [11,12], to predisposition to anxiety disorders [13] and to impaired fear conditioning, fear extinction or safety learning [14-17]. Impaired fear conditioning has in turn, been proposed as a potential mechanism to explain the rapid development and persistence of calorie restriction and fear of unhealthy foods in ON [18].

Despite ON’s high morbidity and mortality, effective treatments are limited, with the best evidence supporting family-based therapy for adolescent ON [19,20]. Better prognosis in this age group is likely due in part to a higher percentage of milder self-limited cases. In chronic adult ON, by comparison, no randomized controlled treatment trial has demonstrated efficacy in achieving remission [21,22]. Furthermore, six month relapse rates following inpatient weight restoration are close to 50% [23] and [24]. Once established, restrictive eating patterns are hard to extinguish and tend to persist [25], and limited dietary variety at discharge in weight-restored patients has been found to predict relapse 1-year post-hospitalization [26]. Surprisingly, however, little research has focused on interventions targeted at relapse prevention in ON following weight restoration.

This review will focus on the relationship between ON and psychological cues for clinical diagnosis (Figure 1). In addition, this review will take a translational approach into addressing the pathophysiology underlying ON and its maintenance, and the potential development of improved treatments for this condition.

Results and Discussion
Successful treatment in addiction involves prevention of a rewarding outcome and abstinence from the drug of abuse, whereas in anxiety disorders treatment requires removal of an aversive outcome and fear extinction. The successful treatment of ON may well require both interruption of excessive exercise and dietary restraint, as well as extinction of the heightened anxiety and meal avoidant behaviors that characterize the disorder. People suffering from ON commonly display the following signs:

a. Prolonged contemplation of healthy food choices (>3 hr/day)
b. Planning advanced menus
c. Continually limiting the number of allowed foods
d. Experiencing a reduced quality of life, social isolation, and depression
e. Critical of other diets
f. Overcome with guilt or self-loathing when they fail at dieting

In this review, we have synthesized existing research to identify what is known about the symptoms, prevalence,
neuropsychological profile, and treatment of ON. An examination of diagnostic boundaries reveals important points of symptom overlap between ON, obsessive-compulsive disorder (OCD), obsessive-compulsive personality disorder (OCPD), somatic symptom disorder, illness anxiety disorder, and psychotic spectrum disorders. Neuropsychological data suggest that ON symptoms are independently associated with key facets of executive dysfunction for which some of these conditions already overlap. Discussion of cognitive weaknesses in set-shifting, external attention, and working memory highlights the value of continued research to identify intermediate, transdiagnostic endophenotypes for insight into the neuropathogenesis of orthorexia. An evaluation of current orthorexia measures indicates a need for further psychometric development to ensure that subsequent research has access to reliable and valid assessment tools. Optimized assessment will not only permit a clearer understanding of prevalence rates, psychosocial risk factors, and comorbid psychopathology but will also be needed to index intervention effectiveness. Though the field lacks data on therapeutic outcomes, current best practices suggest that orthorexia can successfully be treated with a combination of cognitive-behavioral therapy, psychoeducation, and medication.

Conclusion

Recent research in the fields of the neuroscience of learning and psychology of addiction has informed our understanding of ON as a motivated behavioral disorder. Both anxiety disorders and an anxious temperament have long been recognized as highly comorbid with ON; however, only recently has research sought to address the mechanisms underlying this relationship. Work in the neuroscience of learning, fear conditioning, and animal models of ON is contributing to a new understanding of the complex etiology and maintenance of this disorder. A major challenge in severe and chronic ON is the development of effective relapse prevention interventions following hospital-based diet restoration. Treatments targeting anxiety and fear extinction in recently diet restored patients may therefore be of promise. The successful generalization of normal eating behavior and food choice across social settings is likely to be especially important in helping chronically ill patients recover from this serious and debilitating disorder.

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References