



How extreme dieting becomes compulsive: A novel hypothesis for the role of anxiety in the development and maintenance of anorexia nervosa



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A B S T R A C T

The US National Institute of Mental Health's Research Domain Criteria (NIMH RDoC) advocates the study of features common to psychiatric conditions. This transdiagnostic approach has recently been adopted into the study of anorexia nervosa (AN), an illness that can be considered compulsive in nature. This has led to the development of an account of AN that identifies key roles for the heightened reinforcement of starvation, leading to its excessive repetition, and goal-directed system dysfunction. Considering models of illness in other compulsive disorders, we extend the existing account to explain the emergence of reinforcement and goal-directed system abnormalities in AN, proposing that anxiety is central to both processes. As such we emphasise the particular importance of the anxiolytic effects of starvation, over other reinforcing outcomes, in encouraging the continuation of starvation within a model that proposes a number of mechanisms by which anxiety operates in the development and maintenance of AN. We suggest the psychopathology of AN mediates the relationship between the anxiolytic effects of starvation and excessive repetition of starvation, and that compulsive starvation has reciprocal effects on its determinants. We thus account for the emergence of symptoms of AN other than compulsive starvation, and for the relationship between different features of the disorder. By extending and adapting an existing explanation of AN, we provide a richer aetiological model that invites new research questions and could inform novel approaches to prevention and treatment.

Introduction

Anorexia nervosa (AN) is a mental illness whereby a dangerously low body weight is maintained by extreme dietary restriction [1]. The abnormal eating behaviour that is central to AN [2] persists despite its adverse effects on daily and social functioning [3], and physical health [4]. AN affects approximately 1–2% of Western populations, and has the highest mortality rate of any psychiatric illness, this figure approaching 6.0% [5].

AN tends to be chronic, with less than 50% of individuals who develop the illness making a full recovery [6,7]. It is suggested that current pharmacological and psychological therapies cannot address the neurobiological factors or mechanisms responsible for illness development and maintenance because it is unclear what these are [8]. To better understand the aetiology of psychiatric illnesses, Research Domain Criteria (RDoC), resulting from the National Institute of Mental Health (NIMH) 2008 strategic plan [9], encourages a transdiagnostic approach [10,11]. Central to this approach is investigating the causes of

features common to a number of disorders, rather than the causes of symptoms specific to discrete diagnostic categories [12]. Studying the characteristics that AN shares with other psychiatric disorders can allow new and testable theories of AN aetiology to be developed [13]. Potentially causal neural abnormalities that have not previously been considered in aetiological models of AN can be highlighted using this transdiagnostic approach [14].

Compulsivity has been identified as a transdiagnostic trait that is central to obsessive-compulsive disorders and substance and behavioural addictions. Compulsivity describes a tendency to engage in repetitive and stereotyped acts that have unwanted outcomes [15], and arises from a reduced ability to control inflexible yet maladaptive behaviour [16]. Recently compulsive behaviour has been characterised as an imbalance between the influence of the goal-directed system (the ventral medial prefrontal cortex; vmPFC) and the habit system (the dorsal striatum; [17,18]). The habit system guides behaviour based on past outcomes of actions, due to the formation of stimulus-response (S-R) links, a process that occurs when a response produces a favourable

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outcome. S-R links strengthen with behavioural repetition and their establishment allows stimuli to initiate the responses they are paired with automatically, even when these responses are inappropriate [19]. In contrast, the goal-directed system considers predicted outcomes of various actions, and the present value of these outcomes, to elicit behaviour tailored to the current situation [20]. It is suggested that compulsive behaviour arises from a failure of the goal-directed system to override the influence of the habit system when the latter produces maladaptive responses [21].

Steinglass and Walsh [22] and Walsh [2] proposed that the extreme restriction of food intake that epitomises AN is habitual. Later Park and colleagues proposed this behaviour to be compulsive [13,23]. Indeed starvation persists in the face of negative consequences, both immediate, for example interfering with academic/occupational/social interests, and longer-term; the behaviours promoting further, and potentially dangerous, weight-loss. Although individuals with AN often express desires to recover [22], they are seemingly unable to stop engaging in behaviour that contributes to the maintenance of an extremely low weight [24,25].

Godier and Park [13] considered models of compulsivity developed in relation to other disorders to propose the importance of both the reinforcement of starvation, and of a goal-directed system deficit, in the development of compulsive starvation. Greater reinforcement of starvation is suggested to cause excessive repetition of behaviour conducive to caloric restriction. Combined with a reliance on the habit-system for learning and behavioural control, this excessive repetition results in the development of strong S-R habits surrounding dietary restriction that are able to exert a dominant influence over behaviour.

In this paper we consider factors and mechanisms identified as relevant to reinforcement and goal-directed system abnormalities in other compulsive disorders to understand how these develop in AN. Thus we adopt a transdiagnostic approach to extend the aetiological model of AN proposed by Godier and Park [13]. We also adapt the existing account to highlight the particular importance of the anxiolytic properties of dietary restriction, over other potentially reinforcing effects of the behaviour, and explain the emergence of symptoms of AN other than compulsive starvation.

A novel model of anorexia nervosa development and maintenance

In brief, we suggest high levels of anxiety serve to make the anxiolytic effects of dietary restriction more reinforcing, and that anxiety contributes to reduced function of the goal-directed system. Thus, we propose a central role for anxiety in the development of compulsive starvation, with part of the novelty of our hypothesis being in the dual mechanisms by which anxiety is suggested to operate in AN onset. We propose that the reinforcing effects of starvation cause excessive repetition of behaviour via the development of psychological symptoms of AN. We also suggest that starvation becoming compulsive has adverse implications on anxiety, the goal-directed system, and psychological symptoms of AN, to encourage the formation of a vicious cycle that ensures the persistence of extreme dietary restriction. The proposed aetiological model is displayed in Fig. 1 below.

Given the complexity of AN we fully recognise the involvement of factors additional to those included in the proposed model, however we suggest testing the set of central hypotheses proposed here prior to expanding the model further. In the following section we outline each part of the model, and provide evidence to support inclusion of the factor or pathway.

1. Individuals who develop AN experience high levels of anxiety

Clinical observations characterize individuals with AN as highly anxious, and this is supported by empirical studies reporting greater trait anxiety and higher rates of anxiety disorders in AN populations as compared to the general population [26]. Importantly anxious

pathology is consistently documented to precede AN onset [27–30], supporting a role of anxiety in AN development. Notably high levels of anxiety tend to also precede the onset of addiction and OCD [31,32].

2. Dietary restriction is anxiolytic, and the relief of anxiety (or negative reinforcement) provided by dietary restriction increases with anxiety

Engagement in dietary restriction reduces activity of serotonin (5-HT) and noradrenalin (NA) systems that modulate anxiety, due to reduced intake of the dietary precursors of the neurotransmitters (tryptophan for 5-HT, and tyrosine for NA; [33,34]). Indeed, ill AN women have reduced 5-HT metabolites in their cerebral spinal fluid, reduced concentrations of NA in their blood plasma, and excrete reduced NA metabolites, compared to healthy women [35,36]. Recovered AN women have elevated levels of 5-HT metabolites [36], and gene variants linked to more active 5-HT and NA systems are implicated in AN [34,37], supporting the involvement of these neurotransmitter systems in the heightened anxiety that precedes AN. Increased ratios of omega-3:omega-6 fatty acids are suggested to result from a calorie and fat restricted diet, and there is some evidence that this ratio is negatively related to anxiety in AN [38], providing another mechanism by which dietary restriction could ameliorate anxiety.

Anxiety relief is easier to achieve, and more beneficial, for anxious individuals, such as those who develop AN, suggesting starvation has greater anxiolytic effects in these individuals [39,40]. Experimentally induced tryptophan depletion significantly reduced anxiety in women receiving inpatient treatment for AN, and those recovered from the illness, but did not affect the anxiety levels of healthy women [33]. These results can be explained by floor effects given the baseline anxiety of healthy women was comparable to that of current/recovered AN women following tryptophan depletion.

3. Experiencing greater anxiolytic effects of dietary restriction gives rise to the psychological symptoms of AN

The effects of greater reinforcement of starvation (which we propose to consist of anxiety relief) are proposed by O'Hara et al. [41] to result in the induction of a strong drive to starve. The drive to starve in turn results in fears of stimuli/behaviours not conducive to restrictive eating, such as food and weight-gain, and preoccupations with eating and weight [41]. These drives, fears and preoccupations collectively represent AN psychopathology [42].

4. AN psychopathology causes excessive repetition of behaviour that is conducive to starvation

Like O'Hara et al. [41] we suggest AN psychopathology directly encourages the excessive repetition of dietary restriction that results in habit formation. Interestingly individuals with AN may be physiologically more able to engage in starvation over the period of time required for habits surrounding the behaviour to form given enhanced 5-HT activity increases satiety as well as anxiety [43]. The intestinal microbiota of individuals with AN may possess unique characteristics that also contribute to the ability to maintain a diet that is severely calorie restricted [44].

Given the alleviation of anxiety is proposed to promote AN psychopathology we suggest heightened anxiolytic effects of dietary restriction, resulting from greater baseline anxiety, encourages continuation of the dietary restriction, albeit indirectly. Similarly, avoiding an aversive state, and particularly an anxious one, is proposed to motivate continued drug-taking, hair-pulling, gambling and behaviours that become compulsive in OCD [45–48].

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