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On bells, saliva, and abdominal pain or discomfort: Early aversive visceral conditioning and vulnerability for anorexia nervosa

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Abstract

Gastrointestinal (GI) symptoms are common in anorexia nervosa (AN), can predate illness onset, complicate renourishment, and persist after recovery. We explore how, through processes of aversive visceral conditioning, early GI pain and discomfort may increase vulnerability to AN in some individuals. Processes include enhanced preoccupation with the gut resulting from aversive visceral memories and disruptions in the typical acquisition of self-attunement when children learn to map and interpret interoceptive sensations and develop adaptive actions. We question whether a fear of weight gain, in some cases, may be an epiphenomenon of the recapitulation of actual or perceived GI symptoms that is especially relevant during puberty, especially in girls. This conceptualization has immediate clinical implications and offers ideas for future research. We propose that GI discomfort associated with renourishment may reignite prior aversive visceral experiences. We encourage development of a formulation that organizes the individual's current experience of the body with respect to these prior aversive experiences. Our conceptualization underscores the importance of assessment of GI experiences in individuals with AN; the examination of dietary strategies that minimize GI symptoms and enhance renourishment efficacy; and strategies that attempt to alter this aversive visceral conditioning by mapping sensations to meanings and adaptive actions.

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CONFLICT OF INTEREST

Dr. Bulik reports being a grant recipient and member of advisory boards for Shire, a consultant for Idorsia, and a royalty recipient from Pearson (all unrelated to this article). Dr. Zucker has no financial relationships to disclose.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created or analyzed in this study.

Keywords

abdominal pain; anorexia nervosa; avoidant restrictive food intake disorder; bloating; gastrointestinal symptoms; interoception; memory; visceral conditioning

1 INTRODUCTION

Since the time of Pavlov (1957), it has been well recognized that the responses of visceral organs can be entrained to cues both within the body and throughout the external environment. Less understood is how development contributes to the potency of early aversive visceral experiences (Kassab, Hamadneh, Nuseir, ALmomani, & Hamadneh, 2018). In trauma, visceral sensations can become cues that elicit traumatic flashbacks and subsequent avoidance behavior related to a remembered trauma. We query whether early aversive conditioning experiences, particularly those involving the gut, may contribute to the phenomenology and pathophysiology of anorexia nervosa (AN). Although aversive conditioning has an obvious role to play in some presentations of avoidant restrictive food intake disorder (ARFID; Zucker et al., 2019), exploring the role of aversive conditioning in AN may clarify our conceptualization of the disorder for patients, add precision to developmental models of AN, and hopefully, spur research into novel treatments that address the role of aversive visceral conditioning in AN. We propose the following hypotheses (some not new, just re-articulated within a developmental framework) and ways forward to test them. *In this framework, we propose that early gastrointestinal (GI) pain and discomfort experiences may increase visceral vigilance and preoccupation with GI sensations and create learning experiences that heighten risk for AN prior to any fears of weight gain*.

Hypothesis 1

Early pain and discomforting biological events increase vulnerability for the later onset of AN.

GI symptoms are common in AN during the acute phase of the illness, challenge treatment retention, and increasingly, have been shown to persist after recovery (Boyd, Abraham, & Kellow, 2010; Heruc et al., 2018; Mascolo, Geer, Feuerstein, & Mehler, 2017; Norris et al., 2016; Salvioli et al., 2013). Accumulating data suggest that adverse GI symptoms or GI-related autoimmune diseases marked by GI pain and food-related exacerbations (e.g., celiac disease or Crohn's) in childhood are associated with elevated risk for developing disordered eating and eating disorders in adolescence (Hedman et al., 2019; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Marchi & Cohen, 1990; Mårild et al., 2017; Raevuori et al., 2014; Rastam, 1992; Wiklund et al., 2019; Zerwas et al., 2017). These symptoms are also typically seen as normative and expected sequelae of malnutrition and renourishment: Salvioli et al. (2013) reported that over 90% of individuals with AN endorsed GI symptoms, and that while many GI symptoms significantly improved with treatment, abdominal pain and nausea persisted. The presence of GI symptoms necessitating specialty GI care has been shown to mark a more severe disorder course and elevated morbidity (Emmanuel, Stern, Treasure, Forbes, & Kamm, 2004). Combined, the data suggest that GI symptoms are a well-documented and frequent component of the presentation of AN, and an expanding body of

research documents that for a significant subset of individuals, GI symptoms may precede diagnosis and persist following intervention.

Learning models that attempt to explain pain exacerbation may help clarify the contribution of GI symptoms to the course of AN (Leeuw et al., 2007). Early aversive experiences of the GI tract may sensitize pain pathways, leading to amplification, preoccupation, and generalization to innocuous sensations (Labus, Mayer, Chang, Bolus, & Naliboff, 2007). Pain is an important learning signal: individuals efficiently develop behaviors to avoid pain. According to the fear-avoidance model of pain (Leeuw et al., 2007), avoidance would generalize to innocuous sensations that may predict pain exacerbation. For example, while bloating is uncomfortable, it may be conditioned to be experienced as dangerous if it has been a reliable predictor of pain. Furthermore, given the intrusiveness of GI symptoms, hypervigilance and monitoring of GI sensations are well-documented in individuals with GI disease. As such, individuals with a history of GI symptoms in childhood may become preoccupied with gut sensations, which could contribute to increased risk for AN. In partial support of this hypothesis, overanxious disorder (now referred to as generalized anxiety disorder), a disorder characterized by elevated somatic symptoms and related somatic fear as part of a constellation of excessive worry across multiple domains, has been shown to increase the odds of later onset of AN by a factor of 13.4 (Bulik, Sullivan, Fear, & Joyce, 1997). While additional research is needed to further inform the time course of GI symptoms and the psychological sequelae of these symptoms, data so far suggest that early GI symptoms may increase vulnerability for the later onset of AN (Jacobi et al., 2004).

Hypothesis 2

The avoidance of interoceptive sensations precludes accurate mapping of emotional response and valence appraisal.

Children learn to interpret interoceptive sensations and to integrate them into goal-directed activities: a child feels butterflies in her stomach, labels her experience as anxiety, and seeks out a source of support (Hietanen, Glerean, Hari, & Nummenmaa, 2016; Zucker et al., 2017). AN is notable for a disconnect between somatic signals and adaptive responses (e.g., hunger and the eating response). Thus, early attempts to avoid an ever-widening category of sensations may contribute to a lack of self-awareness and self-attunement that may increase vulnerability to AN. We hypothesize that one’s learning history of aversive visceral events, one’s emotional reaction to and assignment of valence to those events (i.e., as dangerous or innocuous), and the degree to which one inhibits their visceral experiences or integrates them into adaptive actions are important pieces to truly understand the experience of the body in AN. This is not a new conceptualization: Bruch proposed that this subversion of interoceptive mapping increased vulnerability for the subsequent need for control of the body (a theme of motivation for weight loss in AN), in part, in response to the somatic volatility of puberty (Bruch, 1980). Minuchin, in turn, focused on how physical vulnerabilities in a child organize a family and maintain symptom expression. These observations were important historical contributions to theoretical models of family therapy and of viewing AN as a psychosomatic disorder embedded in a “psychosomatic family” (Minuchin et al., 1975). In line with these early conceptualizations of AN as a

psychosomatic disorder, the term “low interoception” was employed in the eating disorder field to connote alexithymia, an inability to ascribe meaning to various visceral sensations. While the term interoception is now employed more precisely to distinguish the various phases at which an individual can sense, interpret, and integrate visceral sensations into adaptive actions (Khalsa et al., 2018), this earlier conceptualization of low interoception has been documented as a nonspecific, variable risk factor of low to medium effect (Jacobi et al., 2004). More recently, controlled laboratory studies verified that in ambiguous conditions, individuals with AN have difficulty discriminating adaptive visceral signals from false alarms and experience “visceral illusions” that indicate decreased body awareness (Khalsa et al., 2015; Khalsa et al., 2018). Thus our conceptualization adds to this body of evidence by highlighting the potential importance of early GI events as contributing to subsequent somatic avoidance and confusion.

Hypothesis 3

Early GI events may increase vulnerability to the development of a fear of weight gain specifically and dangerous weight-loss behaviors that have the added motivation of altering GI experience.

Individuals with AN claim to feel better when starved, but what does that actually mean? The field has interpreted it to mean a fear of weight gain, but it may be additionally conceptualized as a conditioned fear of the aversive visceral associations that are associated with eating, bloating, and higher weight. Thus, while models of sociocultural influence of the thin-ideal may influence the form of symptom expression, we propose that alterations in GI experience in sensitive individuals may provide additional sources of reinforcement for dangerous weight-loss behaviors (Rodin, Silberstein, & Striegel-Moore, 1984). Alternatively, or in addition, the presence of early GI symptoms may contribute to a lower body mass index, a specific risk factor for AN (Stice, Gau, Rohde, & Shaw, 2017). What is important is that the early pain experiences may exert influence, even if the symptom has been treated or the memory is not accessible. This may help to explain strong “gut feelings” —strongly held convictions with a distinct visceral component—convictions that often conflict with objective facts. For example, an individual with AN may report that an adaptive behavior such as eating a meal “feels wrong” despite recognizing factually that the behavior of eating a meal is health-sustaining. Thus, learning history is important: we may too quickly dismiss experiences as manifestations of anxiety, when it may be more accurate to ascribe aberrant behaviors as remnants of these visceral memory traces—a more dignifying explanation that acknowledges an individual’s history. Likewise, the dreaded phrase, “I feel fat” has been challenged by cognitive-behavioral practitioners as nonspecific. Ideally, such somatic experiences are used as an opportunity to further explore associated or underlying emotions or aversive learning histories. Such in-depth exploration could provide important clues to precisely those visceral sensations that are threatening to individuals with AN and the associated meanings and related triggers attached to these sensations.

Hypothesis 4

Puberty is high risk for AN onset in females in part due to the onslaught of uncomfortable GI (or interpretable as GI) events.

Puberty in girls is associated with weight gain, changes in fat distribution, and an array of uncomfortable physical sensations related to GI and reproductive organs (e.g., bloating and cramps). The starvation associated with AN eliminates or minimizes menstruation and the attendant GI/abdominal discomfort. One could argue that starvation itself instigates uncomfortable GI sensations (i.e., hunger pangs). However, hunger pangs differ in important ways from the unpredictable and often volatile changes in the GI system that accompany puberty. Post-traumatic stress disorder research indicates that the perceived or actual controllability of the event predicts whether a traumatic event will result in dysfunction (Ehlers & Clark, 2000). Translated to AN, sensations that are “controlled” (i.e., starvation) would be preferred to those that are not (i.e., menstruation-related abdominal discomfort). Starvation is a dominant experience that would provide “controllable” explanations for somatic sensations and thus may provide a valued substitute. With the minimization or suppression of puberty, uncontrollable somatic events are replaced by somatic sensations that can be ascribed directly to one’s behaviors.

Hypothesis 5

Hospital-based renourishment is uncomfortable and recapitulates many of the uncomfortable GI sensations that individuals with AN most fear.

Premature discontinuation of treatment is unsurprising given that our treatment basically prescribes our patients’ pain and discomfort. There is an imperative need to improve the tolerability and acceptability of renourishment. GI discomfort during refeeding is ubiquitously noted. GI consults are frequent, and observed abnormalities such as delayed gastric emptying are typically ascribed to the effects of prolonged starvation. From the standpoint of aversive visceral conditioning, it is hard to imagine a constellation of circumstances that would more perfectly recapitulate learned avoidance. Opportunities for novel research abound here: diets that manipulate rate of gastric emptying, diets that reduce the inflammatory responses to refeeding, diets that reduce gas production—all hold promise. It is crucial to emphasize in light of the role of controllability that the very existence of conversations that give patients agency in the planning of their own weight restoration could itself provide direct treatment benefit, not only in improving aversive conditioning, but also by enhancing the experience of self-attunement and motivation for treatment.

Hypothesis 6

Interventions that involve interoceptive mapping and recontextualize sensations may have promise.

Novel dietary approaches to refeeding are an obvious strategy to improve interventions for AN that do not recapitulate aversive visceral experiences. This conceptualization also points toward the importance of strategies that help individuals relearn (or learn) that visceral sensations are informative and provide important messages about what an individual needs. Such strategies could also aid in ascribing valence to visceral sensations to help distinguish between the innocuous and the truly threatening. Such interventions could aid in helping patients to listen reliably and respond to bodily signals, increasing trust in one’s body and the feeling of safety. In turn, this could increase willingness to try new experiences that may

have unknown effects on the viscera—experiences that were previously avoided. Notably, such interventions could have value irrespective of further confirmatory evidence (or lack thereof) supporting the etiological contribution of early GI events.

2 SUMMARY AND RESEARCH DIRECTIONS

This conceptualization emerges from increasing evidence of histories of aversive childhood GI experiences in individuals with AN. We emphasize the importance of learning and incorporate concepts from both the fields of pain and gastroenterology. Visceral memories can be powerful, and their impacts can be important and enduring shapers of behavior—especially behavioral avoidance. For example, aversive experiences with contaminated foods can create one-trial learning experiences that cement food avoidance for a lifetime (Recall that food you vomited after. Would you consider eating it again?) (Garcia, Kimeldorf, & Koelling, 1955). Other constitutional factors may also contribute to the extent to which visceral conditioning leads to behavioral avoidance. Biologically, individuals who are prone to visceral conditioning may actually have more highly enervated GI systems, amplifying the experience of innocuous GI events. Alternatively, altered neuroplasticity in response to visceral pain may enhance learning—even when pain or associated inflammation has been resolved (Brierley & Linden, 2014).

Regardless of origin, our conceptualization raises important points to consider. First, we emphasize that this formulation is intended to enhance our understanding of a certain facet of AN, somatic experience, and not serve as a model that seeks to explain all of AN phenomenology and pathophysiology. Notwithstanding, this formulation points to the importance of a thorough review of childhood GI experiences in the assessment of eating disorders. Although this is becoming standard in the assessment of ARFID, application to the assessment of all eating disorders may enrich our formulations. Second, the field is ripe for the development of renourishment approaches that reduce GI discomfort. The goal would be to improve treatment tolerability and acceptability, reduce premature treatment discontinuation, and assist with the difficult task of providing adequate energy to re-establish a healthy and maintainable BMI. This emphasis may prove important irrespective of eating disorder diagnosis and thus is consistent with a transdiagnostic framework broadly or for a subset of individuals with somatic complaints. Intriguingly, colleagues in agriculture may be important consultants, as they are frequently under pressure from environmentalists to develop healthy diets that reduce methane production in livestock (Alemu, Vyas, Manafiazar, Basarab, & Beauchemin, 2017). Finally, developing interventions that aid individuals in interoceptive re-mapping may hold promise in helping patients develop more accurate cartography of their internal sensations, their meaning, and their threat (Craske et al., 2011; Plasencia, Sysko, Fink, & Hildebrandt, 2019; Zucker et al., 2017; Zucker et al., 2019). Finding ways to assist our patients to develop a less adversarial relationship with their own physiology is an important treatment target. Our hope is that this proposed conceptualization of AN as a learned response to aversive visceral experiences will provide some guidance to catalyze research and treatment development in this area.

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Abbreviations

AN	anorexia nervosa
ARFID	avoidant restrictive food intake disorder
GI	gastrointestinal

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