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## POSITIVE AFFECT IN AN

Title: The posited effect of positive affect in anorexia nervosa: Advocating for a forgotten piece of a puzzling disease

Short running title: Positive affect in AN

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### Abstract

Anorexia nervosa (AN) is a complex and life-threatening eating disorder. Current models of AN onset and maintenance have largely focused on the role of negative affect, while fewer models have described the role of positive affect (PA). Given that these theoretical models have informed current treatment approaches, and that treatment remains minimally effective for adults with AN, we advocate that targeting PA is one avenue for advancing maintenance models and, by extension, treatment. We specifically propose that AN may arise and be chronically and pervasively maintained as a function of dysregulated PA in response to weight loss and weight loss behaviors (e.g., restriction, excessive exercise), to a degree that is not accounted for in existing models of AN. We present evidence from multiple domains, including biological, behavioral, and self-report, supporting the hypothesis that PA dysregulation in AN contributes to the maintenance of the disorder. We conclude with several specific avenues for treatment development research as well as a call for future work elucidating the biological correlates of PA.

*Keywords:* anorexia nervosa, positive affect, positive affect dysregulation

## I. Introduction

Anorexia nervosa (AN) is a severe disorder for which no gold standard treatment exists for adults (Watson & Bulik, 2013; Zipfel, Giel, Bulik, Hay, & Schmidt, 2015). A growing body of work has conceptualized AN as a disorder of emotion dysregulation (e.g., Haynos & Fruzzetti, 2011). While the evidence for the role of negative affect (NA) in predicting engagement in eating disorder behaviors is growing (Haynos et al., 2017), we concurrently propose that the role of positive affect (PA) in the onset and maintenance of AN is comparatively far less prioritized and incorporated into etiological theories of AN or treatment development efforts for AN. Some early theories of AN have credited PA from weight loss as critical to the onset of the disorder (e.g., Slade, 1982), but have been eclipsed by more recent theories that have posited that cognitive (Fairburn, Cooper, & Shafran, 2003) or behavioral (Walsh, 2013) mechanisms are central to the disorder ( Table 1). There is a vital need to reinvigorate empirical inquiries into conceptualizations of PA as a trigger for, and reinforcer of, AN to improve the specificity and efficacy of treatment.

In this paper, we summarize the limited, but promising, literature on PA in AN from behavioral, psychological, and biological perspectives to explain how this process may contribute to the onset and maintenance of AN. The purpose of putting forth this idea is to highlight the questions that remain unanswered despite what these prior important theories have contributed to our understanding of AN. This holds great contemporary relevance given the advent of novel, advanced methods that have greatly improved the specificity with which affect

is studied. Therefore, we wish to clarify, expand, and encourage the study of PA in AN in light of the new evidence and technologies to study PA with the goal of improving interventions.

## **II. Too much of a good thing?**

PA dysregulation can involve PA experienced in non-adaptive contexts or to an inappropriate degree, which can prompt engagement in a range of risky and disordered behaviors (Gruber, Mauss, & Tamir, 2011). The idea that PA is not an unconditionally beneficial mood state has gained traction in the study of other psychological disorders (e.g., bipolar disorder; Gruber, 2011), but has yet to be applied in the eating disorders field.

The dysregulated PA of relevance to AN appears to be characterized by inappropriate, disorder-congruent PA (e.g., consumption of pro-ana stimuli), which may ultimately positively reinforce weight loss behaviors. Although there are ongoing debates about whether positive and negative reinforcement represent distinct mechanisms (Barberini, Morrison, Saez, Lau, & Salzman, 2012), there is preliminary evidence in AN that PA and NA mechanisms may operate both independently and jointly, thereby simultaneously positively and negatively reinforcing weight loss behaviors (Selby et al., 2015). Importantly, this dysregulated PA is distinct from anhedonia (i.e., diminished/blunted positive affective experience), on which the majority of research on PA in AN has focused (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013). To date, minimal research has examined the degree to which PA may be inappropriately or excessively generated either by eating disorder behaviors (e.g., restriction, compulsive exercise) and/or by environmental cues that reinforce weight loss.

PA dysregulation is a relevant area for study in light of qualitative accounts that suggest that many with AN experience high levels of PA in response to weight loss both before and after AN onset (Nordbø, Espeset, Gulliksen, Skårderud, & Holte, 2006). Individuals with AN also report receiving desirable social responses to weight loss behaviors (Nordbø et al., 2006), such as restriction or over-exercising, suggesting another pathway to positive reinforcement. Although the habit model of AN (Walsh, 2013), which characterizes food restriction as a persistent habitual behavior that occurs outside conscious choice, credits PA as a mechanism of the disorder in its early onset stages due to weight loss success and interpersonal reinforcement, it posits that habit formation replaces PA as the primary maintaining mechanism of restriction over time. Other models suggest insufficient PA (e.g., anhedonia) as a potential mechanism promoting symptoms of AN (Kaye et al., 2013), but have yet to explore dysregulated PA in response to eating disorder behaviors. We propose that PA may influence the onset of AN as the habit model suggests, and also may be chronically maintained by elevated PA in response to disordered eating, to a degree that is not accounted for in existing models.

### III. Current Evidence

There is evidence from multiple domains suggesting that PA dysregulation in AN leads to both onset and maintenance of the disorder:

**Self-report.** Whereas individuals with AN often report lower baseline reward responsivity on temperamental measures (Vervaet, Van Heeringen, & Audenaert, 2004), they report more positive affective reactions to low-calorie foods, low-weight bodies, and physical

activity cues (Cowdrey, Finlayson, & Park, 2013; Fladung et al., 2010; Giel et al., 2013), suggesting that these cues may be among the few to generate PA. This is further corroborated by ecological momentary assessment (EMA) research that measures affect and behaviors in real-time. One EMA study found that diminished ability to distinguish across positive affective states led to PA dysregulation, which motivated and reinforced weight loss behaviors (Selby et al., 2014). Further, daily PA lability has been associated with greater weight loss behaviors, self-weighing, and body-checking among individuals with AN (Selby et al., 2015). Momentary analyses of the same data found elevated PA during and after restrictive eating episodes, compared to normal meals (Fitzsimmons-Craft et al., 2015), and that restrictive eating resulted in increased self-assurance among those with restricting subtype (Haynos et al., 2017). Finally, lower PA preceded days for which restrictive eating was more likely (Engel et al., 2013), lending further evidence that restrictive eating may be utilized to momentarily enhance reward experiencing.

**Behavioral.** Studies have demonstrated that underweight and physical activity cues may elicit PA to a greater extent than monetary incentives in this population. In one study, individuals with AN sacrificed more money in a behavioral task to view bodies that were thinner (Watson, Werling, Zucker, & Platt, 2010). Similarly, on progressive ratio tasks, individuals with AN sacrificed more money and exerted more effort to engage in physical activity compared to control participants (Klein et al., 2010). These results indicate the high salience and behavioral drive for such AN-relevant cues.

**Biological.** Individuals with AN demonstrate elevated skin conductance, decreased eye blink startle, and increased electroencephalogram late positive potential in response to underweight stimuli (Clarke et al., 2016; Horndasch et al., 2018; O'Hara et al., 2016), indicating greater salience and PA responding. This altered dopaminergic functioning and reward response is corroborated by a study showing increased eye blink startle in response to thinness images following dopamine depletion (O'Hara et al., 2016).

There is also evidence of disturbance in the structural and functional organization of reward neural circuitry, as well as the dopaminergic system in AN (Kaye et al., 2013), which may make these individuals vulnerable to PA dysregulation. AN involves heightened activation in key regions of reward circuitry (e.g., striatum) in response to low calorie foods and underweight bodies (Fladung et al., 2010; Foerde et al., Steinglass, Shohamy, & Walsh, 2015), further suggesting that restrictive eating and weight loss induce PA. Several studies have also detected abnormal neural responding in reward regions to palatable food cues; however, the direction of these findings is inconsistent, with some finding enhanced (Frank et al., 2013), and others decreased (Brooks et al., 2011) reward responding compared to controls.

#### **IV. Call to Action: Our Idea Worth Researching**

Several gaps remain in our knowledge of how PA contributes to the onset and maintenance of AN. Thus, we offer the following suggestions to the field for advancing research with respect to theoretical models, assessment, and treatment:

##### **Operationalizing PA**



It may be that those with AN have pervasive difficulty experiencing PA in general, and therefore need to engage in extreme, disorder-syntonic behaviors to elicit PA. Alternatively, those with AN may experience difficulty keeping pervasive NA at bay, and up-regulate PA to do so. Because there is mixed evidence as to whether PA and NA are orthogonal (Diener & Emmons, 1984) or represent two ends of a bipolar spectrum (Russell & Carroll, 1999), further research is needed to address this issue. A third possibility is that individuals with AN may exhibit intact PA regulation at the onset of the disorder, but become over-conditioned to derive an excess of PA from stimuli associated with weight loss as the disorder progresses, akin to the incentive salience hypothesis of drug addiction (Robinson & Berridge, 1993). More research is needed to test these alternate theories to inform more precise and comprehensive mechanistic models.

### **Assessment and methods**

*Longitudinal research.* Although dysregulated PA may contribute to disordered eating behaviors, there is also evidence that increasing non-disorder specific PA is beneficial for treatment outcomes (Cardi et al., 2015), indicating that PA may switch during the illness course from hindering to helping recovery. Longitudinal studies are essential to contextualize these findings and to understand the extent to which dysregulated PA predates illness onset, and whether the link between PA and eating disorder behavior changes as a function of illness stage.

*Biological correlates.* Prior research on the biological correlates of appetitive responding has primarily focused on the construct of reward and corresponding brain regions (e.g., striatum).

Although it is often assumed that reward is synonymous with PA, few of these studies examine how biological responses in reward-associated neural circuitry or psychophysiological indices correspond with subjective affective experience. Further, it is now widely acknowledged that subjective experiences related to PA are regulated by a broader range of brain structures, including some areas that overlap with the processing of NA (Suardi, Sotgiu, Costa, Cauda, & Rusconi, 2016), further suggesting the need for multimethod studies in this area. Additionally, although most prior studies investigating the neurobiology of reward in AN have isolated specific brain regions, novel MRI methods have led to increased interest in measuring structural and functional connectivity to examine how neural circuits function in correspondence. In addition, many prior studies have examined biological responses to passive stimuli, such as pictures of food or underweight bodies, which may not translate directly to engagement in eating disorder behaviors. Therefore, examination of the biological correlates of PA during active decision-making (e.g., food choice; Foerde et al, 2015), perhaps utilizing emerging computational models (Huys & Renz, 2017), may yield more robust and generalizable results. Recent advances in magnetic resonance and computational analysis make this a particularly opportune time to expand the investigation of PA.

*Novel methods.* Other new technologies to pioneer PA research in AN may enhance the external validity of PA. Such methods could include: advanced ambulatory assessment with smart watches and continuous physiological monitoring, integration of computerized reward

assessment tasks (e.g., delay discounting tailored to weight loss), and virtual reality to simulate positive interpersonal comments about weight.

### **Treatment development**

An important question in AN treatment outcome research is whether treatments remain largely ineffective because they fail to act on correctly identified maintenance mechanisms, or whether the mechanisms themselves are incorrectly identified (Jansen, 2016). Although some theories have acknowledged that PA is involved in AN (Table 1), existing treatments fall short of expressly targeting dysregulated PA in AN. For instance, while cognitive-behavioral therapy targets over-evaluation of shape and weight (Fairburn, 2008), which may increase self-esteem by diversifying self-evaluative domains, dysregulated PA is not expressly targeted. The ultimate goal of this proposal is to encourage the development of treatments that more specifically target potentially relevant treatment mechanisms, thereby leading to better treatment outcomes, which are desperately needed in AN. A description of the types of treatment interventions that may be relevant to shape PA in AN is provided in Table 2.

### **V. Conclusion**

We propose that the role of PA in the onset and maintenance of AN is underappreciated and understudied. Dysregulated PA in response to weight loss may contribute to the extreme downward trajectory in weight status characteristic of this disorder. Our idea worth researching calls on the field to understand more clearly the effect of PA in AN to improve disorder prognosis.

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Table 1. Previous theories and treatments targeting positive affect in anorexia nervosa

Theory	Description	Role of positive affect (PA)	PA implicated as primary factor in AN:	
			Onset	Maintenance
<i>Slade (1982)</i>	Dieting emerges out of the need for control over life circumstances given general life dissatisfaction. Subsequent weight loss is reinforced from initial success with weight loss and being in control of self.	After initial weight loss, primary motivation for continued weight loss is through negative reinforcement of negative affect (i.e., wanting to avoid negative feelings resulting from weight gain), not PA.	Yes	No
<i>Vitousek &amp; Hollon (1990)</i>	Anorexia nervosa features strongly entrenched weight-related beliefs about the self (i.e., self-schemas) such that weight is closely tied to personal value and worth.	Emphasizes cognitions over affect in explaining the maintenance of AN, describes those with AN as being “moralistic individuals” able to exert self-control, implying the experience of positive affect is scant, possibly even avoided.	No	No
<i>Fairburn, Cooper, Shafran (2003)</i>	Anorexia nervosa is maintained by an over-evaluation of one’s shape and weight (and being able to control them).	Emphasizes cognitions over affect such that diversifying means by which one evaluates him/herself reduces food restriction. Mood intolerance (typically intolerance of negative affect) gives rise to binge eating and purging behaviors.	No	No
<i>Schmidt &amp; Treasure (2006)</i>	Anorexia nervosa is maintained by a cognitively rigid thinking style, avoidance of negative emotions, pro-anorexia beliefs, and both positive and negative responses from others regarding the physical appearance of the person with the disorder.	Initially, restraint is maintained intrapersonally by positive reinforcement from temporary mood improvement. Pro-anorexia beliefs and positive responses from others also elicit PA.	Yes	No*
<i>Walsh (2013)</i>	Anorexia nervosa is initially maintained through positive reinforcement of weight loss, and is subsequently maintained	Dieting behavior is positively reinforced through successful weight loss and negatively reinforced through successfully alleviating	Yes	No

	<p>through habitual food restriction that persists regardless of weight loss .</p>	<p>negative affect. Eventually, dieting persists regardless of presence or absence of PA from weight loss.</p>
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\*The role of positive affect in disorder maintenance is acknowledged but not as primary

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Table 2. Treatment interventions to target dysregulated positive affect in anorexia nervosa

Technique	Evidence Base
Improve detection and labeling of positive emotions to counter low positive emotion differentiation	Mindfulness module in dialectical behavior therapy (Linehan, 1993, 2015)
Explore and challenge core beliefs underlying stated pro-anorexia beliefs	Maudsley model of anorexia nervosa treatment for adults (MANTRA) (Schmidt & Treasure, 2006; Schmidt, Wade, & Treasure, 2014)
Encourage shift in balance between perceived benefits and drawbacks of the disorder in favor of the latter to elicit change talk and treatment adherence	Motivational interviewing (Miller & Rollnick, 2002)
Promote engagement in non-disorder-related activities that elicit positive emotion	Emotion regulation module in dialectical behavior therapy (Linehan 1993, 2015) Behavioral activation for depression (Jacobson, 2001) Values clarification and committed action acceptance and commitment therapy (Hayes, Strosahl, & Wilson, 2011)
Self-monitoring: <ul style="list-style-type: none"> <li>▪ Self-monitor positive affect <i>in vivo</i> in conjunction with self-monitoring of eating disorder behaviors to track their relationship over time</li> <li>▪ Self monitor use of, and efficacy of, skills to reduce, redirect, or tolerate (without acting on) inappropriate positive affect</li> </ul>	Self-monitoring, as is used in cognitive behavioral therapy for eating disorders (Fairburn, 2008) Diary card, as is used in dialectical behavior therapy (Linehan 1993)
Moderate social media exposure by limiting positive affect-inducing, disorder-promoting content while increasing exposure to positive affect-inducing, disorder-discouraging content	Stimulus control (Steinglass et al., 2018)
Develop effective responses to interpersonal comments (even positive comments) about weight and shape	Interpersonal therapy (Wilfley et al., 1993) Interpersonal effectiveness module in dialectical behavior therapy (Linehan, 1993)
Promote the development of prosocial positive emotions (e.g., compassion) instead of self-focused positive emotions (e.g., pride)	(Gruber et al., 2011; Selby et al., 2014)

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<p>Provide psychoeducation to family members (i.e., parents, siblings, partners) to discourage praise/comments about weight loss or body shape, and to charge the family members with the task of educating the patient's larger social network</p>	<p>Family-based treatment (Lock &amp; Le Grange, 2013)*                  Uniting couples in the treatment of anorexia nervosa (Bulik, Baucom, Kirby, &amp; Pisetsky, 2011)*</p>
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\* This proposed psychoeducation is not expressly addressed in either treatment

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