CLINICAL PSYCHOLOGY
SCIENCE AND PRACTICE

Anorexia Nervosa as a Disorder of Emotion Regulation: Theory, Evidence, and Treatment Implications

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This is a commentary on Haynos and Fruzzetti (2011). The commentary begins with a discussion of how emotion has been included in the conceptualization of anorexia nervosa (AN) for some time, although this has not culminated into an experimental science on somatic-affective arousal in this patient population. Important gaps in our knowledge of how individuals with AN experience signals from the body (including emotion) are highlighted, as is the lack of attention in the Haynos and Fruzzetti model to differences in emotional experience in the acute and chronic stages of the illness. Final comments are made about the focus of AN intervention, specifically how the functional significance of the symptoms can be an afterthought because of the need to restore nutrition, and the utility of identifying common and distinct processes across eating disorder diagnoses.

Key words: **anorexia nervosa, contemporary behavior therapy (DBT, ACT), emotion regulation, interoception, somatosensory, symptom function.** ***[Clin Psychol Sci Prac 18: 208–214, 2011]***

Anorexia nervosa (AN) is one of the most deadly and devastating psychiatric illnesses (Nielsen, 2001; Papadopoulos, Ekbom, Brandt, & Ekselius, 2009). Although sometimes misunderstood as volitional or an illness of vanity, the suffering of individuals with AN is indeed profound, with comorbid anxiety, depression, interpersonal dysfunction/isolation, and high rates of suicide (O'Brien & Vincent, 2003; Steinhausen, 2002). There are no empirically supported treatment options for adults with AN and those for adolescents remain unacceptably sparse (Agras et al., 2004). This, along with the high percentage of individuals who continue to have significant symptoms after treatment (Hudson, Hiripi, Pope, & Kessler, 2007; Steinhausen, 2002), underscores the need for advances in the way that AN is conceptualized and treated. Haynos and Fruzzetti (2011) present a framework for understanding AN as a disorder emerging from a transaction between biological vulnerabilities in emotional experiencing (i.e., experiencing frequent, heightened affect with slower return to baseline) and a sociocultural environment that is invalidating of these experiences. Building upon the Linehan model for borderline personality disorder (Linehan, 1993), the authors provide a tenable description of how these same processes may be relevant and manifest uniquely in AN. They organize a diverse set of data that provide support for the core premises of the model in a logical and clear fashion so that the reader can evaluate the strength of the evidence (which in some cases remains weak). This article draws attention to the need for additional study of emotional experience in AN and highlights the way in which our most current clinical constructs may spawn research that refines understanding and treatment of this disorder.

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CONCEPTUALIZATION OF AN

The author's claim of no attention to emotion in the historical conceptualization of AN is in part an issue of semantics. Although not cast in behavioral terminology, the central etiological role of complex affective experiences has been recognized since the earliest medical reports of AN (e.g., Morton, 1689, depicted AN as an illness caused by "sadness and anxious cares"). Contemporary but early accounts such as that of Hilde Bruch described AN as emerging from feelings of ineffectiveness and lack of self-determination and core symptomatology as an effort to establish a sense of personal autonomy, competence, and self-control (Bruch, 1965, 1981). Bruch also pointed to environmental constituents of this emotional difficulty, asserting (as was in favor at the time) that maternal lack of attunement with the child's independent needs had a profound impact on the experience of the physical boundaries of the child's self (body size overestimation) and the ability to accurately read and respond to internal sensations (e.g., hunger/satiety, emotional states). Crisp (1997) later made sense of typical age of onset for AN and findings that early puberty is a risk factor for the illness

by conceptualizing AN as an attempt to reverse pubertal change and return to the simpler childhood. From this perspective, an inability to respond effectively to developmentally appropriate stress and the emergent complex somatic-affective accompaniments were keys to illness development. Physical changes that resulted from the self-imposed starvation were seen as highly negatively reinforcing, removing the discomfort of sexual development and all that it imposed. Russell (1970) commanded greater attention to the fear that characterizes the illness, specifically fear of fatness. This was later included in the diagnostic criteria, making this phobia of weight gain definitional of AN. Thus, repeatedly, prominent clinicians in the field have provided accounts of AN that highlight the way in which difficult emotional experiences generate symptomatology. This sensibility has been corroborated by decades of patient reports, with narratives of affected individuals consistently describing AN as something that staves off negative affect or promotes positive feelings. Common themes of patient reports are that the illness provides feelings of safety, orderliness, and a sense of pride or specialness and takes away the experience of life chaos, unpredictability, and personal insecurity (Serpell, Treasure, Teasdale, & Sullivan, 1999).

LIMITATIONS AND NEW DIRECTIONS

Bruch's early observations of deficits in the ability to identify and respond to internal sensations eventually translated into an entire body of work on interoceptive awareness in AN (e.g., Fassino, Piero, Gramaglia, & Abbate-Daga, 2004). However, while this research has consistently indicated deficits in emotional clarity and fear regarding emotional experiences, these constructs have been collapsed and measured via self-report, limiting the ability to draw precise conclusions of specific skill deficits (Merwin et al., 2010). Contemporary cognitive-behavioral therapy (e.g., such as that from which Haynos and Fruzzetti emerges, e.g., dialectical behavior therapy; DBT) has highlighted the need to discriminate component skills necessary for effective emotion regulation (e.g., awareness of emotion, acceptance of emotional responses, emotional clarity; Gratz & Roemer, 2004). This sensitivity may facilitate research that allows investigators to further operationalize and refine areas of emotional deficiency among individuals with AN. It may also allow more basic work on emotion and interoception emergent from other areas of psychology (e.g., cognitive neuroscience; LaBar & Cabeza, 2006; Pollatos, Schandry, Auer, & Kaufmann, 2007) to inform AN research. Such advancement may propel new science on the pathophysiology of AN and establish novel treatment strategies.

SOMATIC-AFFECTIVE EXPERIENCE IN AN

It is often inferred that individuals with AN are hyposensitive to somatosensory experience because of the lack of responsivity to signals of hunger or fatigue and apparent denial of affect. However, we know surprisingly little about how individuals with AN experience somatic sensation (e.g., whether they tend to be hyposensitive or hypersensitive to sensory stimulation, intensity of visceral experience). Given that emotions have somatic correlates, individual differences in sensitivity could have implications for the way in which emotions are experienced and ultimately regulated. For example, hyposensitivity might impair one's ability to detect and label an emotion, contributing to ineffective or ill-matched responses. Hypersensitivity, in contrast, might make emotions intolerable and thereby facilitate attempts to avoid or escape these experiences (e.g., avoiding evocative social situations, limiting uncertainty by narrowing food choices). There might also be a synergy between somatic hyposensitivity and hypersensitivity. For example, if an individual is only aware of sensations in the gut and not the other aspects of this emotional experience, this might lead to failure to label this as an emotion (*"I have a stomachache"* rather than *"I feel ashamed"*; referred to as inaccurate expression in Haynos and Fruzzetti, 2011, but not linked to these individual differences). This aberration may perpetuate inappropriate strategies to attenuate this experience, namely restricting food intake, which would be subsequently reinforced by the alleviation of negative arousal (e.g., exerting control over the body may generate a sense of mastery that attenuates shame and relieves the sensitive gut).

Our limited knowledge of somatic-affective arousal in AN is likely due to the dearth of *experimental studies*. The lack of this type of research may be related to complications in measurement specific to AN, including the impact of starvation on peripheral

measurements of arousal (e.g., galvanic skin response), which contributes to reliance on self-report and difficulty discerning preexisting deficits from those secondary to starvation. The study by Pollatos et al. (2008) is a prime example. In this study, individuals with AN demonstrated impaired ability to detect their own heartbeat. The investigators concluded that this reflected poor interoceptive awareness in this population; however, body mass index was significantly related to task performance. Thus, an alternative explanation is that participants' ability to detect their own heartbeat was compromised by a muted signal from the cardiovascular system (i.e., bradycardia associated with low weight; Mitchell & Crow, 2006). If so, this study would have little to say about whether individuals with AN have deficits in the ability to detect somatic signals that facilitate symptom onset. Researchers will have to devise creative solutions to these problems.

The near complete reliance on self-report measures to assess somatic-affective experience (or interoception) in AN has limited the field and may have led to erroneous conclusions. Again consider Pollatos et al. (2008). In this study, AN participants' performance on the heartbeat detection task did not correspond with a commonly used self-report measure of interoceptive awareness (Eating Disorder Inventory–Interoceptive Deficits Subscale; Garner, 2004). This discrepancy suggests that there are differences in what individuals with AN *report* about their ability to detect signals from their body and their *actual skill* in this domain. Refining understanding of how individuals with AN experience signals from their body (including emotions) will require more experimental studies, and longitudinal research identifying individuals early in illness development (or even premorbidly), or extensive cross-sectional study accounting for the influence of age on somatic experience and executive function.

HEIGHTENED EMOTIONAL REACTIVITY?

Sensitivities aside, Haynos and Fruzzetti (2011) propose individuals with AN have a heightened response to emotionally evocative stimuli. However, data are sparse. The authors describe only one study on slow return to baseline, and researchers have relied almost exclusively on pathology-specific stimuli (e.g., body- and food-related words and images). This latter issue is important as sensitivity to these cues says little about overall emotional responsiveness uncomplicated by features of the illness. Certainly, we know that individuals will respond to personally relevant stimuli with greater attention (Riemann & McNally, 1995) and that when someone is starved, food will capture more emotional and cognitive resources (Keys et al., 1950; Mogg, Bradley, Hyare, & Lee, 1998; Polivy, 1996). Thus, increased attention allocation or more subjective arousal in the presence of body- or food-related stimuli may tell us little about the initial *etiological role* of heightened affect. Much more work is needed.

A COMPLEX INTERACTION

The Haynos and Fruzzetti model assumes that with greater illness comes more emotional disruption and increased need for symptoms. This account may oversimplify the complex interaction between somatic-affective arousal and symptoms of AN. In the early stages of illness, individuals may demonstrate heightened emotionality. Later, with prolonged starvation and the strengthening of avoidant repertoire, these same individuals may become less reactive. For example, bradycardia may downgrade the experience of anxiety, or with continued suppression of dysphoria, individuals with AN may become less skillful at noticing and responding to sadness. This would be relevant to detecting not only emotional needs but also signals for food consumption. It is known that with continued neglect of hunger, these signals are muted as a biological adaptation (Wang, Hung, & Randall, 2006). Over time, these adaptations would be expected to increase the need for rigid dietary rules (or rules of emotional responding) to guide action. Recovery from AN may establish new complexities, reinstating hunger cues and promoting emotional volatility. Indeed, it is often only when an individual begins to eat more regularly that he or she recontacts sometimes voracious (and often feared) appetite. The emotion regulation model presented in Haynos and Fruzzetti (2011) might be enriched by including both acute and chronic stage processes, and changes that occur as a result of refeeding, and integrating predispositions toward hyposensitivity or hypersensitivity to somatic signals.

ATTENDING TO FUNCTION OVER FORM

Haynos and Fruzzetti (2011) point to the relative ineffectiveness of cognitive-behavioral therapy (CBT) for AN. They suggest the reason for this limited utility is that the cognitive strategies are less impactful when individuals are emotionally dysregulated. However, it is equally plausible that the shortfall of standard CBT is that it aims to change the topography of the cognitive symptoms (e.g., beliefs about body weight and shape; Garner, Vitousek, & Pike, 1997), rather than addressing the function of this narrow attentional focus or the psychological gain in maintaining such a belief system. The emphasis on the form of the pathology rather than the functional significance of the symptoms is driven perhaps by the evocative nature of AN. With the emaciated frames, egosyntonic cognition, and apparent food and fat phobia, AN symptoms demand notice at the topographical level. Expectedly, and necessarily, clinical tools focus much more on first-order change (i.e., changing dangerous restrictive behavior and increasing food intake), not functional relationships. This is not just an issue for CBT, but other therapies having a similar narrow focus. The most well-established intervention for adolescents with AN is the Maudsley family therapy (MFT; Lock & LeGrange, 2005). MFT manuals focus primarily on articulating conditions that facilitate refeeding (Lock, LeGrange, Agras, & Dare, 2001). Empirical support for this therapy is building, particularly for adolescents with a short duration of the illness (Lock & Fitzpatrick, 2009). However, the question arises whether long-term prognosis could be improved with strategies that block the use of eating disorder symptoms to manage affect and additional skills to promote healthy emotional experience and expression.

The relative lack of emphasis on the emotion regulatory function in AN (and thus lack of training in effective emotional experience and expression in standard AN treatment) contrasts with the approach taken with other eating disorders (Safer, Lock, & Couturier, 2007; Safer, Telch, & Agras, 2001). This might be because emotional and behavioral excesses (e.g., bingeing-purging) tend to be more distressing than deficits. We see this broadly outside the realm of eating disorders with internalizing children being overlooked and those who externalize difficulties more readily identified and treated earlier. Thus, it might be that the typically constricted affect of the AN patient demands less therapeutic attention, although equally problematic as the outpour of emotion from a patient who binges and purges. Greater scientific attention to the functional nature of the symptoms (e.g., the way in which symptoms alter somatic-affective experience, whether reliance on verbal rather than experiential knowledge helps individuals cope with premorbid deficits in implicit learning) could radically alter treatment approach, particularly for adults for whom there are no empirically supported interventions.

COMMON AND DISTINCT PROCESSES IMPLICATED IN PSYCHIATRIC ILLNESS

Anorexia and bulimia nervosa (BN) are often treated as unique disorders with distinctive underlying processes. As a result, there is a relative lack of integration of this literature and as Haynos and Fruzzetti (2011) highlight, a near absence of emotion regulation research in AN as compared to BN. The fact that diagnostic crossover is common (Anderluh, Tchanturia, Rabe-Hesketh, Collier, & Treasure, 2009) suggests that various eating disorders are a reflection of a common etiological process (Fairburn, Cooper, & Shafran, 2003), and the manifestation of the symptoms is influenced by other factors, such as individual differences in biological, psychological, and historical factors. As noted by Haynos and Fruzzetti (2011), the field may advance with parallel research on the emotion regulatory function of symptoms in AN and BN, in conjunction with specification of factors that influence which topography emerges.

FACTORS THAT INFLUENCE SYMPTOM TOPOGRAPHY

Brown and Barlow (2009) recently proposed the inclusion of the dimensions of negative/positive affectivity and behavioral activation/inhibition in diagnostics of emotional disorders such as anxiety and depression. They cite evidence that these temperament characteristics are highly heritable and may be understood as broader phenotypes that manifest as syndromes when activated by stress (the precise nature of the syndrome determined by other factors, e.g., genetic loading for a particular cluster of disorders). Among those with vulnerabilities to eating disorders, these higher-order

constructs might determine illness manifestation. For example, in the context of behavioral inhibition, greater negative affectivity may produce behavior patterns characteristic of AN (emotional and behavioral constriction, avoidance of food) that effectively regulate affect. Individuals with a propensity toward behavioral activation might engage binging and purging as a regulation strategy of choice. Indeed, individuals with AN and BN demonstrate consistent temperamental patterns that roughly correspond with inhibition and activation, respectively (low novelty seeking, constraint, and persistence in AN and high novelty seeking and impulsivity in BN; Cassin & von Ranson, 2005); so much so that there has been discussion about including these traits in future iterations of the *Diagnostic and Statistical Manual of Mental Disorders*.

Support is growing for the central thesis that unhealthy attempts to attenuate difficult or uncomfortable private experiences (including painful emotions) perpetuate psychopathology (Gross, 2002; Hayes, Wilson, Gifford, Follette, & Strosahl, 1996). Articulating both the function of the eating disorder symptoms and traits that influence topographical manifestation could improve diagnostics and potentially point more directly to intervention strategy. Future work might discern factors that distinguish risk for AN as the primary regulation strategy from other somatoform, eating, or anxiety disorders. Factors might include differences in the cardiovascular versus gastrointestinal systems that differentially predispose individuals (e.g., an individual who is more acutely aware of changes in heart rate might be more inclined toward panic disorder than AN; Pollatos, Traut-Mattausch, Schroeder, & Schandry, 2007).

CONCLUSION

This is an exciting time in psychology with increasingly greater attention to the mechanisms by which our interventions produce meaningful change (e.g., Hayes, Luoma, Bond, Masuda, & Lillis, 2006). Emerging data suggest that the ability to accept uncomfortable emotional experience is relevant to AN (Merwin, Zucker, et al., 2010; Wildes, Ringham, & Marcus, 2010). More research is needed to articulate component skill deficits and assess the direct impact of AN symptoms on emotional arousal using experimental methods. Additional study of rule following and concern about mistakes in AN might also be necessary to determine whether these features, consistently associated with the disorder (Bardone-Cone et al., 2007; Crane, Roberts, & Treasure, 2007), can be understood within an emotion regulatory frame. This may help determine whether the process of symptom development and amelioration in AN may be wholly understood as improved emotion regulation capacities or part of a broader change in increased psychological flexibility (Merwin, Timko, et al., 2010). Either way, contemporary CBT that emphasizes willingness to experience difficult thoughts and feelings may usher in a new era of interventions with greater efficacy for this pernicious disorder. This may include not only DBT, discussed in detail by Haynos and Fruzzetti, but also companion treatments that may not specify emotion regulation as target but focus on acceptance of private experience to achieve broader life values and goals (e.g., acceptance and commitment therapy; ACT).

ACKNOWLEDGMENT

Special thanks to Dr. Nancy Zucker, who participated in discussions that contributed to the content of this article.

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Received April 26, 2011; accepted May 11, 2011.