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# Catastrophizing and anxiety sensitivity mediate the relationship between persistent pain and emotional eating

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ABSTRACT

Stress-induced or "emotional eating" contributes to increased caloric intake and weight gain, yet models examining psychosocial factors that promote and sustain this behavior are incomplete. There is a need to identify explicit, clinically-relevant mechanisms of emotional eating behavior. Pain is a common stressor associated with increased weight and, potentially, altered eating behaviors. The present study applies the Fear Avoidance Model (FAM) of pain to examine processes that may explain the relationship between pain and increased weight while also providing the opportunity to examine specific mechanisms that may encourage eating during a variety of stressors.

Our aim is to better understand the impact of pain on eating behavior and the potential for the FAM to improve our understanding of the psychological mechanisms that promote eating during times of duress. A survey of 312 adults explored the link between pain experience and stress-induced eating, further examining the mediating effects of the psychological aspects of the FAM (e.g., anxiety sensitivity, catastrophizing, and pain-related fear). 24% of respondents reported persistent pain, and had significantly higher BMIs than their pain-free peers. All three FAM components were positively correlated with measures of emotional, external, and restrained eating. Anxiety sensitivity and catastrophizing significantly mediated the relationship between persistent pain and emotional eating behavior, while anxiety sensitivity alone mediated the relationship between persistent pain and external eating. Findings suggest pain may be associated with increased likelihood for emotional eating and that characteristics from FAM, in particular anxiety sensitivity and catastrophizing, may mediate the relationship between the presence of persistent pain and emotional eating behavior. Evidence-based treatments targeting anxiety sensitivity and catastrophizing could be useful to address emotional eating in individuals struggling with both weight and chronic pain.



## 1. Introduction

Despite significant efforts to promote prevention and achieve sustained weight loss, obesity continues to be a public health crisis (Flegal, Carroll, Kit, & Ogden, 2012; Ogden, Carroll, Kit, & Flegal, 2012). Stress-induced eating contributes to weight gain (Hays & Roberts, 2008; Konttinen, Männistö, Sarlio-Lähteenkorva, Silventoinen, & Haukkala, 2010), is positively correlated with body mass index (BMI) (Gibson, 2012), and compromises metabolic health (Epel et al., 2004). Recent models of stress-induced eating focus on biological factors, such as stress-related neurobiological dysfunction, to explain hyperphagia in response to stress (Adam & Epel, 2007; Sinha & Jastreboff, 2013). While these models acknowledge an important role for "individual differences" (e.g., psychological factors), they often fail to specify psychological mechanisms that initiate and maintain stress-induced eating (Greeno & Wing, 1994; Sinha & Jastreboff, 2013). Dietary restraint and affect regulation have been targeted as psychological factors that explain stress-induced eating, but findings here are inconsistent and our understanding has been criticized as incomplete, oversimplistic, and unresolved (Greeno & Wing, 1994; Lowe & Kral, 2006; Macht, 2008). Perhaps, more importantly, findings have not

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offered clear guidance for intervention approaches that can be integrated into behavioral treatments for obesity. Researchers and clinicians in the area have called for the identification of explicit, clinically-relevant mechanisms of stress-induced eating (Epel, Stoney, Czajkowski, & Hunter, 2013; Greeno & Wing, 1994).

### 1.1. Stress & eating behavior

While stress alters eating behavior, it does not do so unilaterally. In animal and human laboratory studies, both acute and chronic stress are associated with both decreased and increased caloric intake, the latter particularly when highly palatable foods are available (Adam & Epel, 2007; Gibson, 2012; Moore & Cunningham, 2012; Sinha & Jastreboff, 2013; Torres & Nowson, 2007). These conflicting results likely reflect individual variability in patterns of stress-related eating in the general population (Torres & Nowson, 2007). Prevalence estimates of stress-induced eating suggest approximately one-third to half of all individuals are prone to increases in intake during increasing stress, one-third to two-thirds report decreases in intake, and approximately one-quarter report no change (Gibson, 2012; Macht, 2008). While research has yet to describe characteristics associated with each of these subgroups, the variety in response (increasing consumption, decreasing consumption, no change) helps explains discrepant findings and is consistent with biological mechanisms of eating under stressors of varying intensities (Adam & Epel, 2007; Gibson, 2012; Sinha & Jastreboff, 2013; Torres & Nowson, 2007). Thus, while stress-induced eating has been identified as a risk factor for weight gain and obesity, we have yet to clearly identify the psychological vulnerabilities that promote stress-induced eating in individuals at risk for this behavior.

To date, attempts to characterize psychosocial mediators of stress-induced eating have largely focused on dietary restraint and affect regulation. Experimental evidence suggests a relationship between dietary restraint and stress-induced eating, as it appears those high in restraint appear more likely to engage in stress-induced eating (Conner, Fitter, & Fletcher, 1999; Habhab, Sheldon, & Loeb, 2009; Macht, 2008; O'Connor, Jones, Conner, McMillan, & Ferguson, 2008; Oliver, Wardle, & Gibson, 2000). However, restraint alone does not adequately explain stress-induced eating (Lowe & Kral, 2006). Without a clear understanding of who is vulnerable to the obesogenic effects of stress and, specifically, the mechanisms by which they may become vulnerable, we are insufficiently prepared to craft targeted, behavioral interventions to effectively treat stress-induced eating and reduce the morbidity associated with obesity.

### 1.2. Pain as a stressor that influences eating behavior

While a number of different stressors may trigger eating behavior (Sinha & Jastreboff, 2013), a growing body of evidence links the experience of pain with preference for high-calorie foods and caloric overconsumption (Janke & Kozak, 2012; Meleger, Froude, & Walker, 2014). Pain is a common stressor and a likely risk factor for obesity (Stone & Broderick, 2012), making it a particularly relevant target to examine in relation to stress-induced eating. Evidence supporting sweet- and fat-induced analgesia is robust and replicated, so much so that the administration of sucrose has become accepted standard practice for managing acute procedural pain in NICUs (Holsti & Grunau, 2010). However, far less is known about the impact of pain on eating behavior. If ingesting high-fat, high-sugar foods increases pain tolerance (as research suggests it does), then it is a reasonable hypothesis that humans may seek such foods to help them cope with experienced pain. For example, overweight individuals have demonstrated increases in reported stress and hunger ratings as well as serum levels of cortisol and ghrelin following a cold pressor pain task (Geliebter, Carnell, & Gluck, 2013), and similar findings have been demonstrated in obese samples (Gluck, Geliebter, Hung, & Yahav, 2004; Gluck, Yahav, Hashim, & Geliebter, 2014). However, caloric intake was not examined. Similarly, cross-sectional studies have demonstrated unhealthy patterns of eating including low levels of fruit and vegetable consumption and high intake of sugars and saturated fats in those with chronic pain disorders (Meleger et al., 2014), but such examinations lack experimental controls and detailed measurement.

### 1.3. Fear avoidance model and relevance to eating behavior

The lack of research examining pain's influence on eating behavior is unfortunate. Individuals who become hyperphagic in response to pain may use food to cope when pain or other stressors occur, a pattern that leads to positive energy balance and, ultimately, weight gain, obesity, and increased pain and disability. Examining pain as a specific stressor that contributes to caloric overconsumption presents an additional opportunity to understand psychological mechanisms that encourage stress-induced eating more broadly. Specifically, the Fear-Avoidance Model (FAM) of pain outlines psychological mechanisms relevant to obesity outcomes and behavioral treatment approaches (Crombez, Eccleston, Van Damme, Vlaeyen, & Karoly, 2012; Leeuw et al., 2007; Vlaeyen & Linton, 2000). The successful application of the FAM to understand stress-induced eating would provide insight into mechanisms of weight gain that would allow for the development of more effective intervention approaches.

The Fear-Avoidance Model (FAM) of pain is a leading paradigm for understanding disability and chronicity associated with musculoskeletal pain, and has wide support in cross-sectional, longitudinal, and experimental research (Leeuw et al., 2007; Pincus, Smeets, Simmonds, & Sullivan, 2010). The FAM is a positive-feedback model that suggests two divergent sets of emotional, cognitive, and behavioral responses following acute pain. Depending on which responses are activated, an individual may either enter a cycle of confrontation coping or a cycle of exacerbated and ongoing pain and disability. The first occurs when acute pain is perceived as non-threatening and there is low fear and anxiety for the pain experienced, daily activities continue and functional ability is maintained. The second occurs when acute pain is interpreted catastrophically, pain-related fear and anxiety occur. Fear of pain promotes avoidant/escape behaviors, responses that can increase pain and worsen disability.

Pain catastrophizing, anxiety sensitivity, and pain related fear are often identified as primary concepts in the FAM. Pain catastrophizing is the cognitive element of a fear response during which pain is interpreted as extremely threatening. In the FAM, pain catastrophizing is seen as the precursor of pain-related fear. Catastrophic misinterpretations of the pain experience lead to an excessive fear of pain and fear of activities that may cause pain, fears that eventually may generalize to a fear of physical movement which leads to disuse, disability, and more pain. While many have identified the concept of anxiety sensitivity—a generalized fear of anxiety-related sensations due to the belief they may have harmful consequences—as important to the FAM, there is not yet consensus as to whether it contributes to the pain experience through its effects on fear of pain, through its effect on pain catastrophizing, or through other cognitive processes (Stewart & Asmundson, 2006).

Applying the FAM to examine stress-induced eating in response to pain may prove useful to understanding processes that influence increased caloric consumption in response to a variety of stressors. According to the FAM, psychological vulnerabilities (e.g.,