Internalizing Symptoms in Autistic Young Adults: Comparing the Cognitive and Physiological Components of Emotion Regulation

Sarah Lehman
Marquette University

INTERNALIZING SYMPTOMS IN AUTISTIC YOUNG ADULTS: COMPARING THE COGNITIVE AND PHYSIOLOGICAL COMPONENTS OF EMOTION REGULATION

By

Sarah A. Lehman, B.A.

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ABSTRACT
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Sarah A. Lehman, B.A.

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Autistic people experience co-occurring mental illness at a substantially greater rate compared to their neurotypical counterparts. Of these comorbid psychopathologies, internalizing disorders (anxiety and depression) are among the most prevalent. Emotion dysregulation has been identified as a contributing factor to this phenomenon and potential treatment target. The current study employed cognitive (e.g., use of suppression and use of cognitive reappraisal) and physiological (e.g., respiratory sinus arrhythmia (RSA) and heart period) measures to more holistically capture the multifaceted construct of emotion regulation compared to prior research. In a sample of autistic young adults (N = 63) ages 17-29 (M = 20.14), backward hierarchical regression revealed that cognitive reappraisal significantly predicted symptoms of both anxiety and depression whereas suppression significantly predicted symptoms of only anxiety. The physiological variables were not retained as significant predictors in either model. In sum, the present multi-method study underscores the cognitive component of emotion dysregulation in autistic young adults, particularly as it relates to internalizing symptomatology. These findings have clinical implications for the treatment of anxiety and depression in autistic adult populations. Possible explanations for the null physiological findings and future directions are discussed.

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INTRODUCTION

Autism spectrum disorders (ASDs) are a category of lifelong neurodevelopmental disorders characterized by persistent difficulties with social communication and restricted, repetitive patterns of behavior, interests, or activities (APA, 2013). Symptoms are first present in early childhood and cause clinically significant impairment in social, occupational or other important areas of functioning (APA, 2013). In addition to the array of functional impairments that are often related to ASD, autistic people are at a heightened risk for experiencing co-occurring mental illness (Simonoff et al., 2008). Of all the comorbid psychopathologies diagnosed in the autistic population, internalizing disorders (anxiety and depression) are among the most prevalent (Joshi et al., 2012). At any given point, approximately 27% of autistic people will have an anxiety disorder compared to 12% of the general population and 23% of autistic people will have depression compared to 7% of the general population (Hollocks et al., 2018; Kessler et al., 2003; Kessler et al., 2012). Addressing such staggering rates of internalizing disorders in an already vulnerable population is imperative to promoting the well-being of the autistic community.

Emerging evidence points to emotion dysregulation as a key factor to explicating such elevated levels of internalizing disorders in the autistic population. As such, the present study aims to explore the relationship between the cognitive and physiological aspects of emotion regulation with symptoms of both anxiety and depression in autistic young adults. First, emotion regulation strategy usage and its relation to internalizing symptoms will be reviewed in the general (i.e., neurotypical) and autistic populations. Second, heart rate variability and its relation to internalizing symptoms will be reviewed

in the neurotypical and autistic populations. Third, heart period (i.e., heart rate) will be broadly reviewed along with its relation to symptoms of anxiety and depression. Finally, gaps in the literature will be identified and the current study will be proposed.

Cognitive Components of Emotion Regulation

Emotion regulation is the trans-diagnostic process of managing the presence, intensity and expression of one's own emotions (Cole et al., 1994). Emotion regulation is crucial for directing one's attention, appraising environmental stimuli, and engaging in interpersonal or goal-directed activities during emotion-generating situations (Gross, 1998; Gross & Thompson, 2007). Typically, individuals will accept positive emotions and down-regulate negative emotions using emotion regulation strategies (Gross, 2002). Emotion regulation strategies can be adaptive (e.g., cognitive reappraisal, acceptance, etc.) or maladaptive (e.g., suppression, rumination, avoidance, etc.), depending on how successful they are at reducing negative affect and allowing the individual to engage in behaviors that promote their well-being (e.g., problem solving) (Campbell-Sills et al., 2006). Further, emotion regulation strategies are most effective when employed flexibly, in a manner that is context appropriate (Chesney & Gordon, 2016).

A frequently studied maladaptive emotion regulation strategy is expressive suppression. Expressive suppression involves inhibiting the behavioral expression of one's emotional state (Gross & John, 2003). In contrast, cognitive reappraisal is an adaptive emotion regulation strategy that involves reinterpreting a situation to alter one's emotional state (Gross & John, 2003). These two emotion regulation strategies are often studied in tandem. Overreliance on suppression and ineffective or less utilization of cognitive reappraisal has consistently been associated with higher levels of both anxiety

and depression in the neurotypical population (Aldao et al., 2010; Cai et al., 2018c; Dryman & Heimberg, 2018; Gross & John, 2003; Hofmann et al., 2009; Joormann & Gotlib, 2010; Joormann & Stanton, 2016).

Research on emotion regulation in the autistic population is growing, and comparable findings have been obtained with autistic samples. Several studies have shown that autistic people engage in maladaptive emotion regulation strategies (e.g., expressive suppression) more frequently and adaptive emotion regulation (e.g., cognitive reappraisal) strategies less frequently or less efficiently compared to their neurotypical counterparts (Cai et al., 2018c; Samson et al., 2012; Samson et al., 2014). Further, research with autistic children and adolescents demonstrates that greater usage of expressive suppression and less usage of cognitive reappraisal is associated with increased levels of both anxiety and depression (Cai et al., 2017; Cai et al., 2018a; Mazefsky et al., 2014; Rieffe et al., 2011). Research with autistic adults is less common, however Bruggink et al. (2016) found that maladaptive emotion regulation strategies (e.g., self-blame, catastrophizing, etc.) appeared to be significant predictors of both anxiety and depression. Interestingly, adaptive emotion regulation strategies (e.g., positive reappraisal, acceptance, etc.) were not predictive of either anxiety or depression (Bruggink et al., 2016); a finding that's incongruent with most neurotypical research. The authors attributed this unexpected result to deficits in emotional awareness and cognitive abilities seen in autistic people. Another adult study by Cai et al. (2018b) specifically assessed the relationship between expressive suppression, cognitive reappraisal, and levels of depression. It was found that higher suppression scores and lower cognitive reappraisal scores were associated with increased levels of depression. In contrast to

Bruggink et al. (2016), both emotion regulation strategies were predictive of depression (Cai et al., 2018b). In sum, these results suggest that the relationship between emotion regulation strategy usage and internalizing symptoms continues in some capacity into adulthood for autistic people; however, replication and expansion of these findings is needed for clarification purposes.

Physiological Components of Emotion Regulation

Heart Rate Variability

Research regarding the usage of emotion regulation strategies has relied almost exclusively on the implementation of self-report measures. While self-report measures are critical research tools that garner valuable insight into participants' thoughts, opinions, and views about themselves (Lucas & Baird, 2006), biological or physiological measures of emotion regulation can provide equally important, perhaps more reliable information. Heart rate variability (HRV) has been identified as an objective, non-invasive physiological measure of emotion regulation (Appelhans & Luecken, 2006). HRV is defined as the fluctuation in time interval between heartbeats and is considered an index of autonomic functioning (Shaffer & Ginsberg, 2017). The ability to successfully regulate the experience and expression of one's emotions in an ever-changing environment depends heavily on a flexible autonomic nervous system (ANS) (Porges, 1997; Porges et al., 1994). The ANS can be divided into two primary branches: the sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS). The SNS is associated with increased activity of the hypothalamic-pituitary-adrenal (HPA) axis which releases glucocorticoids (e.g., cortisol) and hormones (e.g., norepinephrine) that prepare the body for action. This heightened physiological state

results in increased respiration, blood pressure, and cardiac output; all of which promote intense muscular action. In contrast, the PNS is associated with decreased respiration, blood pressure, and cardiac output; allowing the body to rest and conserve energy.

Broadly speaking, the SNS and the PNS dynamically interact to produce physiological and emotional responses in accordance with contextual demands (Porges, 1997; Porges et al., 1994). Individuals are typically sympathetic-predominant during stressful or threatening situations (i.e., high physiological arousal) and parasympathetic-predominant during periods of restoration or relaxation (i.e., low physiological arousal). Because both branches of the ANS influence heart rate, HRV measures are considered a valid representation of cardiac autonomic function (Billman et al., 2011; Porges & Byrne, 1992; Shaffer & Ginsberg, 2017), related to an individual's capacity to effectively regulate their emotions in response to environmental stimuli (Appelhans & Luecken, 2006; Thayer & Lane, 2000). Core features of autism have been partially attributed to dysregulation of the ANS (Porges, 2005), making this biopsychological index especially germane for autism research.

Of note, the PNS is regulated in large part by the vagus nerve: the tenth and longest cranial nerve that originates in the medulla (i.e., brainstem) and connects to vital organs all throughout the body (Porges et al., 1994). According to the polyvagal theory, the vagus nerve can be divided into two branches: a myelinated branch that originates in the nucleus ambiguus and an unmyelinated branch that originates in the dorsal motor nucleus (Porges et al., 1994). The vagal branch stemming from the nucleus ambiguus provides direct input to the sinoatrial node of the heart (i.e., the cardiac pacemaker) and thus influences heart rate (Porges et al., 1994). Because the vagus nerve contains both

efferent and afferent fibers, it facilitates rapid, bidirectional communication between the brain stem and numerous organs all over the body, including the heart. When the environment is perceived to be safe, the vagal system is responsible for inhibiting SNS activity (e.g., decreasing heart rate, dampening the HPA axis response, reducing inflammation, etc.) which produces a restorative, homeostatic state that fosters social and emotional engagement (Porges, 2009). This process is operationalized through respiratory sinus arrhythmia (RSA). RSA is the quantification of the inherent respiratory rhythm of the myelinated vagal nerve pathways that originate from the nucleus ambiguus and connect to the sinoatrial node of the heart (Porges et al., 1994). Because the myelinated vagus nerve inhibits sympathetic influences on the heart during periods of safety or rest, RSA is considered an estimate of parasympathetically mediated HRV (Pyetan & Akselrod, 2003). High baseline RSA is associated with a variety of adaptive traits: balanced autonomic functioning, increased ability to engage with the environment (e.g., socially), and emotional or behavioral responding that is flexible (Porges et al., 1994; Porges, 1997; Porges, 2009).

A recent meta-analysis identified RSA as an especially apt HRV frequency-domain measure for researchers working with autistic populations (Cheng et al., 2020). However, there are myriad methods for calculating HRV: time-domain measures (e.g., root mean square of successive differences (RMSSD), standard deviation of the interbeat interval of normal beats (SDNN), etc.), and other frequency-domain measures (e.g., low frequency (LF) band, high frequency (HF) band, LF/HF ratio, etc.). As such, the following review will contain studies that utilized a variety of HRV measures, with particular attention to those that employed RSA with autistic samples.

In the neurotypical population, high HRV is indicative of healthy autonomic function and flexible emotional responding (Appelhans & Luecken, 2006; Thayer & Lane, 2000). In contrast, low HRV suggests an overactivation of the SNS and reduced activity of the PNS (i.e., autonomic imbalance), which contributes to inflexible or inappropriate emotional responding (Thayer & Brosschot, 2005). Research exploring HRV in the autistic population has consistently demonstrated that autistic children and adolescents exhibit lower overall HRV and lower baseline HRV compared to their neurotypical counterparts (Cheng et al., 2020; Lory et al., 2020; Thapa et al., 2020). Similar results have been obtained measuring RSA in autistic children and adolescents (Bal et al., 2010; Guy et al., 2014; Neuhaus et al., 2013; Van Hecke et al., 2009). Because research investigating HRV in the autistic population has focused primarily on children and adolescents, there is a relative gap in the literature with regards to HRV in autistic adults. One study found that autistic adults had decreased resting-state HRV (e.g., RMSSD and HF) compared to a neurotypical control group (Thapa et al., 2019). In contrast, Dijkhunis et al. (2019) found no difference in baseline HRV between autistic young adults and a neurotypical control group using RMSSD as their HRV measure. This finding was counter to the authors' expectations. However, autistic young adults displayed lower HRV reactivity to a stressful social situation compared to participants in the control group as the authors anticipated (Dijkhunis et al., 2019). The authors interpreted these results as an incongruence between emotional awareness (baseline HRV) and arousal regulation (HRV reactivity), which may contribute to overall difficulties with social adaptation seen in autistic people (Dijkhunis et al., 2019). In sum, these studies suggest that low HRV (i.e., low RSA) may be inherent to autistic children

and adolescents, but the extent to which this characteristic continues into adulthood is less understood. Therefore, replication and expansion of these autistic adult HRV findings is required.

With respect to internalizing symptoms, low HRV (i.e., ANS dysregulation) has been associated with an array of anxiety disorders (Chalmers et al., 2014; Gorman & Sloan, 2000; Henje Blom et al., 2010; Klein et al., 1995; Lyonfields et al., 1995; Pittig et al., 2013; Thayer et al., 1996) and depression (Gorman & Sloan, 2000; Henje Blom et al., 2010; Kemp et al., 2010; O'Connor et al., 2002; Van Der Kooy et al., 2006) in the neurotypical population. Similar findings have been obtained in the autistic population. Low RSA has been associated with increased anxiety symptoms in autistic children (Guy et al., 2014; Moskowitz et al., 2013). Further, Neuhaus et al. (2013) found that high baseline RSA in autistic children was associated with fewer symptoms of both anxiety and depression as measured by the Child Behavior Checklist (CBCL) internalizing total score and associated subscale scores (e.g., anxious/depressed, withdrawn/depressed, and somatic complaints). In contrast, Hollocks et al. (2014) did not find a significant relationship between HRV and symptoms of anxiety in autistic children. This study employed the LF/HF ratio as their HRV variable; an indicator of sympathetic arousal (Hollocks et al., 2014). Hollocks et al. (2014) attributed this unexpected result to their small, unbalanced sample; however, further clarification is needed. In essence, these studies suggest that low HRV (i.e., RSA) may be significantly related to anxiety symptoms in autistic children and adolescents. Again, research on how these constructs manifest in adulthood is significantly lacking. Further, less is known about HRV and depression in the autistic population, despite robust findings in the neurotypical literature

(Gorman & Sloan, 2000; Henje Blom et al., 2010; Kemp et al., 2010; O'Connor et al., 2002; Van Der Kooy et al., 2006). Most studies with autistic participants only assessed HRV and anxiety (Guy et al., 2014; Hollocks et al., 2014; Moskowitz et al., 2013). Thus, replication and expansion of the above findings is necessary.

Heart Period (Heart Rate)

An additional physiological measure of ANS functioning is heart period: the mean interbeat interval between successive heart beats. Heart period is inversely related to heart rate (HR) (e.g., low heart period is associated with high HR) and the two are often used interchangeably. In contrast to RSA, a parasympathetic-predominant measure, heart period embodies both sympathetic and parasympathetic influences. Together, RSA and heart period offer a comprehensive operationalization of ANS activity. Outcome differences between heart period and RSA can be theoretically attributed to sympathetic and nonmyelinated vagal factors.

Low resting heart period (i.e., high HR) has been associated with the presence of anxiety disorders (Kemp et al., 2014) and increased risk for cardiovascular disease (Cook et al, 2006) in the neurotypical population. Research exploring heart period (i.e., HR) in the autistic population has consistently demonstrated that autistic children and adolescents exhibit high resting HR (Bal et al., 2010; Ming et al., 2005; Van Hecke et al., 2009). Additionally, high HR and a blunted HR change response have been associated with increased anxiety symptoms in autistic children and adolescents (Hollocks et al., 2014; Hollocks et al., 2016; Moskowitz et al., 2013). Similar gaps in the literature regarding depression and autistic adults exist for heart period. One adult study unexpectedly found no difference in baseline HR or HR reactivity between autistic adults

and their neurotypical counterparts (Dijkhunis et al., 2019). Replication of these contradictory adult HR findings is necessary for elucidation.

Gaps in the Literature

Throughout this review, a striking lack of research regarding usage of emotion regulation strategies, HRV, and their respective relationships to internalizing symptomatology in autistic adults compared to autistic children and adolescents has been delineated. Because adolescent rates of anxiety and depression in the autistic population continue into adulthood (Hollocks et al., 2018), the young adult group cannot be overlooked. Additionally, the transition out of the school system is a significant experience unique to the young adult population. This period is associated with increasingly complex social and occupational scenarios, a loss of ingrained school services, decreased access to age-appropriate services, and slowed progress in the improvement of internalizing behaviors (Tantam, 2003; Taylor & Seltzer, 2010). Understanding which components of emotion dysregulation contribute to symptoms of anxiety and depression is critical to providing more targeted interventions to autistic young adults during this daunting time. Ideally, these supports will help autistic young adults navigate complicated social and occupational situations while equipping them with the skills necessary to effectively manage the experiences and responsibilities associated with adulthood.

A second gap in the literature has been outlined regarding HRV and depression in the autistic population. Physiological arousal (e.g., racing heart, increased respiration, sweaty palms, etc.) is often viewed as more characteristic of anxiety than depression, however due to the robust findings regarding HRV and depression in the neurotypical

population (Gorman & Sloan, 2000; Henje Blom et al., 2010; Kemp et al., 2010; O'Connor et al., 2002; Van Der Kooy et al., 2006), this relationship is worth exploring in the autistic population. Additionally, one must consider the well-established comorbidity of anxiety and depression (Belzer & Schneier, 2004), which implies that participants may likely experience symptomatology related to both disorders. Therefore, to exclude depression from the autism, emotion dysregulation, and anxiety paradigm would be an oversight.

Finally, previous research has relied almost exclusively on either HRV or self-report measures to capture emotion regulation and assess its relationship with internalizing symptoms in the autistic population. Measuring the transdiagnostic, multifaceted construct of emotion regulation in such a narrow way is limiting in terms of the questions researchers can ask as well as the conclusions they can draw. One goal of the current study was to employ multiple methods that captured the cognitive (e.g., usage of emotion regulation strategies) and physiological (e.g., HRV) aspects of emotion regulation, thereby deepening the research scope. Additionally, the present study explored the relationship between the cognitive and physiological aspects of emotion regulation with both social anxiety and depression. Social anxiety was chosen due to the heightened prevalence of this comorbidity in the autistic population compared to other anxiety disorders (Hollocks et al., 2018). Moreover, the manifestation of these variables was explored in autistic young adults.

To our knowledge, only one study to date has explored the relationship between internalizing symptoms, HRV, and the use of expressive suppression and cognitive reappraisal in autistic adults. Cai et al. (2019b) found that HRV indices related to

parasympathetic activity (RMSDD and HF) were associated with autism symptom severity. Interestingly, cognitive reappraisal was found to be a significant predictor of HR, however no relationship was found between expressive suppression and HRV (Cai et al., 2019b). Counter to the authors' expectation, no significant relationship between internalizing symptoms and HRV was found. It should be noted that Cai et al.'s (2019b) sample size was relatively small with 24 autistic participants, meaning the results must be interpreted with caution and replication of these findings is necessary. Further, the current study employed different indices of HRV and internalizing symptoms.

THE PRESENT STUDY

The goal of the present study was to assess the accuracy of emotion regulation strategies (expressive suppression and cognitive reappraisal) and ANS indices (respiratory sinus arrhythmia (RSA) and heart period) at predicting levels of social anxiety and depression in autistic young adults. This direct comparison of methodologies provided valuable insight into what drives the relationship between impaired emotion regulation and internalizing symptoms: a heightened physiological state, maladaptive cognitions, or both. To fulfill this objective, the present study pursued the following aims:

Aim 1. To determine whether measures of emotion regulation (suppression, cognitive reappraisal, respiratory sinus arrhythmia (RSA), and heart period) were significantly associated with internalizing symptoms (social anxiety and depression) in autistic young adults.

a. We hypothesized that greater usage (i.e., overreliance) of suppression and less usage of cognitive reappraisal would be associated with higher levels of both social anxiety and depression.
b. We hypothesized that low RSA and low heart period would be associated with higher levels of both social anxiety and depression.

Aim 2. To assess the cognitive (suppression and cognitive reappraisal) and physiological (RSA and heart period) components of emotion regulation as predictors of social anxiety symptoms in autistic young adults.

a. We hypothesized that the cognitive and physiological components of emotion regulation would together account for unique variance in the occurrence of social anxiety symptoms in autistic young adults.

b. We hypothesized that greater usage of suppression, less usage of cognitive reappraisal, low RSA, and low heart period would be predictive of increased symptomatology of social anxiety.

Aim 3. To assess the cognitive (suppression and cognitive reappraisal) and physiological (RSA and heart period) components of emotion regulation as predictors of depressive symptoms in autistic young adults.

a. We hypothesized that the cognitive and physiological components of emotion regulation would together account for unique variance in the occurrence of depressive symptoms in autistic young adults.
b. We hypothesized that greater usage of suppression, less usage of cognitive reappraisal, low RSA, and low heart period would be predictive of increased symptomatology of depression.

Aim 4. To explore which components of emotion regulation (i.e., suppression, cognitive reappraisal, RSA, and heart period) were the most significant predictors of social anxiety symptoms in autistic young adults.

a. The purpose of this exploratory aim was to ascertain a deeper understanding about the specific attributes of emotion dysregulation that contributed to levels of social anxiety symptoms in autistic young adults. The literature comparing cognitive and physiological components of emotion regulation in autistic participants is sparse. Thus, no directional, specific hypothesis was made for this exploratory aim.

Aim 5. To explore which components of emotion regulation (i.e., suppression, cognitive reappraisal, RSA, and heart period) were the most significant predictors of depressive symptoms in autistic young adults.

a. The purpose of this exploratory aim was to ascertain a deeper understanding about the specific attributes of emotion dysregulation that contribute to levels of depressive symptoms in autistic young adults. The literature comparing cognitive and physiological components of emotion regulation in autistic participants is sparse. Thus, no directional, specific hypothesis was made for this exploratory aim.

METHOD

Participants

To permit the analysis of our aims, an initial prospective power analysis was conducted with 4 tested predictors and 6 total predictors at a power level of .80 using G*Power 3.1. This analysis indicated that a total sample size of at least 85 participants would be necessary to attain adequate power for the statistical approach of Aims 2 and 3.

The present sample consisted of 63 autistic young adults ages 17-29 (M = 20.14), meaning the study was slightly underpowered. With respect to gender, the sample was 17.7% female. With respect to race, the sample was 86.7% White, 5% Black, 5% Asian, 1.7% Middle Eastern, and 1.7% American Indian/Alaskan. The sample was 5.2% Hispanic.

Participants were recruited for the present study through Marquette University's Program for the Education and Enrichment of Relational Skills (PEERS®), a social skills therapy program for adolescents and young adults with autism spectrum disorder and their families. PEERS® is advertised to families through a variety of avenues: the Autism Society of Southeastern Wisconsin, community support groups, and referrals from healthcare providers in the greater Milwaukee area (e.g., neuropsychologists, physicians, social workers, etc.). Acceptance into PEERS® was not contingent on participation in the present study; this was made explicitly clear to all young adults and their caregivers during the informed consent process.

Consenting young adults received a multi-method assessment, including the Autism Diagnostic Observation Schedule (ADOS) and the Kaufman Brief Intelligence Test (KBIT); both of which were administered by trained clinical psychology doctoral

students. The ADOS has high validity, high inter-rate reliability and high inter-item correlation (Lord et al., 2002). This assessment is considered the gold standard for ASD evaluation and is therefore commonly used in both research and clinical settings. To be included in the present study, participants must have obtained an Autism Spectrum classification or higher on the ADOS. The KBIT was chosen for its validity, test-retest reliability and brief length, making it especially useful for a research setting (Bian & Jaspers, 2010). To be included in the present study, participants must have obtained a Verbal IQ of at least 70 on the KBIT. Eligible participants then completed a series of self-report measures related to levels of internalizing symptoms and emotion regulation, and their baseline heart rate variability was collected via a heart rate monitor and electrocardiogram (ECG) electrodes (see below for detailed description of the lab paradigm).

Self-Report Measures

Liebowitz Social Anxiety Scale-Self Report. Participants completed the Liebowitz Social Anxiety Scale-Self Report (LSAS-SR); a 24-item self-report measure that captures the degree of anxiousness a respondent may experience during day-to-day experiences around others (Fresco et al., 2001). Items on the LSAS-SR pertain to specific symptoms of social anxiety: fear/avoidance of social interactions (e.g., talking to people you do not know very well) and fear/avoidance of performance situations (e.g., speaking up in a meeting). For fear items, participants provided fear ratings using a 4-point Likert scale from 0 (*no fear*) to 3 (*severe fear*). For avoidance items, participants provided avoidance ratings using 4-point Likert scale from 0 (*never*) to 3 (*usually*). The LSAS-SR demonstrates good psychometric properties for the total score (the score that will be used

for the current study), with a test-retest reliability of 0.83 (Baker et al., 2002). Internal consistency was excellent for the LSAS ($\alpha = 0.96$) in the present study.

Beck Depression Inventory-II. Participants completed the Beck Depression Inventory-II (BDI-II); a 21-item self-report measure that captures the intensity of depressive symptoms a respondent may be experiencing (Beck et al., 1996). Items on the BDI-II pertain to specific symptoms of depression such as sadness, loss of pleasure, irritability, suicidal thoughts, etc. Participants rated each item using a 4-point Likert scale from 0 to 3, with higher scores indicating more severe symptomatology. Scores from 0-13 indicate minimal depression, scores from 14-19 indicate mild depression, scores from 20-28 indicate moderate depression, and scores from 29-63 indicate severe depression (Beck et al., 1996). The BDI-II exhibits good psychometric properties including a retest reliability ranging from 0.73-0.96 (Wang & Gorenstein, 2013). Internal consistency was excellent for the BDI ($\alpha = 0.91$) in the present study.

Emotion Regulation Questionnaire. Participants completed the Emotion Regulation Questionnaire (ERQ); a 10-item self-report measure that captures respondents' use of two emotion regulation strategies: cognitive reappraisal and expressive suppression (Gross & John, 2003). As such, the ERQ yielded two scores: a reappraisal score and a suppression score. The reappraisal score represents the respondent's tendency to engage in adaptive emotion regulation processes related to cognitive reappraisal (e.g., I control my emotions by changing the way I think about the situation I'm in). The suppression score represents the respondent's tendency to engage in maladaptive emotion regulation processes related to expressive suppression (e.g., I control my emotions by not expressing them). For each item on the ERQ, participants rated how much they agree with the reappraisal or

suppression statement on a 7-point Likert scale from 1 (*strongly disagree*) to 7 (*strongly agree).* The ERQ exhibits strong psychometric properties in both student (Melka et al., 2011) and general community samples (Preece et al., 2019). Both the reappraisal and suppression scores were used for analyses. Internal consistency was acceptable for the reappraisal ($\alpha = 0.78$) and suppression ($\alpha = 0.72$) ERQ subscales in the present study.

Physiological Measures

To collect heart rate data, a BIOPAC ambulatory heart rate monitor and electrocardiogram (ECG) electrodes were used. ECG electrodes were placed on each participant in a standard three-lead configuration (one below the heart, one across the heart near the right collarbone, and one near the right ribcage). Once the ECG electrodes were comfortably secured, the heart rate monitor was started. Participants were instructed to sit quietly for three minutes while baseline heart data was collected. Afterwards, two heart rate indices were calculated offline: respiratory sinus arrhythmia (RSA) and heart period.

Respiratory Sinus Arrhythmia. Respiratory sinus arrhythmia (RSA) is the quantification of the natural fluctuation in heart rate that occurs with typical breathing patterns. RSA is considered a measure of parasympathetically mediated heart rate variability (HRV). To calculate RSA, the Porges' method was implemented with the CardioEdit and CardioBatch programs utilizing data from the frequency band representing spontaneous breathing in adults (i.e., .12-.40 Hz). First, the peak of each R-wave was automatically detected, and the time interval between R-waves (i.e., interbeat interval estimates) was obtained for further processing. The series was then visually inspected for R-wave errors and omissions, and mathematically adjusted for dropped or

combined detection of beats (e.g., skipped beat intervals will be divided, segmented beat intervals will be added, etc.) by a lab member trained and reliable in heart rate editing. Next, three statistical procedures were implemented to calculate RSA: the variance in heart period time associated with spontaneous respiration was removed, the assumption of normality was upheld by taking the natural logarithm of each RSA epoch, and multiple RSA epochs were collected and then averaged to produce a more stable measurement (Porges, 1985; Riniolo & Porges, 2000). This method has proven to be statistically sound compared to alternative techniques (Lewis et al., 2012). Six RSA 30-second epochs were collected for each participant at baseline (with eyes open) and the average of the RSA epochs was used for analyses.

Heart Period. In comparison, heart period is the mean interbeat interval between successive heart beats. Heart period encompasses both sympathetic and parasympathetic activity. Thus, differences in the performance of heart period compared to RSA in the planned analyses can be ascribed to sympathetic factors. Heart period was quantified as the interval (in milliseconds) between consecutive R-waves. Successive heart period estimates were acquired during baseline and then averaged to obtain a mean heart period measure which was used for analyses.

Data Analysis

To test **Aim 1**, Pearson's r coefficients were calculated between 1) suppression and social anxiety, 2) suppression and depression, 3) cognitive reappraisal and social anxiety, 4) cognitive reappraisal and depression, 5) RSA and social anxiety, 6) RSA and depression, 7) heart period and social anxiety, 8) heart period and depression, and 9) social anxiety and depression.

To test **Aims 2 and 3**, two backward hierarchical regression models were conducted to determine which operationalization of emotion regulation (self-report vs. physiological) was stronger at predicting levels of internalizing symptoms in autistic young adults. The first regression model employed suppression, cognitive reappraisal, RSA, and heart period as predictors with social anxiety as the criterion variable (**Aim 2**). The second regression model employed suppression, cognitive reappraisal, RSA, and heart period as predictors with depression as the criterion variable (**Aim 3**). Age, gender, autism traits, and alexithymia were included as covariates for both regression models. Autism traits were assessed via the Autism Quotient (AQ; Baron-Cohen et al., 2001) which had good internal consistency ($\alpha = 0.81$) for the present study. Alexithymia was assessed via the Toronto Alexithymia Scale (TAS; Bagby et al., 1994) which had good internal consistency ($\alpha = 0.81$) for the present study. Next, the squared multiple correlation coefficient (R^2) of each model was evaluated for significance with an F-test and then used to assess the unique variance in social anxiety (**Aim 2**) and depressive (**Aim 3**) symptoms accounted for by the present emotion regulation components.

To test **Aims 4 and 5**, the standardized partial regression coefficients (β) from both regression models were used to assess the significance of individual predictors in each model and compare the predictive power of emotion regulation methods regarding symptoms of social anxiety (**Aim 4**) and depression (**Aim 5**).

RESULTS

Rectifying Outliers

First, data were inspected for outliers by generating boxplots and histograms for each variable. Six upper-bound outliers were identified for BDI, five of which were significant. To rectify the identified BDI outliers, a log transformation was completed to produce a more symmetric distribution (Afifi et al., 2007). Because zeros were present in the dataset, a constant of 1 was added to each BDI value before taking the log. After this transformation, four non-significant lower-bound outliers remained, however skewness and kurtosis values were acceptable (Kline, 2005; Tabachnick & Fidell, 2013). Next, two lower-bound outliers were identified for respiratory sinus arrhythmia (RSA), one upper-bound outlier was identified for heart period (HP), and one upper-bound outlier was identified for the Toronto Alexithymia Scale (TAS). Although none of these four outliers were significant, they were still Winsorized to ensure the variables approximated a normal distribution (Ghosh & Vogt, 2012; Kwak & Kim, 2017). After this process, no outliers remained for either RSA, HP, or the TAS, and all skewness and kurtosis values were acceptable (Kline, 2005; Tabachnick & Fidell, 2013).

Correlations

Aim 1 utilized a series of bivariate Pearson correlations to examine associations among the cognitive variables, physiological variables, and variables of internalizing symptomatology. Results indicated that anxiety symptoms were positively associated with usage of suppression ($r(63) = 0.39$, $p < .01$). Anxiety symptoms were not significantly correlated with usage of cognitive reappraisal, RSA, nor heart period.

Depressive symptoms were not significantly correlated with usage of suppression, usage of cognitive reappraisal, RSA, nor HP.

Regression Models

Next, two backward hierarchical regression models were conducted: one for anxiety (**Aim 2**) and one for depression (**Aim 3**). Usage of suppression, usage of cognitive reappraisal, RSA, and heart period were analyzed as predictors for both models. To preserve power, only covariates that were significantly correlated with anxiety or depressive symptoms were included in the planned analyses. Alexithymia (TAS) was significantly correlated with anxiety ($r(63) = 0.50$, $p < .001$) and depressive ($r(63) = 0.28$, $p < .05$) symptoms; autism symptoms (AQ) were also significantly correlated with anxiety ($r(63) = 0.47$, $p < .001$) and depressive ($r(63) = 0.29$, $p < .05$) symptoms. Thus, both the TAS and AQ were included as covariates for both models.

The first backward regression model with anxiety symptoms as the criterion variable (**Aim 2**) retained usage of suppression ($\beta_1 = 0.33$, $t(57) = 3.18$, $p < .01$), usage of cognitive reappraisal ($\beta_2 = -0.25$, $t(57) = -2.44$, $p < .05$), the TAS ($\beta_3 = 0.32$, $t(57) = 3.01$, $p < .01$) and the AQ ($\beta_4 = 0.28$, $t(57) = 2.71$, $p < .01$) as predictors. The physiological variables (RSA and heart period) did not significantly predict symptoms of anxiety. This regression model accounted for 47.5% of the variance in anxiety symptoms ($R^2 = 0.475$, $F(4, 58) = 13.13$, $p < .001$) for the sample of autistic young adults.

The second backward regression model with depressive symptoms as the criterion variable (**Aim 3**) retained usage of cognitive reappraisal ($\beta_1 = -0.27$, $t(59) = -2.22$, $p < .05$) and the TAS ($\beta_2 = 0.31$, $t(59) = 2.56$, $p < .05$) as predictors. Usage of suppression, RSA, heart period, and the AQ did not significantly predict symptoms of depression. This

regression model accounted for 14.7% of the variance in depressive symptoms (R^2 = 0.147, $F(2, 60) = 5.19$, $p < .001$) for the sample of autistic young adults.

Although **Aims 4 and 5** of the present study sought to compare the significance of cognitive and physiological predictors in both regression models, because none of the physiological variables significantly predicted anxiety nor depressive symptoms, these analyses were not performed.

DISCUSSION

Aim 1 hypothesized that greater usage of suppression, less usage of cognitive reappraisal, low respiratory sinus arrhythmia (RSA), and low heart period (HP) would be associated with higher levels of both anxiety and depressive symptoms. Greater usage of suppression was significantly correlated with increased anxiety symptoms. None of the predictor variables were significantly associated with depressive symptoms.

Despite the lack of significant correlations, all four predictor variables were included in both regression models for **Aims 2 and 3** as this was paramount to the study objectives. It was hypothesized that greater usage of suppression, less usage of cognitive reappraisal, low respiratory sinus arrhythmia (RSA), and low heart period (HP) would significantly predict increased symptoms of both anxiety (**Aim 2**) and depression (**Aim 3**). Findings of the present study indicate that the cognitive components of emotion regulation (i.e., usage of emotion regulation strategies) predicted internalizing symptoms, whereas the physiological components did not. Greater usage of suppression and less usage of cognitive reappraisal significantly predicted increased anxiety symptoms in the sample of autistic young adults, while controlling for alexithymia and autism symptoms severity. Additionally, less usage of cognitive reappraisal significantly predicted increased depressive symptoms in the sample of autistic young adults, while controlling for alexithymia. Because the physiological components of emotion regulation did not predict internalizing symptoms, the current study was unable to address **Aims 4 and 5**: the comparison of strength of prediction between cognitive and physiological components of emotion regulation on internalizing symptoms.

Outcome of Cognitive Variables

These findings add to the literature base in both neurotypical and autistic populations associating greater usage of suppression with increased anxiety symptoms (Aldao et al., 2010; Cai et al., 2018a; Cai et al., 2018c; Dryman & Heimberg, 2018; Hofmann et al., 2009; Mazefsky et al., 2014) and less usage of cognitive reappraisal with increased anxiety and depressive symptoms (Cai et al., 2018a; Cai et al., 2018b; Dryman & Heimberg, 2018; Hoffman et al., 2009; Joormann & Gotlib, 2010; Joormann & Stanton, 2016; Mazefsky et al., 2014). With respect to the cognitive variables, only usage of suppression did not predict symptoms of depression. Although this finding was unexpected given strong evidence in the neurotypical and autism literature bases for increased usage of suppression being associated with increased depressive symptoms (Aldao et al., 2010; Cai et al., 2018b; Cai et al., 2018c; Dryman & Heimberg, 2018; Joormann & Gotlib, 2010; Joormann & Stanton, 2016), there are several prior studies that may elucidate this result. For example, Cai et al. (2018a) found that only reappraisal was significantly associated with depressive symptoms; suppression did not significantly correlate with depression in their sample of autistic adolescents and young adults (ages 14-24). These findings are in line with the results of the present study. Additionally, a recent meta-analysis reviewing 104 neurotypical articles on cognitive reappraisal, suppression, social anxiety disorder (SAD), and major depressive disorder (MDD) revealed mixed findings with respect to suppression and MDD (Dryman & Heimberg, 2018). Findings from this paper indicated that suppression may only be related to depression in specific contexts, such as those with high personal relevance or instances of health-related stressors. For example, if suppression was used during interactions with close friends, this would interrupt the sharing of positive emotions, receiving support

after disclosing negative emotions (e.g., sadness), and the overall development of intimate relationships. In turn, this may heighten feelings of loneliness or sadness, which could worsen the depression. Further, Liverant et al. (2008) found that suppression produced a short-term reduction in sadness for depressed adults, demonstrating the potential temporary benefits of distractive regulatory strategies in response to sadness.

The context or duration of suppression usage was not explicitly accounted for in the present sample of autistic young adults. Participants were not asked whether they used suppression during interactions with close friends or family, or in response to a health-related stressor. They were also not asked to differentiate between their usage of suppression in the short-term vs. long-term. Instead, autistic young adults were asked generally about the frequency of their usage of this emotion regulation strategy which could have impacted the findings. Another possible confounding variable is culture, which can also influence whether suppression is deemed adaptive vs. maladaptive. For example, Soto et al. (2011) found that suppression was only related to adverse psychological functioning for European American participants and not Chinese participants. This study demonstrates how some East Asian cultures encourage the suppression of emotions, and because of this social norm, the mental health consequences typically observed in European Americans may not emerge. These various findings underscore the nuanced nature of suppression; an emotion regulation strategy traditionally considered to be maladaptive but that has proven to be adaptive in specific contexts, for certain durations, and in some cultures.

Nonetheless, suppression positively predicted social anxiety symptoms in the sample of autistic adults, as hypothesized. The relationship between suppression and

symptoms of anxiety appears more robust compared to the relationship between suppression and symptoms of depression (Dryman & Heimberg, 2018). Individuals with social anxiety tend to overregulate their emotions to avoid negative evaluation from others which often has consequences. For example, suppressing positive emotions can result in reduced positive affect and fewer positive social interactions (Framer & Kashdan, 2012). Further, the continuous suppression of negative emotions, such as anger or sadness from social rejection, can perpetuate social anxiety symptoms via rumination (Trew & Alden, 2009).

On the other hand, cognitive reappraisal was shown to be significantly related to internalizing symptoms. More specifically, less usage of cognitive reappraisal by autistic participants predicted higher levels of both anxiety and depressive symptoms. These results are in line with prior neurotypical and autism research which highlight the importance of bolstering the use of adaptive emotion regulation strategies to protect against increased internalizing symptomatology. For example, Cai et al. (2018b) clustered their autistic adolescents and adults into four groups based on their high/low usage of suppression and reappraisal. Interestingly, the high suppression/low reappraisal group demonstrated significantly greater depression symptomatology compared to the high suppression/high reappraisal group. In a later study, Cai et al. (2019a) demonstrated that autistic individuals with high usage of suppression and high usage of reappraisal expressed high positive well-being in addition to low levels of depression compared to those with high usage of suppression and low usage of reappraisal. These data suggest that increased usage of adaptive emotion regulation strategies may *buffer against the negative effects* associated with maladaptive emotion strategy usage. Usage of cognitive

reappraisal being significantly related to both symptoms of anxiety and depression in the present study may provide indirect evidence for this relationship. For example, when usage of cognitive reappraisal low, there is no buffering effect for any maladaptive emotion regulation strategies that the autistic young adult may be employing. Thus, anxiety and depressive symptomatology increases.

In sum, the cognitive findings of the present study add to the growing body of literature examining the usage of emotion regulation strategies in autistic young adults. These data support cognitive reappraisal being an important avenue for treating anxiety and depressive symptoms in this population. The possible buffering effect of this adaptive emotion regulation strategy against consequences from an overreliance on suppression underscore this point. While the relationship between suppression and depressive symptoms is less clear, decreasing the usage of suppression may be clinically relevant for autistic young adults who experience social anxiety. More research with autistic samples is needed to further understand emotion strategy usage in this population. Future work must account for the context of the emotion regulation strategy usage and be grounded in the understanding that these strategies are best used flexibly, in accordance with environmental demands (Aldao et al., 2015).

Outcome of Physiological Variables

Notably, neither respiratory sinus arrhythmia (RSA) nor heart period (HP) significantly predicted social anxiety symptoms nor depressive symptoms. This is counter to the both the neurotypical and autism child/adolescent literature bases that associate low RSA and high heart rate with greater internalizing symptomatology (Chalmers et al., 2014; Gorman & Sloan, 2000; Guy et al., 2014; Henje Blom et al., 2010; Kemp et al.,

2010; Klein et al., 1995; Lyonfields et al., 1995; Moskowitz et al., 2013; Neuhaus et al., 2013; O'Connor et al., 2002; Pittig et al., 2013; Thayer et al., 1996; Van Der Kooy et al., 2006). Broadly speaking, internalizing symptoms are characterized by a hyperarousal of the autonomic nervous system, meaning sympathetic influences are heightened and parasympathetic influences are dampened. Because RSA is conceptualized as a valid, and "pure" measure of parasympathetic function, low RSA should theoretically be related to increased anxiety and depression. There are several possible explanations for this null anxiety finding. First, it's long been theorized that autistic people exhibit chronic autonomic hyperarousal *regardless* of psychopathology, particularly in novel or social scenarios. There are myriad studies demonstrating that autistic people exhibit higher resting heart rate and lower resting heart rate variability compared to their neurotypical counterparts; both of which denote a sympathetic predominance in autonomic function (Bal et al., 2010; Cheng et al., 2020; Ming et al., 2005; Lory et al., 2020, Thapa et al., 2020; Van Hecke et al., 2009). Interestingly, these differences in autonomic function are present as early as 18 months (Sheinkopt et al., 2020). It's plausible that over time, autistic people acclimate to this heightened physiological state resulting in unexpected or blunted patterns of autonomic function in young adulthood. Therefore, patterns we expect to see based on neurotypical literature or autism child/adolescent literature may not emerge.

A second explanation is grounded in the theory of hypoarousal. It's also been theorized that reduced social responsiveness in autistic people is related to a hypoaroused autonomic state, and restrictive or repetitive behaviors (RRBs) serve to stimulate an under aroused system (Lovaas et al., 1987). Some argue that the theories of hyperarousal

and hypoarousal may coexist; there may be *subgroups* of autistic people who exhibit a hyperaroused or a hypoaroused autonomic state. A recent systematic review of sixty studies with autistic children, adolescents, and adults aimed to test each of these theories. Arora et al. (2021) examined studies measuring baseline autonomic arousal in autistic participants via heart rate, heart rate variability, electrodermal activity, and pupil size. Although most findings aligned with the theory of autonomic hyperarousal, the results of the review were quite mixed; the authors also found evidence for autonomic hypoarousal as well as unclear autonomic evidence. Further, Arora et al. (2021) reviewed several studies that grouped their participants based on autonomic response to a task and profiles of hypo- or hyperaroused participants emerged. The authors posed that collapsing these subgroups may have resulted in past null findings, *as may have been the case for the current study*. The Arora et al. (2021) review highlights the complex and variable nature of autonomic function in autistic populations, making it difficult to accurately predict how physiological variables will manifest in young adulthood and relate to other variables such as internalizing symptomatology.

As previously mentioned, Cai et al (2019b) was the only study to date that examined HRV, usage of cognitive reappraisal, usage of suppression, and internalizing symptomatology in autistic *adults*. The authors employed three HRV indices: SDNN, RMSSD, and HF. All three HRV measures were associated with usage of cognitive reappraisal and usage of medication. Two HRV indices were associated with ASD symptomatology. Of note, none of the HRV indices were significantly correlated with usage of suppression, anxiety symptoms, or depressive symptoms and thus were not included in any of the regression models. Interestingly, cognitive reappraisal significantly

predicted all three HRV indices. The authors connected this finding with prior neuroimaging research demonstrating that individuals who report greater usage of cognitive reappraisal may have more efficient prefrontal cortex function, which has historically been associated with higher resting HRV. These data emphasize the significance of cognitive reappraisal in regulating emotions at the physiological level, while highlighting the variable nature of HRV in its relation to both suppression and internalizing symptoms in autistic adults.

In sum, differences in profiles of resting state autonomic function (e.g., hyperaroused vs. hypoaroused) may have implications for how autistic people respond to and engage with their environment, thus contributing to the heterogeneity of the neurodevelopmental disorder (Charman et al., 2005). It's possible that collapsing these subgroups in the present study, especially with participants experiencing clinically significant symptoms of anxiety or depression, contributed to the unexpected physiological findings. Further, HRV patterns and their relation to internalizing symptoms observed in autistic children and adolescents may manifest differently in autistic adults (Cai et al., 2019b). Future HRV research should aim to increase the representation of autistic adults and categorize autistic participants as hyper- or hypoaroused to better contextualize physiological analyses.

CLINICAL IMPLICATIONS

Results from the present study have implications for autistic people and for those working with autistic populations in clinical settings. Because less usage of cognitive reappraisal was associated with higher levels of both anxiety and depression, bolstering the use of this adaptive emotion regulation strategy might hold clinical significance. The buffering effect of cognitive reappraisal against the consequences associated with an overreliance on suppression underscores this approach (Cai et al., 2018b; Cai et al., 2019a). Additionally, given that greater usage of suppression predicted higher levels of anxiety, learning about the different contexts in which usage of suppression could be especially detrimental to one's mental health (e.g., during interactions with friends or close family members) and when it may be beneficial (e.g., during public interactions with peripheral acquaintances or in certain cultures) could be useful for autistic young adults. Emotion Regulation Therapy (ERT) (Mennin & Fresco, 2014) is one therapeutic approach that includes psychoeducation on adaptive emotion regulation strategies with an emphasis on practicing these skills. This intervention also includes elements of Cognitive Behavior Therapy (CBT) such as exposures to emotion-generating scenarios. Of note, EFT has been shown to be effective for those with GAD and MDD (Mennin et al., 2015), however its effectiveness with autistic clients has not been studied directly. Therefore, it would be imperative that autistic clients participate in EFT with a knowledgeable, neurodivergent-affirming provider.

LIMITATIONS

There are several limitations to the present study that must be noted. First, the Beck Depression Inventory-II (BDI-II) data was not normally distributed, thus violating one of the key assumptions of regression analyses. Although a log transformation was implemented to ameliorate this issue (Afifi et al., 2007), the results for the depression regression may have been impacted by the four non-significant lower-bound outliers. Further, an a priori G*Power 3.1 analysis indicated that a total sample size of at least 85 participants would be necessary to attain adequate power for the present study. With 63 autistic young adults participating, the current study was slightly underpowered thus increasing the likelihood of making a Type II error. Future research should aim to collect a larger sample size, which would increase power and the likelihood that variables of interest would achieve normal distributions. Another statistical limitation pertains to using linear analyses. It is possible that the relationship between physiology and mental health outcomes in autistic young adults is nonlinear; therefore, analyzing data in a linear fashion may have washed out any predictive power for the physiological components of emotion regulation. Future work should explore potential nonlinear patterns of predictive relations amongst these variables.

Another possible limitation is the usage of a baseline, or resting, HRV. Despite evidence from prior literature on the significance of resting HRV with internalizing symptoms in autistic populations (Guy et al., 2014; Moskowitz et al., 2013; Neuhaus et al., 2013), neither baseline respiratory sinus arrhythmia (RSA) nor baseline heart period (HP) were retained in either regression model. Of note, autistic people often experience more dysregulation or feelings of anxiety in unfamiliar environments (i.e., lab settings)

compared to neurotypical participants. It's likely that the baseline HRV measure used in the present study was not a "true" baseline measure, but a measure of participants aroused pre-task state. It's also been argued that an ideal baseline HRV measure does not exist, due to imprecise and variable methods (Laborde et al., 2017). For example, one's baseline HRV can vary greatly depending on their body posture (e.g., palms of the hand facing up or facing down while sitting) or the instructions given by the examiner (e.g., deep breathing with eyes closed or deep breathing with eyes open looking at a neutral stimulus). For the present study, participants were instructed to sit comfortably, and not given specific instructions for how their legs, arms, hands, etc. should be positioned. Participants were instructed when to have their eyes open vs. closed. Additionally, the time of day and level of water or food consumption can also influence one's baseline HRV. This was not assessed in the present study.

Given that emotion regulation, one's ability to flexibly manage their emotions in response to environmental stimuli, was a target variable, perhaps a reactivity HRV measure would have captured this concept more comprehensively. For example, a reactivity HRV variable would account for the change in HRV from baseline to an event, thus illuminating participant's physiological response to an emotion-generating task. A future extension of the present study should aim to calculate an HRV reactivity variable in response to an emotion-generating task to better capture the physiological component of emotion regulation. Future research must also obtain information on usage of antidepressants, which was not accounted for in the present study. This notable, given the impact usage of antidepressants has on one's heart rate and heart rate variability (Kemp et al., 2010).

A final, but notable, limitation to the present study is the sample being predominately White and male. Although improvements have been made in recent years to expand access to diagnostic services and update conceptualizations of autism beyond White males (Maenner et al., 2023), a significant lack of diversity in research samples still exists. Along with prior studies, these data should be interpreted with caution as their generalizability to female and racially minoritized populations may be limited.

CONCLUSIONS

This muti-method approach was essential to ascertaining what specific components of emotion dysregulation are contributing to rates of social anxiety and depression in autistic young adults. Results from the current study emphasize the importance of the cognitive component of emotion regulation and its impact on internalizing symptoms in this population. Importantly, findings from the present study have the potential to inform treatment procedures for autistic young adults who are experiencing symptoms of social anxiety and/or depression while navigating the unique demands associated with this critical transition period (e.g., increasingly complex social scenarios, decreased access to age-appropriate services, etc.). For example, this research adds to the growing body of autism literature supporting the decrease, or flexible use, of suppression and increase of cognitive reappraisal as meaningful points of intervention. Enhancing adaptive emotion regulation strategies such as cognitive reappraisal may hold particular clinical significance. Given the null findings for the physiological component of emotion regulation, the present study may challenge the usage of baseline HRV data with autistic young adult samples. Future extensions of the current study should examine the usage of an HRV reactivity measure, the categorization of autistic participants as hyper- or hypoaroused, and the presence of nonlinear relationships. In sum, these findings may be used to further target the treatment of emotion dysregulation and internalizing disorders in autistic young adults and perhaps inform future physiological autism research.

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