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DO INDIVIDUALS WITH FUNCTIONAL GASTROINTESTINAL SYMPTOMS EXPERIENCE EMOTIONS DIFFERENTLY? AN EXAMINATION OF EMOTIONAL RESPONDING DURING A SERIES OF EMOTION INDUCTION TASKS

A Dissertation

presented in partial fulfillment of requirements

for the degree of Doctor of Philosophy

in Clinical Psychology

The University of Mississippi

Sara Michelle Witcraft

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ABSTRACT

Functional gastrointestinal disorders (FGIDs) are disorders of the brain-gut axis characterized by physiological and emotional disturbances, including heightened comorbidity with psychiatric disorders. Individuals with FGIDs experience difficulties with emotional processing, including the awareness, identification, and regulation of emotions. Although emotional processing is implicated in the maintenance and exacerbation of FGID pathology, literature examining these associations is limited. The current study aimed to enhance understanding of the emotional processing of individuals with FGIDs. We recruited individuals with and without FGID symptoms and examined the: (1) association between gastrointestinal distress and psychopathology; (2) association between gastrointestinal distress and emotional and physiological responsivity; (3) awareness, identification, and intensity of emotional experiences; and (4) distress, emotion regulation abilities, and deployment of specific emotion regulation strategies during emotion inductions. In total, 291 university students ($M_{age} = 20.59$; SD = 5.50; 72.5% female; 82.5% White) completed an online battery of self-report questionnaires. A portion of participants (n = 52) engaged in a neutral induction and a series of experimental emotion inductions to elicit anxiety, disgust, and sadness. Gastrointestinal distress was positively correlated with psychopathology and physiological and emotional responsivity, but not use of emotion regulation strategies. Throughout emotion inductions, participants in the FGID and control groups did not differ in their differentiation of negative emotions, distress, or emotion regulation. One significant within-subjects main effect of induction emerged, such that SUDS following the emotion inductions were higher than following the neutral induction, and that

anxiety and disgust resulted in greater SUDS than the sadness induction. Consistent with extant literature, findings suggest that college students may be at risk for FGID symptoms, and that gut symptoms are associated with both heightened emotional and physiological reactivity. Significant and null findings must be considered in the light of several limitations, including a nonclinical sample of participants with relatively low gastrointestinal distress, novel videoconferencing methodology, and data collection during a global pandemic. Future studies should replicate and extend this study by using a larger, in-person sample of individuals with verified FGIDs to determine whether functional gastrointestinal symptoms are indeed associated with difficulties distinguishing and regulating emotions.

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CHAPTER 1

INTRODUCTION

Gastrointestinal symptoms are common in the general population (Drossman et al., 1993; Thompson et al., 2002) and rates are even higher among individuals with psychological disorders, such as anxiety and depression (Hillila & Farkkila, 2004; Van Oudenhove et al., 2016). Gastrointestinal disorders that are psychosocial in nature are called functional gastrointestinal disorders (FGIDs). These disorders are characterized by visceral hypersensitivity (Mertz, 2003) and symptom perception rather than structural or biochemical abnormality (Drossman, 2016; Farrokhyar et al., 2006). The comorbidity of FGIDs and emotional disturbance results from a connection between the brain and gut, which allows bidirectional communication between the brain (i.e., central nervous system) and gastrointestinal tract (i.e., enteric nervous system; Drossman, 2016). Given the influence of neurophysiological, social and/or environmental, and psychological aspects in the manifestation of FGIDs, a biopsychosocial model is commonly used to understand FGIDs (Van Oudenhove et al., 2016). The dysregulation of the central and enteric nervous systems not only contributes to gastrointestinal distress bidirectionally, but also deficits in emotional processing (Elsenbruch et al., 2010; Fichna & Storr, 2012; Van Oudenhove et al., 2016). Specifically, individuals with FGIDs experience high levels of alexithymia, or the inability to understand and express emotions (Larsen et al., 2003; Porcelli et al., 1999; Portincasa et al., 2003), as well as difficulties identifying (Fournier et al., 2018) and regulating emotions (Mazaheri, 2015; Stanculete et al., 2015; Zvolensky et al., 2018).

Despite these proposed associations, there is limited research investigating emotion regulation processes within this population, including use of specific emotion regulation strategies and underlying emotion regulation potential. The current study describes the integration of these concepts and contributes to this body of knowledge by using an experimental approach to further understand responses to emotions among individuals with FGID symptoms.

Gastrointestinal Disorders

From the neonatal environment and continuing throughout the lifespan, gastrointestinal health is paramount to overall health (Hollister et al., 2014) and is generally considered to be a requirement for well-being (Drossman, 2016). In addition to aiding digestion and colon health (Guarner & Malagelada, 2003), a healthy gastrointestinal system contributes to immune system regulation (Cebra, 1999; Hansen et al., 2012; Kindt et al., 2009), prevention of allergic disease (Bisgaard et al., 2011), nondigestible nutrient absorption (Hooper et al., 2002), prevention of multisystem organ failure (Guarner & Malagelada, 2003), fewer physical (e.g., lower blood pressure) and mental health (e.g., depression) comorbidities, and greater overall health (Claesson et al., 2012). As such, dysregulation of the gastrointestinal tract results in myriad acute and chronic health issues (Aziz et al., 2013).

Gastrointestinal dysregulation is classified within three domains: organic gastrointestinal disorders, motility disorders, and functional gastrointestinal disorders (FGIDs; Drossman, 2016). Organic gastrointestinal disorders consist of those which result from organs with structural abnormalities, such as inflammatory bowel disease (IBD; i.e., Crohn's disease, ulcerative colitis) and gastroesophageal reflux disease (GERD), and may be identified by gut pathology through means such as endoscopy. Next, motility disorders, such as gastroparesis, are characterized by disturbances in organ functioning which result in altered motility in the gut and heightened

sensitivity to visceral sensations. Lastly, FGIDs, such as irritable bowel syndrome (IBS), are not caused by structural or biochemical abnormalities, but instead are characterized by the interpretation and experience of gastrointestinal symptoms (Drossman, 2016). As such, these symptoms cannot be seen by blood work or other tests such as endoscopies or x-rays, but rather, are diagnosed based on self-reported symptoms.

In comparing organic gastrointestinal diseases and disorders of motility with FGIDs, there are notable discrepancies in the prevalence and impact of these disorders. Patients with IBD spend more days hospitalized for their symptoms, often require surgery due to complications of their disease (e.g., hemorrhaging, perforation, intestinal obstructions), and experience higher risk of colorectal cancer contributing to increased rates of mortality (Ananthakrishnan et al., 2008; Barton & Ferguson, 1990; Berg et al., 2002; Pohl et al., 2000; Witte et al., 2000). Although FGIDs do not typically result in such severe morbidity or mortality, they occur more frequently in the general population and are associated with their own costs and consequences.

As many as 62 - 69% of individuals in the general population experience at least one FGID in any given three-month period (Drossman et al., 1993; Thompson et al., 2002). Relative to individuals without FGIDs, individuals with FGIDs utilize primary care and gastroenterology clinics more frequently, have a greater number of missed or less productive work days, and engage in fewer leisure activities (Farrokhyar et al., 2006). IBS is the most commonly diagnosed FGID and occurs in 11.2% of the worldwide population and between 5 - 25% of gastroenterological patients, accounting for 36% of all gastroenterologist visits (Chang, 2004; Lovell & Ford, 2012). In contrast to FGIDs, organic gastrointestinal and motility disorders occur at much lower rates. For instance, IBD affects less than 1% of individuals worldwide (Ng et al.,

2017), GERD between 5 – 20% (Dent et al., 2005; Fujiwara et al., 2005), and gastroparesis 6.3% (Jung et al., 2009).

While the physiological underpinnings of organic and motility disorders lend to a straightforward causal explanation, the etiology of FGIDs is more nuanced with diverse influences. FGIDs can affect numerous locations throughout the gastrointestinal tract, including the esophagus (e.g., functional heartburn; Aziz et al., 2016), gastroduodenal region (e.g., functional indigestion; Stanghellini et al., 2016), bowels (e.g., IBS; Lacy et al., 2016), gallbladder and sphincter (e.g., pain in this region that may result in nausea and vomiting; Cotton et al., 2016), and anorectal region (e.g., fecal incontinence; Rao et al., 2016). Additionally, centrally mediated disorders are a class of FGIDs that are independent of motility disturbances, and are instead are associated with disinhibition of pain signals that result in near constant abdominal pain which may be produced by digestive or non-digestive organs (e.g., urinary or gynecologic systems; Keefer et al., 2016). Although there are numerous FGIDs, they all have one commonality in that they are all disorders of brain-gut interaction (Drossman, 2016).

The brain-gut axis is characterized by the bidirectional relationship of the brain and gastrointestinal tract through the central nervous system (CNS) and the enteric nervous system (ENS), which control the functions of the brain and gastrointestinal tract, respectively (Cryan & O'Mahony, 2011; Drossman, 2016; Vanner et al., 2016). Importantly, the ENS, which is part of the autonomic nervous system, has both motor (i.e., efferent information) and sensory (i.e., afferent information) functions, and therefore is uniquely able to function without input from the CNS (Rao & Gershon, 2016). The CNS conveys information regarding emotional (e.g., fear, anger) and cognitive (e.g., pain regulation) stimuli through neurotransmitters to the ENS, which can cause gut dysfunction including decreased motility, diarrhea, and gut pain. Conversely, the

ENS delivers information regarding increased motility or visceral inflammation, amplifying visceral pathways to the CNS and thereby increasing the experience of pain, which may then alter mental functioning to beget anxiety and depression (Drossman, 2016). Notably, colloquialisms such as "I find this hard to swallow," "I can't stomach it," "I have butterflies in my stomach," and "my stomach is in knots" convey this relationship between the gut and stress and emotion (Drossman, 2016). As such, the brain-gut axis is critical for gut health, including regulation of food intake and digestion, gut sensations, and bowel movements, as well as psychological health and overall well-being (Fichna & Storr, 2012).

Stress plays a critical role in the bidirectional relationship of the CNS and ENS, which may result in FGID symptoms. Increased sympathetic activity of the autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis are the main systems responsible for stress response in humans (Fichna & Storr, 2012). Specifically, emotional arousal such as stress excites the HPA axis, which produces and releases cortisol (Vanner et al., 2016), the hormone responsible for increased allostatic load (Braveman & Gottlieb, 2014). The influx of cortisol due to stress contributes to increased autonomic activity as well as exaggerated HPA response, which lends to altered (i.e., decreased) gastrointestinal motor and immune functioning, leading to visceral signaling. Together, these systems contribute to worse gastrointestinal symptoms (Vanner et al., 2016; Van Oudenhove et al., 2016). As such, the HPA axis and ANS are thought to be the mechanisms through which brain and gut communicate (Van Oudenhove et al., 2016). Although the brain-gut axis is a critical component of FGIDs, it is important to consider other factors that contribute to the etiology and maintenance of these symptoms. Consistent with other chronic health conditions (see Sallis et al., 2015), a biopsychosocial conceptualization of FGIDs provides further understanding of relevant etiological and maintenance factors.

Biopsychosocial Model of FGIDs

As illustrated by the biopsychosocial model, the biological, psychological, and social components of FGIDs are reciprocally related to one another, in that they each affect and are affected by one another (Van Oudenhove et al., 2016). For instance, environmental factors such as culture and parental behavior alter CNS structure and function (e.g., modulation of visceral signals, fear conditioning) and can influence psychological (e.g., anxiety, hypervigilance) and gut factors (e.g., visceral sensation, motility). Through the brain-gut axis, altered CNS functioning and psychological morbidities can influence gut physiology and sensations, and vice versa. The reciprocal relations that the environment, brain, and gut have with one another contribute to the clinical presentation exhibited by individuals with FGIDs, including symptoms and disease severity, comorbidities, and resulting behavior. This resultant symptom presentation adversely affects quality of life and results in increased health care utilization, which in turn worsens symptom severity and other psychological and health-related comorbidities (Levy et al., 2006; Van Oudenhove et al., 2016).

Psychosocial factors are thought to underlie FGID pathophysiology (Van Oudenhove et al., 2016), such that symptomatology is centered around psychosocial stressors and symptoms, rather than physical or functional abnormalities as found in organic gastrointestinal and motility disorders (Drossman, 2016; Farrokhyar et al., 2006). In chronic health conditions, pathological fear and behavior negatively affect psychosocial functioning, which in turn contribute to the maintenance and exacerbation of pathophysiology. Reciprocally, elevations in autonomic activity may contribute to avoidant behavior through processes such as catastrophization and perceived inability to cope with aversive physiological sensations (e.g., pain; Aue et al., 2013; Carver & Blaney, 1977; Norton & Asmundson, 2003), further exacerbating fear of physiological

(e.g., gut) sensations. Development of fear is posited to be threefold: 1) respondent conditioning of the feared stimulus, which causes the initial development of the fear; 2) operant conditioning, which maintains the fear through negative reinforcement of avoidant behavior; and 3) habitual avoidance, in which avoidance is maintained long-term *without* contact with the reinforcer (i.e., reduction in fear; LeDoux et al., 2017). Because contact with the feared, conditioned stimulus is necessary for extinction to take place (Craske et al., 2008, 2014; Foa & Kozak, 1986), extinction is unable to occur due to the processes involved in habitual avoidance (LeDoux et al., 2017).

In FGIDs, fear is introduced when an unconditioned gastrointestinal stimulus, such as pain or visceral sensations, occurs and is misappraised, which results in the unconditioned response of fear or threat of something bad occurring (e.g., an accident). Over time, gastrointestinal sensations become associated with a threat response, and the two become conditioned to one another. In order to prevent a feared outcome from occurring, an individual may engage in avoidance behaviors each time gastrointestinal sensations occur. Consequently, when the already unlikely feared outcome does not occur, the reason for this absence is misattributed to the avoidance, thereby negatively reinforcing this behavior. Through long-term avoidance, the individual becomes preoccupied with gastrointestinal symptoms and believes that they must be avoided, which reinforces avoidance even when symptoms are not present. Thus, extinction and new learning are disallowed as avoidance occurs without reinforcing properties (i.e., it becomes habitual). Principles of respondent and operant conditioning are exhibited throughout each domain of the biopsychosocial model.

Biological Influences

As disorders of brain-gut interaction, FGIDs are characterized by a combination of the following physiological and neuropsychological dysfunctions: disturbance in motility, altered

mucosal and immune functioning, altered gut microbiota, visceral hypersensitivity, and CNS dysregulation (Drossman, 2016; Drossman & Hasler, 2016; Tillisch et al., 2011). Stress and anxiety influence motility in the stomach (Geeraerts et al., 2005; Van Oudenhove & Aziz, 2013) and colon (Fukudo et al., 1998), and through activation of the HPA axis stress also has a deleterious effect on colonic mucosal functioning (Piche et al., 2007; Vanuytsel et al., 2014). Additionally, a healthy enteric microbiome is paramount to the development of the CNS and ENS and subsequently brain-gut communication, as well as the maintenance of homeostasis and physiological functioning throughout the lifetime (Carabotti et al., 2015). As such, disruption of the microbiome results in CNS (Cryan & Dinan, 2012; Stilling et al., 2014) and gastrointestinal dysfunction (Clarke et al., 2012), and increases stress reactivity through its effect on the HPA axis (Carabotti et al., 2015). Together, these psychophysiological processes influence emotion, cognition, and behavior by increasing anxiety, heightening attention toward visceral sensations, and conditioning avoidant responding, respectively.

Beyond physiological dysfunctions occurring in the gut, dysregulation of CNS activity also has detrimental effects on gastrointestinal health (Tillisch et al., 2011). For a typical individual, interoceptive gut cues that are communicated to and from the CNS to the ENS are not consciously perceived. However, individuals with FGIDs are hypersensitive to these signals (Boeckxstaens et al., 2016; Vanner et al., 2016; Van Oudenhove et al., 2016). For those with FGIDs, when a noxious (or sometimes, non-noxious) stimulus is introduced in the gut, the ENS signal is modulated by cognitive and affective neurocircuits in the brain, which results in perception of pain or feelings of discomfort in the gut, known as visceral hypersensitivity. The presence of psychopathology, such as anxiety and depression, results in greater visceral pain in IBS patients, which suggests that CNS processing of visceral signals may be modulated by

symptoms of anxiety and depression, resulting in increased pain perception (Elsenbruch et al., 2010; Van Oudenhove & Aziz, 2013). In addition, the brain is able to upregulate (i.e., increase pain) or downregulate (i.e., decrease pain) neural signals that contribute to the perception of pain. Individuals with FGIDs are not as effective at downregulating this sensory information, which results in greater gastrointestinal pain. In addition, the experience of psychosocial distress decreases the ability to downregulate further, contributing to the experience of even more pain (Drossman, 2016).

Social and Environmental Influences

Although individualized patient presentations are central to FGIDs, there are numerous cultural, social, and environmental factors that also influence one's perception of and symptoms related to FGIDs (Francisconi et al., 2016). For instance, perception and localization of pain related to IBS differs in Westerners compared to non-Westerners (Gerson et al., 2008; Zola, 1966). In addition, there are regional and cultural differences in the language used to describe symptoms (Callister, 2003). For example, the word "bloating" is often used by English speakers to describe the sensation that one's abdomen is distended, but this particular word does not translate to other languages. Instead, words such as "swelling" and phrases such as "feeling blown up like a balloon" are used to describe the same sensations of abdominal distention (Francisconi et al., 2016; Gwee et al., 2009; Quigley et al., 2012). Moreover, one's culture influences which foods are or are not eaten as well as attributions bestowed upon certain food and food groups, both of which may activate circuits of the CNS and contribute to functional symptoms (Mayer et al., 2006).

In addition to one's larger culture, familial and parental factors and early life stressors influence the presence, perception, and symptomatology of FGIDs. Although IBS has higher

concordance rates among monozygotic compared with dizygotic twins (Levy et al., 2001; Morris-Yates et al., 1998), there is also evidence that the presence of IBS is highly influenced by parental IBS, particularly through social learning (Levy et al., 2001). For instance, parents with IBS tend to pay more attention to their children's gastrointestinal symptoms, thereby positively reinforcing illness behavior and increasing the child's report of stomachaches and school absences (Levy et al., 2004). Ultimately, this pattern contributes to greater health-care seeking for both non-gastrointestinal and gastrointestinal symptoms (Levy et al., 2000). In a similar vein, through modeling and/or reinforcement, children's abdominal symptoms may correspond with their parents' complaints of anxiety, depression, and somatic catastrophization of abdominal pain (for a review, see Van Oudenhove et al., 2016).

In addition to parental influences, early life stressors such as physical, emotional, and especially sexual abuse are reported at higher rates among individuals, particularly women, with IBS relative to those without FGIDs (Bradford et al., 2012; Drossman et al., 1996). These adverse childhood events are associated with increased severity of FGID symptoms, psychopathology and psychosocial deficits, health-care utilization, and worse overall functioning in adulthood (Drossman, 2011; Lackner & Gurtman, 2004). As such, chronic stress associated with these adverse outcomes can result in poorer treatment outcomes and worse gastrointestinal symptom severity (Bennett et al., 1998). Extant literature purports that exposure to psychological trauma may have a deleterious effect on the CNS, whereby traumatic exposure causes dysregulation of the HPA axis resulting in increased release of cortisol and inflammatory substances (Drossman, 2011). The release of these factors may result in visceral hypersensitivity and inhibition of neural signals that downregulate pain, thereby resulting in enhanced experience of gastrointestinal distress and pain. Through the dysregulation of CNS functioning and increased visceral pain, psychosocial distress such as anxiety and depression become more severe (Drossman, 2011; Drossman et al., 1996; Vaccarino et al., 2009).

Psychological Influences

Psychological distress is proposed to be both a vulnerability to and outcome of FGIDs (Van Oudenhove et al., 2016). A prospective population-based study found that individuals with FGIDs without anxiety at baseline were significantly more likely to develop symptoms of anxiety over a 12-year span, and similarly, individuals who had anxiety without FGIDs at baseline had a higher incident rate of FGIDs at follow-up (Koloski et al., 2012). Between 40% and as many as 90% of FGID patients at gastroenterology clinics experience psychological comorbidities, relative to 25% of patients with organic gastrointestinal conditions and 20% of individuals without FGIDs (Drossman et al., 2002). Although depression is common (e.g., 17.2%; Hillila & Farkkila, 2004) among individuals with FGIDs, anxiety disorders are the most common psychiatric comorbidity, with an approximate 30 - 50% overlap in co-occurrence (Van Oudenhove et al., 2016) relative to the 21.3% annual prevalence rate in the general population (Kessler et al., 2012). Such psychopathology may contribute to altered visceral processing and heightened activation of the ANS and HPA axis (e.g., cortisol release; Lembo et al., 1999; Wu, 2012), increasing sensitivity to gut pain (Elsenbruch et al., 2010) and accounting for the heightened stress response seen in patients with bowel symptoms (i.e., flare-ups; Dickhaus et al., 2003; Posserud et al., 2004). This alarmingly high co-occurrence could be due to shared cognitive and affective mechanisms such as hypervigilance of bodily sensations, which is exacerbated through processes such as catastrophization and contributes to use of maladaptive coping (e.g., avoidance; Lee et al., 2009; Van Oudenhove et al., 2016).

Visceral sensitivity, or gastrointestinal-specific anxiety, describes the cognitive, affective, and behavioral responses that occur as a result of gastrointestinal sensations or symptoms, or in the presence of contexts that elicit such sensations (Labus et al., 2007). Visceral sensitivity is purported to be a key predictor of IBS above worry and anxiety sensitivity (Hazlett-Stevens et al., 2003) and distinguishes IBS patients from individuals with IBS who have not sought medical aid as well as controls (Labus et al., 2007). Individuals with heightened visceral sensitivity, such as those with FGIDs (Mertz, 2003), have the propensity to label gastrointestinal sensations negatively, experience lower tolerance of these sensations (Naliboff et al., 1997), and are more reactive to low levels of abdominal and bowel sensations and pain (vs. healthy controls; Bradette et al., 1994). Furthermore, visceral sensitivity has been shown to have an even greater effect on quality of life, health-care utilization, and costs than the gastrointestinal symptoms themselves (Chang, 2004).

Visceral hypersensitivity is posited to contribute to avoidant behavior through catastrophization. Specifically, individuals with FGIDs may misinterpret the consequences of their gut sensations and underestimate their ability to cope with feared outcomes (Hunt et al., 2009), which contribute to worse psychosocial outcomes (Hunt et al., 2009) such as increased pain and worry (Lackner & Quigley, 2005; Van Oudenhove et al., 2016). At the same time, avoidance of situations (e.g., restaurants) or stimuli (e.g., certain foods) that may elicit gut sensations further reinforces the belief that gut sensations are dangerous and should be avoided. This habitual avoidance prevents extinction of the undesirable avoidant behavior (Bonnert et al., 2018; LeDoux et al., 2017). In the long-term, this avoidance leads to reduced quality of life (Van Oudenhove et al., 2016) and maintains gastrointestinal symptom severity (Bonnert et al., 2018) through prevention of emotional processing and new learning (e.g., 'this sensation does not always mean that I will have an accident;' Foa & Kozak, 1986). Given the role of avoidance in maintaining FGID pathology (Bonnert et al., 2018) and the frequent co-occurrence of emotional difficulties (Drossman et al., 2002), further exploration of the impact of emotions is critical for understanding how they perpetuate FGIDs.

Emotion and Its Regulation

Although "gut feelings" are frequently described as indicators of emotional responses to environmental cues, research examining these experiences in the context of FGIDs is limited. Historically, emotion research has attempted to understand the affective (Cannon, 1927), physiological (James, 1884; Lange, 1922), neurological (Davidson, 1984; Ekman et al., 1983), cognitive (Lang, 1979; LeDoux, 1984; Schachter & Singer, 1962), and behavioral (Panksepp, 1982) underpinning of emotions. These seminal works have paved the way for contemporary emotion research, which integrates these components to emphasize the physiological and affective correlates of discrete emotions (Gilchrist et al., 2016; Kragel & LaBar, 2013; Levenson et al., 2017; Prkachin et al., 1999). Consistent with the current conceptualization of FGIDs, the biopsychosocial model underscores the interlinking of the neurophysiological and affective influences upon FGID symptom presentation.

Regardless of valence, emotions are associated with increased autonomic arousal, such as increased heart rate and electrodermal activity (Levenson et al., 2017), which over time may contribute to dysregulation of the sympathetic nervous system and parallel stress-related response of the HPA axis (Charmandari et al., 2005), leading to worse FGID symptomatology and associated outcomes (Vanner et al., 2016; Van Oudenhove et al., 2016). However, discrete emotions differ in specific physiological responding (Kragel & LaBar, 2013; Prkachin et al., 1999). For instance, cardiovascular output is typically higher (e.g., increased systolic blood

pressure, lower stroke volume) in negative emotions relative to positive, and specifically, higher for sadness relative to fear and anger, but bears no relation to disgust (Prkachin et al., 1999). Rather than autonomic activation, disgust is characterized by lower cardiovascular activity (e.g., lower systolic blood pressure, higher stroke volume) and respiration (Gilchrist et al., 2016). That is, emotions such as anxiety, fear, and anger are associated with sympathetic nervous system activation ('fight or flight'; Lench et al., 2011), while disgust is associated with parasympathetic nervous system activation. In addition to the established effect of sympathetic nervous system arousal on FGIDs, lower activity of the parasympathetic nervous system has also been implicated, which may account for greater sympathetic activation and worse gastrointestinal symptom severity (Manabe et al., 2009; van Orshoven et al., 2006).

Another indication of the discrete nature of emotions is differences in experiential components, such as behavior and cognition (Roseman et al., 1994). Although negatively valenced emotions such as anxiety, sadness, and disgust are associated with behavioral avoidance, they differ in presentation and function (Levenson et al., 2017). Avoidance related to anxiety aims to escape from anxiety-provoking situations (Beck et al., 2010), sadness is characterized by avoidance of activity through behavioral inhibition (Hong, 2007), and avoidance of disgust functions to protect from disease and bodily harm (Oaten et al., 2009). Regarding cognition, those relevant to anxiety concern the future (i.e., worry, catastrophization), sadness concern the past (i.e., rumination), while disgust relates to repulsion (Roseman et al., 1994). Individuals with FGIDs frequently engage in avoidance behaviors (Bonnert et al., 2018), catastrophize gut pain and social consequences of gastrointestinal symptoms (Hunt et al., 2009, 2014; Lackner & Quigley, 2005), and experience worse psychosocial outcomes after ruminating (Martin & Chapman, 2010).

Despite broad physiological and behavioral similarities, there are clearly distinct differences in the processes by which emotional information is internalized and expressed in general and within FGIDs. Emotion differentiation, or the ability to make distinctions between discrepant emotions and physiological sensations (Oh & Tong, 2020), appears to be particularly salient to HPA axis activation (Hua et al., 2014), suggesting that difficulties distinguishing emotional experiences increase cortisol production. Given individuals with FGIDs may experience difficulty with identifying and being aware of emotions (Fournier et al., 2018) and the role of HPA axis activity and heightened cortisol levels in FGID symptomatology (Butler et al., 2019), further examination of emotion processes in FGIDs is warranted.

Beyond the physiological regulation of emotion information, a growing area of literature has emphasized emotion regulation as a psychological process. Emotion regulation, defined as the ability to be aware of, identify, and regulate or respond to emotions, is paramount to emotional wellbeing (Aldao et al., 2010) and physical health (Smyth & Arigo, 2009). Two prominent models of emotion regulation have been used to inform an integrative conceptualization of emotion regulation (Gratz et al., 2018). At a micro level, the process of identifying and expressing an emotion is a function of the particular emotion regulation strategy employed (i.e., process or strategies model; Gross, 2015a). More broadly, the dispositional potential to understand and respond to an emotional experience is considered one's emotion regulation ability (i.e., abilities model; Gratz & Roemer, 2004).

With regard to the strategies model, emotion regulation focuses on the timing and type of emotion regulation strategy selected in a given situation in the service of one's goal(s), and the emotional consequences of selecting a particular strategy (Gross, 1998, 2015a; Gross et al., 2011). The specific strategy selected may have adaptive or maladaptive consequences on future

strategy selection and resultant emotional experiences. One commonly studied maladaptive strategy is expressive suppression, described as the intentional avoidance of emotional expression and experience, which paradoxically increases sympathetic nervous system activation and the intensity and frequency of the undesired emotion (Gross, 1998). Conversely, cognitive reappraisal is considered an adaptive regulation strategy that reduces the emotional impact of a situation by engaging in thought restructuring, effectively reducing physiological activation in emotionally salient situations (Gross, 1998). Emotion regulation strategies can be used to either upregulate or downregulate both positive and negative emotions, such that the intensity, duration, and quality of an emotion are altered (Gross, 2015a). Accordingly, there are multiple steps to regulating one's emotions wherein individuals identify, select, and implement a specific emotion regulation strategy to achieve an identified goal (Gross, 2015b). Selected strategies may be subsequently modified in accordance to one's physiological and behavioral responding in a given situation, resulting in alterations in cognition, behavior, and emotional experience both immediately and across time (Gross, 2015a; Gross & John, 2003).

The abilities model of emotion regulation is a higher-level approach to the understanding of emotion regulation that emphasizes the dynamic nature of emotions in various different situations, and focuses on the adaptive response to such emotions rather than the use of specific skills (Gratz et al., 2018). Gratz and Roemer (2004) posit that adaptive emotion regulation involves the ability to modulate one's emotional experience to inhibit unhelpful, impulsive behavior and instead act in line with one's goals in a given situation despite the occurrence of negative emotions. There are hypothesized to be six dimensions of the ability to regulate emotions: 1) awareness of emotions and emotional responding, 2) knowledge of the nature of emotions, 3) acceptance of and reaction to emotions, 4) flexible use of non-avoidant situation-

specific strategies, 5) ability to inhibit impulsive behavior when experiencing negative emotions, and 6) ability to engage in goal-directed behavior when experiencing negative emotions (Gratz & Roemer, 2004). Accordingly, the response to an emotional experience affects the trajectory and consequences of that emotion (Gratz et al., 2018).

In the integration of these models, emotion regulation abilities are posited to influence the particular strategies that are selected in any given situation and result in the generation of an emotional state consistent with the situational demands and goals, which may be either adaptive or maladaptive in the short- and/or long-term (Tull & Aldao, 2015). For example, an individual who is non-accepting of emotional experiences may be more likely to rigidly engage in avoidant strategies regardless of the situation, whereas an individual with awareness and acceptance of emotions may be more likely to stay attuned to emotions. On the other hand, emotion regulation strategies may also influence emotion regulation abilities. Repeated, inflexible use of a particular emotion regulation strategy, such as situational or experiential avoidance, regardless of the context may lead to a reduction in the ability to understand and distinguish emotional experiences. However, flexible and situationally appropriate use of emotion regulation strategies may increase self-efficacy for emotion regulation abilities by fostering awareness and acceptance of emotions. Taken together, the integration of the two emotion regulation models yields a positive (i.e., adaptive) or negative (i.e., maladaptive) feedback loop (Tull & Aldao, 2015). The positive feedback loop occurs when the chosen emotion regulation strategy (e.g., reappraisal) enhances underlying abilities (e.g., promoting acceptance and awareness of emotions), while the negative feedback loop occurs when the chosen strategy (e.g., suppression) undermines emotion regulation potential (e.g., reinforcing non-acceptance of emotions).

Numerous studies provide evidence that the inability to regulate emotions is associated with greater use of maladaptive or avoidant regulation strategies (e.g., experiential or situational avoidance, escape behaviors, expressive suppression) across a broad array of psychopathology (e.g., Briere et al., 2010; Gratz, 2007; Iverson et al., 2012). Difficulties in selecting appropriate regulatory strategies and the underlying ability to regulate emotions are considered key transdiagnostic features underlying numerous psychological syndromes (Aldao et al., 2010; Gratz et al., 2018) including depression (Brockmeyer et al., 2012; Ehring et al., 2010), anxiety (Amstadter, 2008; Vujanovic et al., 2008), posttraumatic stress disorder (Tull et al., 2007), borderline personality disorder (Baer et al., 2012; Gratz et al., 2013), and maladaptive behaviors such as substance use (Fox et al., 2008; Weiss et al., 2012) and disordered eating (Svaldi et al., 2012).

Additionally, difficulties in emotion regulation and use of avoidant strategies may also exacerbate outcomes related to chronic health conditions due to the association with increased HPA axis activity (Gratz et al., 2018; Gross & Levenson, 1997; Lam et al., 2009). However, studies examining the relationship between emotion regulation difficulties and health outcomes are limited. Emotion regulation difficulties have been implicated as a risk factor for worse psychological and physiological outcomes among various health populations including those with HIV/AIDS (Brandt et al., 2013), chronic pain (Kökönyei et al., 2014), and FGIDs (Mazaheri, 2015; Zvolensky et al., 2018). Individuals with gastrointestinal distress and pain may experience difficulties understanding, expressing, and regulating emotions (Fournier et al., 2018), which contribute to worse psychosocial distress (Zvolensky et al., 2018) and abdominal pain (Mazaheri, 2015). Therefore, further empirical work is needed to enhance the understanding of emotional identification, expression, and regulation among individuals with FGIDs, as well as the role these factors play in psychophysiological outcomes.

Emotion and Its Regulation in Relation to FGIDs

As described, the extant literature indicates that emotional processes may underlie the maintenance and exacerbation of FGID pathology; however, there are few studies investigating these associations. The brain-gut connection is attributed to shared underlying structures and processes involved in the cognitive-affective processes of emotional responding (e.g., arousal, conditioning, catastrophization, negative affect) and visceral perception. Specifically, the brain regions involved in the processing of visceral information are also involved in the generation and regulation of emotions and cognition (Van Oudenhove & Aziz, 2009). Given that the processing of visceral information is altered in individuals with FGID (Elsenbruch et al., 2010; Lembo et al., 1999; Posserud et al., 2004; Wu, 2012), it would follow that emotional processing would also be disrupted.

Research examining emotional processes in FGIDs has primarily emphasized alexithymia. Alexithymia means "without words for emotions," and is characterized by a reduction in the experience of and ability to verbalize emotions, absence of thinking about emotions, and difficulty identifying emotions (Larsen et al., 2003). Although empirical evidence suggests that alexithymia and emotion regulation are distinct (Edwards & Wupperman, 2017; Pandey et al., 2011), they overlap in that they are both associated with difficulties in identification and awareness of emotions (Swart et al., 2009). The prevalence of alexithymia among individuals with FGIDs ranges from 43.0% – 75.9% (Porcelli et al., 1999, 2004; Portincasa et al., 2003), which is markedly higher than among IBD patients (38.0%; Porcelli et al., 1999), the general population (9.9% – 25.7%; Mattila et al., 2006; Tolmunen et al., 2011),

and healthy controls (4.5%; Mazaheri et al., 2012; Porcelli et al., 1999). Alexithymia in FGIDs is associated with worse gastrointestinal symptom severity (Mazaheri et al., 2012) and psychological treatment outcomes (Porcelli et al., 2003; Portincasa et al., 2003). Additionally, despite the critical role of visceral sensitivity in FGIDs, alexithymia may be a more robust predictor of gastrointestinal symptom severity and treatment success than visceral sensitivity (Porcelli et al., 2014, 2017). Together, this body of literature indicates that individuals with FGIDs experience broad deficits in cognitive processing of emotions that may be characterized by the inability to identify, describe, and express emotions, which in turn worsens gastrointestinal symptom severity.

Similar to alexithymia, awareness of emotions is central to the ability to regulate emotions (Gratz & Roemer, 2004). Individuals with IBS experience difficulty identifying emotions, particularly during heightened stress, leading to increased experience of negative emotions such as sadness and anger (Fournier et al., 2018). The identification and expression of emotions is just one, albeit important, component of emotion regulation; there are far fewer studies examining other emotion regulation strategies or abilities within FGIDs. The studies that do exist suggest that these individuals engage in more avoidant coping styles than their healthy counterparts (Jones et al., 2006; Stanculete et al., 2015), and that this avoidance is associated with worse outcomes (Rutter & Rutter, 2002; Sugawara et al., 2017) relative to use of adaptive coping strategies (Torkzadeh et al., 2019). Individuals with FGIDs were also found to engage in rigid use of coping strategies regardless of the situation and frequent monitoring of internal and external stimuli, thereby exacerbating co-occurring psychological symptoms (Cheng et al., 2000). With regard to emotion regulation abilities, nonacceptance of emotions, emotion-driven impulse control difficulties, difficulty engaging in goal-directed behavior, and limited access to emotion regulation strategies have been associated with psychological symptoms among patients with FGIDs (Mazaheri, 2015). Additionally, emotion dysregulation accounted for the relationship between visceral sensitivity and psychopathology among individuals with current or past gastrointestinal symptoms (Zvolensky et al., 2018). Cumulatively, these studies provide preliminary evidence for the connection between emotion regulation processes and FGIDs, while also highlighting the need for additional research.

Current Study

The underlying neurophysiological mechanisms and the biopsychosocial model demonstrate numerous connections between FGIDs and emotion (e.g., Cryan & O'Mahony, 2011; Drossman, 2016; Vanner et al., 2016). As emotions are associated with increased autonomic arousal (Levenson et al., 2017), their dysregulation prolongs the autonomic stress response (Vanner et al., 2016; Van Oudenhove et al., 2016) and adversely affects physiological syndromes. As individuals with FGIDs experience deficits in the understanding and awareness of their emotional experiences (Fournier et al., 2018; Mazaheri et al., 2012), they are at increased risk for even worse autonomic reactivity, exacerbating their gastrointestinal distress. Research demonstrates a high co-occurrence between emotional disorders and FGIDs (Drossman et al., 2002); however, a more nuanced examination of emotional processes is needed to elucidate the connections between these disorders and identify potential targets for intervention. Specifically, further research is needed to examine how individuals with FGIDs distinguish emotions, regulate emotions across contexts, and whether or not they select and utilize regulation strategies flexibly or rigidly.

To address this gap in the literature, the current study examined emotional processing among individuals with current FGID symptoms. Specifically, this study used an experimental

induction and compared individuals with and without FGID symptoms with regard to their: (1) underlying propensity to regulate emotions; (2) awareness, identification, and intensity of emotional experiences; and (3) deployment of specific emotion regulation strategies. A series of three experimental emotion inductions were conducted to induce anxiety, disgust, and sadness, in addition to a control (neutral) induction. The primary dependent variables in the current study were: 1) psychological and emotional correlates of FGID symptoms, 2) characterization of emotional experiences, and 3) underlying emotion regulation abilities and use of specific strategies throughout emotion inductions. The study tested the following hypotheses:

- As demonstrated by previous research, individuals with FGID symptoms would report higher levels of depression, anxiety, and stress than those without FGID symptoms. *COVID-19 Update* (see below for additional details): Depression, anxiety, and stress would be significantly positively associated with gastrointestinal symptoms.
- 2. Individuals with FGID symptoms, relative to those without, would report higher emotional responsivity and poorer awareness of emotions, and given the role of physiological responding in emotion, greater physiological sensation awareness. *COVID-19 Update* (see below for additional details): Heightened emotional responsivity, difficulties understanding emotions, and greater physiological sensation awareness would be significantly positively associated with gastrointestinal symptoms.
- Throughout a series of emotion induction trials, individuals with FGID symptoms would exhibit significantly lower negative emotion differentiation abilities than individuals without FGID symptoms.

- Differences in distress and emotion regulation strategies and abilities would be observed between individuals with and without FGID symptoms across emotion induction trials relative to the neutral trial.
 - Individuals with FGID symptoms, relative to those without, would evidence significantly greater distress during emotion inductions compared to the neutral trial.
 - Individuals with FGID symptoms, relative to those without, would evidence greater deficits in the ability to regulate emotions during emotion inductions compared to the neutral trial.
 - c. Individuals with FGID symptoms, relative to those without, would evidence greater use of maladaptive emotion regulation strategies and limited use of adaptive emotion regulation strategies during emotion inductions compared to the neutral trial.

CHAPTER 2

METHOD

Participants

In total, 291 individuals enrolled in the study, of which 52 completed the experimental procedures. The sample included university students ($M_{age} = 20.59$, SD = 5.50; 72.5% female) enrolled at a large, Southeastern public university. The full sample was comprised of individuals who primarily identified as White (82.5%) and non-Latinx (93.5%), heterosexual (86.9%), freshmen (51.9%), and not currently employed (64.3%). Participants who completed experimental procedures were undergraduate students ($M_{age} = 19.33$, SD = 1.88; 82.4% female; 67.3% freshmen) who primarily identified as White (63.5%) and non-Latinx (94.1%). See Table 1 for complete demographic information.

Eligibility and Recruitment

Eligibility was based on a screening questionnaire assessing current gastrointestinal discomfort (GSRS; see below). To be eligible, participants had to: 1) be at least 18 years of age; 2) be an enrolled student; and 3) report at least one gastrointestinal symptom consistent with FGIDs (FGID group; Vivier et al., 2020), or no current gastrointestinal symptoms (control group). Exclusionary criteria included mild to moderate discomfort in the absence of more severe symptoms, or current gastrointestinal symptoms due to primary organic (e.g., IBD) or motility (e.g., gastroparesis) gastrointestinal disorders. Eligible participants were invited via email to schedule an experimental study session.

Baseline Measures

Demographics and Medical History

Participants reported their age, sex and gender, race and ethnicity, and year in school. Regarding gastrointestinal-related diagnoses, the presence of organic gastrointestinal disorders, motility gastrointestinal disorders, and other medical conditions that impact or influence gut sensations (e.g., endometriosis, stomach flu) were assessed. Women indicated whether or not gastrointestinal distress was experienced as a result of their menstrual cycle (e.g., cramping, bloating). Appointments with a medical provider and use of over-the-counter medication for gastrointestinal pain were also assessed. Additionally, engagement in avoidance behaviors (e.g., avoiding eating at restaurants) related to gastrointestinal symptoms was evaluated. See Appendix A for the Demographic and Medical Questionnaire.

Gastrointestinal Symptoms

The Gastrointestinal Symptom Rating Scale (GSRS; Svedlund et al., 1988) is a 15-item self-report measure of common gastrointestinal symptoms that occur throughout the digestive tract, such as stomachache and bloat. Additional instruction specified for females to only consider gastrointestinal symptoms when they are *not* menstruating. Symptoms are clustered into five subscales: abdominal pain, reflux, diarrhea, indigestion, and constipation (only the total score was used in the current study). Severity of symptoms are rated on a 7-point Likert-type scale from 1 ("no discomfort") to 7 ("very severe discomfort"), and items are averaged to comprise the total score and individual subscale scores. Higher scores are indicative of more severe gastrointestinal symptom severity. The original GSRS was developed as a clinician-administered interview (Svedlund et al., 1988), and was subsequently adapted to self-report and has since been widely used and validated among a variety of gastrointestinal disorders such as dyspepsia (Kulich et al., 2008), GERD (Revicki et al., 1998), stomach ulcers (Dimenäs et al.,

1995), Celiac disease (Lohiniemi et al., 2000), and IBS (Wiklund et al., 2003). The GSRS is negatively correlated with measures of psychological and physical wellbeing and each of the five subscales demonstrate acceptable internal consistency ($\alpha s = .61 - .83$; Revicki et al., 1998). In the current study, the GSRS was used to identify presence, quality, and severity of gastrointestinal symptoms for FGID symptom characterization and group assignment. In the current sample, Cronbach's α was excellent ($\alpha = .91$). See Appendix B for the GSRS.

Psychopathology Symptoms

The 21-item version of the Depression, Anxiety, and Stress Scale (DASS-21; Lovibond & Lovibond, 1995) is a self-report measure of depression ("I felt down-hearted and blue") and the physiological (anxiety; "I felt I was close to panic") and cognitive (stress; "I tended to over-react to situations") symptoms of anxiety. Items are rated on a 4-point Likert-type scale from 0 ("did not apply to me at all") to 3 ("applied to me very much, or most of the time"), with higher scores indicating greater levels of distress. Although not validated in a sample of individuals with FGIDs, patients diagnosed with IBS scored an average of 14.8 (moderate severity) on the depression and anxiety subscales, and an average of 20.2 (moderate severity) on the stress subscale (Mazaheri, 2015). The three subscales demonstrate good ($\alpha_{anx} = .81$; $\alpha_{stress} = .89$) to excellent ($\alpha_{dep} = .91$) internal consistency among undergraduate students, and the depression subscale is highly correlated with other measures of anxiety (Lovibond & Lovibond, 1995). In the current sample, the DASS-21 subscales ($\alpha_{dep} = .93$; $\alpha_{anx} = .88$; $\alpha_{stress} = .88$) demonstrated good to excellent internal consistency. See Appendix C for the DASS-21.

Emotional Intensity

The Emotional Intensity Scale – Revised (EIS-R; Geuens & de Pelsmacker, 2002) is a 17-item self-report measure of trait affective intensity with two subscales consisting of positive and negative emotional experiences. Items are hypothetical situations (e.g., "someone complements me," "someone I know is rude to me"), and respondents are asked to identify how they would typically feel in that situation. Items are rated on a 5-point Likert-type scale and summed, with higher scores indicating more emotional intensity. The EIS-R is adapted from the original 30-item EIS (Bachorowski & Braaten, 1994), which has a factor structure that is unable to yield emotional intensity of both positive and negative emotions. Relative to other measures of emotional intensity (e.g., Affect Intensity Measure), the EIS is a "pure" measure of emotional intensity as it does not measure frequency of emotions and only measures intensity regardless of frequency (Geuens & de Pelsmacker, 2002). The total score and two subscales have demonstrated good to excellent internal consistency ($\alpha s > .80$) and test-retest reliability ($\rho s >$.80). Additionally, the subscales also demonstrate adequate convergent validity in that both subscales are positively related to affect intensity ($r_{pos} = .80$; $r_{neg} = .62$) and the negative emotionality subscale is related to neuroticism (r = .68; Geuens & de Pelsmacker, 2002). In the current study, the EIS-R total score (α = .79) and two subscales (α_{pos} = .78; α_{neg} = .78) demonstrated acceptable internal consistency. See Appendix D for the EIS-R.

Alexithymia

The 20-item Toronto Alexithymia Scale (TAS-20; Bagby, Parker, et al., 1994; Bagby, Taylor, et al., 1994) is a self-report questionnaire that measures the cognitive experience of alexithymia. The TAS-20 has three subscales that assess difficulty identifying and describing feelings as well as externally-oriented thinking (only the total score was used in the current study). Items, which range from 1 ("strongly disagree") to 5 ("strongly agree"), are summed and higher scores indicate more severe alexithymia. A clinical cutoff indicative of alexithymia is \geq 61, while non-alexithymia is measured by scores \leq 51 (Taylor et al., 1997). The TAS-20 demonstrates good internal consistency overall ($\alpha = .81$) and acceptable consistency in each subscale ($\alpha s = .66 - .78$) across nonclinical and psychiatric patient samples (Bagby, Parker, et al., 1994; Loas et al., 2001). Further, the TAS-20 is positively correlated with measures of anxiety, depression, and neuroticism, and negatively associated with the propensity to be open and experience positive emotions (Bagby, Taylor, et al., 1994). The TAS-20 total score achieved good internal consistency ($\alpha = .85$) in the current sample. See Appendix E for the TAS-20.

Emotion Regulation Strategies

The Emotion Regulation Questionnaire (ERQ; Gross & John, 2003) is a 10-item selfreport questionnaire that assesses the tendency to regulate one's emotions by using expressive suppression or cognitive reappraisal. Items are rated on a 7-point Likert scale from 1 ("strongly disagree") to 7 ("strongly agree") and those from the suppression and reappraisal subscales are summed separately to yield two scores. The test-retest reliability (both $\rho s = .69$) and internal consistency of the two subscales is acceptable ($\alpha_{sup} = .73$; $\alpha_{re} = .79$). Both the suppression and reappraisal subscales are positively correlated with measures of perception of successful emotion regulation, demonstrating adequate convergent validity overall. Further, suppression was positively associated with measures of inauthenticity and venting and negatively associated with openness and extraversion, while reappraisal was positively associated with reinterpretation and negatively associated with rumination (Gross & John, 2003). In the current study, the rumination subscale had good internal consistency ($\alpha = .86$), while suppression was acceptable ($\alpha = .73$). See Appendix F for the ERQ.

Emotion Regulation Abilities

The Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004) is a 36item self-report measure assessing underlying difficulties with awareness, acceptance, and modulation of emotions. Items are rated on a 5-point Likert-type scale from 1 ("almost never [0-10%]") to 5 ("almost always [91-100%]"), with higher scores indicating more difficulties regulating emotions. The DERS consists of six subscales, which reflect the six domains of emotion regulation abilities proposed by Gratz & Roemer (2004), including nonacceptance of emotional responses (nonaccept), difficulties engaging in goal-directed behavior (goals), impulse control difficulties (impulse), lack of emotional awareness (awareness), limited access to emotion regulation strategies (strategies), and lack of emotional clarity (clarity). The DERS total score displays excellent internal consistency (Cronbach's $\alpha = .93$) and good test-retest reliability overall ($\rho = .88, p < .01$), while internal consistency within each subscale is good (α s > .80; Gratz & Roemer, 2004). Available data on psychometric properties of the DERS in FGID populations is lacking. However, the DERS has shown adequate to excellent internal consistency in other psychosomatic populations such as psychogenic nonepileptic seizures ($\alpha = .94$; Roberts et al., 2012) and chronic pain (α s > .70; Kökönyei et al., 2014). The DERS total score demonstrated excellent internal consistency in this sample ($\alpha = .91$). See Appendix G for the DERS.

Visceral Sensitivity

The Visceral Sensitivity Index (VSI; Labus et al., 2004; Labus et al., 2007) is a 15-item self-report scale measuring gastrointestinal-specific anxiety. Respondents indicate to what extent items correspond to their experience of abdominal discomfort on a 6-point Likert-type scale from 1 ("strongly agree") to 6 ("strongly disagree"). Items are then reverse scored (i.e., 1-6 becomes

5-0) and summed to comprise a total score wherein higher scores indicate worse hypersensitivity to gastrointestinal symptoms (range 0 – 75). Internal consistency was excellent ($\alpha > .90$) among individuals with IBS (Labus et al., 2004) and non-selected undergraduate students (Labus et al., 2007). Further, the VSI is positively correlated with measures of anxiety sensitivity (r = .66), trait anxiety (r = .73), and gastrointestinal symptom severity (r = .49), suggesting strong convergent validity (Labus et al., 2004). Cronbach's α for the VSI in this sample was excellent ($\alpha = .95$). See Appendix H for the VSI.

Perception of Body Sensations

The Body Perception Questionnaire Short Form (BPQ-SF; Cabrera et al., 2018) is a 46item self-report measure that assesses the degree to which individuals are aware of physiological sensations and processes (26 items) as well as ANS reactivity (20 items). Body awareness is one factor, while ANS reactivity is comprised of two sub-factors reflected by organ systems above (i.e., supradiaphragmatic) and below (i.e., subdiaphragmatic) the diaphragm. Items are rated on a 5-point Likert-type scale from 1 ("never") to 5 ("always") then dichotomized (0 = "never", 1 "occasionally or more often") and summed, with higher scores indicating higher body awareness and ANS reactivity. One item ("I feel like vomiting") is included on both ANS subscales. Across three independent samples of community members and undergraduate students, internal consistency measured by the categorical omega coefficient was excellent for the body awareness factor ($\omega = .92 - .96$) and adequate to excellent for the supradiaphragmatic ($\omega = .88 - .94$) and subdiaphragmatic ($\omega = .77 - .87$) subfactors of the ANS reactivity subscales (Cabrera et al., 2018). All BPQ-SF subscales are associated with measures of stress reactivity and hypersensitivity to psychosomatic sensations (Cabrera et al., 2018). Internal consistency across the subscales was good to excellent ($\alpha_{aware} = .73$; $\alpha_{supra} = .88$; $\alpha_{sub} = .81$) in the current study. See Appendix I for the BPQ-SF.

Experimental Measures

The following measures assess state psychological characteristics and were administered after the neutral induction and each emotion induction trial.

Emotion Identification, Intensity, and Differentiation

The Positive and Negative Affect Scale (PANAS; Watson et al., 1988) is a 20-item measure wherein participants identify emotions or affective states (e.g., excited, upset) they are currently experiencing, as well as the intensity of these states. An additional item, "disgusted," was included in the PANAS in order to assess the experience of disgust, totaling 21 items. Each item is rated on a 5-point Likert-type scale from 1 ("very slightly or not at all") to 5 ("extremely"), and two subscales of positive and negative affect are yielded ("disgusted" was included in the negative affect subscale). Only the negative affect subscale was used in the current study. Regarding internal consistency, the PANAS is able to reliably measure general, trait-level affect ($\alpha_{pos} = .88$; $\alpha_{neg} = .87$) as well as in-the-moment, state affect ($\alpha_{pos} = .89$; $\alpha_{neg} = .89$) .85; Watson et al., 1988). Further, the positive affect subscale is negatively correlated with psychopathology, while the negative affect scale is positively correlated with depression, demonstrating convergent and divergent validity among a college student sample (Watson et al., 1988). Additionally, the inverse relationship of positive and negative affect under stressful or aversive states has been implicated among chronic health populations (Davis et al., 2004), which is demonstrated by the lack of relationship between the two subscales in a sample of individuals with chronic kidney disease (de Sousa et al., 2016). Across the four inductions, internal

consistencies for the negative affect subscale ranged from poor ($\alpha_{neutral} = .67$), adequate ($\alpha_{disgust} = .78$), to good ($\alpha_{anxiety, sadness} = .86$). See Appendix J for the PANAS.

State Emotion Regulation Strategies

The Strategies Questionnaire (SQ) is a 4-item measure that assesses in-the-moment use of emotion regulation strategies that was developed for use in a previously published study (Ehring et al., 2010). Items are measured on a Likert-type scale from 0 ("strongly disagree") to 6 ("strongly agree"). The SQ assesses the degree to which an individual engages in suppression and/or reappraisal as a way to regulate emotions during an experimental paradigm, and as such, is designed to be administered prior to and following an experimental trial. In the study the SQ was created for, the suppression and reappraisal items had good (α = .84) and adequate (α = .70) internal consistency, respectively (Ehring et al., 2010). In the current study the SQ was used to assess use of emotion regulation strategies in the experimental paradigm. Across emotion inductions, the suppression ($\alpha_{neutral}$ = .76, $\alpha_{anxiety}$ = .94, $\alpha_{sadness}$ = .88, $\alpha_{disgust}$ = .85) and reappraisal ($\alpha_{neutral}$ = .69, $\alpha_{anxiety}$ = .85, $\alpha_{sadness}$ = .89, $\alpha_{disgust}$ = .79) subscales evidenced poor to excellent internal consistency. See Appendix K for the SQ.

State Emotion Regulation Abilities

The State Difficulties in Emotion Regulation Scale (S-DERS; Lavender et al., 2017) is a 21-item self-report questionnaire developed to measure difficulties in emotion regulation across repeated assessment over brief periods of time. Respondents answer items on a 5-point Likert-type scale from 1 ("not at all") to 5 ("completely"), with higher scores indicating poor ability to regulate emotions at the moment of assessment. The S-DERS is comprised of the total score and four subscales reflecting nonacceptance of current emotions (nonacceptance), difficulties regulating emotional and behavioral responses in the moment (modulate), limited awareness of

current emotions (awareness), and limited understanding of current emotional experiences (clarity). Overall, the S-DERS demonstrates good internal consistency ($\alpha = .86$), while the four subscales range from poor (clarity; $\alpha = .65$), acceptable (awareness; $\alpha = .79$), good (modulate; $\alpha = .85$), and excellent (nonacceptance; $\alpha = .92$). Lavender and colleagues (2017) attest that because the clarity scale is comprised of only two items and that the two items are moderately correlated (r = .48, p < .001), the internal consistency should be considered acceptable. Additionally, the S-DERS is highly correlated with the trait DERS (Gratz & Roemer, 2004) as well as experimental measures of emotional reactivity (Lavender et al., 2017). The S-DERS was utilized in the current study to identify difficulties modulating and identifying one's emotions during an experimental paradigm. The total score evidenced good internal consistency across inductions ($\alpha_{anxiety, disgust} = .83$, $\alpha_{neutral, sadness} = .84$). See Appendix L for the S-DERS.

Current Distress

The Subjective Units of Distress Scale (SUDS; Wolpe, 1958) is a brief assessment of the severity of an individual's level of emotional discomfort or distress in a given situation. Distress is rated from 0 ("no distress") to 10 ("most severe distress ever experienced"). An initial SUDS rating is typically established as the baseline level of distress in a particular situation and is periodically reassessed to monitor changes in distress (McCabe, 2015; Wolpe, 1958). SUDS ratings are highly correlated with measures of state negative affect including anxiety and depression (McCabe, 2015). In the current study, SUDS was used as an assessment of current distress or discomfort following emotion inductions.

Manipulation Check and Mood Induction Questions

Participants responded to manipulation check questions through a Qualtrics survey. In addition, individual PANAS items (i.e., nervous, upset, disgusted) served as induction-congruent

manipulation checks for each emotion induction. Open-ended questions assessed participants' perception of the purpose of each task, and then yes/no and multiple choice (e.g., not at all, a little, moderately, very much) questions assessed the intensity of participants' typical reactions to anxiety-, sadness-, and disgust- eliciting stimuli. Regarding the anxiety induction, questions assessed the believability of the speech deception and typical emotional response to having to give a speech. Sadness induction items asked about familiarity with the show *This Is Us* and sadness following film clips depicting death, while the disgust induction inquired about watching disgusting videos (e.g., pimple popping, botched surgery television shows). See Appendix M for a full list of the manipulation check items.

Modifications Due to Coronavirus Disease 2019 (COVID-19)

A priori power analyses using G*Power version 3.1 (Erdfelder et al., 1996; Faul et al., 2009) were conducted to determine the necessary sample size to be fully powered to test all hypotheses. The power analysis revealed that 78 participants would be needed to achieve a medium to large effect ($d \ge .65$) and adequate power (1 - $\beta = 0.8$). Data collection began at the start of the Fall 2020 semester. However, the ongoing COVID-19 pandemic disrupted data recruitment through the Psychology Department's research pool (Sona Systems). Multiple efforts were made to increase enrollment in the study, such as increasing the number of experimenter time slots, sending regular reminder emails, and adding a raffle wherein participants had a 10% chance of being selected to receive a \$25 Amazon gift card. However, these efforts did not substantially increase enrollment.

Prior to the Spring 2021 semester, a survey-only study option was added to Sona Systems (in addition to the original study) that was open to all students (vs. invitation only) to complete online through Qualtrics. This new study included the aforementioned self-report battery of

baseline measures. In an additional effort to enroll participants, this online survey was also available to students participating in a university-wide survey panel managed by the University's Office of Institutional Research, Effectiveness, and Planning (IREP). Students aged 18 years or older were eligible for the online survey, and the eligibility criteria relating to the presence or absence of gastrointestinal symptoms were not applied to participants recruited through these mechanisms. After participants completed this online study, those eligible for the experimental protocol as described above (see "Eligibility and Recruitment") were able to enroll to receive additional credit (Sona Systems) or as a volunteer (IREP). Of note, no participants recruited through IREP elected to participate in the experimental study procedures. Accordingly, questionnaire (hypotheses 1 and 2) and experimental data (hypotheses 3 and 4) were handled separately. In light of these changes, hypotheses 1 and 2 were updated to reflect these continuous data as follows:

- 1. Depression, anxiety, and stress would be significantly positively associated with gastrointestinal symptoms.
- Heightened emotional responsivity, difficulties understanding emotions, and greater physiological sensation awareness would be significantly positively associated with gastrointestinal symptoms.

Procedure

Self-Report Online Study Procedures

Sona Systems and IREP participants provided informed consent and completed the battery of baseline questionaries online through Qualtrics, which included the demographic and medical questionnaire, GSRS, DASS-21, EIS-R, TAS-20, ERQ, DERS, VSI, and BPQ-SF. Sona

Systems participants received course credit for their participation in the study. Participation in the IREP survey panel was voluntary and no incentive was provided.

Experimental Session Procedures

There were no differences in the procedures for the FGID and control group. Given the occurrence of COVID-19 at the time of data collection, the experimental portion of the study was conducted over the Zoom videoconferencing platform. An experimenter reviewed consent procedures and individuals provided informed consent. Next, participants were oriented to SUDS, instructed how to rate SUDS, and asked for their typical (i.e., baseline) SUDS. Participants then watched a video of a nature scene, which served as a control induction. Then, participants completed the baseline battery of self-report questionnaires through Qualtrics (i.e., demographic and medical questionnaire, GSRS, DASS-21, EIS-R, TAS-20, ERQ, DERS, VSI, and BPQ-SF). Participants who had previously completed these measures through the surveyonly study option did not complete them again. Next, participants completed the series of three emotion inductions designed to elicit anxiety, disgust, and sadness. Each induction lasted approximately three minutes, with a minimum 3-minute distraction period (i.e., nature videos) between trials and following the final trial. The order of trials was counterbalanced across participants to decrease the potential for order effects. To achieve this, the sample was divided into thirds, with each third completing the three emotion induction trials in a different order. Each participant number corresponded with the trial order *a priori*.

To induce anxiety, a speech anticipation task was used. Deception was used to lead participants to believe that they may have to present a speech and that a coin flip would determine whether or not they would present the speech. In reality, the coin flip was rigged, and no participants were asked to complete a speech task. The anticipation of presenting a speech has

been shown to elicit a significant anxiety response, such as increased physiological arousal and subjective anxiety (Macatee et al., 2017; Waugh et al., 2010). In the current study, participants were told that they would receive three minutes to prepare a speech on the topic "Why are you a good friend?", which has been successful at inducing anxious response in previous studies (Macatee et al., 2017; Waugh et al., 2010). Participants were told that two virtual coin flips would determine if and when they would be presenting their speech, and if selected to present, that the speech would be recorded for later evaluation on its clarity, coherence, and persuasiveness. The first coin flip followed the 3-minute anticipation period, and participants were told that it would determine if they would present the speech immediately or wait for the second coin flip following completion of a short battery of questionnaires, which would determine whether they gave the speech at that time or not at all. In line with the deception, all participants received the feedback that they would not give the speech immediately, and after completing the experimental questionnaires, were again told that they would not be giving the speech. Altogether, participants anticipated giving the speech for approximately five minutes. A 5-minute speech anticipation period demonstrates a robust influence on stress response, and is suggested to be the most ideal amount of time (Labuschagne et al., 2019). Additionally, digital or virtual administration of speech tasks designed to elicit anxiety is sufficient in producing comparable levels of cortisol response as in-person administration, particularly for individuals under the age of 25 (Helminen et al., 2019).

To induce sadness, participants viewed a 3-minute film clip that addressed themes of loss and grief. Reviews suggest that using film clips depicting themes of loss can be a potent elicitor of sadness (Gross & Levenson, 1995; Joseph et al., 2020). However, the majority of clips used to induce sadness originated in the 1980s and 1990s; therefore, in attempt to increase the salience of

these clips to current undergraduate students, a survey of sad movie themes and examples of specific movies was provided to a group of nine undergraduate research assistants who rated how sad each theme and movie would make them. Based on the survey results, a scene from the television program *This Is Us* depicting the loss of a loved one was chosen as the sadness induction. Prior to the film clip, a brief 30-second introduction summarized the plot of the television show, as providing context has been shown to amplify the emotional impact (Palomba & Stegagno, 1993).

To induce disgust, participants viewed a 3-minute compilation of disgusting video clips. The video clips featured disgusting videos that correspond with the various facets of disgust (Rozin et al., 2008) such as autopsy and surgery scenes, maggot infestations, vomiting, and consuming unusual food (i.e., insects).

Following each induction, participants completed the experimental questionnaires (i.e., SUDS, PANAS, SQ, S-DERS). Completion of each induction was followed by the 3-minute distraction task to allow for return to baseline SUDS. After the distraction, participants again provided their SUDS rating and if distress had not yet returned to their baseline, they continued to watch the nature video for another three minutes. Lastly, the manipulation checks and mood induction questions were administered.

After study procedures were completed, participants were debriefed about the purpose of the study, and participants were informed that they were provided with false information pertaining to the possibility of delivering a speech task. All participants provided informed consent a second time after they were debriefed on this deception and were credited for their participation.

Data Analysis

Data Cleaning Procedures

All statistical analyses were conducted using SPSS version 26. Alpha was set at p < .05. Due to the novel and exploratory nature of the current study, hypotheses 3 and 4 were considered the primary analyses. Data obtained through the baseline survey battery and experimental protocol were separately screened for accuracy errors, missing data, outliers, and assumptions including non-normality, skewness, and kurtosis. Participants missing more than 5% of their data were removed. Missing data were imputed using linear trend at point regression for those missing fewer than 5% data, which takes the scale average within and item average across participants. Tukey's outlier labeling rule (Hoaglin et al., 1986) identified three outliers in the survey sample and three outliers in the experimental sample. However, the outliers were not removed or altered given that this is a novel pilot study and the full range of emotional and physiological functioning within this population is not yet understood.

Of the 370 original survey participants, 30 were removed for not having completed the primary gastrointestinal symptom measure (GSRS), 28 were removed for missing more than 5% of their data, and 21 were removed for failing an attention check. Three participants were missing one item on the EIS-R due to experimenter error, and the single missing item was imputed for each participant using the previously described imputation procedures. All data met assumptions of normality, multicollinearity, linearity, homogeneity, and homoscedasticity. The final sample consisted of 291 participants, 52 of which participated in the experimental protocol. Of these 52 experimental participants, 53.8% (n = 28) were classified in the FGID group and 46.2% (n = 24) were classified in the control group.

Preliminary Analyses and Sample Characteristics

Demographic variables were examined to identify potential between-group differences in those with and without FGID symptoms. Given the low frequency of racial minorities in the sample, race was collapsed into White and non-White. Descriptive statistics were conducted for each variable to characterize the sample and responses to the emotion inductions. Responses to the manipulation checks and potential between-group differences were evaluated.

Examination of Hypotheses 1 and 2

Pearson correlations were used to test hypotheses 1 and 2 in the full sample of participants (N = 291). Specifically, these analyses tested the associations between gastrointestinal symptoms (GSRS) and 1) depression, anxiety, and stress (DASS-21; hypothesis 1); and 2) emotional and physiological responsivity and awareness (EIS-R, TAS-20, ERQ Reappraisal, ERQ Suppression, DERS, VSI, and BPQ-SF; hypothesis 2).

Examination of Hypothesis 3

Hypothesis 3 was tested in the experimental sample (n = 52). Negative emotion differentiation was characterized by variability in affective experience throughout the emotion inductions. Prior literature has used the PANAS to calculate the degree to which individuals are able to adequately distinguish emotional or affective states (Edwards & Wupperman, 2017; Pond et al., 2012). Standard deviations were calculated between negative emotions across emotion induction trials (anxiety, sadness, and disgust) for each participant. Higher scores are interpreted as little difficulty in differentiating negative emotions relative to lower scores that indicate greater difficulty differentiating negative emotions (de Sousa et al., 2016). To examine group differences in emotion differentiation during emotion inductions, an independent samples *t*-test was computed with group status (FGID, control) entered as the independent variable and negative emotion differentiation entered as the dependent variable.

Examination of Hypothesis 4

Hypothesis 4 was tested in the experimental sample (n = 52). To test hypothesis 4, a series of 2 (Condition: FGID, control) X 4 (Induction: neutral, anxiety, sadness, disgust) mixed factorial ANOVAs were computed, with SUDS, SQ suppression, SQ reappraisal, and S-DERS from each induction entered as the dependent variables in each respective model. Post-hoc planned contrasts were used to examine interaction effects, while pairwise comparisons examined main effects.

CHAPTER 3

RESULTS

Preliminary Data Analysis

Across FGID and control groups, there were no significant differences in sex ($X^2 = 1.84$, p = .18), age (t = -1.35 (49), p = .18), or race ($X^2 = 0.81$, p = .37). Experimental participants were counterbalanced to one of three induction orders: A (anxiety, sadness, disgust), B (disgust, anxiety, sadness), or C (sadness, disgust, anxiety). Participants were relatively evenly counterbalanced across the three induction orders (34.6%, 32.7%, and 32.7%, respectively).

Manipulation checks were employed to assess differences across groups in SUDS, negative affect, and manipulation-congruent emotions following each induction. Regarding the anxiety induction, 21.2% of participants reported not believing the speech task deception (yes/no response choice); therefore, additional information was examined to evaluate how believability affected distress and negative affect. Results revealed no significant differences between those who reported believing and not believing the deception in SUDS (t = 0.94 (50), p = .35), negative affect (t = 0.33 (16.09), p = .75), or nervousness (t = 1.12 (50), p = .27) following the anxiety induction. Given this consistent pattern of findings, it appears that the speech anticipation task was successful in eliciting distress and anxiety regardless of deception believability.

Regarding the clip of the television program used for the sadness induction, 30.8% of participants had seen the television program before and 19.2% of participants had seen the

specific episode depicted in the sadness induction. There were no significant differences in SUDS (t = -0.21(50), p = .84; t = 0.48(14), p = .69), negative affect (t = 0.60(50), p = .55); t = 1.85(14), p = .09), or feeling upset (t = .39 = (50), p = .70; t = 1.85(14), p = .09) between participants who had seen the show and/or the exact episode and those who had not, respectively. Therefore, participants' prior knowledge of the television program did not appear to significantly affect experience of sadness or related distress during the sadness induction.

Lastly, 48.1% of participants reported typically watching disgusting videos but did not evidence differences in SUDS (t = -0.56 (50), p = .57), negative affect (t = -1.50 (50), p = .14), or disgust (t = 0.75 (50), p = .46) following the disgust induction, relative to those who did not watch disgusting videos. Therefore, participants' previous exposure to disgusting stimuli did not appear to significantly affect experience of disgust or related distress during the disgust induction. Based on these data, all data were included in primary study analyses given that external factors did not appear to differentially affect participants' responses to the anxiety, sadness, and disgust inductions.

Participant Characteristics

Across the entire sample, most participants had never been diagnosed with a gastrointestinal disorder (91.8%, n = 267), while 4.5% (n = 13) self-reported having been diagnosed with IBS and 3.7% (n = 11) an organic gastrointestinal condition (e.g., GERD, lactose intolerance). Of the 14.8% (n = 43) of the sample that had reported seeking medical attention for gastrointestinal problems, 15.5% saw a general practitioner, 7.6% saw a specialist, and 1.7% saw a mental health provider. Regarding behavior aimed to reduce or prevent gut symptoms, participants reported avoiding specific foods (65.3%), avoiding eating prior to an event or activity (54.0%), and purposely eating small portions of food (50.2%). Regarding

psychopathological characteristics, the average levels of depression (M = 8.73, SD = 9.82), anxiety (M = 7.40, SD = 8.29), and stress (M = 10.95, SD = 9.36) across the sample were within the normal range. See Table 1 for additional participant demographic and gastrointestinal characteristics, as well as clinical cutoffs regarding emotional difficulties.

Among the participants who completed the experimental procedures, the structure of the FGID and control groups were as expected, with those in the FGID group endorsing higher gastrointestinal symptoms (M = 2.54, SD = 1.05) and visceral sensitivity (M = 25.93, SD = 20.12) than the control group ($M_{GSRS} = 1.45$, $SD_{GSRS} = 0.56$; $M_{VSI} = 7.29$, $SD_{VSI} = 11.08$). See Table 2 for means and standard deviations of psychological and physiological trait characteristics across FGID and control groups.

Examination of Study Hypotheses

Relationship Between Gastrointestinal Distress and Emotional and Physiological

Characteristics (Hypotheses 1 and 2)

Psychopathology. In concordance with hypothesis 1, significant positive correlations were observed between gastrointestinal distress and depression (r = .31, p < .001), anxiety (r = .47, p < .001), and stress (r = .52, p < .001). See Table 3 for zero-order correlations and descriptive statistics.

Emotional and Physiological Reactivity. Partially supporting hypothesis 2, significant positive correlations were observed between gastrointestinal distress and emotion intensity, alexithymia, emotion regulation abilities, visceral sensitivity, and body awareness and ANS reactivity (rs = .20 - .69, ps < .001). Stronger correlations were observed for the physiological reactivity variables as compared to the emotional reactivity variables. Inconsistent with

hypothesis 2, there were no significant correlations between gastrointestinal distress and reappraisal or suppression (ps > .30).

Group Differences in Negative Emotion Differentiation Across Inductions (Hypothesis 3)

Standard deviation of negative affect during the three emotion inductions was on average $0.93 \ (SD = 0.39)$ and ranged from 0 to 1.65 (see Table 4 for negative affect means and standard deviations across groups following each induction). Unexpectedly, there was not a significant difference across the FGID and control groups in negative emotion differentiation, $t \ (50) = 1.47$, p = .15. Therefore, hypothesis 3 was not supported.

Group Differences in Emotional Characteristics Across Inductions (Hypothesis 4)

Distress. Participants' SUDS were examined across each of the four induction trials (neutral, anxiety, sadness, disgust) and two groups (FGID, control). Mauchly's test (p = .01) indicated assumptions of sphericity were not met, therefore a Huynh-Feldt correction ($\varepsilon > .75$) was used. There was not a significant interaction of induction by group on SUDS, F(2.78,139.12) = 0.28, p = .83 (see Table 5 for post-hoc planned contrasts for each dependent variable). However, there was a significant within-subjects main effect of induction (F[2.78,139.12] = 36.12, $\eta^2 = .42$, p < .001). SUDS following the anxiety, sadness, and disgust inductions were significantly higher than SUDS following the neutral induction, and SUDS following the anxiety and disgust inductions were significantly higher than SUDS following for each dependent variable). There was no statistical difference in SUDS between the anxiety and disgust inductions. There was not a significant difference in SUDS across the FGID and control groups, F(1,50) = 0.34, p = .56.

Emotion Regulation Abilities. Participants' state-level emotion regulation abilities were examined across each of the four inductions (neutral, anxiety, sadness, disgust) and two groups (FGID, control). Mauchly's test (p < .001) indicated assumptions of sphericity were not met, therefore a Huynh-Feldt correction ($\varepsilon > .75$) was used. There was not a significant interaction of induction by group on emotion regulation abilities, F(2.44, 122.00) = 1.94, p = .14, nor were there significant main effects of induction (F[2.44, 122.00] = 1.03, p = .37) or group (F[1,50] = 1.84, p = .18).

Emotion Regulation Strategies. Participants' state-level emotion regulation strategies were examined across each of the four inductions (neutral, anxiety, sadness, disgust) and two groups (FGID, control). Participants' use of cognitive reappraisal and suppression were each examined separately. Regarding cognitive reappraisal, Mauchly's test (p = .06) indicated that assumptions were met. There was not a significant interaction of induction by group on use of cognitive reappraisal, F(3,150) = 1.35, p = .26, nor were there significant main effects of induction (F[3,150] = 2.03, p = .11) or group (F[1,50] = 0.22, p = .64). Regarding suppression, Mauchly's test (p = .73) indicated that assumptions were met. There was not a significant interaction of induction by group on use of suppression, F(3,150) = 0.06, p = .98, nor were there significant main effects of induction (F[3,150] = 1.19, p = .32) or group (F[1,50] = 0.57, p = .45). Overall, hypothesis 4 was not supported.

CHAPTER 4

DISCUSSION

Although individuals with FGID symptoms are known to experience heightened emotional responsivity (Drossman, 2016; Fournier et al., 2018), the empirical literature examining these associations is sparse. The current study aimed to deepen this knowledge by examining trait and state emotional and physiological responses in the context of gastrointestinal distress. With regard to the characterization of FGID symptoms in the current sample, gastrointestinal symptoms were generally less severe than in clinical patients with emotional and/or functional gastrointestinal disorders (Grzesiak et al., 2014; Simrén et al., 2018); yet, gut symptoms commonly affected participants' routine and eating behaviors. Nearly one-third of participants avoided eating at restaurants, approximately one in five avoided sex and exercise, and although avoiding specific food items (e.g., dairy) may be adaptive in some cases, over half of the sample did so to prevent gut symptom exacerbation. These findings are consistent with studies suggesting FGIDs and related impairment are common among samples of college students (up to 26.9%; Goyal et al., 2020; Jiang et al., 2019) and are comparable to the general population (Chang, 2004).

Consistent with FGID presentations among other university (Dong et al., 2013; Goyal et al., 2020; Jiang et al., 2019) and clinical samples (Lee et al., 2017), study participants with FGID symptoms experienced more symptoms of depression, anxiety, and stress relative to healthy controls. However, the FGID group reported overall normal to mild levels of symptoms, with

only a minority endorsing clinically significant symptoms of depression (14.8%), anxiety (18.5%), and stress (3.7%). In contrast, levels of depression and anxiety among FGID populations are typically much higher, with severity scores falling between means observed in psychiatric populations and healthy control groups (Çakmak et al., 2018; Drossman, 1999; Hartono et al., 2012). Although convenience samples such as college students may serve as a more easily accessible proxy population to FGID patients, future studies may consider overselecting for more severe gut and emotional symptoms to more accurately represent patient populations.

Despite the generally low level of gut and emotional distress in the current sample, FGID symptoms evidenced a small association with depression and moderate associations with anxiety and stress symptoms. This finding supports hypothesis 1 and the broader link between gut and emotional symptoms. These associations may reflect the integral role of the ANS and HPA axis in the reciprocal link between psychopathology, particularly stress and anxiety, and alterations in motility and visceral signaling, which together contribute to FGIDs (Elsenbruch et al., 2010; Fukudo et al., 1998; Geeraerts et al., 2005; Van Oudenhove & Aziz, 2013; Van Oudenhove et al., 2016). Chronic stress is also a major risk factor for depression; therefore, depression is linked to HPA axis excitation through the experience of stress (Saveanu & Nemeroff, 2012). In a related vein, recent reviews have implicated dysbiosis of the gut microbiome in the etiology of anxiety and depression by way of proinflammatory bacteria (Rivet-Noor & Gaultier, 2020; Simpson et al., 2021). Stress may be responsible for alterations in the microbiota, which has direct ties to depression (Rivet-Noor & Gaultier, 2020) and gut pain (Mayer et al., 2014). In turn, gut inflammation is thought to disrupt the HPA axis causing increased release of cortisol and triggering the above-mentioned process (Rivet-Noor & Gaultier, 2020; Simpson et al., 2021).

Incorporating the gut microbiome into future work examining the links between emotions and the gastrointestinal tract is important for developing a complete understanding of these associations.

Consistent with ties to emotional distress, FGID symptoms evidenced small associations with heightened emotion intensity, alexithymia, and greater difficulties regulating emotions, which provides support for hypothesis 2. Through the HPA axis, deficits in the processing and expression of emotions as well as emotional hyperreactivity may contribute to heightened visceral perception and chronic gut symptoms (Aldao et al., 2010; Carrozzino & Porcelli, 2018; Fournier et al., 2018; Panayiotou et al., 2021). Therefore, emotion-related deficits may serve as an amplifier of distress contributing to somatic and visceral sensitivities through excitation of the HPA axis, but additional work is needed to test this hypothesis. Contrary to hypothesis 2, the tendency to use reappraisal and suppression was not associated with gut symptoms. Given the low levels of emotional distress and gastrointestinal symptoms reported by the FGID group, the selection of emotion regulation strategies may not be as salient to this group relative to clinical populations. Among clinical FGID samples, research has shown connections between the selection of emotion regulation strategies and gastrointestinal symptoms. For instance, behavioral avoidance of gastrointestinal symptoms contributes to worse gut symptom severity (Bonnert et al., 2018), whereas positive emotion regulation strategies such as mindfulness have been found to ameliorate gut symptoms (Aucoin et al., 2014; Mazaheri, 2015). Additional empirical work examining the role of emotion regulation strategies among patients with FGIDs experiencing higher levels of emotional distress is warranted to truly understand these associations.

Robust associations were observed between gastrointestinal symptoms and broad awareness of visceral (i.e., internal organs) and somatic (i.e., skin, muscles, soft tissue) sensations, which provides further support for hypothesis 2. Through alteration of gut motility and CNS signaling, visceral sensitivity contributes to both gastrointestinal symptoms (Simrén et al., 2018) and stress (Mertz, 2003). In a vicious cycle, stress reciprocally contributes to visceral sensitivity in those with FGIDs. More broadly, ANS reactivity associated with the ENS (i.e., the gut; Cabrera et al., 2018) is particularly relevant to FGID symptoms compared to reactivity in the upper body cavity (e.g., esophagus), which is linked with autonomic inhibition of the parasympathetic nervous system. Taken together with the current literature, these correlational findings implicate physiological reactivity and sensitivity in the gut as having a robut influence on FGID symptoms.

Inconsistent with hypothesis 3, the findings revealed those with FGID symptoms did not differ in their ability to differentiate negative emotions following emotion-provoking stimuli relative to healthy peers. Greater ability to differentiate negative emotions is associated with more effective selection of emotion regulation strategies (and decreased problematic behavior; e.g., substance use) and improved emotion regulation abilities (Barrett et al., 2001; Kalokerinos et al., 2019; Kashdan et al., 2015). Given that those with FGIDs are hypothesized to be vulnerable to negative emotion and exhibit poor emotion regulation relative to healthy peers (Fournier et al., 2018), it is unexpected that the FGID group was not worse at differentiating emotions. Notably, low levels of emotion differentiation were observed in both groups across emotion inductions. Adolescents exhibit poor emotion differentiation (Nook et al., 2018); therefore, it is possible that participants, who were on average 19 years of age, may have struggled to distinguish discrete emotions during emotion inductions. College students overall

may lack the ability to differentiate discrete emotions when encountering multiple emotionprovoking stimuli, making them an inopportune group to test this hypothesis. Examining emotion differentiation abilities among adult populations experiencing FGIDs would shed light on an important skill that may buffer, or contribute to, detrimental consequences of negative affect (Smidt & Suvak, 2015).

With regard to hypothesis 4, distress, emotion regulation strategies, and emotion regulation abilities did not differ between the FGID and control groups across emotion inductions, which is contrary to the burgeoning literature in this area (Cheng et al., 2000; Fournier et al., 2018; Jones et al., 2006; Mazaheri, 2015; Stanculete et al., 2015). Of note, SUDS during the emotion inductions were generally low, ranging from 1.18 to 4.29 (out of 10) in the FGID group; yet, these data are comparable to outcomes observed in previous studies using clinical analogue samples (e.g., 2.42 – 4.83; Cloutier et al., 2019; Franklin et al., 2012; Schatten et al., 2015). Chronic or heightened levels of distress may be more salient to FGID presentations than short-term, acute emotional distress; therefore, the current sample of individuals experiencing low levels of distress would not be ideal for detecting such emotional differences. Future research is needed to further evaluate relevant mood induction paradigms in this population and identify circumstances under which it is possible to detect differences in laboratory-induced emotions between individuals with FGID symptoms and healthy controls. For instance, prolonged emotion induction paradigms or idiographic mood inductions, such as participant-selected video or music clips or recitation of an emotionally salient autobiographical event, may be necessary for evoking complex emotional responses, such as processes of psychopathology and emotion regulation (Ellard et al., 2012; Kuo et al., 2014).

It is possible that alexithymia has a more precise relationship with FGIDs than emotion regulation, an arguably broader construct that alexithymia may be encompassed within (Panayiotou et al., 2021), potentially explaining the null results of hypothesis 4. Notably, the FGID group reported clinical levels of dispositional alexithymia at two times the rate of the control group, a finding consistent with the current literature (Porcelli et al., 1999, 2004; Portincasa et al., 2003). Alexithymia is posited to be a somatosensory amplifier associated with deficits in cognitive-emotional processing and increased sensitivity towards physical sensations (see Kano & Fukudo, 2013). Those high in alexithymia experience hyperinteroceptive arousal, especially when physiologically or emotionally stimulated, such that objectively low stimulation is attended to and experienced as intensely painful (Kano & Fukudo, 2013). This somatosensory amplification process may partially explain FGIDs, in that there is no biochemical or structural abnormality, yet, typical gut sensations are experienced as painful. One potential but understudied pathway linking alexithymia to illnesses such as FGIDs is common third variables (Lumley et al., 2008), and although the mechanism is still unknown, studies implicate emotion regulation system deficits and activation of the ANS and HPA axis (Kano et al., 2018; Kano & Fukudo, 2013). There is need to understand the unique, yet inextricable roles of alexithymia and emotion regulation in activation of the HPA axis and other excitatory systems in the body that may affect the brain-gut axis.

In addition to hypothesis-specific explanations, factors associated with the study sample and methodology may have also contributed to the null findings of hypotheses 3 and 4. First, the FGID group was not necessarily diagnosed with FGIDs, and participants generally experienced little gastrointestinal and emotional distress. Given the high co-occurrence of psychopathology and FGIDs observed in clinical samples (Elsenbruch et al., 2010; Hillila & Farkkila, 2004; Lee et

al., 2009; Van Oudenhove et al., 2016; Wu, 2012), this sample may not be an appropriate proxy population with which to understand FGIDs and the disruption of the brain-gut axis more broadly. Secondly, the emotion inductions may not have been sufficient elicitors of anxiety, sadness, and disgust. One main effect of induction on SUDS emerged, such that the anxiety and disgust inductions elicited a greater level of distress than the sadness induction, and that all three emotion inductions were associated with greater distress than the neutral induction. However, this difference in distress was not seen across groups, nor were there differences among state-level emotion regulation strategies or abilities, suggesting that the inductions may not have been salient to this sample. Lastly, the robustness of inductions may have been weakened by the remote administration, such that completing the study in participants' home environment may have led to lower distress than might be seen in a laboratory setting (see "Videoconferencing Considerations" section below for more considerations of remote administration).

Limitations

These results should be interpreted in the context of several limitations. First, the specificity and sensitivity of the eligibility screening is unclear. In some cases, the screening may have been over-restrictive (e.g., excluded people with co-occurring organic gastrointestinal disorders and FGIDs), while in other cases it may have been under-restrictive by not effectively identifying participants with organic disorders (e.g., not reporting organic disorders at screening but subsequently reporting such disorders). More generally, the inclusion criteria may have contributed to issues in group composition given that there are no strict guidelines on how to categorize GSRS responses. Participants included in the FGID group did not necessarily have FGIDs, but rather, endorsed at least one moderate to very severe gut symptom. This may have resulted in average levels of minor to mild gastrointestinal symptoms, which are lower than other

student (Chen et al., 2021) and clinical samples (Porcelli et al., 2020). Nevertheless, this classification is consistent with previous studies that have used college student samples (Norton et al., 1999; Vivier et al., 2020), which examine gut sensations on a continuum of severity rather than dichotomously due to frequent fluctuations in severity. Although it may be prudent to recruit persons diagnosed with FGIDs for future work, understanding the impact of emotional reactivity in gastrointestinal distress among college samples is important as students are vulnerable to both emotional distress (Auerbach et al., 2016, 2018) and functionally impairing gut sensations (Chen et al., 2021; Dong et al., 2013; Gulewitsch et al., 2011; Zvolensky et al., 2018).

Second, the sample was primarily young ($M_{age} = 20.59$), White (82.5%), female (72.5%) college students. Although certain characteristics are reflective of those observed in FGID patients, greater heterogeneity in demographic characteristics is needed to fully understand this population. For instance, college students remain an important target population as approximately one-third of students experience at least one mild to moderate gastrointestinal symptom at a given time (Vivier et al., 2020). Yet, expanding the age range is essential as FGIDs occur across the lifespan, with different disorders peaking at different times (i.e., childhood vs. adulthood) and in concurrence with life events (e.g., pregnancy; Houghton et al., 2016). FGIDs are also more common among females relative to males (Sperber et al., 2021), with one study suggesting women experience IBS at a rate of 53% to 82% times greater than men (Card et al., 2014). This imbalance may be partially due to societal expectations of women (e.g., bodily function is shameful) and attractiveness standards (e.g., bloating contributing to body image difficulties), both of which result in the experience of greater gut pain and emotional duress (Houghton et al., 2016; Longstreth & Wolde-Tsadik, 1993). The complete pathophysiology

underlying higher prevalence in women is unknown, but it is likely multifaceted and may include several factors such as autonomic and immune dysregulation, health care seeking behaviors, and genetic predispositions (Houghton et al., 2016). Lastly, as FGIDs are prevalent across the world (Sperber et al., 2012, 2021) and different cultures (Francisconi et al., 2016), future studies should take care to enroll ethnically diverse samples.

Third, while hypotheses 3 and 4 were tested with an experimental design, the survey data are cross-sectional and temporal assumptions are precluded. To further strengthen the methodology, future studies may consider building upon the current design by incorporating a longitudinal component to assess long-term associations between FGID and emotional symptoms. Given the frequency of gut sensations in college students (Vivier et al., 2020), recruiting at-risk students and following them throughout college may present opportunities to understand the trajectory of gastrointestinal and emotional symptoms by identifying risk and protective factors. As a fourth limitation, the study relied on retrospective self-report measures to assess emotional responding and presence and severity of FGID symptoms. Despite the ANS being integral to the brain-gut axis, the psychophysiology literature is underdeveloped in the context of FGIDs. Psychophysiological assessments (e.g., facial expression analysis, electrodermal activity, heart rate variability) could be incorporated in future work to further characterize emotional responding in this population. Additionally, utilization of medical providers, hospital records, or clinical interviews to verify FGID symptoms would more closely approximate FGID patients.

Fifth, the emotion inductions may not have been potent enough to elicit an adequate amount of distress across participants. Although the anxiety induction used in this study has been previously validated (Macatee et al., 2017; Waugh et al., 2010), a recent review indicated that

virtual administrations, evidencing moderate effect sizes, are not as effective at eliciting stress as in-person administrations (Helminen et al., 2019). Additionally, while the sadness and disgust inductions were based on prior research (Al-Shawaf et al., 2019; Gross & Levenson, 1995; Joseph et al., 2020; Stark et al., 2005), the psychometric properties regarding the ability to evoke desired emotional responses have not been previously studied. Although there are existing validated film clips that serve as sadness inductions, the most commonly used films premiered between the years of 1979 and 1998 (Joseph et al., 2020), and most college students in the 2020 – 2021 academic year (i.e., the period data collection occurred) were born after these films premiered. Consequently, these film clips may not be as relevant to current and upcoming generations of college students. Furthermore, in addition to the limited availability of suitable emotion inductions, no studies have examined nor validated emotional inductions within a FGID sample. In order to understand emotional responsivity as it relates to FGIDs, standardization and validation efforts are needed for these instruments within FGID samples to identify stimuli that are effective elicitors of discrete emotions.

Lastly, the COVID-19 pandemic caused disruptions to recruitment of study participants, resulting in the experimental sample being underpowered (n = 52, relative to the sample size of 78 that experimental hypotheses were powered for). Due to the underpowered nature of the experimental sample, hypotheses 3 and 4 should be interpreted cautiously. However, as the relationship between power and sample size is asymptotic (Field, 2018), it is possible that the sample may be sufficiently powered to detect significant results. It is always a possibility that, regardless of issues with power, null results accurately reflect reality in that there truly are not associations between study variables. In addition to affecting recruitment, the pandemic may have also altered typical responding. A multitude of articles suggest that the COVID-19

pandemic has altered mental health globally (e.g., Alzueta et al., 2021; Vindegaard & Benros, 2020; Wang et al., 2020a), and notably, among college students (Husky et al., 2020; Wang et al., 2020b). Recent data collected from this university suggests that nearly 80% of undergraduate students relocated to their family homes due to COVID-19-related closures (Dixon et al., in press). As many participants likely completed study procedures in their home environment, these data may not capture typical responding and therefore it is unclear how findings may generalize to other time periods (i.e., responding prior to the pandemic). Overall, it is difficult to know how the pandemic may have altered participant responding in psychological studies such as this one.

Videoconferencing Considerations

Social distancing guidelines during the pandemic demanded that data collection occur in a novel, virtual environment. Thus, the experimental procedures of this study were conducted via the videoconferencing platform, Zoom Video Communications Inc. (Zoom), representing a novel contribution to the empirical literature. Inherent to any new methodology, there are yet to be published (and peer-reviewed) guidelines on best practices for empirical data collection via videoconference. Zoom adaptations to the experimental design were based primarily on published qualitative literature and American Psychological Association-sponsored recommendations (Archibald et al., 2019; Gray et al., 2020; Lourenco & Tasimi, 2020; Su & Ceci, 2021; Wood, 2021).

In our experience and established through the literature, there are advantages and disadvantages to conducting research via Zoom, as well as important considerations that are unique to conducting research remotely. The first benefit of using videoconferencing technology in research is that it can provide access to typically hard to reach populations (e.g., rural; Lourenco & Tasimi, 2020), and participants can access the study from anywhere, such as their

family homes. This is particularly relevant in Mississippi, and in this sample, one-fourth of participants who completed the experimental procedures reported being from rural locations. Second, methodologies that emphasize the viewing of stimuli may be a good fit for online data collection, especially data collection that is conducted to inform future in-person studies (Wood, 2021). Virtual study sessions may be advantageous for identifying and addressing methodological issues in the pilot phase before dedicating more time and resources to recruit inperson participants.

Although remote methods may allow typically underrepresented persons access to participate in research, it also limits individuals with poor or no internet connection from participating, particularly Black and Latinx populations (Lourenco & Tasimi, 2020). In this study, data collection via Zoom may have restricted participation to students physically located on campus or in family homes with access to internet (e.g., suburban or urban locations) and/or necessary equipment (i.e., computers with audio and video capability). A second limitation to using Zoom is the lack of control over the study environment. Many participants completed the study from their dorm room with roommates present, while others were in public spaces (e.g., library). These environments inherently introduce more confounds (e.g., distractions, presence of other people) but may also represent a more ecologically valid environment relative to a typical laboratory setting. Lastly, online or remote studies that are not theoretically related to online or virtual behavior may lack generalizability to in-person behaviors (Wood, 2021). Participants' responses to emotion-evoking stimuli delivered via Zoom may not be representative of their inperson behavioral or affective responding. Despite these limitations, data collected in the current study may provide a preliminary understanding of emotional and physiological responding of

students with FGID symptoms relative to healthy controls, and the context for identifying methodological modifications for future in-person studies.

Researchers must carefully consider whether samples used in remote research are accurate representations of the target population of interest and not artifacts of remote data collection (Wood, 2021). A priori methodological considerations must be put into place such as attention and manipulation checks to screen out inaccurate responding and individuals who may not belong to the target population, respectively. It is also prudent to compare responding to that of in-person studies to understand how well remote designs capture in-person phenomena. Regarding the current study, much of the current emotion induction literature focuses on physiological responses (e.g., heart rate variability, cortisol, electrodermal responding) associated with emotion-provoking stimuli rather than the affective responses measured in the current study, making responses difficult to compare. Regarding emotion variables, relative to a sample of depressed and never-depressed participants, participants in the current study evidenced greater use of suppression and reappraisal in response to emotion inductions (Ehring et al., 2010). Notably, Ehring and colleagues (2010) averaged responses following a series of emotion inductions and following a rest period, rather than only after inductions as in the current study, making this impossible to compare responses to the current study. There are no known studies utilizing emotion inductions among individuals with FGIDs to provide context for in-person responding within this population. In sum, Zoom provides access to populations that would otherwise not be accessible and the opportunity to fine-tune the design of studies for in-person data collection, but it is crucial to assess whether findings are comparable to published in-person studies.

Future Directions

Although the primary study hypotheses resulted in null findings, the current study provides a novel contribution to the FGID literature, particularly through its methodology, and underscores the need for future in-person experimental studies to uncover the relationship between emotion processes and the gut. Researchers may consider inducing a wider range of emotions to further understand which discrete emotions impact FGID symptoms. Additionally, it would be prudent to understand the associations between gastrointestinal symptoms, emotion dysregulation, and alexithymia. For instance, instructing participants with FGID symptoms to use either suppression or reappraisal during emotion inductions (e.g., Ehring et al., 2010) may facilitate understanding of how specific emotion regulation strategies (e.g., avoidance) influence gut sensations. Alexithymia is thought to arise from deficits in emotion regulation, although there is contrasting evidence for which components of the emotion regulation process are associated with alexithymia (e.g., perception vs. regulation of emotions; van der Velde et al., 2015). Behavioral tasks (e.g., emotion inductions, vignettes, computer tasks) and use of psychophysiological equipment to measure and differentiate emotion regulation and alexithymia may shed light on how these emotion constructs differentially impact individuals with FGID symptoms.

Equally important is developing a more thorough understanding of physiological responding and sensitivity among individuals with FGIDs. Beyond its role in the stress response (Fichna & Storr, 2012), the ANS has wide-reaching effects throughout the body including visceral responses to CNS activity (Tougas, 2000) and differential responding to emotional stimuli (Kreibig, 2010). Future work may attempt to elucidate which components of the ANS are responsible for contributing to gut symptoms, and how these associations reciprocally affect

emotional responding. Researchers could attempt to induce gut responding through physiological or psychological stressor tasks while measuring sympathetic (e.g., cardiovascular, electrodermal, respiratory activity) and parasympathetic (e.g., heart rate variability) activity.

As more information about disorders of the brain-gut axis is unveiled, we become closer to refining intervention and identifying clinical implications for this vulnerable population. Due to the heterogeneous nature and relatively unknown pathophysiological mechanisms of FGIDs, pharmaceuticals used to treat these conditions are limited and only moderately effective (Craig & Quigley, 2011). Further, FGID patients frequently report dissatisfaction with and perceived invalidation from health care providers because of the poorly understood and functional nature of FGIDs (Chang et al., 2006). Considering FGIDs are viewed through a biopsychosocial lens and co-occur frequently with emotional disorders (Drossman et al., 2002), psychological interventions may be an effective alternative. Burgeoning literature suggests emotion awareness and regulation may improve both gastrointestinal and psychological symptoms (Doroui et al., 2017; Thakur et al., 2013). Such transdiagnostic interventions contribute to reduction in gastrointestinal symptoms through increases in cognitive reappraisal and reductions in emotional suppression and behavioral avoidance (Bonnert et al., 2018; Mazaheri et al., 2014; Mohsenabadi et al., 2018). Thus, changes in emotion regulation may be a critical component of intervention for FGID symptoms. Clinical case studies are warranted to provide more empirical support for existing evidence-based psychological interventions for FGIDs and to inform the next phases of clinical research, including the implementation of randomized controlled trials. As with emotional disorders (Donker et al., 2009) and other chronic pain conditions (Salvetti et al., 2012), it would also be pertinent for investigators to identify whether providing psychoeducation

regarding the interplay of emotions, behavior, and the gut may serve as a standalone treatment for FGIDs.

Conclusions

The current study examined the role of emotion processes in FGIDs using a novel experimental design, wherein a series of emotion inductions were administered through a videoconferencing platform. Although the results do not indicate that there are differences in distress or emotion differentiation or regulation across those with and without FGID symptoms, they do suggest that heightened affective and physiological reactivity are correlated with gastrointestinal distress. Results also show that gut symptoms are relatively common among college students, a population that is vulnerable to emotional duress (Auerbach et al., 2016, 2018). Given the link between emotions and the gut, college students experiencing psychological distress are potentially at risk for developing gut sensitivities. Future studies are needed to examine specific emotional-behavioral factors that may confer risk for developing or worsening gut symptoms, as well as investigating whether individuals with FGIDs have trouble identifying, differentiating, and modulating emotions relative to their healthy counterparts.

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LIST OF APPENDICES

APPENDIX A: DEMOGRAPHICS AND MEDICAL QUESTIONNAIRE

- 1. With what gender do you identify?
 - a. Male
 - b. Female
 - c. Transgender
 - d. Non-binary
 - e. Other
- 2. What was your sex at birth?
 - a. Male
 - b. Female
- 3. Age (Free response)
- 4. With what race do you identify?
 - a. White/Caucasian
 - b. Black/African American
 - c. Native American/Alaska Native
 - d. Asian/Pacific Islander
 - e. Multiracial (specify)
 - f. Other (specify)
 - g. Prefer not to answer
- 5. With what ethnicity do you identify?
 - a. Hispanic/Latinx
 - b. Non-Hispanic/Latinx
- 6. Identify which best represents your housing situation:
 - a. Dormitory
 - b. Greek housing
 - c. With friends or roommates in apartment/condominium/house
 - d. With family in apartment/condominium/house
 - e. Live alone
- 7. Are you employed?
 - a. Yes, part-time (< 30 hours per week)
 - b. Yes, full-time (\geq 30 hours per week)
 - c. No
- 8. Are you a first-generation college student?
 - a. Yes
 - b. No
- 9. What location best describes where you were raised?
 - a. Urban
 - b. Rural
 - c. Suburban
 - d. Other (specify)
- 10. How do you self-identify?
 - a. Gay
 - b. Lesbian
 - c. Bisexual
 - d. Queer
 - e. Questioning
 - f. Heterosexual/Straight

- g. Asexual
- h. Other (specify)
- 11. Year in college
 - a. Freshman $(1^{st} year)$
 - b. Sophomore (2nd year)
 - c. Junior (3rd year)
 - d. Senior (4th year)
 - e. Other (specify)
- 12. What is your current height in inches? One-foot equals 12 inches. (free response)
- 13. What is your current weight in pounds? (free response)
- 14. About how often did you smoke cigarettes in the past year?
 - a. Never
 - b. One time
 - c. Monthly or less
 - d. 2-4 times a month
 - e. 2-3 times a week
 - f. 4 or more times a week
- 15. About how often did you use smokeless tobacco products (e-cigarettes (e.g., Juul), chewing tobacco) *in the past year*?
 - a. Never
 - b. One time
 - c. Monthly or less
 - d. 2-4 times a month
 - e. 2-3 times a week
 - f. 4 or more times a week

16. About how often did you use cannabis (i.e., marijuana) in the past year?

- a. Never
- b. One time
- c. Monthly or less
- d. 2-4 times a month
- e. 2-3 times a week
- f. 4 or more times a week

17. Have you seen any of the following *specifically* for gastrointestinal or stomach pain or discomfort? (multiple responses)

- a. General practitioner/family doctor
- b. Specialists (e.g., gastroenterologist)
- c. Complementary and alternative medicine
- d. Psychologist/Psychiatrist
- e. Other (specify)

18. Which of the following over the counter medications do you use to help alleviate gastrointestinal or stomach pains? (multiple responses)

a. Antacids (e.g., Tums, Pepto-Bismol, milk of magnesia, Alka-Seltzer)

b. Diuretics

c. Laxatives (Metamucil, Dulcolax)

- d. Gas relief (e.g., Beano)
- e. Other (specify)

- 19. Which of the following dietary guidelines/diets do you follow? (multiple responses)
 - a. Vegetarian
 - b. Vegan
 - c. Pescatarian
 - d. FODMAP
 - e. Gluten free
 - f. Lactose free
 - g. Keto
 - h. Other (specify)

19a. How stringently do you follow these dietary guidelines/diets? (multiple responses)

- a. Every day
- b. Weekdays only
- c. Weekends only
- d. Specific times related to upcoming events (e.g., social event, sporting event)
- e. Other (specify)
- 20. Has a medical provider diagnosed you with any of the following? (multiple responses)
 - a. Celiac disease
 - b. Crohn's disease
 - c. Ulcerative colitis
 - d. Gastroesophageal reflux disease (GERD)
 - e. Irritable bowel syndrome
 - f. Gastroparesis
 - g. Gastritis
 - h. Diverticulitis
 - i. Other (specify)

21. Do you have other, non-gastrointestinal medical conditions that may cause gastrointestinal distress, pain, or discomfort, such as endometriosis, or temporary illnesses that would cause gastrointestinal distress, such as the stomach flu? (free response).

22. Do you experience/are you currently experiencing gastrointestinal distress *as a result of menstruation* (e.g., cramping, bloating)?

- a. Yes
- b. No/I am male

23. Do you do any of the following to avoid gastrointestinal or stomach pain or discomfort? (multiple responses)

- a. Avoiding meals at certain times of day (specify)
- b. Avoiding or restricting intake of food
- c. Avoiding restaurants
- d. Avoiding certain foods (specify)
- e. Avoiding situations without easy access to a restroom
- f. Checking for location of bathrooms
- g. Relying on medications when traveling
- h. Avoiding public transportation

i. Avoiding exercise (e.g., cardiovascular activities, lifting weights (squatting), abdominal exercises

APPENDIX B: GASTROINTESTINAL SYMPTOM RATING SCALE

<u>Please read this first</u>: This survey contains questions about how you have been feeling and what it has been like DURING THE PAST WEEK. Mark the choice that best applies to you and your situation. If you are female, please only consider gastrointestinal symptoms that occur when you are <u>NOT</u> menstruating. Response scale: (1) No discomfort at all. (2) Minor discomfort. (3) Mild discomfort. (4) Moderate discomfort. (5) Moderately severe discomfort. (6) Severe discomfort. (7) Very severe discomfort.

- 1. Have you been bothered by STOMACH ACHE OR PAIN during the past week? (Stomach ache refers to all kinds of aches or pains in your stomach or belly.)
- 2. Have you been bothered by HEARTBURN during the past week? (By heartburn we mean an unpleasant stinging or burning sensation in your chest.)
- 3. Have you been bothered by ACID REFLUX during the past week? (By acid reflux we mean regurgitation or flow of sour or bitter fluid into your mouth.)
- 4. Have you been bothered by HUNGER PAINS in the stomach or belly during the past week? (This hollow feeling in the stomach is associated with the need to eat between meals.)
- 5. Have you been bothered by NAUSEA during the past week? (By nausea we mean a feeling of wanting to throw up or vomit.)
- 6. Have you been bothered by RUMBLING in your stomach or belly during the past week? (Rumbling refers to vibrations or noise in the stomach.)
- 7. Has your stomach felt BLOATED during the past week? (Feeling bloated refers to swelling often associated with a sensation of gas or air in the stomach.)
- 8. Have you been bothered by BURPING during the past week? (Burping refers to bringing up air or gas through the mouth.)
- 9. Have you been bothered by PASSING GAS OR FLATUS during the past week? (Passing gas or flatus refers to the release of air or gas from the bowel.)
- 10. Have you been bothered by CONSTIPATION during the past week? (Constipation refers to a reduced ability to empty the bowels.)
- 11. Have you been bothered by DIARRHEA during the past week? (Diarrhea refers to a too frequent emptying of the bowels.)
- 12. Have you ever been bothered by LOOSE STOOLS during the past week? (If your stools have been alternately hard and loose, this question only refers to the extent you have been bothered by the stools being loose.)
- 13. Have you been bothered by HARD STOOLS during the past week? (If your stools have been alternately hard and loose, this question only refers to the extent you have been bothered by the stools being hard.)
- 14. Have you been bothered by an URGENT NEED TO HAVE A BOWEL MOVEMENT during the past week? (This urgent need to go to the toilet is often associated with a feeling that you are not in full control.)
- 15. When going to the toilet during the past week, have you had the FEELING OF NOT COMPLETELY EMPTYING YOUR BOWELS? (This feeling of incomplete emptying means that you still feel a need to pass more stool despite having exerted yourself to do so.)

APPENDIX C: DEPRESSION, ANXIETY, STRESS SCALE – 21

Please read each statement and choose the number which indicates how much the statement applied to you over the <u>past week</u>. There are no right or wrong answers. Do not spend too much time on any statement. The rating scale is as follows:

- 0 = Did not apply to me at all
- 1 = Applied to me some degree, or some of the time
- 2 = Applied to me a considerable degree, or a good part of the time
- 3 = Applied to me very much, or most of the time
- _____1. I found it hard to wind down.
- _____2. I was aware of dryness in my mouth.
- 3. I couldn't seem to experience any positive feeling at all.
- 4. I experience breathing difficulty (e.g., excessively rapid breathing, breathlessness in the absence of physical exertion).
- 5. I found it difficult to work up the initiative to do things.
- 6. I tended to over-react to situations.
- _____7. I experienced trembling (e.g., in the hands).
- 8. I felt that I was using a lot of nervous energy.
- 9. I was worried about situations in which I might panic and make a fool of myself.
- 10. I felt that I had nothing to look forward to.
- _____11. I found myself getting agitated.
- _____ 12. I found it difficult to relax.
- _____13. I felt down-hearted and blue.
- 14. I was intolerance of anything that kept me from getting on with what I was doing.
- _____15. I felt I was close to panic.
- _____16. I was unable to become enthusiastic about anything.
- 17. I felt I wasn't worth much as a person.
- 18. I felt that I was rather touchy.
- 19. I was aware of the action of my heart in the absence of physical exertion (e.g.,

sense of heart rate increase, heart missing a beat).

- _____ 20. I felt scared without any good reason.
- _____ 21. I felt that life was meaningless.

APPENDIX D: EMOTIONAL INTENSITY SCALE – REVISED

Imagine yourself in the following situations and then choose the answer that best describes how you usually feel.

- 1. Someone compliments me. I feel:
 - a. It has little effect on me.
 - b. Mildly pleased.
 - c. Pleased.
 - d. Very pleased.
 - e. Ecstatic on top of the world.
- 2. I am happy. I feel:
 - a. It has little effect on me.
 - b. Mildly happy.
 - c. Happy.
 - d. Extremely happy.
 - e. Euphoric so happy I could burst.
- 3. Someone I am very attracted to asks me out for coffee. I feel:
 - a. Ecstatic on top the world.
 - b. Very thrilled.
 - c. Thrilled.
 - d. Mildly thrilled.
 - e. It has little effect on me.
- 4. I am at a fun party. I feel:
 - a. It has little effect on me.
 - b. A little lighthearted.
 - c. Lively.
 - d. Very lively.
 - e. So lively that I almost feel like a new person
- 5. Something wonderful happens to me. I feel:
 - a. Extremely joyful exuberant.
 - b. Extremely glad.
 - c. Glad.
 - d. A little glad.
 - e. It has little effect on me.
- 6. I have accomplished something valuable. I feel:
 - a. It has little effect on me.
 - b. A little satisfied.
 - c. Satisfied.
 - d. Very satisfied.
 - e. So satisfied it's as if my entire life was worthwhile.
- 7. A person with whom I am involved prepares me a candlelight dinner. I feel:
 - a. It has little effect on me.
 - b. Slightly romantic.
 - c. Romantic.
 - d. Very romantic.
 - e. So passionate nothing else matters.
- 8. I am involved in a romantic relationship. I feel:

- a. So consumed with passion I can think of nothing else.
- b. Very passionate.
- c. Passionate.
- d. Mildly passionate.
- e. It has little effect on me.
- 9. Someone surprises me with a gift. I feel:
 - a. It has little effect on me
 - b. A little grateful
 - c. Grateful
 - d. Very grateful
 - e. So grateful that I want to run out and buy them a gift in return
- 10. Something frustrates me. I feel:
 - a. It has little effect on me.
 - b. A little frustrated.
 - c. Frustrated.
 - d. Very frustrated.
 - e. So extremely tense and frustrated that my muscles knot up.
- 11. I say or do something I should not have done. I feel:
 - a. It has little effect on me.
 - b. I twinge of guilt.
 - c. Guilty.
 - d. Very guilty.
 - e. Extremely guilty.
- 12. Someone criticizes me. I feel:
 - a. It has little effect on me.
 - b. I am a bit taken aback.
 - c. Upset.
 - d. Very upset.
 - e. So extremely upset I could cry.
- 13. I have an embarrassing experience. I feel:
 - a. It has little effect on me.
 - b. A little ill at ease.
 - c. Embarrassed.
 - d. Very embarrassed.
 - e. So embarrassed I want to die.
- 14. Someone I know is rude to me. I feel:
 - a. So incredibly hurt I could cry.
 - b. Very hurt.
 - c. Hurt.
 - d. A little hurt.
 - e. It has little effect on me.
- 15. I see a sad movie. I feel:
 - a. So extremely sad that I feel like weeping.
 - b. Very sad.
 - c. Sad.
 - d. A little sad.

- e. It has little effect on me.
- 16. I am involved in a situation in which I must do well, such as an important exam or job interview. I feel:
 - a. It has little effect on me.
 - b. Slightly anxious.
 - c. Anxious.
 - d. Very anxious.
 - e. So extremely anxious I can think of nothing else.
- 17. I am in an argument. I feel:
 - a. It has little effect on me.
 - b. Mildly angry.
 - c. Angry.
 - d. Very angry.
 - e. So incredibly angry I find it difficult to remain composed.

APPENDIX E: TORONTO ALEXITHYMIA SCALE

Rate the following items from 1, strongly disagree, to 5, strongly agree.

- 1. I am often confused about what emotion I am feeling.
- 2. It is difficult for me to find the right words for my feelings.
- 3. I have physical sensations that even doctors don't understand.
- 4. I am able to describe my feelings easily.
- 5. I prefer to analyze problems rather than just describe them.
- 6. When I am upset, I don't know if I am sad, frightened, or angry.
- 7. I am often puzzled by sensations in my body.
- 8. I prefer to just let things happen rather than to understand why they turned out that way.
- 9. I have feelings that I can't quite identify.
- 10. Being in touch with emotions is essential.
- 11. I find it hard to describe how I feel about people.
- 12. People tell me to describe my feelings more.
- 13. I don't know what's going on inside me.
- 14. I often don't know why I am angry.
- 15. I prefer talking to people about their daily activities rather than their feelings.
- 16. I prefer to watch "light" entertainment shows rather than psychological dramas.
- 17. It is difficult for me to reveal my innermost feelings, even to close friends.
- 18. I can feel close to someone, even in moments of silence.
- 19. I find examination of my feelings useful in solving personal problems.
- 20. Looking for hidden meanings in movies or plays distracts from their enjoyment.

APPENDIX F: EMOTION REGULATION QUESTIONNAIRE

We would like to ask you some questions about your emotional life, in particular, how you control (that is, regulate and manage) your emotions. The questions below involve two distinct aspects of your emotional life. One is your emotional experience, or what you feel like inside. The other is your emotional expression, or how you show your emotions in the way you talk, gesture, or behave. Although some of the following questions may seem similar to one another, they differ in important ways. For each item, please answer using the following scale:

1 =strongly disagree ----- 2 ----- 3 ----- 4 =neutral ----- 5 ----- 6 ----- 7 =strongly agree

- 1. When I want to feel more *positive* emotion (such as joy or amusement), I *change what I'm thinking about*.
- 2. I keep my emotions to myself.
- 3. When I want to feel less *negative* emotion (such as sadness or anger), I *change what I'm thinking about.*
- 4. What I am feeling *positive* emotions, I am careful not to express them.
- 5. When I'm faced with a stressful situation, I make myself *think about it* in a way that helps me stay calm.
- 6. I control my emotions by *not expressing them*.
- 7. When I want to feel more *positive* emotion, I *change the way I'm thinking* about the situation.
- 8. I control my emotions by *changing the way I think* about the situation I'm in.
- 9. When I am feeling *negative* emotion, I make sure not to express them.
- 10. When I want to feel less *negative* emotion, I *change the way I'm thinking* about the situation.

APPENDIX G: DIFFICULTIES IN EMOTION REGULATION SCALE

Please indicate how often the following statements apply to you by filling in the appropriate numbered bubble from the scale below:

	Almost never	Sometimes	About half the time	Most of the time	Almost always
1. I am clear about my feelings.	1	2	3	4	5
2. I pay attention to how I feel.	1	2	3	4	5
3. I experience my emotions as overwhelming and out of control.	1	2	3	4	5
4. I have no idea how I am feeling.	1	2	3	4	5
5. I have difficulty making sense out of my feelings.	1	2	3	4	5
6. I am attentive to my feelings.	1	2	3	4	5
7. I know exactly how I am feeling.	1	2	3	4	5
8. I care about what I am feeling.	1	2	3	4	5
9. I am confused about hwo I feel.	1	2	3	4	5
10. When I'm upset, I acknowledge my emotions.	1	2	3	4	5
11. When I'm upset, I become angry with myself for feeling that way.	1	2	3	4	5
12. When I'm upset, I become embarrassed for feeling that way.	1	2	3	4	5
13. When I'm uspet, I have difficulty getting work done.	1	2	3	4	5
14. When I'm upset, I become out of control.	1	2	3	4	5
15. When I'm uspet, I believe that I will remain that way for a long time.	1	2	3	4	5
16. When I'm upset, I believe that I'll end up feeling very depressed.	1	2	3	4	5

1=almost never (0-10%); 2=sometimes (11-35%); 3=about half the time (36-65%); 4=most of the time (66-90%); 5=almost always (91-100%).

17. When I'm upset, I believe					
that my feelings are valid and	1	2	3	4	5
important.	1	2	3	4	5
18. When I'm upset, I have	1	2	3	4	5
difficulty focusing on other	1	Ζ	3	4	3
things.					
19. When I'm upset, I feel out	1	2	3	4	5
of control.					
20. When I'm upset, I can still	1	2	3	4	5
get things done.			_		_
21. When I'm upset, I feel					
ashamed with myself for	1	2	3	4	5
feeling that way.					
22. When I'm uspet, I know					
that I can find a way to	1	2	3	4	5
eventually feel better.					
23. When I'm uspet, I feel	1	2	3	4	5
like I am weak.	1	2	5	4	5
24. When I'm upset, I feel					
like I can remain in control of	1	2	3	4	5
my behaviors.					
25. When I'm upset, I feel	1	2	2	4	-
guilty for feeling that way.	1	2	3	4	5
26. When I'm upset, I have	1	2	2	4	-
difficulty concentrating.	1	2	3	4	5
27. When I'm upset, I have					
difficulty controlling my	1	2	3	4	5
behaviors.	-	_	C C		C
28. When I'm upset, I believe					
that there is nothing I can do	1	2	3	4	5
to make myself feel better.	1	2	5	•	5
29. When I'm upset, I become					
irritated with myself for	1	2	3	4	5
feeling that way.	1	2	5	-	5
30. When I'm upset, I start to					
feel very bad about myself.	1	2	3	4	5
31. When I'm upset, I belive					
	1	2	3	4	5
that wallowing in it is all I can do	1	۷	3	4	3
32. When I'm upset, I lose	1	2	3	4	5
control over my behaviors.					
33. When I'm upset, I have	1	2	2	A	E
difficulty thinking about	1	2	3	4	5
anything else.					

34. When I'm upset, I take					
time to figure out what I'm	1	2	3	4	5
really feeling.					
35. When I'm upset, it takes	1	r	2	1	5
me a logn time to feel better.	1	2	5	4	5
36. When I'm upset, emotions	1	r	2	1	5
feel overwhelming.	1	2	3	4	5

APPENDIX H: VISCERAL SENSITIVITY INDEX

Below are statements that describe how some people respond to symptoms or discomfort in their belly or lower abdomen. These may include pain, diarrhea, constipation, bloating, or sense of urgency. Please answer "how strong you agree or disagree" with each of these statements, AS THEY RELATE TO YOU. Answer all of the statements as honestly and thoughtfully as you can.

0=strongly agree, 1=moderately agree, 2=mildly agree, 3=mildly disagree, 4=moderately disagree, 5=strongly disagree

- 1. I worry that whenever I eat during the day, bloating and distension in my belly will get worse.
- 2. I get anxious when I go to a new restaurant.
- 3. I often worry about problems in my belly.
- 4. I have a difficult time enjoying myself because I cannot get my mind off of discomfort in my belly.
- 5. I often fear that I won't be able to have a normal bowel movement.
- 6. Because of fear of developing abdominal discomfort, I seldom try new foods.
- 7. No matter what I eat, I will probably feel uncomfortable.
- 8. As soon as I feel abdominal discomfort I begin to worry and feel anxious.
- 9. When I enter a place I haven't been before, one of the first things I do is to look for a bathroom.
- 10. I am constantly aware of the feelings I have in my belly.
- 11. I often feel discomfort in my belly could be a sign of a serious illness.
- 12. As soon as I awake, I worry that I will have discomfort in my belly during the day.
- 13. When I feel discomfort in my belly, it frightens me.
- 14. In stressful situations, my belly bothers me a lot.
- 15. I constantly think about what is happening inside my belly.

APPENDIX I: BODY PERCEPTION QUESTIONNAIRE – SHORT FORM

I. Body Awareness

Please rate your awareness on each of the characteristics described below. Select the answer that most accurately describes you. (Scale: Never, Occasionally, Sometimes, Usually, Always)

During most situations I am aware of:

- 1. Swallowing frequently
- 2. An urge to cough to clear my throat
- 3. My mouth being dry
- 4. How fast I am breathing
- 5. Watering or tearing of my eyes
- 6. Noises associated with my digestion
- 7. A swelling of my body or parts of my body
- 8. An urge to defecate
- 9. Muscle tension in my arms and legs
- 10. A bloated feeling because of water retention
- 11. Muscle tension in my face
- 12. Goose bumps
- 13. Stomach and gut pains
- 14. Stomach distention or bloatedness
- 15. Palms sweating
- 16. Sweat on my forehead
- 17. Tremor in my lips
- 18. Sweat in my armpits
- 19. The temperature of my face (especially my ears)
- 20. Grinding my teeth
- 21. General jitteriness
- 22. The hair on the back of my neck "standing up"
- 23. Difficulty in focusing
- 24. An urge to swallow
- 25. How hard my heart is beating
- 26. Feeling constipated
- II. Autonomic Nervous System Reactivity

The autonomic nervous system is the part of your nervous system that controls your cardiovascular respiratory, digestive, and temperature regulation systems. It is also involved in the experience and expression of emotions. The autonomic nervous system functions differently among people. This scale has been developed to measure how your autonomic nervous system reacts.

Please rate yourself on each of the statements below:

- 27. I have difficulty coordinating breathing and eating
- 28. When I am eating, I have difficulty talking
- 29. My heart often beats irregularly

- 30. When I eat, food feels dry and sticks to my mouth and throat
- 31. I feel shortness of breath
- 32. I have difficulty coordinating breathing with talking
- 33. When I eat, I have difficulty coordinating swallowing, chewing, and/or sucking with breathing
- 34. I have a persistent cough that interferes with my talking and eating
- 35. I gag from the saliva in my mouth
- 36. I have chest pains
- 37. I gag when I eat
- 38. When I talk, I often feel I should cough or swallow the saliva in my mouth
- 39. When I breathe, I feel like I cannot get enough oxygen
- 40. I have difficulty controlling my eyes
- 41. I feel like vomiting
- 42. I have 'sour' stomach
- 43. I am constipated
- 44. I have indigestion
- 45. After eating I have digestive problems
- 46. I have diarrhea

APPENDIX J: POSITIVE AND NEGATIVE AFFECT SCALE

This measure consists of a number of words and statements that describe different feelings and emotions. Please rate each word or statement using the scale below. Please rate each item based on how you are feeling <u>right now, at this moment</u>.

l Very slightly or not at all	2 A little	3 Moderately	4 Quite a bit	5 Extremely
interested distressed excited upset strong guilty scared hostile enthusiastic proud disgusted	с		irritable alert ashamed inspired nervous determined attentive jittery active afraid	

APPENDIX K: STRATEGIES QUESTIONNAIRE

Rate the following items on a scale from 0 (strongly disagree) to 6 (strongly agree).

- 1. I tried not to let my feelings show
- 2. I tried to suppress my emotions3. I thought about the [stimulus] in a way that helps me to experience less emotion
- 4. I tried to adopt an unemotional attitude toward the [stimulus]

APPENDIX L: STATE – DIFFICULTIES IN EMOTION REGULATION QUESTIONNAIRE

Instructions: Please read each statement and indicate how much it applies to <u>YOUR</u> <u>EMOTIONS RIGHT NOW</u>.

1	2	3	4	5			
Not at all	Somewhat	Moderately	Very much	Completely			
1) I feel gu	ilty for feeling this	way.					
2) I am pay	ing attention to ho	w I feel.					
3) I feel out	t of control.						
4) I am eml	parrassed for feelin	ng this way.					
5) I am feel	ling very bad abou	t myself.					
6) I am ack	nowledging my en	notions.					
7) I have no	o idea how I am fe	eling.					
8) I feel ash	named with myself	for feeling this wa	ay.				
9) I am hav	9) I am having difficulty doing the things I need to do right now.						
10) I believ	10) I believe that I will continue feeling this way for a long time.						
11) I care a	_ 11) I care about what I am feeling.						
12) I am an	12) I am angry with myself for feeling this way.						
13) I am ha	13) I am having difficulty controlling my behaviors.						
14) I am co	_ 14) I am confused about how I feel.						
15) I believ	15) I believe that I am going to end up feeling very depressed.						
16) I am tal	16) I am taking time to figure out what I am really feeling.						
17) My em	17) My emotions feel out of control.						
18) I am irr	18) I am irritated with myself for feeling this way.						
19) I believ	19) I believe that my feelings are valid and important.						
20) I feel li	20) I feel like I'm a weak person for feeling this way.						
21) My em	otions feel overwh	elming.					

APPENDIX M: MANIPULATION CHECK ITEMS

- 1. What do you believe the purpose of the three tasks was? (open ended)
- 2. What do you believe the intended emotion of the first task was? (open ended)
- 3. What do you believe the intended emotion of the second task was? (open ended)
- 4. What do you believe the intended emotion of the third task was? (open ended)
- 5. Do you typically watch videos that some people would find disgusting (e.g., pimple popping, botched surgery television shows)?
 - a. Yes
 - b. No
- 6. To what extent do you enjoy watching these videos or shows?
 - a. Not at all
 - b. A little
 - c. Moderately
 - d. Very much
- 7. To what extent do you experience disgust or get "grossed out" when watching these videos or shows?
 - a. Not at all
 - b. A little
 - c. Moderately
 - d. Very much
- 8. To what extend are you saddened by watching television shows that depict death such as the death of a spouse or loved one?
 - a. Not at all
 - b. A little
 - c. Moderately
 - d. Very much
- 9. Have you seen the television show *This Is Us*?
 - a. Yes
 - b. No
- 10. Do you like the television show *This Is Us*?
 - a. Yes
 - b. No
- 11. Have you seen the episode that was shown earlier?
 - a. Yes, and I had the same reaction as the first time I saw it
 - b. Yes, and I had a completely different reaction than the first time I saw it
 - c. No
- 12. How did your reaction differ watching it this time? (open ended)
- 13. When having to give a speech, do you typically become anxious, nervous, or uncomfortable?
 - a. Yes
 - b. No
- 14. Did you believe that you would have to give the speech you prepared for?
 - a. Yes
 - b. No

LIST OF TABLES

		n (%)
Age $(M \pm SD)$		20.59 ± 5.50
Sex	Female	211 (72.5)
	Male	80 (27.5)
Race	White	240 (82.5)
	Black	28 (9.6)
	Multiracial	10 (3.4)
	Asian	9 (3.1)
	Other	3 (1.0)
	Preferred not to answer	1 (0.3)
Hispanic/Latinx		19 (6.5)
Year in college	Freshman	151 (51.9)
U	Sophomore	57 (19.6)
	Junior	38 (13.1)
	Senior	29 (10.0)
	Other	16 (5.5)
First generation student		46 (15.8)
Self-reported GI	Irritable bowel syndrome	13 (4.5)
diagnoses	Gastroesophageal reflux disease	5 (1.7)
diagnoses	Gastroparesis	1(0.3)
	Celiac disease	1(0.3)
	Other (e.g., lactose intolerance, leaky gut)	4 (1.2)
	Crohn's disease, ulcerative colitis, gastritis, and	0(0.0)
	diverticulitis	
Medical providers seen	General practitioner	45 (15.5)
for GI distress	Specialist (e.g., gastroenterologist)	22 (7.6)
	Mental health provider	5 (1.7)
	Other (e.g., emergency department)	2 (0.7)
OTC medication taken	Antacids	83 (28.5)
for GI distress	Laxatives	37 (12.7)
	Gas relief	26 (8.9)
	Diuretics	4 (1.4)
	Other (e.g., acetaminophen)	8 (2.7)
GI avoidance behaviors	Avoiding specific food	190 (65.3)
	Avoid eating before specific event/activity	157 (54.0)
	Eating small portions	146 (50.2)
	Avoiding eating certain times of day	123 (42.3)
	Checking for location of public restroom	58 (31.6)
	Carrying medication, food, or water	91 (31.3)
	Avoiding restaurants or eating outside of the home	91 (31.3)
	Relying on medication when traveling	65 (22.3)
	Avoiding sexual activity	59 (20.3)

Table 1. Overall sample demographic, gastrointestinal, and clinical indicators of emotional

characteristics (N = 291).

	Avoiding situations without private restroom	58 (19.9)
	Avoiding exercise	54 (18.6)
	Avoiding public transportation	48 (16.5)
Depression	Normal	183 (62.9)
	Mild	25 (8.6
	Moderate	48 (16.5
	Severe	13 (4.5
	Extremely Severe	22 (7.6)
Anxiety	Normal	177 (60.8)
	Mild	17 (5.8)
	Moderate	45 (15.5)
	Severe	23 (7.9)
	Extremely Severe	29 (10.0)
Stress	Normal	200 (68.7
	Mild	31 (10.7
	Moderate	31 (10.7
	Severe	24 (8.2)
	Extremely Severe	5 (1.7)
Alexithymia	Clinical	63 (21.6)
-	Sub-clinical	73 (25.1)

 $\overline{Note. \text{ GI} = \text{gastrointestinal; OTC} = \text{over-the-counter.}}$

Table 2. Means and standard deviations for trait characteristics between FGID and control

participants (n = 51).

	FGID Group ($n = 27$)	Control Group ($n = 24$)
	M(SD)	M(SD)
GI symptom severity	2.5 (1.1)	1.5 (0.6)
Depression	9.0 (9.8)	5.8 (8.1)
Normal ^a	19 (70.4)	19 (79.2)
Mild ^a	1 (3.7)	1 (4.2)
Moderate ^a	3 (11.1)	2 (8.3)
Severe-Extremely Severe ^a	4 (14.8)	2 (8.4)
Anxiety	8.4 (6.1)	2.8 (4.8)
Normal ^a	11 (40.7)	21 (87.5)
Mild ^a	4 (14.8)	1 (4.2)
Moderate ^a	7 (25.9)	1 (4.2)
Severe-Extremely Severe ^a	5 (18.5)	1 (4.2)
Stress	13.4 (6.7)	6.9 (8.1)
Normal ^a	15 (55.6)	19 (79.2)
Mild ^a	5 (18.5)	3 (12.5)
Moderate ^a	6 (22.2)	1 (4.2)
Severe-Extremely Severe ^a	1 (3.7)	1 (4.2)
Emotion Intensity	61.03 (5.95)	56.67 (8.91)
Alexithymia ^a	14 (51.9)	6 (25.0)
Reappraisal	29.19 (7.89)	28.04 (7.21)
Suppression	14.59 (5.27)	15.67 (5.82)
ER Difficulties ^a	15 (29.4)	12 (23.5)
Visceral Sensitivity	25.96 (20.12)	7.29 (11.08)
Body Awareness	15.19 (6.70)	8.83 (7.17)
ANS-Supra	4.04 (2.88)	2.63 (3.29)
ANS-Sub	3.48 (1.99)	1.33 (1.79)

Note. Alexithymia = sub-clinical and clinical levels included; ANS-Sub = autonomic nervous system reactivity in the subdiaphragmatic region; ANS-Supra = autonomic nervous system reactivity in the supradiaphragmatic region; ER = emotion regulation; GI = gastrointestinal. an (%) Table 3. Descriptive statistics and Pearson correlations of the relation between gastrointestinal

	GSRS	Mean (SD)	Observed	Possible
		· · /	Range	Range
Hypothesis 1				
Depression	.305*	8.73 (9.82)	0 - 42	0 - 42
Anxiety	.468*	7.40 (8.29)	0 - 38	0 - 42
Stress	.518*	10.95 (9.33)	0 - 42	0 - 42
Hypothesis 2				
Emotion Intensity	.288*	58.65 (8.16)	32 - 85	17 - 85
Alexithymia	.204*	50.51 (11.76)	26 - 75	20 - 100
Reappraisal	007	27.17 (6.62)	6 - 41	6 - 42
Suppression	.061	15.36 (4.73)	4 - 28	4 - 28
ER Abilities	.310*	87.74 (26.10)	39 - 161	36 - 180
Visceral Sensitivity	.631*	15.22 (16.83)	0 - 75	0 - 75
Body Awareness	.487*	13.98 (7.85)	0 - 26	0 - 26
ANS-Supra	.463*	3.95 (3.94)	0 - 15	0 - 15
ANS-Sub	.692*	2.32 (2.07)	0 - 6	0 - 6

symptoms and emotional and physiological symptoms (N = 291).

Note. ANS-Sub = autonomic nervous system reactivity in the subdiaphragmatic region; ANS-Supra = autonomic nervous system reactivity in the supradiaphragmatic region; ER = emotion regulation; GSRS = Gastrointestinal Symptom Rating Scale. *p < .001

Table 4. Means and standard deviations for induction-related emotional responding between FGID and control participants (n = 52).

	FGID Group $(n = 28)$			Control Group ($n = 24$)				
	Neutral	Anxiety	Sadness	Disgust	Neutral	Anxiety	Sadness	Disgust
SUDS	1.18 (1.44)	4.11 (2.39)	3.00 (1.94)	4.29 (2.61)	0.98 (1.42)	4.21 (2.50)	2.63 (1.56)	3.81 (2.36)
Negative Affect	13.04 (2.62)	20.39 (7.94)	16.68 (6.44)	17.57 (6.42)	11.83 (2.96)	16.54 (5.24)	14.50 (4.24)	16.96 (6.55)
State Reappraisal	3.43 (2.12)	4.36 (3.94)	3.75 (3.09)	3.68 (3.59)	4.33 (3.38)	4.75 (3.86)	2.88 (3.69)	4.63 (4.01)
State Suppression	4.64 (2.82)	4.54 (4.25)	3.86 (3.34)	3.86 (3.65)	3.88 (3.14)	4.17 (3.64)	3.25 (3.44)	3.42 (3.59)
State ER Difficulties	34.57 (7.53)	34.64 (8.27)	31.50 (5.82)	32.11 (5.72)	35.79 (9.68)	35.63 (9.68)	36.21 (10.10)	36.08 (9.77)
<i>Note</i> . ER = emotion regulation; FGID = functional gastrointestinal disorder; SUDS = subjective units of distress.								

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	W 14 10D	D 1 1 CE	MOE		10	
	Weighted SE	Pooled SE	MSE	t	df	<i>p</i>
SUDS	0.155	0.910	5.882			
C1				-0.316	1	.403
C2				0.183	1	.442
C3				0.286	1	.411
Reappraisal	0.155	2.696	17.420			
C1				0.312	1	.404
C2				1.084	1	.237
C3				-0.025	1	.492
Suppression	0.155	2.663	17.208			
C1				0.314	1	.403
C2				1.091	1	.236
C3				-0.026	1	.492
ER Difficulties	0.155	18.423	119.038			
C1				0.055	1	.482
C2				-0.813	1	.283
C3				-0.642	1	.318

Table 5. Post-hoc planned contrasts examining interaction effects of group (FGID, control) and induction (anxiety, sadness, disgust) relative to neutral induction (n = 52).

Note. C1 = anxiety compared to neutral contrast; C2 = sadness compared to neutral contrast; C3 = disgust compared to neutral contrast; ER = emotion regulation; MSE = mean squared error; SE = standard error; SUDS = subjective units of distress.

VITA

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EDUCATION

2021-	Predoctoral Psychology Residency/Internship (APA-Accredited) The Charleston Consortium (Medical University of South Carolina [MUSC] and Ralph H. Johnson Veterans Affairs Medical Center [VAMC]), Charleston, SC Track: Adult Psychopathology <u>Research Preceptor</u> : Daniel F. Gros, Ph.D.
2016–	Doctor of Philosophy, Clinical Psychology (expected 2022; APA-Accredited) University of Mississippi, University, MS <u>Dissertation</u> (defended 8/25/2021): <i>Do individuals with functional</i> gastrointestinal symptoms experience emotions differently? An examination of emotional responding during a series of emotion induction tasks (Chair: Laura J. Dixon, Ph.D.)
2018	Master of Arts, Clinical Psychology (APA-Accredited) University of Mississippi, University, MS <u>Thesis</u> : Nonmedical use of prescription drugs among young adults: An examination of anxiety sensitivity, distress tolerance, and emotion-driven impulse control difficulties (Chair: Laura J. Dixon, Ph.D.)
2014	Bachelor of Arts, Psychology, <i>Cum Laude</i> University of Miami, Coral Gables, FL <u>Honors Thesis</u> : <i>The relationship between traumatic life events and hoarding</i> <i>symptoms</i> (Chair: Kiara R. Timpano, Ph.D.)

PEER-REVIEWED PUBLICATIONS

*denotes mentored undergraduate student author

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- Dixon, L. J., & Witcraft, S. M. (2020). Anxiety and skin disease. In S. Richards (Volume Ed.) & L. M. Cohen (Series Ed.), *Wiley encyclopedia of health psychology, volume III: Clinical health and behavioral medicine* (1st ed., pp. 451–458). John Wiley & Sons.
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GRANT EXPERIENCE

Misophonia Research FundRole: Research Assistant02/08/2021-07/23/2021Advancing the Characterization and Assessment of Misophonia through Laboratory and

Population-based Research

Health and Anxiety Research and Treatment Lab, University of Mississippi, University, MS Principle Investigator: Laura J. Dixon, Ph.D.

1R34-MH099218-01A1Role: Research Coordinator08/15/2014-07/01/2016National Institute of Mental Health3/3 Dose Timing of D-Cycloserine to Augment CBT for Social Anxiety Disorder

Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX Principle Investigator: Jasper A. J. Smits, Ph.D. (with Stefan G. Hofmann, Ph.D. & Mark H. Pollock, M.D.

AWARDS AND HONORS

2020	Graduate Research Achievement Award, University of Mississippi, Psychology Department
2019	Outstanding Paper Presentation Award, 9 th Annual Graduate Research Symposium, University of Mississippi
2019	Graduate Student Council Travel Award (\$300), University of Mississippi
2016-2020	Graduate Honors Fellowship (\$3,000 per year), University of Mississippi
2016–2020	Psychology Department Travel Award (\$400 per year), University of Mississippi
2014	B.A. Awarded Cum Laude, Departmental Honors in Psychology, University of
	Miami
2014	First Place, Research Creativity and Innovation Forum: Social Sciences Division,
	University of Miami
2013	Student Travel Scholarship, University of Miami
2013	College of Arts and Sciences Summer Program for Underrepresented Minorities and
	Women (\$2,500), University of Miami
2013	Psychology Research Initiatives Mentorship Experience (PRIME) Summer Research
	Scholarship (\$2,000), University of Miami
2010-2014	President's Honor Roll, Provost's Honor Roll, and Dean's List, All semesters,
	University of Miami

SYMPOSIA AND ORAL PRESENTATIONS

- 10. Witcraft, S. M., & Dixon, L. J. (2020, November). Examining the mediating role of emotion regulation difficulties in behavioral avoidance of gastrointestinal symptoms. In L. Dixon & A. Lee (Chairs), *Extending the impact of cognitive and behavioral therapies through the integration of health outcomes: A closer look at emotion regulation processes* [Symposium]. Association for Behavioral and Cognitive Therapies 54th Annual Convention, Philadelphia, PA, United States.
- Dixon, L. J., Schadegg, M. J., Boullion, G. Q., Witcraft, S. M., & Perry, M. M. (2019, November). Obsessive-compulsive related disorders, emotion regulation, and quality of life in adults with skin disease. In J. McCann (Chair), *Change that matters: What, why, and how meaningful change happens in CBT for anxiety-related disorders* [Symposium]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
- 8. Witcraft, S. M., & Dixon, L. J. (2019, April). *Psychological sensitivities in dental anxiety: An examination of sensitivities to anxiety, disgust, and pain* [Data blitz]. University of Mississippi Psychological Research Day 6th Annual Meeting, University, MS, United States.

- Witcraft, S. M., & Dixon, L. J. (2019, March). The role of emotion-driven impulsivity in inpatients with anxiety and prescription drug abuse [Paper presentation].** University of Mississippi Graduate Student Council Research Symposium 9th Annual Meeting, University, MS, United States.
 **This presentation received an award for being among the top three presentations in its category.
- Boullion, G. Q., Dixon, L. J., Perry, M. M., & Witcraft, S. M. (2018, November). Emotion regulation difficulties and depression among individuals with dermatological and body dysmorphic concerns. In B. Mathes & B. Summers (Chairs), *Recent advances in OC spectrum disorders: A transdiagnostic and translational perspective* [Symposium]. Association for Behavioral and Cognitive Therapies 52nd Convention, Washington, D.C., United States.
- Vrijsen, J. N., Dainer-Best, J., Witcraft, S. M., Papini, S., Müller, B., Hertel, P., Beevers, C. G., Becker, E. S., Tendolkar, I., & Smits, J. A. J. (2018, July). Retrieval-based memory bias modification for depression. In M. Weymar (Chair), *Current research and emerging directions in emotional memory: Evidence from healthy functioning, psychopathology, and interventions* [Symposium]. International Conference of the European Society for Cognitive and Affective Neuroscience 4th Annual Meeting, Leiden, The Netherlands.
- 4. Witcraft, S. M., & Dixon, L. J. (2018, April 13). *Examining emotion regulation differences in prescription drug abusers and non-users* [Data blitz]. University of Mississippi Psychology Research Day 5th Annual Meeting, University, MS, United States.
- Dixon, L. J., & Witcraft, S. M. (2017, November). Anxiety sensitivity and quality of life among adults with dermatological conditions. In L. Dixon (Chair), *Expanding the AS horizon: Recent advances in the study of anxiety sensitivity among individuals with medical conditions* [Symposium]. Association for Behavioral and Cognitive Therapies 51st Annual Meeting, San Diego, CA, United States.
- Witcraft, S. M., & Dixon, L. J. (2017, September). Prescription drug use and trait characteristics of psychopathology in an undergraduate sample. In M. Perry (Chair), *The age of anxiety: Exploring and assessing anxiety and its problematic health correlates* [Symposium]. Mississippi Psychological Association 68th Annual Conference, Biloxi, MS, United States.
- 1. Witcraft, S. M., & Dixon, L. J. (2017, April). *Prescription opioid and anxiolytic use and trait characteristics of anxiety in an undergraduate sample* [Data blitz]. University of Mississippi Psychology Research Day 4th Annual Meeting, University, MS, United States.

POSTER PRESENTATIONS

*denotes mentored undergraduate student author

- 20. Witcraft, S. M., *Niehaus, L. K., *Schruff, M. A., & Dixon, L. J. (2021, November). Do gut reactions matter? How individuals with and without gastrointestinal symptoms respond to emotional stimuli [Poster accepted for presentation]. Association for Behavioral and Cognitive Therapies 55th Annual Convention, New Orleans, LA, United States.
- *Schruff, M. A., *Niehaus, L. K., Witcraft, S. M., & Dixon, L. J. (2021, September 29 October 1). *Emotion regulation strategies among those with gastrointestinal symptoms: Findings from an experimental study* [Poster accepted for presentation]. Mississippi Psychological Association 72nd Annual Conference, Biloxi, MS, United States.

- Witcraft, S. M., Dixon, L. J., & Lee, A. A. (2020, November). Concerns about physical symptoms are associated with overuse of healthcare and short-acting medication among individuals with asthma [Poster presentation]. Association for Behavioral and Cognitive Therapies 54th Annual Convention, Philadelphia, PA, United States.
- Witcraft, S. M., Schadegg, M. J., Boullion, G. Q., Perry, M. M., & Dixon, L. J. (2019, November). What sensitivities matter in dental anxiety? Investigating sensitivity to anxiety, pain, and disgust [Poster presentation]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
- 16. Schadegg, M. J., Witcraft, S. M., Perry, M. M., Boullion, G. Q., & Dixon, L. J. (2019, November). An aggressive reaction to sound: The interactive effects of anxiety sensitivity and misophonia on facets of aggression [Poster presentation]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
- Boullion, G. Q., Perry, M. P., Witcraft, S. M., Schadegg, M. J., & Dixon, L. J. (2019, November). Social anxiety and loneliness: The indirect effect of emotion regulation difficulties [Poster presentation]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
- Perry, M. M., Boullion, G. Q., Schadegg, M. J., Witcraft, S. M., & Dixon, L. J. (2019, November). *Examining interpersonal and intrapersonal emotion regulation, social anxiety, and aggression among college students* [Poster presentation]. Association for Behavioral and Cognitive Therapies 53rd Annual Convention, Atlanta, GA, United States.
- LaRosa, K. N., Niel, K., Klages, K. L., Mandrell, B. N., Merchant, T. E., Wise, M. S., Witcraft, S. M., Hancock, D., Caples, M., & Crabtree, V. M. (2019, June). *Comparison of actigraphy to polysomnography in the measurement of sleep in children treated for craniopharyngioma* [Poster presentation]. SLEEP 33rd Annual Meeting, San Antonio, TX, United States.
- *Ellison, L., *York, M., Witcraft, S. M., & Dixon, L. J. (2019, April). *It's not just "skin deep:"* Social anxiety and anxiety sensitivity in adults with psychodermatological disorders [Poster presentation]. University of Mississippi Psychological Research Day 6th Annual Meeting, University, MS, United States.
- 11. *Long, M. M., *Sappington, L. M., *Seale, N. M., Witcraft, S. M., & Dixon, L. J. (2019, April). Use of safety behaviors in predicting social anxiety in adults with dermatological conditions [Poster presentation]. University of Mississippi Psychological Research Day 6th Annual Meeting, University, MS, United States.
- Witcraft, S. M., Perry, M. M., Boullion, G. Q., & Dixon, L. J. (2018, November). *The moderating role of anxiety sensitivity social concerns in stress and quality of life among adults with skin disease* [Poster presentation]. Association for Behavioral and Cognitive Therapies 52nd Annual Convention, Washington, D.C., United States.
- Perry, M. M., Boullion, G. Q., Witcraft, S. M., Viana, A., & Dixon, L. J. (2018, November). *The importance of a mother's perceived ability to regulate emotions in postpartum maternal quality of life and parenting distress* [Poster presentation]. Association for Behavioral and Cognitive Therapies 52nd Annual Convention, Washington, D.C., United States.

- *Olson, S., Witcraft, S. M., & Dixon, L. J. (2018, April 13). Examination of dental anxiety in relation to anxiety sensitivity, pain sensitivity, and distress tolerance [Poster presentation]. University of Mississippi Psychology Research Day 5th Annual Meeting, University, MS, United States.
- *Cantrell, A. N., *Young, G. K., Witcraft, S. M., & Dixon, L. J. (2018, April 13). Prescription stimulants and polysubstance use among college students [Poster presentation]. University of Mississippi Psychology Research Day 5th Annual Meeting, University, MS, United States.
- Witcraft, S. M., Dixon, L. J., Perry, M. M., Gratz, K. L., & Tull, M. T. (2017, November). *Correlates of nonmedical use of prescription drugs among patients with co-occurring anxiety and substance use disorders* [Poster presentation]. Association for Behavioral and Cognitive Therapies 51st Annual Convention, San Diego, CA, United States.
- Witcraft, S. M., Davis, M. L., Baird, S. O., & Smits, J. A. J. (2016, October). Increased use of negative emotion words during public speaking exposures predicts greater decreases in social anxiety symptoms [Poster presentation]. Association for Behavioral and Cognitive Therapies 50th Annual Convention, New York, NY, United States.
- 4. Baird, S. O., Davis, M. L., **Witcraft, S. M.**, & Smits, J. A. J. (2016, April). *Linguistic analysis as a correlate of fear activation and social anxiety change* [Poster presentation]. Anxiety and Depression Association of America 36th Annual Conference, Philadelphia, PA, United States.
- Witcraft, S. M., Powers, M. B., Gillespie, E. M., Kauffman, B. Y., Becker, E., Cuijpers, P., Van Ameringen, M. & Smits, J. A. J. (2015, November). *A meta-analysis of psychological and pharmacological treatments for generalized anxiety disorder (GAD)* [Poster presentation]. Association for Behavioral and Cognitive Therapies 49th Annual Convention, Chicago, IL, United States.
- Witcraft, S. M., Davis, M. L., Julian, K., Beard, C., Schmidt, N. B., Powers, M. B., & Smits, J. A. J. (2015, April). *Correlating attention bias to social anxiety symptom severity: A second look using Trial Level Bias Score* [Poster presentation]. Anxiety and Depression Association of America 35th Annual Conference, Miami, FL, United States.
- Witcraft, S. M., Shaw, A. M., Pedersen, E. J., & Timpano, K. R. (2013, October). *The relationship between traumatic life events and hoarding symptoms* [Poster presentation]. Society for Advancement of Chicano and Native Americans in Science 40th Annual Conference, San Antonio, TX, United States.

RESEARCH POSITIONS AND EMPLOYMENT

- 2016–2021 **Graduate Research Assistant** Health and Anxiety Research and Treatment Lab, University of Mississippi, University, MS <u>Supervisor</u>: Laura J. Dixon, Ph.D.
- 2017–2018 **Graduate Research Assistant** Mississippi Contextual Psychology Lab, University of Mississippi, University, MS <u>Supervisors</u>: Kelly G. Wilson, Ph.D. & K. Kate Kellum, Ph.D., BCBA-D
- 2017–2018 Graduate Research Assistant St. Jude Children's Research Hospital, Department of Psychology, Memphis, TN

Supervisor: Valerie M. Crabtree, Ph.D.

- 2014–2016 **Project Coordinator** Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX <u>Supervisors</u>: Jasper A. J. Smits, Ph.D. & Mark B. Powers, Ph.D.
- 2012–2014 Undergraduate Research Assistant Program for Anxiety, Stress, and OCD, University of Miami, Coral Gables, FL Supervisor: Kiara R. Timpano, Ph.D.
- 2011–2012 **Undergraduate Research Assistant** Schizophrenia Family Lab, University of Miami, Coral Gables, FL <u>Supervisor</u>: Amy Weisman de Mamani, Ph.D.

CLINICAL EXPERIENCE

2021– Predoctoral Resident/Intern

The Charleston Consortium, Charleston, SC

Ralph H. Johnson VAMC, Mental Health Service Line <u>Rotation</u>: Cognitive Behavioral Therapy Clinic for Emotional Disorders <u>Supervisor</u>: Daniel F. Gros, Ph.D.

MUSC, Department of Psychiatry and Behavioral Sciences <u>Rotation</u>: Sleep and Anxiety Treatment and Research Program <u>Supervisor</u>: Allison K. Wilkerson, Ph.D.

<u>Rotation</u>: Women's Health and High Risk OB Clinic (February – July 2022) <u>Supervisor</u>: Amber Jarnecke, Ph.D.

<u>Rotation</u>: Health and Wellness Institute (February – July 2022) <u>Supervisor</u>: Alyssa A. Rheingold, Ph.D.

2020–2021 **Opioid Replacement Therapist** Willow Pain and Wellness, Oxford, MS <u>Supervisor</u>: John Young, Ph.D.

March- Outpatient Therapist

- June 2020 Communicare, Lafayette County Office Region 2 Community Mental Health Center, Oxford, MS <u>Supervisors</u>: Dixie Church, M.A., LMFT & Scott Gustafson, Ph.D., ABPP
- 2019–2020 **Primary & Transition Residential Program Therapist** Communicare's Chemical Dependency Unit: The Haven House Region 2 Community Mental Health Center, Oxford, MS <u>Supervisors</u>: Dixie Church, M.A., LMFT, Scott Gustafson, Ph.D., ABPP, & Terri Hall, LCSW
- 2019–2020 **Psychological Assessor** Psychological Assessment Clinic, University of Mississippi, University, MS Supervisor: Scott Gustafson, Ph.D., ABPP

2018–2019 Therapist

Education and Behavior Support, The Baddour Center, Senatobia, MS Supervisor: Joshua C. Fulwiler, Ph.D. & Deborah McNamee, BCBA

2017–2018 **Dialectical Behavior Therapy Skills Group Co-Leader** Psychological Services Center, University of Mississippi, University, MS <u>Supervisor</u>: Laura J. Dixon, Ph.D.

2016–2021 Graduate Therapist

Psychological Services Center, University of Mississippi, University, MS <u>Supervisors</u>: Laura J. Dixon, Ph.D., Alan M. Gross, Ph.D., Scott A. Gustafson, Ph.D., ABPP, & John Young, Ph.D.

2015–2016 Clinical Interviewer & Assessor

Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX <u>Supervisors</u>: Jasper A. J. Smits, Ph.D. & Mark B. Powers, Ph.D.

2014–2016 **Protocol Therapist – Social Anxiety Disorder RCT** Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX Supervisor: Jasper A. J. Smits, Ph.D.

2014–2016 Clinic Coordinator

Anxiety & Stress Clinic, University of Texas-Austin, Austin, TX <u>Supervisors</u>: Jasper A. J. Smits, Ph.D. & Mark B. Powers, Ph.D.

- 2013 Group Facilitator for Self-Directed Cognitive Behavioral Therapy for Hoarding Disorder Program for Anxiety, Stress, and OCD, University of Miami, Coral Gables, FL Supervisor: Kiara R. Timpano, Ph.D.
- 2011–2014 **Practicum Clinic Office Assistant** Psychological Services Center, University of Miami, Coral Gables, FL <u>Supervisor</u>: Saneya Tawfik, Ph.D.

TEACHING EXPERIENCE

- 2020–2021 **Instructor of Record** University of Mississippi, University, MS <u>Course</u>: Introduction to Psychology (online)
- 2017, 2020 **Teaching Assistant** University of Mississippi, University, MS <u>Course</u>: Abnormal Psychology (web-based)

2016–2018 **Guest Lecturer** University of Mississippi, University, MS <u>Courses</u>: *Introduction to Psychology*, "Abnormal Psychology: Anxiety, Stress, and Depressive Disorders"

Abnormal Psychology, "Generalized Anxiety Disorder"; "Obsessive-Compulsive Spectrum Disorders"

MENTORING EXPERIENCE

- 2020-2021 Mentor for Undergraduate Psychology Majors Department of Psychology, University of Mississippi, University, MS
- 2020-2021 **Graduate Student Peer Mentor** Department of Psychology, University of Mississippi, University, MS
- 2017-2020 **Graduate Student Mentor: Undergraduate Honors Thesis Projects** Health and Anxiety Research and Treatment Lab, University of Mississippi, University, MS Through the screen: Examining peer relationships, social anxiety, loneliness, and social media in undergraduates Lindsay Sappington, Defended: April 29, 2020

It's not just "skin deep": Social anxiety and anxiety sensitivity in adults with psychodermatological disorders Lauren Ellison, Defended: May 2, 2019

Examination of dental distress and anxiety-related vulnerability factors Sydney Olson, Defended: May 3, 2018

- 2017-2021 **Undergraduate Research Assistant Supervisor** Health and Anxiety Research and Treatment Lab, University of Mississippi, University, MS
- 2014-2016 **Research Assistant Training Supervisor and Mentor** Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX
- 2012-2014 **Undergraduate Student Peer Mentor** Counseling Outreach and Peer Education, University of Miami, Coral Gables, FL

SERVICE AND ADMINISTRATIVE ROLES

2020	Diversifying Psychology Day Panelist
	Department of Psychology, University of Mississippi, University, MS
2020-2021	Assistant to Director of Undergraduate Studies
	Department of Psychology, University of Mississippi, University, MS
2019–2020	Graduate Student Advisory Committee Member
	Department of Psychology, University of Mississippi, University, MS
2019–2020	Communications Committee Member
	Department of Psychology, University of Mississippi, University, MS
2016-2021	Website Design Coordinator
	Health and Anxiety Research and Treatment Lab, University of Mississippi,
	University, MS
2014-2016	Website Design & Unkeen

website Design & Upkeep

Anxiety & Health Behaviors Lab, Anxiety & Stress Clinic, University of Texas-Austin, Austin, TX

ATTENDED WORKSHOPS AND CLINICAL TRAININGS

09/2020	Diversity, Equity, and Inclusion Training
	University of Mississippi, University, MS
05/2020	Provider Training for Cognitive Behavioral Therapy of Insomnia (CBT-I), 6.5 hour
	Continuing Education training
	Medical University of South Carolina
03/2020	American Psychological Association Telepsychology Best Practice 101, 8 hour
	Continuing Education training
	American Psychological Association
02/2020	Delis-Kaplan Executive Function System (D-KEFS) Workshop
	Psychological Assessment Clinic, University of Mississippi, University, MS
	Instructor: Alexis K. Liberto, M.A.
02/2020	Sexual Paraphilias and 'Kink-Aware Therapy'
	University of Mississippi, University, MS
	Instructors: Sarah A. Bilsky, Ph.D. & Carrie V. Smith, Ph.D.
11/2019	Functional Analysis in Process-based CBT
	Association for Behavioral and Cognitive Therapies Annual Convention, Atlanta, GA
	Instructors: Stefan G. Hofmann, Ph.D. & Steven C. Hayes, Ph.D.
11/2019	Dimensions and Diagnosis of Autism Spectrum Disorder
	University of Mississippi, University, MS
	Instructor: Joshua C. Fulwiler, Ph.D.
05/2019	Making Your Job Easier – Using Applied Behavior Analysis
	The Baddour Center, Senatobia, MS
	Instructor: Molly Campbell Arana
05/2019	Trauma Informed Care – Understanding How the Past can Affect Future Behavior
	The Baddour Center, Senatobia, MS
	Instructor: Stephen Bell, Ph.D.
10/2018	Introduction to Mediation, Moderation, and Conditional Process Analysis, two-day
	training workshop
	University of Mississippi, University, MS
	Instructor: Andrew F. Hayes, Ph.D.
03/2016	Exposure Therapy for Generalized Anxiety Disorder, specialized training
	Institute for Mental Health Research, University of Texas-Austin, Austin, TX
00/001	Instructor: Eni S. Becker, Ph.D.
09/2015	Prolonged Exposure Therapy for PTSD, two-day training seminar
	Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX
07/0015	Instructor: Mark B. Powers, Ph.D.
07/2015	SCID-101 for DSM-IV Training Series
	Anxiety & Health Behaviors Lab, University of Texas-Austin, Austin, TX

AD HOC REVIEWING EXPERIENCES/ACTIVITIES

Behaviour Research and Therapy Children's Health Care Clinical Practice in Pediatric Psychology Clinical Psychology Review Cognitive Therapy and Research Frontiers in Psychology Journal of Clinical Sleep Medicine Journal of Consulting and Clinical Psychology Journal of Neuro-Oncology Journal of Nervous and Mental Disease Pediatrics Sleep Sleep Health: Journal of the National Sleep Foundation Sleep Medicine The Journal of General Psychology

PROFESSIONAL AFFILIATIONS

2015– Association for Behavioral and Cognitive Therapies