Anorexia nervosa (AN) is uniquely placed in the spectrum of psychiatric nosology as the only disorder in which an actualization of the core symptomatic fear (ie, weight gain) is directly targeted as a primary treatment goal. While treatment outcomes are generally indexed along dimensions of both weight-based and cognitive symptomatology, the initial focus on immediate weight restoration raises an important question relating to how the fear of weight gain might be abated throughout treatment when this primary symptomatic fear (ie, weight gain) is targeted and confirmed. Here, we address an important gulf between extinction theory and the practice of exposure therapy for AN.

Recent fear extinction literature posits that the amelioration of acquired fear does not operate solely according to habituation processes, such that exposure to feared stimuli alone is insufficient in ensuring long-term fear reduction. Rather, fear reduction more centrally operates via mechanisms of inhibitory learning, which is an active learning process in which the violation of expected feared outcomes results in the new learning of nontreat associations relating to the originally feared stimulus, which in turn serves to inhibit anxiety (Figure). As such, exposure therapies are now tailored toward maximally violating fear expectancies, rather than aiming for habituation, per se, with long-term consolidation of fear extinction being centrally linked to treatment outcome.

However, this theoretical framework is predicated on the clear distinction drawn between feared cues/stimuli and feared outcomes, which, in turn, may be violated throughout treatment to generate inhibitory learning. This is particularly relevant in the context of AN, for which, despite the inherent centrality of exposure processes, sparse empirical evidence has precisely deconstructed core fear associations, which may nullify the most salient pathway of fear extinction.

Disentangling Feared Stimuli and Feared Outcomes in Anorexia Nervosa

Fear of weight gain in the context of malnourishment is perhaps the hallmark feature of AN, although it remains unclear whether the phobic content of weight gain is experienced as a feared stimulus/cue or as a feared outcome/consequence. This distinction is crucial in determining optimal learning pathways that capitalize on advances in the anxiety disorder field. Thus, while exposure principles may be implied in classic cognitive behavioral approaches to AN, a corresponding dissection of feared stimuli vs feared outcomes remains understudied.

Food as a material object is typically not the feared stimulus in AN. Recent reports have postulated that the “broken cognition” in AN is a dysfunctional food intake-weight gain connection, in that food consumption is expected to cause catastrophic weight gain, which is therefore mitigated by the avoidance or restriction of food intake. Certainly, many patients describe an array of “fear foods,” which are typically calorie-dense foods deemed subjectively more likely to result in weight gain, although, in this formulation, the magnitude of weight gain deemed catastrophic, in either absolute or relative terms, remains unclear. Clinical observation suggests that these are linked, in that a small degree of weight gain in individuals with AN typically evokes a fear of continued, uncontrollable weight gain. Thus, there may be a chained anxiogenic process in which consumption, particularly of food types or quantities associated with weight gain, yields a fear of gaining any amount of weight, and small positive weight changes, in turn, stimulate a feared expectancy of exponential weight gain.

Alternatively, another putative cue-outcome threat association in AN involves weight normalization as the stimulus and self-concept-related feared consequences as the outcome. Specifically, patients may fear that weight normalization will portend intolerable affective states or violate their experience of themselves as highly controlled, unique, or adherent to the culturally endorsed thin-body ideal.

These hypothesized cue-outcome relationships are not mutually exclusive and underscore the complex nature of AN. However, the precise configuration of these feared cue-outcome expectancies may significantly impact, or potentially undermine, treatment efficacy because it is unlikely that a uniform treatment approach may effectively violate all potential configurations of fear-based expectancies.

The Impact of This Conundrum on Treatment Mechanisms

The treatment of AN, by virtue of directly targeting the primary phobic content (ie, weight gain), inherently centralizes exposure-driven processes. Thus, a comprehensive distillation of the core fear is both novel and necessary in optimizing the potency of treatments. For instance, if food consumption is conceptualized as the core feared stimulus, and weight gain as the feared outcome, it is possible that the introduction of previously eliminated/fearred foods alongside the necessary weight gain throughout weight restoration may confirm the notion that these foods are indeed “dangerous” as weight increases. In this instance, inhibitory learning may technically only commence after achieving weight restoration, when feared food consumption (in normal volumes) will not necessarily result in further weight gain. Alternatively, if one conceptualizes weight gain as the feared cue and aversive self-concept-related consequences as the feared outcome, treatment focusing...
entirely on food and weight gain may not provide adequate exposure training for violation of this fear. In this instance, a broader focus of treatment is likely warranted in promoting learning experiences beyond the scope of food consumption, per se, as feared self-concept-related consequences are violated.

Crucially, the potential scope for differential fear associations within presentations of AN may underpin differential treatment responses. For instance, the process of weight restoration may likely impart different cognitive outcomes depending on whether a patient’s core fear association relates to food consumption resulting in weight gain (confirmed through weight restoration) or weight gain resulting in self-concept-related aversive outcomes (likely violated during weight restoration, depending on the patient’s nonfood-related exposures). Furthermore, the small preponderance of patients demonstrating “nonfat phobic AN” underscores the need to delineate the core fear associations in AN and serves to caution against a one-size-fits-all approach to the treatment of a diverse array of fear associations within AN.

Certainly, the study of extinction learning holds much promise in the treatment of AN, and approaches such as computational psychiatry may likely shed light on the elusive pathways of cognitive AN symptomatology by examining the well-defined biomarkers of fear extinction throughout treatment of AN. However, with the surging interest in exposure-based therapies for AN, a key endeavor lies in disentangling feared stimuli and feared outcomes, such that treatments can be more precisely oriented toward violating core symptomatic fear expectancies. We propose that individualized exposure exercises, based on patient-specific configurations derived from a broader model of cue-outcome fear associations in AN, will promote better treatment outcomes. It is only after the precise delineation of core fear associations in AN that targeted efforts to promote the long-term consolidation of their extinction may be effectively undertaken and tested. The application of exposure therapy without clearly delineating which fear-based expectancy one is attempting to violate is, at best, nonspecific or, at worst, contraindicated.

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