School of Population Health

Changes and Persistence in Heart Rate Variability Before and During Social Stress: A Comparison of Individuals With Social Anxiety Disorder Before and Following Cognitive Behaviour Therapy Versus Controls.

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Declaration

To the best of my knowledge and belief, this thesis contains no material previously published by any other person except where due acknowledgment has been made. This thesis contains no material which has been accepted for the award of any other degree or diploma in any university.

The research presented and reported in this thesis was conducted in accordance with the National Health and Medical Research Council National Statement on Ethical Conduct in Human Research (2007) – updated in March 2014.

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Chapter 1: General Introduction

This chapter provides a very brief overview of heart rate variability that has been examined in relation to social anxiety disorder. This introduction will be brief to prevent repetition as the following two empirical chapters provide comprehensive background information in their respective introductions. Social anxiety disorder (SAD) is the third most prevalent mental health disorder, with 8% of Australians meeting the diagnosis within their lifetime (Kessler et al., 2005; Aderka et al., 2012). SAD often has a severe impact on everyday functioning, educational attainment, and ability to maintain relationships (Aderka et al., 2012). Individuals with SAD hold negatively biased mental representations of themselves (Heimberg et al., 2010). These negative mental representations of self can be informed by past traumas in social situations, misinterpreted social information, and beliefs that others are critical evaluators (Heimberg et al., 2010). A distorted image of self and others is a significant maintaining factor for SAD and is targeted by theory-driven treatments like cognitive behavioural therapy to help change problematic thinking styles and patterns (Heimberg et al., 2010). SAD is associated with various physiological symptoms (e.g., blushing, sweating, trembling; Aderka et al., 2013). Thus, gaining a better understanding of physiological processes associated with SAD may help to better understand the maintenance of the disorder, complement cognitive behavioural models and inform adjunctive or alternative interventions to current evidence-based treatments (Hyett et al., 2018).

According to Wong and Rapee (2016), individuals with SAD often engage in anticipatory processing, which is the process of worrying about a social-evaluative threat (i.e., negative evaluation from others) before the situation has occurred. Anticipatory anxiety often results in the performance of 'safety behaviours', which are behaviours designed to prevent feared social outcomes from occurring and thus provide short-term relief from anxiety (Clark & Wells, 1995; Morrison & Heimberg, 2013). These behaviours prevent the individual from disproving their negatively biased beliefs about themselves and others in social-evaluative situations, as the non-occurrence is attributed to the use of

the safety behaviour rather than the fear being unfounded. Therefore, negative beliefs and social anxiety are maintained (Clark & Wells, 1995; Leigh & Clark, 2018)

Porges' (2003) polyvagal theory is an influential model that outlines the crucial relationship between the autonomic nervous system (ANS) and an individual's social and emotional behaviour. The theory proposes that an individual's physiological state limits their social behaviours and psychological experience (Porges, 2003). Anxiety disorders can affect an individual's ability to control maladaptive autonomic processes, as they are unable to successfully identify a safe environment and as a result, they remain consistently hypervigilant (Porges, 2003; Brosschot et al., 2018). A better understanding of physiological processes and how these processes respond to psychological intervention may lead to a more complete understanding of anxiety.

The sympathetic nervous system (SNS) and the parasympathetic nervous system (PNS) are separate branches of the ANS arousal (i.e., fight or flight) and regulation (i.e., rest and digest), respectively (Kushiki et al., 2013). Heart rate variability (HRV) represents variations in time between heartbeats (Kristal-Boneh et al., 1995) and is frequently used as a measure of ANS functioning. HRV can be measured by frequency domain (signal energy of heartbeats within different frequency bands) and time-domain indices (amount of HRV at different periods of time; Shaffer & Ginsberg, 2017). High frequency is an index of parasympathetic tone and a frequency domain measure, while the root mean square of successive differences between heartbeats (RMSSD) is a time-domain measure that quantifies the variability in the time intervals between successive beats (Shaffer & Ginsberg, 2017). These indices have previously been utilised as a sensitive biomarker of emotional control (Thayer & Lane 2000). Thus, lower HRV indicates less ability to regulate and adapt, whereas higher HRV reflects better flexibility and adaption (Quintana et al., 2016).

The first line of treatment for many anxiety disorders (Andrews et al., 2018), and for SAD in particular (National Institute for Health and Care Excellence [NICE], 2013), is cognitive behavioural

therapy (CBT). CBT focuses on modifying cognitive and behavioural maintaining factors to facilitate symptom relief while minimising the influence of influence psychophysiology in the maintenance of the disorder (Hoffman, 2007; Andrews et al., 2018). Thus, observing the impact of CBT on physiological indices (HRV) may help to better understand SAD and processes of maintenance and change and identify additional targets for interventions, leading to more tailored treatments for individuals with SAD. Thus, it is important to understand the patterns of psychophysiological activation and change that characterise SAD during psychological treatments like CBT.

Structure and Aims of the Thesis

This thesis aims to extend the limited research investigating HRV in SAD by examining changes and persistence in HRV in anticipation of and during social stress between individuals with and without SAD. This thesis is comprised of four chapters. Chapter two presents Study One, which examines differences in HRV between individuals with and without SAD at pre-treatment across four phases of the Trier Social Stress Test (TTST; a systematic method for assessing the effects of stress at baseline, preparation, speech task and social interaction phases; Kirschbaum et al., 1993). Chapter three presents Study Two, which uses the same sample as Study 1 to investigates HRV in SAD by examining differences in HRV across four phases of the TSST (baseline, preparation, speech task and social interaction) between (a) the sample with SAD at pre-treatment versus post-treatment and (b) the sample with SAD at post-treatment versus a non-SAD sample. Chapter four provides a general discussion synthesising the key findings from the two studies, theoretical and clinical implications, study strengths and limitations and future research directions. Please note that the two studies were prepared as manuscripts for submission to a journal, thus, there is some overlap in the reviewed literature, although descriptions of common methods are only described in relation to Study 1.

Chapter 2: Study 1

Abstract

Individuals with social anxiety disorder (SAD) experience significant and persistent fear of social situations as they anticipate rejection, scrutiny, and embarrassment. Given that physiological reactions to social situations may shape emotional experience in SAD, understanding psychophysiological changes operating in SAD may be important to address this potentially key perpetuating factor. This study compared the patterns of change (via linear mixed models) and persistence (via autoregressive models) of two indices of heart rate variability (HRV; Root Mean Square of Successive Differences between normal heartbeats, and High Frequency absolute units), as physiological measures of emotion regulation, between individuals with SAD (n = 94) and without (n = 59) using the Trier Social Stress Test phases (TSST). Results revealed that the SAD group increased their need to regulate their emotions (peak HRV) during the preparation (i.e., anticipation) phase, particularly among females, whereas HRV peaked for the non-SAD group during the social-evaluative context. The non-SAD group demonstrated no significant persistence of HRV between some TSST phases, whereas the SAD group showed significant persistence across all phases, however no between-group differences were found. These findings provide novel evidence of similarities and differences in HRV between individuals with and without SAD while anticipating and encountering social-evaluative contexts.

Changes and Persistence in Heart Rate Variability Before and During Social Stress: A Comparison of Individuals With and Without Social Anxiety Disorder.

1. Introduction

Individuals with social anxiety disorder (SAD, also known as social phobia) experience significant and persistent fear of social situations within which they anticipate rejection, scrutiny, and embarrassment (American Psychiatric Association [APA], 2013). SAD can have a severe impact on individuals' everyday functioning, educational attainment, and ability to work productively and maintain relationships (Aderka et al., 2012). Psychophysiological processes of SAD have received relatively little research attention but may help increase understanding of interactions with cognitions, behaviours, and emotions that maintain SAD (Hyett et al., 2018). Identifying and understanding the physiological mechanisms underlying SAD may help inform and guide evidence-based treatments of SAD (Hyett et al., 2018). The first critical step, however, is to increase our understanding of the nature of the physiological changes that occur for people with SAD in anticipation of, and during exposure to, social situations to inform treatment enhancements.

According to cognitive behavioural theory (Heimberg et al., 2014), individuals with social anxiety anticipate social threats (e.g., negative evaluation from others) before encountering social situations. This anticipated threat increases subjective anxiety, physiological arousal, and self-focused attention on perceived performance deficits (Heimberg et al., 2014; Wong & Rapee, 2016). Anticipatory processing and overactive physiological responding guides a mental representation of the self that falls short of self-perceived standards. A common coping behaviour is then social withdrawal and avoidance, precluding social fears from being directly tested and ultimately contributing to their maintenance. While the impact of these thus social-evaluative concerns has been well explored (Wong & Rapee,

2016), few studies have directly examined how the impacts of anticipation of, and exposure to, social contexts on psychophysiology differ between individuals with and without SAD.

The Generalised Unsafety Theory of Stress (GUTS; Brosschot et al., 2018) and the Polyvagal Theory (Porges, 2003) suggest that socially anxious individuals maintain a constant physiological stress response unless in an environment they perceive to be safe. According to these theories, socially anxious individuals have difficulty controlling maladaptive autonomic processes due to an inability to identify socially safe environments (Brosschot et al., 2018). While models implicate a higher trait level of physiological arousal, with less variability for individuals with social anxiety, this has yet to be fully explored. Thus, our understanding of the interaction between context and psychophysiological responding is limited.

The sympathetic nervous system (SNS) is a branch of the autonomic nervous system (ANS) that has an excitatory effect, increasing the heart rate to allow the body to respond to stress/danger ("fight or flight", Kushki et al., 2013). In contrast, the parasympathetic nervous system (PNS) is activated after stress to inhibit heart rate and allow for an adaptive response to stimuli (Kushki et al., 2013). A common measure of this adaptability of response is heart rate variability (HRV), which reflects the fluctuations of time intervals between heartbeats (Kristal-Boneh et al., 1995). HRV is controlled by the balancing action of the SNS and PNS branches, allowing the heart to react quickly to various circumstances and needs (Wu, et al., 2019). As such, higher HRV is thought to reflect an increased biological capacity to respond to stress (Quintana et al., 2016). As HRV is an index of flexibility and physiological self-regulation (i.e., vagally mediated cardiac control), which can influence overall mental health and well-being, it would be beneficial to understand how individuals with anxiety disorders react in situations that may provoke distress (Kimhy et al., 2013; Perna et al., 2020). This is particularly important for SAD, where anticipatory anxiety is an important maintaining factor (Clark & Wells, 1995).

HRV has been indexed by frequency-domain and time-domain indices (Shaffer & Ginsberg, 2017). Frequency-domain indices calculate the signal energy of heartbeats within different frequency bands, while time-domain indices measure the amount of HRV at different periods of time (Shaffer & Ginsberg, 2017). There are various frequency domain measures of HRV, including high frequency (an index of parasympathetic tone) and root mean square of successive differences between heartbeats (RMSSD; a time-domain measure of HRV that indicates the beat-to-beat variance in heart rate; Shaffer & Ginsberg, 2017). While both parameters primarily reflect PNS activity (see Table 1), RMSSD emphasizes short-term variability in heart rate, while HFabs provides insight into the absolute power of PNS-driven HRV within the high-frequency band (Shaffer & Ginsberg, 2017). They complement each other in assessing ANS dynamics and physiological responses to stress and relaxation. RMMSD is highly preferred due to its strong statistical properties, robustness, and as it primarily reflects parasympathetic activation (a strong measure of vagally mediated fluctuations in heart rate; Pham et al., 2021); which is why it was chosen for this study and especially for the design. HFabs was chosen due to its well-established relevance and sensitivity to the parasympathetic branch of the ANS (Shaffer & Ginsberg, 2017). HFabs is widely used in psychophysiological research as it is established as an index of the parasympathetic modulation of heart rate (Pham et al., 2021). Including these measures in this study ensures that our findings are comparable to existing literature, enhancing the generalizability and impact of our results.

Table 1

Heart Rate Variability Parameters

Physiological Index	Domain	Definition	ANS Branch
RMSSD	Time-domain	Root mean square of successive differences between heartbeats.	PNS dominance. Higher RMSSD indicates greater parasympathetic modulation and flexibility in heart rate responses (Vreijling et al., 2020).
HFabs	Frequency-domain	High frequency power.	PNS dominance. Higher values indicate increased parasympathetic modulation associated with a clam and relaxed physiological state (Shaffer & Ginsberg, 2017).

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; PNS = parasympathetic nervous system.

Trait HRV is typically measured at 'baseline' when an individual is at rest, assessing the individual's overall ANS functioning in the absence of stress. State HRV instead measures HRV in anticipation of and during exposure to a stressor, assessing how the individual's ANS reacts in response to changing stress conditions. State HRV assesses the balance and interplay between the SNS and PNS and can, therefore, assess whether the individual exhibits inflexible or otherwise maladaptive HRV patterns under social stress (Laborde et al., 2017; Thayer et al., 2000). Typically, higher state HRV is associated with a healthy and adaptive autonomic response, indicating greater parasympathetic (vagal) activity and better stress resilience to changing conditions, while lower state HRV is often linked to reduced vagal activity and higher sympathetic dominance, which can be indicative of stress and anxiety (Gullett et al., 2023). However, higher state HRV is not necessarily an indicator of the absence of anxiety; instead, it can suggest an increased effort to regulate their physiological state in anticipation of stress (Segerstorm & Nes, 2007). This aligns with empirical evidence that HRV can reflect both the presence of anxiety and the effort to manage it (Segerstorm & Nes, 2007). From a theoretical perspective, the interpretation of state HRV in this study is grounded in the understanding that HRV serves as a dynamic marker of ANS function and flexibility (Gullett et al., 2023).

The role of HRV in the physiological self-regulation of a stress response implies that individuals without SAD would show an increase in HRV (decrease in SNS activity) during social stress relative to individuals with SAD (Clark & Wells, 1995; Thayer et al., 2000). Studying HRV provides insight into these autonomic responses because HRV reflects the balance between the sympathetic and parasympathetic branches of the autonomic nervous system (Gullett et al., 2023). Somatic symptoms of SAD (e.g., blushing, sweating, trembling) are indicative of unsuccessful regulation of the ANS (Aderka et al., 2012). By examining HRV, we can quantify the physiological dysregulation (Gullett et al., 2023) in individuals with SAD, thus linking the observed physiological symptoms to underlying autonomic dysfunctions. This connection not only validates the significance of HRV as a measure in SAD research but also underscores

the potential for HRV as a target for therapeutic interventions aimed at improving autonomic regulation and, consequently, reducing the physiological symptoms of anxiety. Thus, investigating HRV in anticipation of and during social stressors may help to explain psychophysiological drivers of social disengagement, stress dysregulation and behavioural problems in people with SAD (Alvares et al., 2013).

In one of the first investigations of HRV in social anxiety, Alvares et al. (2013) identified that individuals with SAD had lower HRV compared to the healthy controls at rest and that lower HRV was associated with higher self-reported social interaction anxiety. These findings are consistent with the GUTS (Brosschot et al., 2018) and Porges' (2003) theory, which implicate maladaptive autonomic functioning as a key factor in SAD. Klumbies et al. (2014) aimed to extend these findings by assessing HRV both at rest and during social interaction but found no difference between socially anxious individuals and healthy controls in either condition. Another study that was inconsistent with Klumbies et al. (2014) was a study by Tolin et al. (2021) which examined changes in psychophysiological arousal from baseline to a stressor phase in individuals with SAD, panic disorder and generalised anxiety disorders compared to healthy controls. This study found that compared to the healthy controls, individuals with SAD showed heightened psychophysiological reactivity, while individuals with panic disorder and generalised anxiety disorder showed decreased reactivity. However, both these studies only explored HRV at baseline and during a social stressor, which cannot adequately capture the role of physiological self-regulation in anticipatory stress with SAD. Given that individuals with high social anxiety engage in anticipatory processing, it is particularly important to understand how HRV changes from rest to anticipation of social stress (anticipatory processing) and from anticipation through to direct exposure to social stressors, which the current study will do for the first time. A more complete picture of psychophysiological responding in SAD requires a comparison between individuals with and without SAD across baseline, anticipation, speech, and social interaction tasks, particularly in light of discrepant findings in previous studies.

The aim of this present study was to extend the limited research investigating HRV in SAD by examining differences in HRV between individuals with and without SAD and across baseline, preparation (anticipation), speech task and social interaction phases of the Trier Social Stress Test (TSST; Kirshbaum et al., 1993). Our first hypothesis was that if individuals with SAD have less trait biological capacity to respond to stress than individuals without SAD then we should observe lower trait HRV at baseline compared to individuals without SAD. Our second hypothesis was that if individuals with SAD are, on average, less physiologically flexible when under social stress, then we should observe smaller mean changes in state HRV when anticipating and experiencing social stress compared to individuals without SAD. Our third hypothesis was that HRV will be more persistent (i.e., less flexible) across the TSST phases in individuals with versus without SAD. This finding would indicate that any change in HRV in one phase persists more (indicating less flexibility) into subsequent phases for individuals with SAD compared to individuals without SAD.

2. Methods

2.1 Participants

The SAD data came from the baseline of a randomised control trial of Cognitive Behavioural Therapy (CBT) for SAD (McEvoy et al., 2022a, 2022b). Participants were recruited from a community mental health clinic for SAD treatment who were over 18 years of age, on stable medications for over a month and willing to be randomised (see Table 2). Clinical diagnosis of SAD was assessed according to the criteria of the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5; APA, 2013), using the Structured Clinical Interview for DSM-5 (SCID-5; First et al., 2016). Participants were excluded if they had current or past bipolar disorder, psychosis, or substance abuse disorder on the SCID-5, were experiencing CBT for SAD elsewhere or were considered highly suicidal or high self-harm risk. The non-SAD sample was recruited from a University School of Psychology research participant pool, over 18 years old, and had no history of SAD (confirmed by the SCID-5).

2.2 Apparatus/Measures

2.2.1 Social Interaction Anxiety Scale and Social Phobia Scale

The Social Interaction Anxiety Scale (SIAS) and the Social Phobia Scale (SPS) are 20-item self-report measures of social interaction and performance anxiety, respectively (Mattick & Clarke, 1998). Items are answered on a Likert-type scale from 0 ("Not at all characteristic of me") to 4 ("Extremely characteristic of me"). The SIAS consists of items such as "when mixing socially, I am uncomfortable." The SPS items include physical symptoms like "I worry my head will shake or nod in front of others" and cognitive symptoms such as "I feel self-conscious if I have to enter a room where others are already seated." The SPS and SIAS have good discriminant validity and are internally consistent with good test-retest reliability (Mattick & Clarke, 1998). Cronbach's alphas were high for the SIAS and SPS in the SAD (.82 and .94, respectively, McEvoy et al., 2022b) and non-SAD samples (.81 and .92, respectively).

Table 2

Participant Characteristics

Sample Characteristics	Non-SAD	SAD	χ^2/t	*df*	*p*	*d*
Sex *n* (%)						
Male	21 (36)	49 (52)	3.99	1	0.46	
Female	38 (64)	45 (48)				
Education Level *n* (%)						
Less than Year 12	1 (2)	11 (12)	23.5	3	<.001	
Year 12	47 (80)	38 (40)				
Technical/Trade	7 (12)	23 (25)				
Tertiary	4 (7)	22 (23)				
Employment Status *n* (%)						
Employed	45 (76)	52 (45)	14.7	1	<.001	
Unemployed	14 (24)	42 (55)				
Additional disorders *n* (%)						
Major Depressive Disorder	18 (31)	38 (40)	1.54	1	0.22	
Generalised Anxiety Disorder	6 (10)	37 (39)	15.3	1	<.001	
Age M (SD)	21.56 (0.45)	28.97 (1.22)	4.68	151	<.001	.78
SIAS M (SD)	38.00 (12.60)	57.70 (9.21)	11.13	150	<.001	1.85
SPS M (SD)	28.75 (8.10)	46.30 (16.14)	7.68	139	<.001	1.31

Note. t = t-statistic; df = degrees of freedom; *p* = two-tailed p-value; *d* = effect size (Cohen's d). Non-SAD *n* = 59. SAD *n* = 94.

2.2.2 Trier Social Stress Test

The purpose of the TSST is to systematically elicit moderate levels of psychological social stress (Kirschbaum et al., 1993). This procedure is widely used and has been shown to increase heart rate and various stress hormones (Kirschbaum et al., 1993). The four phases of the TSST used in this study were:

1) A five-minute baseline phase, which involves participants resting quietly with no stress induction tasks (Allen et al., 2017).
2) A three-minute preparation phase, where participants are tasked to prepare a speech explaining why they are the best applicant for the mock job of their choice.
3) A three-minute speech phase, where the participant presents the speech to the researcher
4) A five-minute social interaction phase, where the participant engaged in conversation with the researcher, answering questions such as "What do you appreciate about your friends?"

The TSST was audio and video recorded. The TSST also includes an arithmetic assessment phase but was excluded as the speech and social interaction tasks were sufficient to assess responding in social-evaluative contexts.

2.2.3 Measuring equipment and physiological data

A video camera was used to record each speech. The ECG signal was acquired using 4 mm Vermed electrodes, which contain 10% chloride gel to ensure good conductivity. The electrodes were placed below the left and right clavicles and medial to the anterior-superior iliac spine to obtain a clear and consistent signal. The ECG data were sampled at a high frequency of 2000 Hz, which exceeds the minimum requirement of 300 Hz, thus providing a robust dataset for subsequent HRV calculations. A 3-lead electrocardiography using a BioPac MP150 data acquisition and analysis system running AcqKnowledge software collected HRV data. For the SAD group, 152/979 (15.52%) electrocardiogram files and 85/979 (8.68%) electrodermal files were concluded as having substantial artefact and were treated as missing for all analyses. Heartbeat data were checked for outliers and artefacts using visual

inspection and MATLAB (Higham et al., 2016). For the non-SAD group, artefacts were determined through visual inspection. Of the original 216 files, 34 were removed due to excessive artefacts (15.74%) and 98 were corrected for minor artefacts (45.37%). HFabs and RMSSD were calculated using established electrocardiogram processing software (Artiifact; Kaufmann et al., 2011).

2.3 Procedure

This study was approved by the University Human Research Ethics Committee (HR87/2016-13). The SAD group for this study is from McEvoy et al.'s (2022b) randomised control trial of CBT for social anxiety. All participants received an information sheet and provided informed consent. Once eligibility was determined and informed consent was obtained the self-report measures (SIAS and SPS) were prior to the TSST. The researcher placed an electrode on each participant's left hipbone, with an additional two under each collarbone to measure HRV (McEvoy et al., 2022b). While seated in front of a video camera, the TSST commenced, and participants were left for five minutes in a room for the baseline phase. Participants were informed not to use mobile devices and to remain relaxed with their eyes open. Participants then moved on to the preparation, speech and social interaction phases (McEvoy et al., 2022a, 2022b). Participants were asked to treat the social interaction as a general conversation. Both the SAD and non-SAD groups were seated during the whole experiment with HRV being recorded at each phase of the TSST.

2.4 Data Analysis

2.4.1 Hypothesis 1

Hypothesis one was tested using independent-samples t-tests with group (SAD versus non-SAD) as the between-group factor and baseline RMSSD and HFabs as dependent variables.

2.4.2 Hypothesis 2

Hypothesis two was tested using linear mixed models on Jamovi (version 2.3, The Jamovi Project, 2022) with group and phase (TSST phases: baseline, preparation, speech task, social interaction)

as fixed factors, participants as a random factor, and RMSSD and HFabs as dependent variables to examine within- and between-group differences in HRV between individuals with and without SAD and across four phases of the TSST. Greater mean increases in HRV in the non-SAD group compared to the SAD group would suggest that, on average, the non-SAD group was more physiologically flexible and adaptive to social evaluative contexts.

2.4.3 Hypothesis 3

Hypothesis three was then tested using multi-group autoregressive panel models in M*plus* (version 7.4, Muthèn & Muthèn, 1988-2015) to compare patterns of persistence of HRV between consecutive TSST phases. Bootstrapped confidence intervals with 1000 resamples were calculated for all parameters (Cline, 2019). In the first model (AR1), social interaction HRV was regressed on speech HRV, which was regressed on preparation HRV, which was regressed on baseline HRV. In the second model (AR2), each phase was also regressed on two phases prior, so that speech task HRV was regressed on baseline HRV and social interaction HRV was regressed on preparation HRV. AR1 and AR2 models were both bested because it was considered plausible that HRV would persist for one or two phases.

Higher parameters in these models indicate greater persistence of HRV across the phases, such that a larger proportion of an HRV increase in one phase persists into the next phase(s) and thus indicates less flexibility and adaptability to new contexts. For example, an autoregressive parameter of .50 indicates that if an individual's HRV is one standard deviation above the mean, then HRV would remain 50% of the standard deviation above the mean at the next time point. Goodness-of-fit was evaluated using the chi-square test, comparative fit index (CFI; values should be ≥.95), Tucker–Lewis index (TLI; values should be ≥.95), root mean square error of approximation (RMSEA; ≤.06), and the standardized root mean square residual (SRMR; values should be ≤.08; Hu & Bentler, 1999). The sample size can affect the chi-square statistic thus, it is a less useful measure of fit (Hu & Bentler, 1999).

2.4.4 Analysis Assumptions

To address missing data, Full Information Maximum Likelihood estimation was used for all analyses (Muthén & Muthén, 1998-2015; The Jamovi Project, 2022). Little's MCAR test revealed that the data were missing completely at random (p = .224; Li, 2013). A total of 11.1% of HRV data from the overall sample was missing from the baseline phase, 10.5% from the preparation phase, 11.8% from the speech phase and 12.4% from the social interaction phase. Inspection of histograms revealed extreme outliers, which were winsorised to 3.29 standard deviations from the mean (Tabachnick & Fidell, 2013).

An a priori power analysis using G*Power 3.1 (Faul et al., 2009) revealed that a sample size of 28 was sufficient, assuming an alpha of .05, power of .80, and a medium effect size (minimum difference in slopes between consecutive phases within the autoregressive model = 0.09). To account for the multi-group analysis across the three slopes assessing the persistence of HRV (baseline-preparation, preparation-speech, speech-social interaction), the recommended sample size was tripled ($N \geq 84$). The sample size exceeds this estimate with 59 healthy controls (non-SAD) and 94 participants with a diagnosis of SAD (SAD group; N = 153).

In summary, the linear mixed models investigated *group*-level changes in HRV, with a focus on between-group average changes over time. The AR models tell us about how HRV persists for *individuals* across each phase, that is, how much HRV severity at one time-point causes HRV severity at the next time-point, capturing the influence and relationship of past values on future values (Falkenström et al., 2022). These models provide novel information about physiological flexibility across contexts at the group and individual levels. A summary of findings is reported in the main manuscript, with an extended results section in the appendices.

3. Results

Independent samples t-tests revealed that, as expected, the SAD group scored significantly higher than the non-SAD group on both the SIAS and SPS and SPS (see Table 2). There was a significant

difference in age and sex between the SAD (and non-SAD groups, with the non-SAD group being younger and having a high proportion of females. Bivariate correlations for HRV measures can be seen in Table 3. All correlations were statistically significant, positive, and at least moderate magnitude. For the below results, it should be noted that a negative value indicates an increase and a positive value indicates a decrease in HRV.

Table 3

Bivariate Correlations for HRV indices

Variable	1	2	3	4	5	6	7
1. RMSSD Baseline	-						
2. RMSSD Preparation	.65*	-					
3. RMSSD Speech	.60*	.51*	-				
4. RMSSD Social	.58*	.66*	.72*	-			
5. HFabs Baseline	.87*	.52*	.56*	.49*	-		
6. HFabs Preparation	.68*	.87*	.53*	.56*	.66*	-	
7. HFabs Speech	.60*	.43*	.85*	.58*	.55*	.47*	-
8. HFabs Social	.57*	.53*	.62*	.86*	.54*	.55*	.60*

Note. N = 153. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *$p \leq .001$.

3.1 Hypothesis One

Independent samples t-test found no differences between-groups at the baseline phase for RMSSD (trait HRV; $p = 0.15$) or HFabs (trait HRV; $p = 0.33$).

3.2 Hypothesis Two: Mean Comparisons

3.2.1 RMSSD

Age was not a significant covariate and did not alter the significance or pattern of any effects and was therefore removed as a covariate from the analyses. Sex, however, was observed to influence the pattern of effects significantly and was included as a factor. There was a significant main effect of phase, group by phase interaction effect, and a three-way interaction effect between group, phase, and sex (see Table 4 and Figure 1a). Estimated marginal means, 95% confidence intervals, and standard errors are reported in Table 4. We explored the three-way interaction effect for group by phase by sex between consecutive phases.

Between the baseline and preparation phases, there was a significant main effect of phase and a significant three-way interaction (see Table 4). Between these phases for females, there was a significant main effect of phase, $F(1,62.2) = 69.81$, $p < .001$, group, $F(1,71.4) = 6.82$, $p = .01$, and group by phase interaction, $F(1, 62.2) = 7.44$, $p = .01$, whereby females with SAD increased to a greater extent, $t(127) = -9.22$, $p < .001$, $\Delta = -20.28$, than females without SAD, $t(133) = -3.91$, $p < .01$, $\Delta = -10.24$. For males, there was a significant main effect of phase, $F(1,59.8) = 35.91$, $p < .001$, a non-significant main effect of group, $F(1,65.4) = .03$, $p = .86$, and a non-significant group by phase interaction, $F(1,59.8) = .13$, $p = .72$.

Between the preparation and speech phases, there were no significant main effects or three-way interactions (see Table 4); however, there was a significant group by phase interaction, $F(1,126) = 9.31$, $p < .01$, whereby the SAD group showed a reduction, $t(128) = 2.52$, $p = .08$, $\Delta = 4.59$, while the non-SAD group showed an increase in HRV, $t(135) = -1.93$, $p = .29$, $\Delta = -4.74$. Between the speech and social

interaction phases, there was a significant main effect of phase; however, no significant two-way or three-way interactions (see Table 4; see Appendix A for full comparisons).

These findings indicate that the interaction was driven by a larger increase in RMSSD between the baseline and preparation phase for the SAD group compared to the non-SAD group, and specifically for the females within the SAD group relative to the males (see Figure 1a), followed by relative stability across the remaining three phases in the SAD group. In contrast, the non-SAD group continued to increase from preparation to speech phases and then reduced RMSSD during the social interaction. Thus, the HRV of females within the SAD group rapidly increased when anticipating the speech task and to a lesser extent for males with SAD, whereas HRV for the non-SAD group 'peaked' during the speech task before starting to return to baseline (see Figure 1a).

3.2.2 HFabs

Similar to RMSSD, age was not observed to be a significant covariate and did not alter the significance or pattern of any effects and was therefore removed from the analyses. Sex, however, was observed to influence the pattern of effects significantly and was retained as a factor. There was a significant main effect of phase, group by phase interaction effect and a significant three-way interaction effect (see Table 4 and 5, Figure 1b). We explored the three-way interaction effect for group by phase by sex between consecutive phases.

Between the baseline and preparation phases, there was a significant main effect of phase and no significant three-way interactions (see Table 4); however, there was a significant phase by sex interaction, $F(1,126) = 5.85$, $p = .02$, whereby the females increased to a greater extent, $t(130) = -5.62$, $p < .001$, $\Delta = -239.2$, than males, $t(127) = -1.74$, $p = .26$, $\Delta = -83.9$. Between the preparation and speech phases, there was a significant group by phase interaction and group by phase by sex interaction (see Table 3). Between these phases for females, there was a significant group by phase interaction, $F(1, 64.3) = 15.55$, $p < .001$, whereby females with SAD showed a reduction, $t(130) = 2.72$, $p = .19$, $\Delta = 193.95$,

while females without SAD showed an increase in HRV, $t(131) = -3.06$, $p = .08$, $\Delta = -235.54$. For the males, there was a non-significant main effect of phase, $F(1,62.6) = 2.64$, $p = .11$, a non-significant main effect of group, $F(1,66.7) = .58$, $p = .45$, and a non-significant group by phase interaction, $F(1,62.6) = .08$, $p = .78$. Between the speech and social interaction phases there was a significant main effect of phase, $F(1,126) = 13.19$, $p < .001$, however, no significant two-way or three-way interactions (see Table 4; see Appendix A for full comparisons between phases).

Similar to RMSSD, these findings indicate that interaction was driven by the larger increase in HFabs between the baseline and preparation phase for the SAD group compared to the non-SAD group, and specifically for the females within the SAD group relative to the males (see Figure 1b), followed by relative stability across the remaining three phases in the SAD group. The non-SAD group and males with SAD continued to increase from the preparation to speech phases and then reduced during the social interaction phase.

Table 4

Fixed Effect Omnibus tests

Measure	RMSSD				HFabs			
	F	Num df	Den df	*p*	*F*	Num df	Den df	*p*
Overall								
Group	0.41	1	145	0.52	4.38	1	147	1.00
Phase	49.81	3	381	<.001	14.93	3	385	<.001
Sex	0.94	1	145	0.33	1.45	1	147	0.23
Group * Phase	4.41	3	381	0.01	3.27	3	385	0.02
Group * Sex	2.29	1	145	0.13	0.39	1	147	0.53
Phase * Sex	1.09	3	381	0.35	1.44	3	385	0.23
Group * Phase * Sex	2.92	3	381	0.03	2.94	3	385	0.03
Baseline to Preparation								
Group	3.45	1	137	0.07	1.44	1	141	0.23
Phase	100.51	1	122	<.001	25.32	1	126	<.001
Sex	0.36	1	137	0.55	0.88	1	141	0.35
Group * Phase	2.84	1	122	0.10	1.75	1	126	0.19
Group * Sex	2.60	1	137	0.11	1.21	1	141	0.27
Phase * Sex	3.23	1	122	0.08	5.85	1	126	0.02
Group * Phase * Sex	4.85	1	122	0.03	3.46	1	126	0.07
Preparation to Speech								
Group	0.19	1	136	0.66	0.04	1	137	0.84
Phase	0.00	1	126	0.96	2.10	1	127	0.15
Sex	0.00	1	136	0.97	0.05	1	137	0.83
Group * Phase	9.31	1	126	<.01	8.12	1	127	0.01
Group * Sex	4.13	1	136	0.04	0.83	1	137	0.37
Phase * Sex	0.68	1	126	0.41	0.88	1	127	0.35
Group * Phase * Sex	3.19	1	126	0.08	5.93	1	127	0.02

Speech to Social Interaction								
Group	0.27	1	136	0.61	1.30	1	136	0.26
Phase	16.76	1	123	<.001	13.19	1	125	<.001
Sex	1.06	1	136	0.31	1.30	1	136	0.26
Group * Phase	2.22	1	123	0.14	1.31	1	125	0.26
Group * Sex	1.13	1	136	0.29	0.01	1	136	0.94
Phase * Sex	0.00	1	123	0.97	0.00	1	125	0.97
Group * Phase * Sex	0.29	1	123	0.59	0.40	1	125	0.53

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *F* = F-value; Num df = numerator degrees of freedom; Den df = denominator degrees of freedom; *p* = two-tailed p-value. Non-SAD *n* = 59. SAD *n* = 94.

Table 5

Means for each group across the Trier Social Stress Test phases

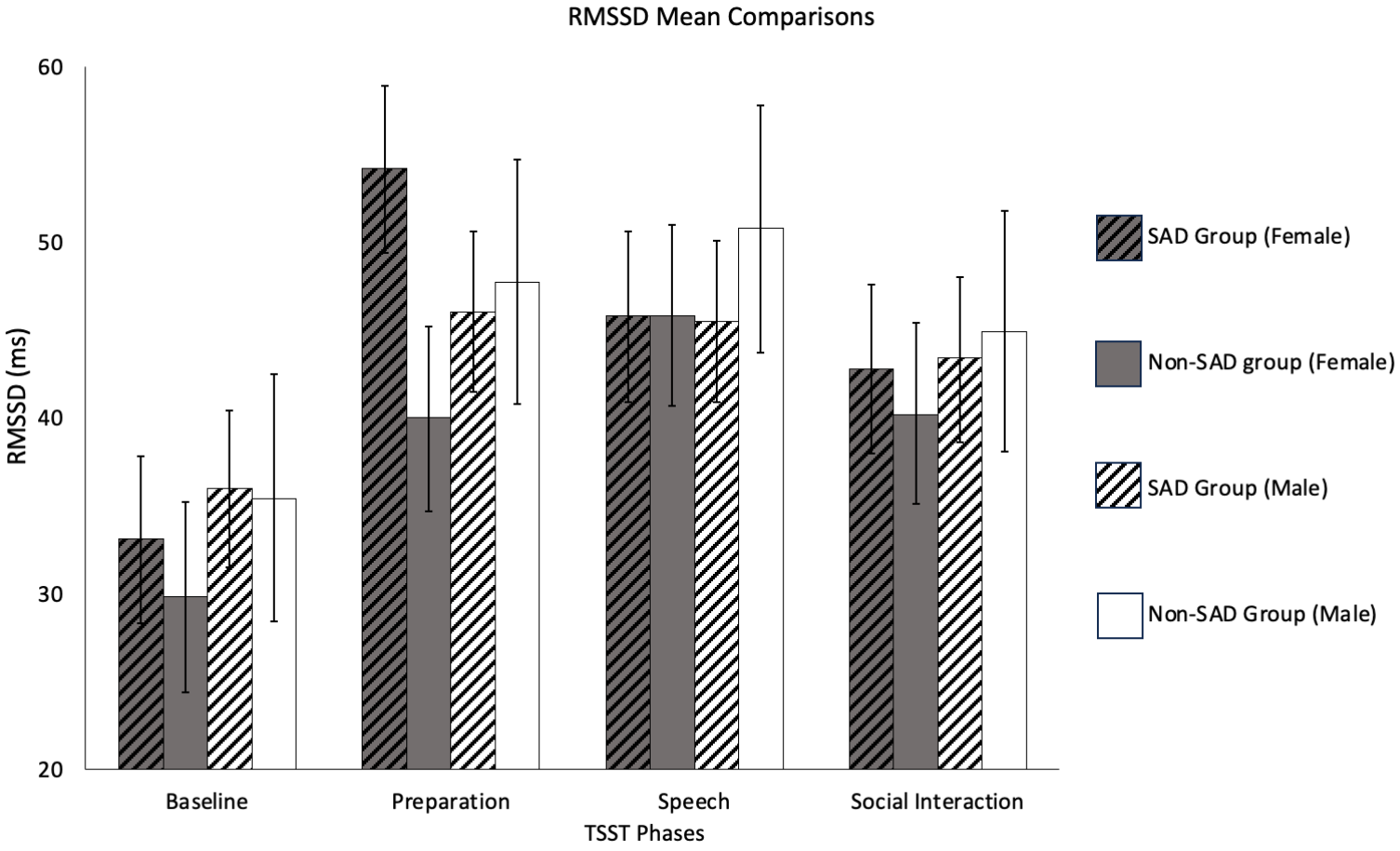
	SAD			Non-SAD		
Measure	*M*	95% CI	S.E.	*M*	95% CI	S.E.
RMSSD						
Female						
Baseline	33.1	28.3, 37.8	2.41	29.8	24.4, 35.2	2.75
Preparation	54.2	49.4, 58.9	2.41	40.0	34.7, 45.2	2.68
Speech	45.8	40.9, 50.6	2.46	45.8	40.7, 51.0	2.62
Social Interaction	42.8	38.0, 47.6	2.44	40.2	35.1, 45.4	2.62
Male						
Baseline	36.0	31.5, 40.4	2.27	35.4	28.4, 42.5	3.58
Preparation	46.0	41.5, 50.6	2.31	47.7	40.8, 54.7	3.53
Speech	45.5	40.9, 50.2	2.33	50.8	43.7, 57.8	3.58
Social Interaction	43.4	38.6, 48.0	2.38	44.9	38.1, 51.8	3.49
HFabs						
Female						
Baseline	399	274, 524	63.4	351	208, 494	72.8
Preparation	755	630, 880	63.5	489	350, 629	70.8
Speech	568	440, 695	65.0	719	584, 855	68.9
Social Interaction	509	382, 636	64.4	525	390, 661	68.8
Male						
Baseline	525	408, 642	59.4	519	333, 705	94.4
Preparation	595	476, 715	60.8	647	465, 830	93.0
Speech	675	555, 796	61.2	743	557, 929	94.4
Social Interaction	572	448, 696	63.0	607	426, 797	91.6

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *M* = mean estimate; CI = confidence intervals; S.E. = standard erorr. Non-SAD *n* = 59. SAD *n* = 94.

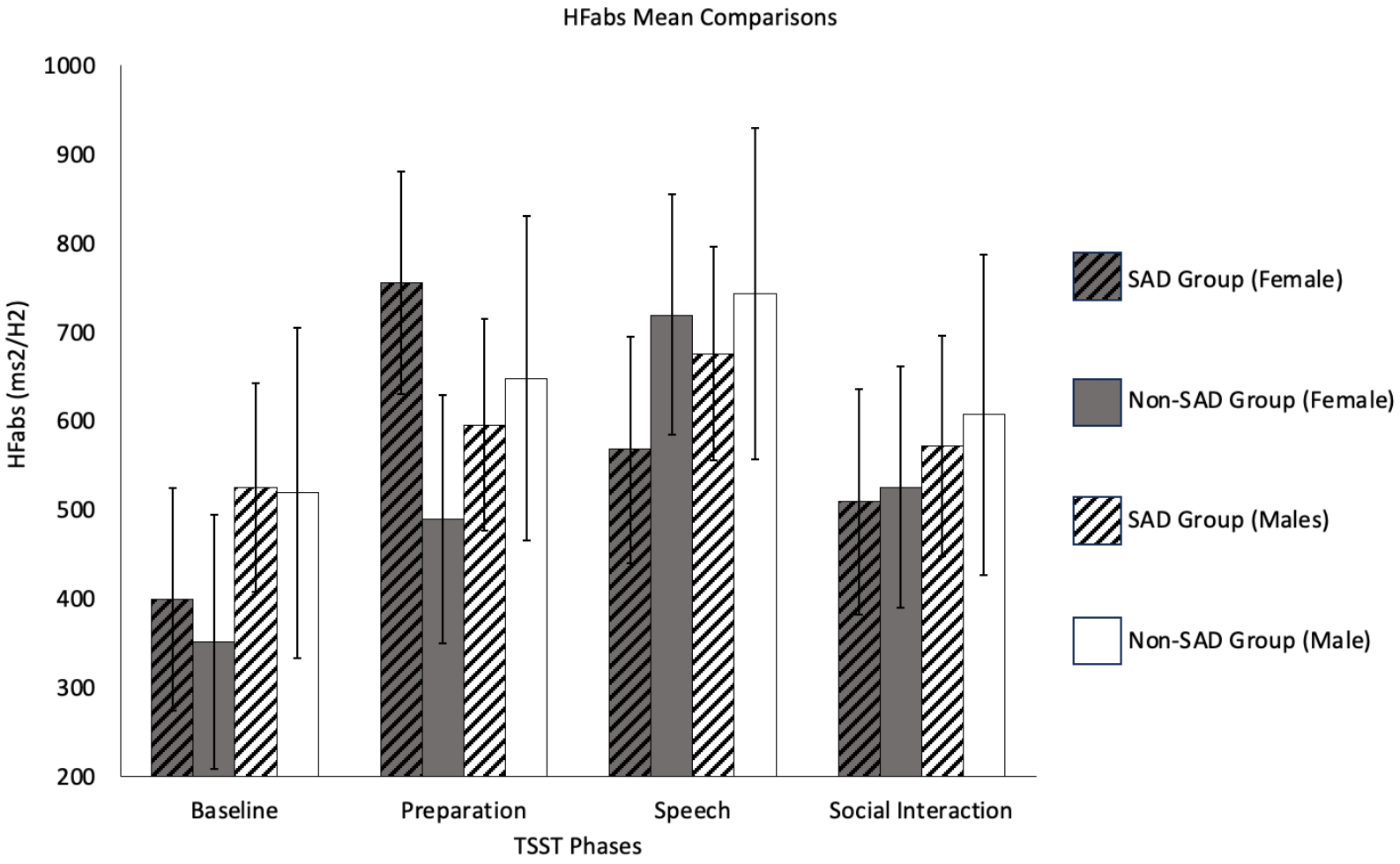
Figure 1

Mean Comparisons with 95% confidence intervals between the SAD and Non-SAD Participants (Female and Male) during Four Phases of the TSST

a) RMSSD



b) HFabs


HFabs Mean Comparisons
1000
900
800
700
600
500
400
300
200
HFabs (ms2/H2)
Baseline
Preparation
Speech
Social Interaction
TSST Phases
SAD Group (Female)
Non-SAD Group (Female)
SAD Group (Males)
Non-SAD Group (Male)

3.3 Hypothesis Three: Autoregressive Models

3.3.1 RMSSD

Age but not sex was a significant covariate in the autoregressive models, so only models controlling for age are reported. The autoregressive model for RMSSD regressing each phase on to the phase immediately prior (AR1) was a poor fit to the data (see Table 6). We then ran an autoregressive model regressing each phase on the preceding two phases (AR2), which provided an excellent fit on the CFI and SRMR, an adequate fit on the TLI, and a borderline fit for the RMSEA (see Table 6). Modification indices did not identify additional substantive sources of model misfit. A chi-square difference test indicated that the final AR2 model fit significantly better than the AR1 model, χ^2_{diff} (4) = 54.95, $p < .05$.

Results for within-group comparisons showed that all autoregressive pathways for the SAD group were significantly different from zero (*ps* < .05), indicating HRV persistence. The autoregressive pathways for the non-SAD group were also all significant (*ps* < .05), except between the preparation and speech phase (β = .07, 95%CI = -.17, .52, *p* =.69; see Table 7). No autoregressive parameters significantly differed between-groups (*ps* >.05, see Table 7).

3.3.2 HFabs

The AR1 provided a poor fit to the data for HFabs (see Table 6). Similar to RMSSD, the AR2 model was an excellent fit on CFI and SRMR and adequate on the TLI and the RMSEA (see Table 6). A chi-square difference test indicated that the AR2 model fit significantly better than the AR1 model, χ^2_{diff} (4) = 31.16, $p < .05$. Results for within-group comparisons showed that all autoregressive pathways for the SAD group were significantly different from zero (*ps* < .05; see Table 7). The autoregressive parameters for the non-SAD group were also all significant (*ps* < .05), except between the preparation and speech phases (β = .13, 95%CI = -.14, .54, *p* =.46) and between the preparation and social interaction phases (β = .34, 95%CI = .00, .70, *p* =.06). No autoregressive parameters significantly differed between-groups (*ps* >.05; see Table 7).

Table 6

Goodness of Fit of Autoregressive Models

	χ^2	*df*	CFI	ΔCFI	TLI	SRMR	RMSEA [90%CI]
RMSSD							
AR1	58.91	6	.81		.35	.16	.34 [0.26, 0.42]
AR2	3.96	2	.99	.18	.93	.02	.11 [0.00, 0.28]
HFabs							
AR1	34.86	6	.85		.51	.11	.25 [0.17, 0.33]
AR2	3.16	2	.99	.20	.94	.02	.09 [0.00, 0.26]

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; AR = autoregressive; CFI = comparative fit index; TLI = Tucker–Lewis Index; SRMR = standardized root mean square residual; RMSEA = root mean square error of approximation; *df* = degrees of freedom; CI = confidence interval; Δ = difference.

Table 7

Autoregressive Model 2 – Within- and between-groups

	SAD			Non-SAD			Group Comparisons		
HRV	β	[95% CI]	*p*	β	[95% CI]	*p*	β	[95% CI]	*p*
RMSSD									
Baseline → Preparation	1.05	[0.82, 1.26]	<.001	0.94	[0.52, 1.40]	<.001	0.12	[-0.48, 0.60]	.70
Baseline → Speech	0.45	[0.08, 0.70]	.01	0.65	[0.07, 0.96]	.01	-0.20	[-0.73, 0.46]	.50
Preparation → Speech	0.30	[0.10, 0.51]	.01	0.07	[-0.17, 0.42]	.69	0.23	[-0.25, 0.58]	.29
Preparation → Social Interaction	0.31	[0.09, 0.44]	<.001	0.32	[0.14, 0.47]	<.001	-0.01	[-0.29, 0.22]	.93
Speech → Social Interaction	0.52	[0.31, 0.67]	<.001	0.52	[0.29, 0.70]	<.001	<.001	[-0.30, 0.27]	.98
HFabs									
Baseline → Preparation	0.84	[0.64, 1.06]	<.001	0.82	[0.52, 1.40]	<.001	0.02	[-0.59, 0.40]	.94
Baseline → Speech	0.43	[0.05, 0.76]	.01	0.51	[-0.04, 0.98]	.04	-0.08	[-0.68, 0.50]	.78
Preparation → Speech	0.27	[0.02, 0.53]	.04	0.13	[-0.14, 0.54]	0.46	0.14	[-0.32, 0.50]	.51
Preparation → Social Interaction	0.23	[0.02, 0.40]	.02	0.34	[<.001, 0.70]	.06	-0.12	[-0.51, 0.30]	.60
Speech → Social Interaction	0.39	[0.21, 0.57]	<.001	0.39	[0.10, 0.62]	<.001	<.001	[-0.29, 0.36]	.99

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; β = beta estimate; CI = confidence intervals; *p* = two-tailed p-value. Non-SAD *n* = 59. SAD *n* = 94.

4. Discussion

This study aimed to extend the limited research investigating HRV in SAD by examining differences in HRV between individuals with and without SAD across four phases of the TSST. Previous studies have explored HRV as a predictor of SAD at rest and during stress, but no studies have examined HRV across the four phases of the TSST included in this study, which allowed us to examine responding at baseline, while anticipating a speech task (preparation), and during two social-evaluative tasks relating to performance (speech) and social interaction. Our first hypothesis was that individuals with SAD have less trait biological capacity to respond to stress than individuals without SAD, which would be demonstrated by lower trait HRV at baseline compared to individuals without SAD. This hypothesis was not supported as there were no significant baseline (trait HRV) differences between the SAD and non-SAD groups on either measure of HRV and no main effects of group (SAD versus non-SAD) in any of the linear mixed models. Our second hypothesis was that individuals with SAD are less flexible when under social stress, which would be demonstrated by the SAD group showing less variation in HRV on average across the phases than the non-SAD group. This hypothesis was only partially supported, and the pattern of findings was somewhat more complex. Specifically, HRV for the SAD group increased to a greater degree from baseline to the preparation phases compared to the non-SAD group. Interestingly, this effect appeared to be driven by females with SAD, who increased more from the baseline to the preparation (anticipation) phase compared to the males with SAD and the non-SAD groups. The non-SAD group increased HRV more from the preparation to speech task phases compared to the SAD group.

Our third hypothesis was that individuals with SAD are more persistent (i.e., less flexible) with their HRV, and therefore a larger proportion of the changes in HRV at one timepoint would persist (and thereby have a greater influence on) HRV at the subsequent timepoint, than those without SAD. This hypothesis was not supported as perseverance did not significantly differ between the groups between any phase on either HRV index. However, there was some evidence in the AR models that the groups

differed with respect to the 'within-group persistence' of HRV, such that the non-SAD group demonstrated better emotion regulation between some phases, whereas the SAD group showed persistence across all phases. However, the absence of statistically significant between-group differences means we cannot confidently conclude that the level of persistence differed across the groups. The lack of persistence and increase in HRV for both HRV indices between the preparation and speech phases may reflect that, in addition to emotional control, the non-SAD group may be more successful in planning (feeling more prepared) and conducting the speech task.

Overall, these findings suggest that compared to the SAD group, the non-SAD group were more physiologically flexible between the preparation and speech phases as they moved from anticipation to delivery of their speech task with respect to changes in mean HRV level and persistence. However, as stated previously, there were no significant between-group differences observed in the AR models, so we cannot conclude that differences in the pattern of within-group effects reflect reliable differences between the groups.

The between-group differences (for AR models and linear mixed models) for both RMSSD and HFabs seem to be inconsistent with the study by Alvares et al. (2013), which found that individuals with SAD showed lower levels of HRV compared to healthy controls. However, the between-group results from the autoregressive models and mean differences are consistent with Klumbies et al.'s (2014) study, which reported no between-group differences in RMSSD between individuals with and without social phobia at rest or during social stress. Klumbies et al. (2014) only explored the baseline and social interaction phases of TSST and not the preparation phase, which is necessary for examining levels of anticipatory anxiety, a purported maintaining factor of SAD (Wong & Rapee, 2016). This study, therefore, extends previous research and provides a more complete picture of psychophysiological responding in SAD by comparing individuals with and without SAD across the baseline, preparation (anticipation), speech and social interaction phases of the TSST.

The mean HRV and the autoregressive parameters did not differentiate between individuals with and without SAD across any of the TSST phases. However, examination of the mean changes between successive phases provided evidence that overall parasympathetic activity may have differed between the two groups between the preparation and speech phases. The findings demonstrate that the SAD group, and specifically females with SAD, have higher HRV during the preparation phase, which could be taken as evidence that they increased their need to regulate their emotions (during anticipation) to a greater degree than the non-SAD group.

These results may be explained in part by gender differences in cognitive processes, specifically rumination and repetitive negative thinking styles. Rumination involves engaging in behaviours and thoughts that passively and repetitively focus on one's symptoms of distress, causes and consequences (e.g., thinking about how unmotivated one feels and how this will affect their day negatively; Nolen-Hoeksema & Jackson, 2001). This may be contributing to the observed pattern, as rumination has been associated with increased anticipatory anxiety as it tends to be driven by recollections of past failures and social interactions (Mellings & Alden, 2000). This then results in increased attentional focus on social threat and the use of 'safety behaviours' to avoid or prevent negative social outcomes (Leigh & Clark, 2018), which, in turn, prevents violation of beliefs about social threat and thereby maintains SAD (Leigh & Clark, 2018). According to the response styles theory, women are more likely to ruminate on their distress in comparison to men (Johnson & Whisman, 2013). Worry and rumination (two forms of repetitive negative thinking) are significantly correlated with each other and increase the vulnerability to anxiety and depressive disorders, which are highly comorbid (McEvoy et al., 2013). Thus, this theory may offer a plausible explanation for the heightened increase in HRV during the preparation phase for the females in the SAD group, as rumination may amplify their anticipatory anxiety.

These findings seem to align with previous research demonstrating that women are more likely to engage in rumination and have higher rates of anxiety disorders (McLean et al., 2011). Understanding

the underlying mechanisms associated with gender differences in SAD may contribute to more tailored interventions for individuals with SAD. In contrast, the non-SAD group demonstrated higher HRV within the social-evaluative context (speech) but not to the same degree during anticipation of the event. It is possible that the attenuated increase in HRV in anticipation of the social-evaluative stressor for the non-SAD group reflects a lower perception of threat as compared to the females with SAD and, thus, less need to deploy psychophysiological emotion regulation.

Although between-group differences were not detected for the AR models, the non-SAD group's HRV was not persistent between the preparation and speech phases, whereas the SAD group's HRV was persistent between these phases. Therefore, not only does the SAD group experience a greater increase from the baseline to the preparation phase compared to the non-SAD group, but this increase persists into the speech phase for the SAD group. In contrast, for the non-SAD group, HRV severity during the preparation phase did not persist (or significantly influence) HRV during the speech task. These findings are consistent with the idea that the SAD group experiences greater perceived threat during the preparation phase. Indeed, the consistency of HRV levels between the preparation and speech phase could imply that anticipation is experienced to be as intimidating as the social-evaluative context itself. This pattern of psychophysiological arousal is entirely consistent with cognitive behavioural theories, which argue that social anxiety is triggered in anticipation of social evaluative situations and that this persists into the situation itself (Wong & Rapee, 2016). In contrast, individuals without SAD demonstrate peak HRV during the actual social-evaluative context, suggesting possible 'optimisation' of the timing of their psychophysiological regulation, and prior HRV levels do not appear to significantly influence HRV during exposure to social evaluation, thereby indicating greater flexibility.

The findings can be explained by the Polyvagal Theory (Porges, 2003) and the GUTS (Brosschot et al., 2018), which suggests that when an individual identifies a safe environment, vagal outflow increases, promoting social behaviours and a relaxed state. Under these theories, the SAD group

showing higher HRV at the preparation phase (context sensitivity) may be due to individuals with SAD being less able to distinguish between 'safe' (preparation) and 'unsafe' (the actual social-evaluative context) situations. In contrast, the non-SAD group did not appear to fully activate emotion regulation until they were within the social-evaluative situation. The lack of 'further increase' in HRV in the speech context might indicate that individuals with SAD are unable to harness greater levels of psychophysiological flexibility (i.e., vagal activation) within threatening contexts, which would help them to regulate. In contrast, the non-SAD group may recognise the speech task as a context requiring a higher level of regulation than the anticipation/preparation phase and, thus, increase further to that point, whereas the SAD group remains as activated in the social evaluative context. This suggests that group differences in psychophysiology do not lie in general trait HRV but rather in context-dependent state differences (flexibility between contexts). Some of these effects were apparent in the mean differences in the magnitudes of HRV change but not in the AR models, suggesting that the HRV patterns in individuals with and without SAD may be more influenced by situational factors rather than trait characteristics. This finding has important theoretical implications, suggesting that HRV may be influenced by context-specific factors, such as the anticipation of social-evaluative contexts.

The results of the current study show some inconsistencies with both the Polyvagal Theory (Porges, 2003) and the GUTS (Brosschot et al., 2018). These theories suggest that disorders related to social dysfunction (such as SAD) are associated with a reduced ability to control maladaptive autonomic processes (ANS arousal) reflected by lower HRV (PNS activation). This study did not reveal any significant baseline differences and few other differences in HRV indices between individuals with and without SAD, therefore challenging the association between anxiety and reduced parasympathetic influence on HRV as posited by Porges' (2003) model. The absence of significant between-group differences in this study also suggests that the patterns of HRV in individuals with SAD do not consistently align with the

GUTS. The findings suggest that the relationship between SAD and HRV may be more complex and 'context sensitive' than these theories suggest.

Cognitive-behavioural theories offer an additional explanation for our many non-significant differences between the groups on objective measures of physiological responding. Specifically, these models suggest that socially anxious individuals hold situation-specific thoughts and negatively biased mental representations of themselves and how they are viewed by others. These mental representations are guided, in part, by awareness of physiological symptoms of anxious arousal (Clark & Wells, 1995; Heimberg et al., 2010; Rapee & Heimberg, 1997). It may be that subjective hypervigilance and hypersensitivity to internal physiological cues is, therefore, a more important between-group difference that maintains SAD than objective psychophysiological responding. Future research investigating both psychophysiological and self-report measures will be valuable in contributing to more effective treatments for SAD.

Understanding the context-dependent nature of HRV in people with SAD may have important clinical implications. Our findings indicate the potential value of interventions that focus on regulating emotions and physiological arousal (e.g., biofeedback, Lehrer et al., 2000), specifically in anticipation of social-evaluative contexts, particularly for women with SAD. By targeting this crucial phase (anticipation), clinicians may be able to enhance outcomes by increasing the capacity to control physiological responses and, in turn, emotion regulation within social situations. The absence of consistent between-group differences suggests that strategies that reduce hypervigilance or hypersensitivity to bodily cues in individuals with SAD may also be particularly important, such as attention training procedures that reduce self-focused attention (e.g., Wells, 2009).

Challenging misappraisals of physiological reactions (e.g., self-perceived prominence of sweating and blushing, etc.) is commonly used in evidence-based cognitive behavioural interventions to reduce the perceived cost of internal physiological reactions (e.g., McEvoy et al., 2022b). Our findings of

similarities between people with and without SAD may help to challenge perceptions of critical differences in psychophysiology between these groups. Interoceptive exposure, in conjunction with behavioural experiments, may be a powerful way to challenge social fears relating to symptoms of SAD (e.g., 'my anxiety will be obvious to others and will lead to rejection'). Additionally, understanding whether the few differences we identified between the SAD and non-SAD groups disappear after individuals with SAD receive evidence-supported interventions such as CBT may have significant clinical implications. If these psychophysiological differences remain, and if they limit treatment gains or serve as a risk factor for relapse, adjunctive interventions that target physiological responding may enhance longer-term outcomes from existing evidence-based treatments. For example, biofeedback that increases control over vagal (PNS) activity, and thus HRV, may result in greater adaptability and lower anxiety in response to social-evaluative stress (Lehrer et al., 2020). Therapists may be able to utilise the positive effects of biofeedback with HRV to improve emotion regulation and decrease emotional and autonomic activity. Our findings suggest that targeting physiological responding in the transition from anticipation to encountering social-evaluative situations may be particularly fruitful.

This study has several strengths, such as being one of the first to examine psychophysiological responding across four phases of the TSST in both the SAD and non-SAD groups. However, several limitations also need to be considered. A range of external factors that can impact HRV, including physical exercise, caffeine, smoking, alcohol, sleep quality, and medication use Alvares et al., 2013; Tiwari et al., 2021). We were interested in identifying differences in HRV between individuals with SAD and without SAD, and one challenge with comparing these groups is that they differ on many of these external factors, which are meaningful parts of the SAD 'syndrome' rather than confounds. For example, individuals with SAD often use alcohol as a social lubricant and use more alcohol than individuals without SAD (Bulley et al., 2016). Therefore, controlling for all of these factors may have removed meaningful differences between the groups. Whilst this reduced our capacity to attribute group

differences to social anxiety per se, it does allow us to identify naturalistic differences between the groups. Nonetheless, it is important it is important to acknowledge the limitation that while our approach may have increased generalisability to naturalistic SAD and non-SAD groups, it does limit the capacity to identify which of the factors (SAD per se and/or the additional factors that are part of the syndrome) uniquely contributed to these differences. Future studies should investigate the impact of other factors that are known to moderate HRV (Alvares et al., 2013; Tiwari et al., 2021). It is important to note that we had intended to partially out medication use, but unfortunately, there was an administrative loss of data for medication use in the non-SAD sample. Despite this limitation, the significant findings of the present study offer valuable insights into the physiological responses associated with SAD and its treatment while highlighting the need for comprehensive approaches in future research. Additionally, future research should explore both HRV and state repetitive negative thinking when anticipating an impeding stressor to examine whether trait or state repetitive negative thinking accounts for heightened HRV in women during such anticipatory periods. These findings highlight the need for higher-powered research to capture absolute differences between groups and differences in the profiles of HRV reactivity. This may lead to a more refined understanding of the intricate mechanisms underlying HRV patterns in individuals with SAD, which may in turn, help to inform the development of more targeted interventions for SAD.

The present study demonstrated that HRV in anticipation of social stressors may be an effective biomarker for SAD, particularly for females. Overall, the SAD group tended to activate regulation to a greater degree than the non-SAD group in anticipation of the social-evaluative context, whereas the non-SAD group only fully activated emotion regulation during the actual stressor (speech). This 'context-sensitivity' was the only key difference observed between the groups, with no differences in baseline/trait levels of HRV observed. The SAD group showing higher HRV at the preparation phase (context sensitivity) may be due to females with SAD being less able to distinguish between 'safe'

(preparation) and 'unsafe' (the actual social-evaluative context) situations (Porges, 2003; Brosschot et al., 2018). While no between-group differences were observed in the AR models, within-group effects indicated that the non-SAD group demonstrated less evidence of persistence between some phases, whereas the SAD sample showed persistence across all phases. The absence of significant group differences means that the differing patterns of these effects should be regarded with caution. These findings are broadly consistent with cognitive behavioural and psychophysiological models and suggest that targeting emotion regulation in anticipation of social stressors may be critical for treating SAD. The absence of between-group differences in mean HRV and the AR models also suggests there may be an important role for hypervigilance or hypersensitivity to bodily cues in people with SAD, which is also consistent with cognitive behavioural theory. Given the inconsistencies in observed effects across studies, more research is clearly required to replicate these findings in larger samples and develop a more comprehensive understanding of the complex relationship between HRV, social stress, and SAD. Future research should also examine how evidence-supported treatments influence HRV indices and whether adjunctive interventions targeting psychophysiological parameters will improve outcomes.

Study one investigated pre-treatment differences between SAD and the non-SAD groups and identified some distinct patterns. However, it is unknown whether these differences persist following CBT for SAD. This question may be critical for identifying psychophysiological differences between people with SAD versus non-SAD that are not impacted by existing treatments, and that may serve as vulnerability factors for relapse. Therefore, the aim of study two was to identify psychological differences that remain between the SAD relative to the non-SAD samples following a 12-session group CBT intervention for SAD.

Chapter 3: Study 2

Abstract

Individuals with social anxiety disorder (SAD) experience intense and persistent fear of social situations where rejection, negative evaluation, and embarrassment may occur. It is crucial to understand patterns of psychophysiological activation that characterise SAD during existing evidence-based treatments to provide a more complete understanding of the disorder. This study investigated whether observed patterns of change (via linear mixed models) and persistence (via autoregressive models) of two indices of heart rate variability (HRV; Root Mean Square of Successive Differences between normal heartbeats and High-Frequency absolute units) at pre-treatment change or are maintained at post-treatment between individuals with and without SAD across four phases of the Trier Social Stress Test (baseline, preparation, speech task and social interaction). The mean HRV results of this study revealed that at post-treatment, individuals with SAD started to demonstrate similar patterns of HRV to the non-SAD participants, especially with respect to earlier phases (in both HRV indices). The SAD group demonstrated more flexibility across the phases, while the non-SAD group demonstrated less persistence (i.e., better adaptability) between some phases; however, no significant between-group differences were found. These findings provide evidence for the impact of cognitive behavioural therapy on HRV patterns between individuals with and without SAD.

Changes and Persistence in Heart Rate Variability Before and During Social Stress: Impacts of Group Cognitive Behaviour Therapy for People With Social Anxiety Disorder.

1. Introduction

Individuals with social anxiety disorder (SAD, also known as social phobia) experience intense and persistent fear of social situations within which rejection, negative evaluation, and embarrassment may occur (American Psychiatric Association [APA], 2013). Individuals with SAD often experience distressing physiological symptoms such as sweating, trembling, and blushing during social situations (Aderka, 2012), which further exacerbate social-evaluative concerns (Kushki et al., 2013). There is limited research exploring the nature of the physiological changes that occur for individuals with SAD in anticipation of, and during exposure to, social evaluative contexts to inform treatment enhancements that target such processes. A greater understanding of these psychophysiological processes may provide insights into the underlying mechanisms contributing to SAD and inform improvements to current evidence-based interventions for SAD. However, it is first important to understand how psychophysiology changes, or indeed does not change, during existing evidence-based treatments before enhancements targeting these processes can be developed. Understanding physiological changes in response to evidence-based treatments is critical for advancing our understanding of SAD and paves the way for more tailored interventions.

According to Wong and Rapee (2016), individuals with SAD frequently engage in anticipatory processing, which is the process of worrying about social threats before experiencing actual social situations. Anticipatory processing is driven by recollections of past failures and predictions about future poor performance in social situations, thus leading to repetitive thinking and anxiety (Mellings & Alden, 2000). Wong and Rapee (2016) suggested that anticipatory processing can impede an individual's ability to effectively engage with other people as it causes the individual's adaptive system to overreact. This contributes to self-focus on internal cues (e.g., physical symptoms of anxiety) and external cues (e.g.,

ambiguous social cues and expected negative evaluation from others), which lead the individual to infer they are falling short of others' expectations, and in turn, to even higher levels of social anxiety. This process often results in the use of 'safety behaviours' within social situations, which are strategies used to cope with and/or avoid feared outcomes (Leigh & Clark, 2018). Safety behaviours such as limiting eye contact and over-preparing for social situations can maintain social anxiety as they prevent the individual from learning that their feared situation was unlikely to occur in the absence of the safety behaviour (Leigh & Clark, 2018). Moreover, safety behaviours such as self-monitoring and internal focus on physiological symptoms can distract from current social interactions and make individuals appear as though they lack social skills, which may paradoxically elicit social evaluation, thereby becoming a self-fulfilling prophecy (Leigh & Clark, 2018). Physiological responding in anticipation of, and during, social situations is therefore theorised to play a critical role in maintaining social anxiety.

The sympathetic nervous system (SNS) is a branch of the ANS and increases heart rate in response to perceived danger (excitatory effect/fight or flight; Kushki et al., 2013). The parasympathetic nervous system is another branch of the ANS that is activated after stress and which aids in relaxation and regulated responses to stimuli by reducing heart rate (Kushiki et al., 2013). Heart rate variability (HRV) represents variations in time intervals between heartbeats (Kristal-Boneh et al., 1995) and is frequently used to assess autonomic functioning as it is regulated by the balancing action of SNS and PNS branches. Measures of HRV are commonly obtained from frequency domain and time domain indices (Shaffer & Ginsberg, 2017). Time-domain indices assess the quantity of HRV at various time points, whereas frequency-domain indices calculate the signal energy of heartbeats across various frequency bands (Shaffer & Ginsberg 2017). High frequency is a frequency domain measure of HRV and an index of parasympathetic tone, whereas root mean square of successive differences between heartbeats (RMSSD) is a time-domain measure of HRV that indicates the beat-to-beat variance in heart rate (Shaffer & Ginsberg, 2017). HRV is considered to be a psychophysiological index of adaptability and

self-regulation (i.e., vagally mediated cardiac control) where higher HRV under stress would be associated with lower SNS activity and more flexibility. State HRV offers insights into how an individual's ANS responds in anticipation of and when exposed to a stressor and is, therefore, an index of an individual's ability to regulate and adapt under social stress (Laborde et al., 2017). Trait HRV is typically measured at rest (baseline) and provides an index of an individual's overall ANS functioning. Given that the responsivity of HRV to changing environmental demands may be critical to understanding psychophysiological factors that contribute to social disengagement, stress dysregulation and behavioural issues in SAD, it is important to understand both trait levels and state changes in HRV in anticipation of (i.e., anticipatory processing) and during social stressors (Alvares et al., 2013).

According to the Generalised Unsafety Theory of Stress (GUTS), individuals with SAD lack the ability to distinguish between a safe and unsafe environment (Brosschot et al., 2018). Thus, individuals with SAD often experience chronic feelings of stress, unsafety, and hypervigilance in social situations, as they assume an environment is unsafe as a default (Brosschot et al., 2018). Such persistent perceptions of unsafety may contribute to maladaptive ANS functioning, including reduced parasympathetic influence on HRV and increased sympathetic activation (Brosschot et al., 2018). Porges' (2003) Polyvagal Theory suggests that vagal tone, a measure of vagus nerve activity, is essential for regulating HRV. Individuals with SAD may demonstrate changes in vagal tone and disrupted ANS functioning, which results in dysregulation of HRV rhythms and emotion regulation (Porges, 2003). The physiological symptoms of SAD, such as an elevated heart rate and decreased HRV amid social stressors, may be influenced by these changes. Therefore, examining how cognitive behavioural therapy (CBT) affects HRV may offer valuable insights into how CBT modulates vagal tone and ANS functioning. With this information, we may be able to develop more tailored interventions by learning how to improve HRV patterns in individuals with SAD. Additionally, adjunctive interventions that target the vagus nerve and

psychophysiological regulation, such as biofeedback and mindfulness-based practices, may improve treatment efficacy by directly influencing HRV and ANS functioning.

CBT is recommended by the clinical practice guidelines as an evidence-based treatment for SAD (Andrews et al., 2018). CBT aims to modify maladaptive thoughts and reduce symptoms by understanding and modifying emotions, thoughts, and behaviours through graded psychoeducation, behavioural experiments, and cognitive reappraisal strategies (Andrews et al., 2018). A study by Hyett et al. (2018) used a modified version of the TSST to compare indices of HRV before and after single-session group treatments. Three groups were compared; two groups diagnosed with SAD who received different types of CBT (verbally-based and imagery-enhanced CBT), which were compared to a waitlist control group. Verbally-based CBT incorporated verbal-linguistic techniques without referencing imagery-based techniques, while imagery-enhanced CBT incorporated mental imagery-based techniques (McEvoy et al., 2022a). This study found that emotion regulation, indexed by HRV, demonstrated a greater increase during social stress following imagery-enhanced CBT compared to other groups (Hyett et al., 2018). This finding highlights greater physiological flexibility following imagery-enhanced CBT in comparison to the alternative treatment and waitlist control (Hyett et al., 2018). CBT leads to large changes in self-reported symptoms (e.g., McEvoy et al., 2022a; Rapee et al., 2009); however, a substantial minority of individuals with SAD still fail to respond to treatment (De Castella et al., 2014).

Alvares et al. (2013) conducted one of the first studies exploring HRV in social anxiety, comparing HRV levels of individuals diagnosed with SAD and healthy controls at rest. According to this study, people with SAD had lower HRV compared to healthy controls, and lower HRV was linked to higher levels of self-reported social interaction anxiety. The GUTS (Brosschot et al., 2018) and Porges' (2003) theory are supported by this data, which suggests that there may be a trait difference in HRV between people with and without SAD that contributes to the maintenance of the disorder. Another study by Klumbies et al. (2014) investigated physiology in individuals with social phobia compared to

healthy controls at baseline and social interaction. In contrast with Alvares et al.'s (2013) study, Klumbies et al. (2014) found that HRV did not differ at baseline or during the social stress task between socially anxious people and healthy controls, indicating that there were no significant group differences in parasympathetic functioning. However, individuals with social phobia displayed high levels of subjective stress (Klumbies et al., 2014). Socially anxious individuals may, therefore, be more sensitive to feelings of anxiety than to actual physiological responses.

A more complete picture of psychophysiology in SAD responding was provided in study one of this thesis, which examined differences in HRV between individuals with and without SAD and across four phases of the Trier Social Stress Test (TSST; baseline, preparation, speech task and social interaction; Kirschbaum et al., 1993). Study one reported no significant between-group differences in HRV, which is inconsistent with Alvares et al. (2013) but consistent with Klumbies et al. (2014), which reported no between-group differences in HRV between individuals with and without social phobia at rest or during social stress. Study one found that individuals with SAD increased their need to regulate their emotions during the preparation phase (anticipation), especially among women, whereas HRV peaked for individuals without SAD during the social interaction phase. Individuals with SAD showed significant persistence of HRV across all phases; that is, a larger proportion of an HRV increase in one phase persists into the next phase(s) and thus indicates a lack of context-specific self-regulation across different contexts. In contrast, the individuals without SAD showed non-significant persistence in HRV between some TSST phases. The findings from study one can be explained by the GUTS (Brosschot et al., 2014), where individuals with SAD may be showing higher HRV during the preparation phase (context sensitivity) as individuals with SAD are less able to identify 'safe' (preparation) and 'unsafe' (actual social-evaluative stressor) situations. Thus, the findings from the first study showed that HRV may be influenced by context-specific factors, whereby individuals with SAD are less able to distinguish relatively "safe" (i.e., anticipation of social-evaluative situations) from unsafe (i.e., actual social-

evaluative situation) contexts, and once HRV is activated it persists in individuals with SAD, indicating a lack of context-specific regulation. Study one provides novel evidence of similarities and differences in HRV between individuals with and without SAD across the various phases. It is crucial to understand the processes of psychophysiological change during CBT but also potentially identify signals that might represent risk factors for relapse if differences remain following CBT. Therefore, the present study extends this research by investigating whether HRV changes during CBT and whether differences observed at pre-treatment in study one were maintained following CBT for SAD.

Monitoring the impact of CBT on physiological indices such as HRV is critical for understanding the impact of evidence-based psychological interventions on psychophysiology and potentially identifying additional targets for intervention, but few studies have assessed HRV before and after psychological interventions (Hofmann, 2007). There is an emerging field of adjunctive interventions targeting psychophysiological parameters that could enhance outcomes. For example, biofeedback is a self-regulation process that involves increasing awareness and regulation of physiological responses (e.g., heart and breath rate) as a method to increase HRV and, in turn, reduce symptoms of anxiety (Lehrer et al., 2020). However, before exploring the benefits of adjunctive interventions with individuals with SAD, it is crucial to first understand the patterns of psychophysiological activation that characterise SAD during existing evidence-based psychological treatments.

The aim of this present study was to extend the limited research investigating HRV in SAD by examining differences in HRV across four phases of the TSST (baseline, preparation, speech task and social interaction) between (a) the sample with SAD at pre-treatment versus post-treatment and (b) the sample with SAD at post-treatment versus a non-SAD sample. These comparisons are important for understanding the effects of CBT on HRV and processes of psychophysiological change. Hypotheses one to three relate to the SAD group pre-treatment versus post-treatment comparison, whereas hypotheses four to six relate to the post-treatment SAD versus non-SAD comparison.

The hypotheses comparing the pre- versus post-treatment SAD group were:

Hypothesis one: Individuals with SAD at pre-treatment will have less trait biological capacity to respond to stress compared to individuals with SAD at post-treatment. If this is the case, then we should observe lower trait HRV at pre-treatment baseline compared to post-treatment baseline.

Hypothesis two: Individuals with SAD will be less physiologically flexible when under social stress at pre-treatment. If this is the case, then we should observe smaller mean changes in state HRV when anticipating and experiencing social stress at pre-treatment compared to post-treatment. This would indicate that, on average, individuals with SAD who underwent CBT are able to adapt better to stressful situations as they are more physiologically flexible in comparison to pre-treatment.

Hypothesis three: Individuals with SAD will be less flexible in their HRV across contexts at pre-treatment. If this is the case, then HRV will be more persistent across the TSST phases at pre-treatment compared to post-treatment.

The hypotheses comparing the post-treatment SAD group versus the non-SAD group mirror hypotheses one to three and were:

Hypothesis four: Individuals with SAD at post-treatment will have less trait biological capacity to respond to stress than individuals without SAD. If this is the case, then we should observe lower trait HRV at baseline for individuals with SAD compared to individuals without SAD.

Hypothesis five: Individuals with SAD at post-treatment will be less physiologically flexible when under social stress, if this is the case, then we should observe smaller mean changes in state HRV when anticipating and experiencing social stress compared to individuals without SAD.

Hypothesis six: Individuals with SAD at post-treatment will be less flexible in their HRV across contexts. If this is the case, then HRV will be more persistent across the TSST phases in individuals with versus without SAD.

1. Methods

2.1 Research Design

For this study, we examined a group that had completed a group CBT intervention for SAD (SAD group) and a non-SAD control group. To test hypotheses one to three, there were two independent variables: time with two levels (pre- versus post-CBT) and TSST phases with four levels (baseline, preparation, speech and social interaction). To test hypotheses four to six, there were two independent variables being group with two levels (SAD and non-SAD) and TSST phases. The dependent variables of HRV included Root Mean Square of Successive Differences between normal heartbeats (RMSSD) and High Frequency absolute units (HFabs). Group-level mean changes in HRV over time (main effects and interactions) were examined using linear mixed models. Greater mean increases in HRV indicate more physiological flexibility and adaptability to social evaluative contexts. An autoregressive panel model design was employed to examine the patterns of persistence of HRV during the four TSST phases. Greater persistence of HRV across the phases would suggest that individuals are less flexible and adaptable to new contexts. Together, these analyses provide novel information about physiological flexibility across contexts at the group and individual levels.

2.2 Participants

The post-treatment SAD data came from a randomised control trial of Cognitive Behavioural Therapy (CBT) for SAD (McEvoy et al., 2022a, 2022b). Those recruited by McEvoy et al. (2022a, 2022b) were patients seeking treatment for SAD at a community health clinic who were over 18 years of age (see Table 2 and McEvoy et al., 2022a, 2022b). Inclusion criteria were SAD according to the DSM-5 (APA,

2013) using the Structured Clinical Interview (SCID-5; First et al., 2016); stable medication for over a month; willingness to be randomised; no current or past bipolar disorder, psychosis, substance abuse disorder; not undergoing CBT for SAD elsewhere; not currently expressing suicidal intent. The non-SAD group was recruited from a University School of Psychology research participant pool. This sample consisted of individuals over 18 years of age with no history of SAD (confirmed by the SCID-5).

2.3 Apparatus Measures

2.3.1 Social Interaction Anxiety Scale and Social Phobia Scale

The Social Interaction Anxiety Scale (SIAS) and the Social Phobia Scale (SPS) are 20-item self-report measures of social interaction and performance anxiety, respectively (Mattick & Clarke, 1998). A Likert-type scale is used to rate responses, with 0 being “Not at all characteristic of me” and 4 being (Extremely characteristic of me.” Items on the SIAS included “When mixing socially, I am uncomfortable.” The SPS items cover both somatic symptoms, like “I worry my head will shake or nod in front of others”, and cognitive symptoms, like “I feel self-conscious if I have to enter a room where others are already seated.” The SIAS and SPS exhibit good discriminant validity and are internally consistent with good test-retest reliability (Mattick & Clarke, 1998). Both scales have high Cronbach’s alphas in the SAD (.82 and .94, respectively, McEvoy et al., 2022) and non-SAD groups (.81 and .92, respectively).

2.3.2 Trier Social Stress Test

According to Kirschbaum et al. (1933), the TSST is designed to systematically elicit moderate levels of psychological social stress. HRV was measured throughout the five-minute baseline phase, followed by three minutes where participants were able to prepare for a speech describing why they are the best applicant for the mock job of their choice (preparation phase), a three-minute speech to the researcher (speech task), and finally, a five-minute social interaction task where participants

communicated with the researcher and answered questions like “What do you appreciate about your friends?” Audio and Video were recorded during the TSST. This procedure is widely used and has been shown to raise heart rate and other stress hormones (Kirschbaum et al., 1993).

2.4 Treatments and Clinicians

All treatment sessions were audiotaped. Participants were randomly assigned to imagery-enhanced or verbally-based CBT. Both treatments targeted social and performance anxiety. Throughout the sessions, imagery-enhanced CBT included mental imagery-based techniques, while verbally-based CBT concentrated on ‘thoughts’ without referencing imagery/imagery-based techniques (except for video feedback). Both treatments consisted of 12 2-hour weekly group sessions, a group follow-up at 1-month, and individual 1- and 6-month follow-ups with the trial assessor. Two clinicians facilitated each group. No substantive or consistent differences between the two treatments were found on self-report measures (McEvoy et al., 2022b) or psychophysiological indices used in this study (McEvoy et al., 2022), so data were combined across the treatment conditions for this study. The intervention has demonstrated large effect sizes on the SIAS ($d \geq 2.00$, McEvoy et al., 2022a).

Assessing clinicians were blind throughout the trial and were required to conduct two practice SCIDs with concordant principal and comorbid diagnoses prior to this study. The treating clinicians were clinical psychologists with master's or doctoral degrees who had undergone extensive training from the protocol's developers and principal investigators. In addition to co-facilitating at least one group with one of the protocol developers or licenced therapists, the training required reading thorough treatment manuals with detailed therapist instructions, scripts, and client handouts.

2.5 Procedures

The Human Research Ethics Committee of the local Health Department approved the study protocol (Approval # 04_2016) and University (HR87_2016), and the full protocol is detailed in McEvoy

et al. (2022b). For the SAD participants, the SIAS, SPS and SCID-5 were administered at the 1- and 6-month follow up, and principal and comorbid diagnoses were re-assessed at each follow-up (McEvoy et al., 2022b). The non-SAD also completed the self-report measures SIAS and SPS. The researcher placed an electrode on each participant's left hipbone, with an additional two under each collarbone to measure HRV (McEvoy et al., 2022b). Consistent with chapter one, the ECG signal acquisition procedure was the same, both the SAD and non-SAD groups were seated in front of a video camera during the whole experiment with HRV being recorded at each phase of the TSST.

2.6 Data Analysis

2.6.1 Hypotheses 1 and 4

Hypothesis one was tested using paired sample t-tests with group (individuals with SAD pre- versus post-treatment) as the between-group factor and baseline RMSSD and HFabs as dependent variables. Hypothesis four was tested using independent sample t-tests with group (post-treatment SAD versus non-SAD) as the between-group factor and baseline RMSSD and HFabs as dependent variables.

2.6.2 Hypotheses 2 and 5

Both hypotheses were tested using linear mixed models in Jamovi (version 2.3, The Jamovi Project, 2022) with group (individuals with SAD pre- versus post-treatment [H2] and post-treatment SAD versus non-SAD [H5]) and phase (baseline, preparation, speech, social interaction) as fixed factors, participants as a random factor, and RMSSD and HFabs as dependent variables to investigate within- and between-group differences in HRV.

2.6.3 Hypotheses 3 and 6

Both hypotheses were tested using multi-group autoregressive panel models in M*plus* (version 7.4, Muthèn & Muthèn, 1988-2015) to compare patterns of persistence of HRV between consecutive TSST phases. Bootstrapped confidence intervals with 1000 resamples were calculated for all parameters (Cline, 2019). In the first model (AR1), social interaction HRV was regressed on speech HRV, which was

regressed on preparation HRV, which was regressed on baseline HRV. In the second model (AR2), each phase was also regressed on two phases prior, so that speech task HRV was regressed on baseline HRV and social interaction HRV was regressed on preparation HRV. AR1 and AR2 models were both bested because it was considered plausible that HRV would persist for one or two phases.

Higher parameters in these models indicate greater persistence of HRV across the phases, such that a larger proportion of an HRV increase in one phase persists into the next phase(s) and thus indicates less flexibility and adaptability to new contexts. For example, an autoregressive parameter of .60 indicates that if an individual's HRV is one standard deviation above the mean, then HRV would remain 60% of a standard deviation above the mean at the next time point. The AR models tell us about how HRV persists for individuals across each phase, that is, how much HRV severity at one time-point causes HRV severity at the next time-point, capturing the influence and relationship of past values on future values; Falkenström et al., 2022). Goodness-of-fit was evaluated using the chi-square test, comparative fit index (CFI; values should be ≥.95), Tucker–Lewis index (TLI; values should be ≥.95), root mean square error of approximation (RMSEA; ≤.06), and the standardized root mean square residual (SRMR; values should be ≤.08; Hu & Bentler, 1999). The sample size can affect the chi-square statistic thus, it is, a less useful measure of fit (Hu & Bentler, 1999). A summary of findings is reported in the main manuscript, with an extended results section provided in the Appendices.

2.6.4 Analysis Assumptions

To handle missing data, Full Information Maximum Likelihood estimation for all analyses (Muthén & Muthén, 1998-2015; The Jamovi Project, 2022). We screened the data for missing values using Little's MCAR test, which was significant $p < .001$ (Li, 2013). A total of 38.6% of HRV data from the overall sample was missing from the baseline phase, 38.6% from the preparation phase, 38.6% from the speech phase and 38.6% from the social interaction phase. Inspection of histograms revealed no extreme outliers.

An a priori power analysis using G*Power 3.1 (Faul et al., 2009) revealed that a sample size of 28 was sufficient, assuming an alpha of .05, power of .80, and a medium effect size (minimum difference in slopes between consecutive phases within the autoregressive model = 0.09). To account for the multi-group analysis across the three slopes assessing the persistence of HRV (baseline-preparation, preparation-speech, speech-social interaction), the recommended sample size was tripled ($N \geq 84$). The sample size exceeds this estimate, with 59 healthy controls (non-SAD group) and 94 participants with a diagnosis of SAD (SAD group; N = 153).

3. Results

There was a significant difference in age and sex (SAD = 48% females, non-SAD = 64% females) between the SAD and non-SAD groups (See Table 2). Bivariate correlations for HRV measures, age and sex can be seen in Table 8 for the combined SAD group post-treatment and non-SAD group. All intercorrelations for HRV measures were statistically significant and of at least moderate magnitude. For the below results, a negative value indicates an increase, and a positive value indicates a decrease in HRV.

Table 8

Bivariate Correlations for HRV indices for the Post-treatment SAD and non-SAD groups

Variable	1	2	3	4	5	6	7	8	9
1. RMSSD Baseline	-								
2. RMSSD Preparation	.89*	-							
3. RMSSD Speech	.85*	.87*	-						
4. RMSSD Social	.89*	.87*	.87*	-					
5. HFabs Baseline	.95*	.82*	.81*	.84*	-				
6. HFabs Preparation	.83*	.96*	.83*	.84*	.80*	-			
7. HFabs Speech	.79*	.83*	.96*	.82*	.78*	.82*	-		
8. HFabs Social	.82*	.82*	.84*	.95*	.81*	.84*	.85*	-	
9. Age	-.12	-.15	-.06	-.03	-.04	-.08	.01	.04	-
10. Sex	-.05	.01	.01	-.03	.02	<.01	.02	-.06	

Note. N = 153. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *$p \leq .001$.

3.1 Hypothesis One: TSST Baseline Differences between Pre- versus Post-Treatment SAD Groups

Paired samples t-test revealed that there were no significant differences between-groups at the baseline phase for RMSSD (trait HRV; p = .24) or HFabs (trait HRV; p = .28).

3.2 Hypothesis Two: Mean Comparisons across TSST Phases between Pre- versus Post-Treatment SAD Groups

3.2.1 RMSSD

Age was not a significant covariate and did not alter the significance or pattern of any effects; therefore, it was removed as a covariate. Sex, however, did significantly influence the pattern of effects and was retained as a factor. changed significantly from pre-treatment to post-treatment (overall reduction in HRV; see Table 10), regardless of the specific phase. The post-hoc comparisons revealed that for females, there was a significant increase between the baseline and preparation phases, $t(447) = -11.28$, $p \leq .001$, $\Delta = -20.03$, a significant reduction between the preparation and speech phases, $t(444) = 3.02$, $p = .04$, $\Delta = 5.43$, and a non-significant reduction between the speech and social interaction phases, $t(445) = 1.14$, $p = 1.00$, $\Delta = 2.04$. For males, there was a significant increase between the baseline and preparation phases, $t(446) = -5.57$, $p \leq .001$, $\Delta = -9.43$, a non-significant reduction between the preparation and speech phases, $t(444) = 0.44$, $p = 1.00$, $\Delta = 0.74$, and a non-significant reduction between the speech and social interaction phases, $t(444) = 1.47$, $p = 1.00$, $\Delta = 2.51$. Estimated marginal means, 95% confidence intervals, and standard errors are reported in Table 10 (see also Figure 2a)

3.2.2 HFabs

Similar to RMSSD, age was not significant covariate and did not alter the significance or pattern of any effects and was therefore removed as a covariate from the analyses. Sex, however, significantly influence the pattern of effects and was retained as a factor. There was a significant main effect of phase, a significant main effect of time, and a significant two-way phase by sex interaction, but no three-way interactions (see Table 9). The post-hoc comparisons revealed that for females, there was a

significant increase between the baseline and preparation phases, $t(447) = -7.11$, $p \leq .001$, $\Delta = -329.60$, non-significant reduction between the preparation and speech phases, $t(445) = 2.72$, $p = .15$, $\Delta = 127.93$, and a non-significant reduction between the speech and social interaction phases, $t(446) = 0.38$, $p = 1.00$, $\Delta = 17.75$. For males, there was a non-significant increase between the baseline and preparation phases, $t(446) = -1.56$, $p = 1.00$, $\Delta = -69.10$, a non-significant increase between the preparation and speech phases, $t(444) = -1.14$, $p = 1.00$, $\Delta = -50.46$, and a non-significant reduction between the speech and social interaction phases, $t(444) = 2.25$, $p = .45$, $\Delta = 100.52$. Estimated marginal means, 95% confidence intervals, and standard errors are reported in Table 10 (see also Figure 2b).

Table 9

Fixed Effect Omnibus Tests Pre- and Post-treatment SAD Group

Measure	RMSSD				HFabs			
	F	Num df	Den df	*p*	*F*	Num df	Den df	*p*
Phase	53.55	3	441.9	<.001	14.66	3	443.1	<.001
Time	6.01	1	470.3	0.02	7.71	1	475.1	0.01
Sex	0.08	1	90.1	0.77	0.77	1	91.1	0.38
Phase * Time	0.93	3	441.5	0.42	0.75	3	442.7	0.52
Phase * Sex	6.31	3	441.9	<.001	6.08	3	443.1	<.001
Time * Sex	0.36	1	470.3	0.55	1.04	1	475.1	0.31
Phase * Time * Sex	1.39	3	441.5	0.25	1.46	3	442.7	0.23

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *F* = F-value; Num df = numerator degrees of freedom; Den df = denominator degrees of freedom; *p* = two-tailed p-value.

Table 10

Means for each group across the Trier Social Stress Test phases

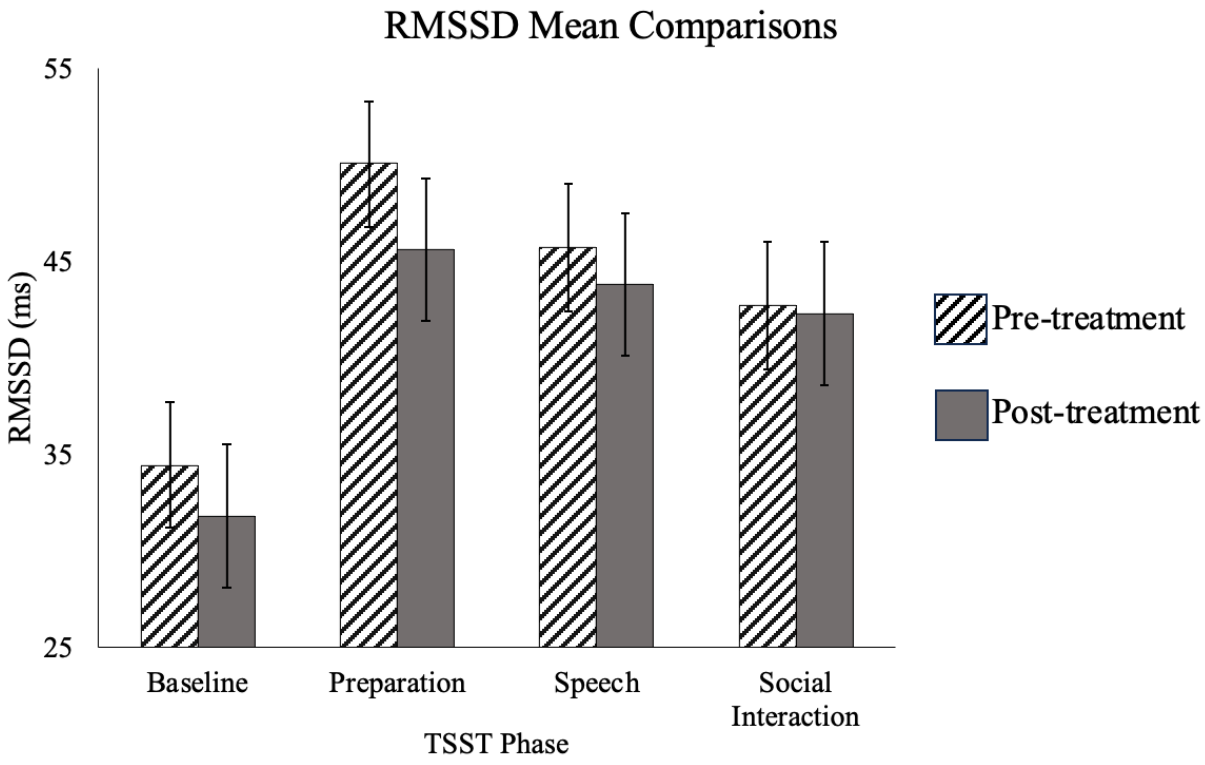
	Post-treatment SAD			Non-SAD		
Measure	*M*	95% CI	S.E.	*M*	95% CI	S.E.
RMSSD						
Baseline	31.8	28.0, 35.7	1.94	31.7	27.8, 35.7	2.01
Preparation	45.6	41.8, 49.5	1.95	42.8	38.9, 46.6	1.96
Speech	43.5	39.7, 47.3	1.95	47.6	43.8, 51.4	1.94
Social Interaction	42.3	38.5, 46.1	1.93	41.9	38.1, 45.7	1.92
HFabs						
Female						
Baseline	314	177, 451	69.6	390	264, 516	63.9
Preparation	604	463, 744	71.4	532	408, 657	63.3
Speech	534	394, 675	71.4	746	624, 868	62.0
Social Interaction	568	431, 705	69.6	474	354, 593	60.7
Male						
Baseline	480	344, 616	69.2	422	255, 590	85.1
Preparation	546	411, 680	68.4	603	444, 762	80.7
Speech	560	425, 694	68.3	657	498, 816	80.7
Social Interaction	484	349, 618	68.3	637	478, 796	80.7
Time						
RMSSD						
Pre-treatment	43.2	40.5, 45.9	1.37			
Post-treatment	40.9	37.9, 43.8	1.48			
HFabs						
Pre-treatment	576	510, 642	33.3			
Post-treatment	506	434, 578	36.4			

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *M* = mean estimate; CI = confidence intervals; S.E. = standard erorr. Non-SAD *n* = 59. SAD *n* = 94.

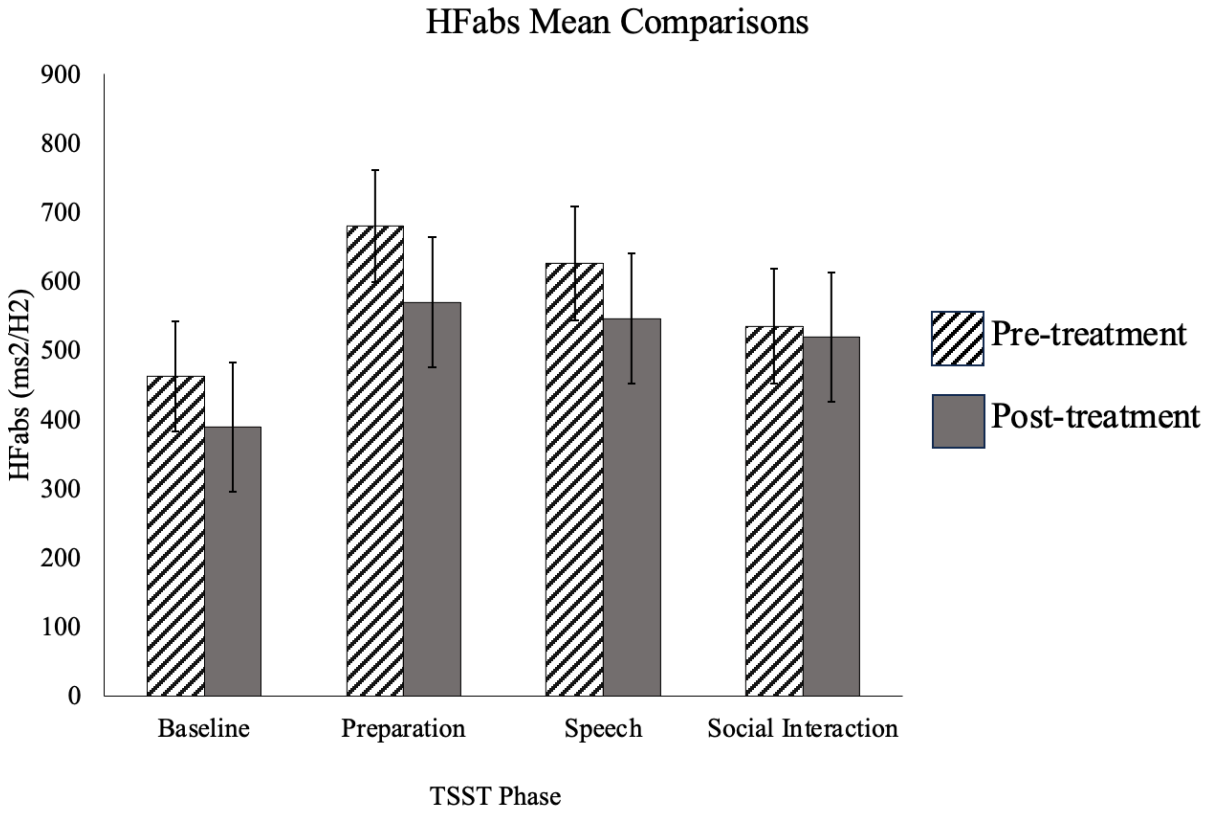
Figure 2

Mean Comparisons with 95% confidence intervals between the Pre- and Post-treatment SAD Participants during Four Phases of the TSST

a) RMSSD



b) HFabs


HFabs Mean Comparisons
900
800
700
600
500
400
300
200
100
0
HFabs (ms2/H2)
Baseline
Preparation
Speech
Social Interaction
TSST Phase
Pre-treatment
Post-treatment

3.3 Hypothesis Three: Autoregressive Models Comparing Pre- versus Post-treatment SAD Groups

3.3.1 RMSSD

Consistent with study one, we ran an autoregressive model regressing each phase on the preceding two phases (AR2), which provided an excellent fit on the CFI, SRMR, TLI, and RMSEA (see Table 11).

Results for within-group comparisons showed that all autoregressive pathways for the pre-treatment SAD group were significantly different from zero (*ps* < .05), indicating HRV persistence. The autoregressive pathways for the post-treatment SAD group were also all significant (*ps* < .05), except between the preparation and social interaction phases (β = .13, 95%CI = -0.06, 0.32, *p* =.18; see Table 12). Results revealed no autoregressive pathways significantly differed between-groups (ps >.05), except for between the baseline and preparation phases (β = -.29, 95%CI = -0.57, -.02, *p* =.04; see Table 12). This could indicate a possible treatment effect where there was a reduction in HRV persistence at post-treatment compared to pre-treatment (see Table 12).

3.3.2 HFabs

The AR2 model provided an excellent fit on CFI, SRMR, TLI and RMSEA (see Table 11). Results for within-group comparisons showed that all autoregressive pathways for the pre-treatment SAD group were significantly different from zero (*ps* < .05), indicating HRV persistence. The autoregressive pathways for the post-treatment SAD group were also all significant (*ps* < .05), except between the baseline and speech phases (β = .20, 95%CI = -0.06, 0.45, *p* =.13; see Table 12). Results revealed that no autoregressive pathways significantly differed between-groups for HFabs (ps >.05; Table 12).

Table 11

Goodness of Fit of Autoregressive Models

	χ^2	*df*	CFI	TLI	SRMR	RMSEA [90%CI]
Pre- versus post-treatment SAD group						
RMSSD						
AR2	15.54	2	1.00	1.00	.02	<.01 [<.001, 0.21]
HFabs						
AR2	0.28	2	1.00	1.00	.01	<.01 [<.001, 0.11]
SAD versus non-SAD group						
RMSSD						
AR2	4.36	2	.99	.86	.02	.14 [<.001, 0.33]
HFabs						
AR2	0.53	2	1.00	1.00	.02	<.01 [<.001, 0.15]

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; AR = autoregressive; CFI = comparative fit index; TLI = Tucker–Lewis Index; SRMR = standardized root mean square residual; RMSEA = root mean square error of approximation; *df* = degrees of freedom; CI = confidence interval; Δ = difference.

Table 12

Autoregressive Model 2 – Within- and Between-groups (Pre- versus Post-treatment SAD groups)

	Pre-Treatment SAD			Post-treatment SAD			Group Comparisons		
HRV	β	[95% CI]	*p*	β	[95% CI]	*p*	β	[95% CI]	*p*
RMSSD									
Baseline → Preparation	1.08	[0.88, 1.28]	<.001	0.53	[0.15, 0.92]	.01	-0.29	[-0.57, -0.02]	.04
Baseline → Speech	0.45	[0.13, 0.76]	.01	0.38	[0.14, 0.62]	<.01	-0.05	[-0.38, 0.28]	.75
Preparation → Speech	0.27	[0.05, 0.50]	.02	0.34	[0.11, 0.57]	<.01	0.02	[-0.36, 0.39]	.93
Preparation → Social Interaction	0.32	[0.16, 0.47]	<.001	0.13	[-0.06, 0.32]	.18	-0.28	[-0.57, 0.01]	.06
Speech → Social Interaction	0.51	[0.33, 0.70]	<.001	0.74	[0.54, 0.94]	<.001	0.20	[-0.05, 0.45]	.12
HFabs									
Baseline → Preparation	0.87	[0.67, 1.07]	<.001	0.50	[0.26, 0.74]	<.001	0.02	[-0.44, 0.07]	.16
Baseline → Speech	0.43	[0.13, 0.73]	.01	0.29	[-0.06, 0.45]	.13	-0.08	[-0.53, 0.19]	.36
Preparation → Speech	0.26	[0.01, 0.52]	.04	0.41	[0.17, 0.65]	<.01	0.14	[-0.23, 0.48]	.50
Preparation → Social Interaction	0.25	[0.06, 0.43]	.01	0.24	[0.01, 0.46]	.04	-0.12	[-0.44, 0.21]	.49
Speech → Social Interaction	0.38	[0.21, 0.56]	<.001	0.63	[0.43, 0.83]	<.001	<.001	[-0.15, 0.41]	.36

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; β = beta estimate; CI = confidence intervals; *p* = two-tailed p-value. Pre-treatment *n* = 57. Post-treatment *n* = 94.

3.4 Hypothesis Four: TSST Baseline Differences between SAD vs. Non-SAD Groups

Independent samples t-test revealed that there were no differences between-groups at the baseline phase for RMSSD (trait HRV: $p = 0.76$) and HFabs (trait HRV: $p = 0.88$).

3.5 Hypothesis Five: Mean Comparisons across TSST phases across SAD vs. non-SAD Groups

3.5.1 RMSSD

Age and sex were not significant covariates and did not alter the significance or pattern of any effects and were therefore removed from RMMSD analyses. There was no significant main effect for group, a significant main effect of phase, and no significant two-way or three-way interactions (see Table 13 and Figure 3a). As seen in Figure 3a, the post-hoc comparisons revealed that there was a significant increase between the baseline and preparation phases, $t(304) = -9.16$, $p < .001$, $\Delta = -12.41$, no significant increase between the preparation and speech phases, $t(304) = -1.01$, $p = .31$, $\Delta = -1.35$, and a significant reduction between the speech and social interaction phases, $t(302) = 2.62$, $p = .03$, $\Delta = 3.43$. Estimated marginal means, 95% confidence intervals, and standard errors are reported in Table 11.

Therefore, RMSSD increased between the baseline and preparation phases and between the preparation and speech phases, then reduced between the speech and social interaction phases (see Appendix B for full comparisons between phases). Importantly, in contrast to the pre-treatment comparisons (study one), the interaction effect was not significant, indicating that the groups did not significantly differ in the degree to which they physiologically responded to each phase.

3.5.2 HFabs

Age was not observed to be a significant covariate and did not alter the significance or pattern of any effects and was therefore removed as a covariate from the analyses. Sex, however, was observed to significantly influence the pattern of effects and was retained as a covariate. There was a significant main effect of phase and a significant three-way interaction effect for group by phase by sex (see Table 13). We explored the three-way interaction effect for group by phase by sex between consecutive

phases. Between the baseline and preparation phases, there was a significant main effect of phase and a significant three-way interaction (see Table 13). Between these phases for females, there was a significant main effect of phase, $F(1,46.4) = 36.18$, $p < .001$, and group by phase interaction, $F(1, 46.4) = 7.55$, $p = .01$, whereby the females with SAD increased to a greater extent $t(95.1) = -4.76$, $p < .001$, $\Delta = -304.30$, than females without SAD, $t(100.2) = -2.10$, $p = .84$, $\Delta = -126.85$. For males, there was a significant main effect of phase, $F(1,93.3) = 24.69$, $p < .001$, no significant main effect of group, $F(1,103.3) = .08$, $p = .78$, and a non-significant group by phase interaction, $F(1,93.3) = .20$, $p = .65$.

Between the preparation and speech phases, there were no significant main effects, two-way or three-way interactions (see Table 13). Between the speech and social interaction phases, there was a significant main effect of phase and a significant three-way interaction (see Table 13). Between these phases for females, there was a significant main effect of phase, $F(1,56.7) = 7.41$, $p = .01$, and group by phase interaction, $F(1, 56.7) = 14.11$, $p < .001$, whereby the SAD females showed an increase, $t(103) = -0.68$, $p = 1.00$, $\Delta = -42.04$, while females without SAD decreased, $t(102) = 4.93$, $p < .001$, $\Delta = 264.01$. For males, there were no significant main effects or group by phase interactions.

These findings indicate that the interaction was driven by a larger increase in HFabs between the baseline and preparation phase for the SAD group compared to the non-SAD group, specifically for the females with SAD group relative to the males (see Figure 3b, followed by relative stability across the remaining three phases in the SAD group. In contrast, the non-SAD group, specifically females, continued to increase from the preparation to speech phases and then reduced to the social interaction phase (see Appendix B for full comparisons between phases).

Table 13

Fixed Effect Omnibus tests SAD versus non-SAD

Measure	RMSSD				HFabs			
	F	Num df	Den df	*p*	*F*	Num df	Den df	*p*
Overall								
Group	5.08	1	108	0.98	0.70	1	109	0.40
Phase	41.46	3	295	<.001	13.14	3	297	<.001
Sex	0.01	1	108	0.93	0.26	1	109	0.61
Group * Phase	1.38	3	295	0.25	2.07	3	297	0.11
Group * Sex	1.05	1	108	0.31	0.08	1	109	0.77
Phase * Sex	1.44	3	295	0.23	1.13	3	297	0.34
Group * Phase * Sex	2.33	3	295	0.07	3.36	3	297	0.02
Baseline to Preparation								
Group	0.51	1	99.5	0.48	0.03	1	100.1	0.86
Phase	80.95	1	90.5	<.001	27.20	1	89.3	<.001
Sex	0.48	1	99.5	0.49	1.04	1	100.1	0.31
Group * Phase	0.62	1	90.5	0.43	0.22	1	89.3	0.64
Group * Sex	0.67	1	99.5	0.42	0.03	1	100.1	0.86
Phase * Sex	1.38	1	90.5	0.24	1.86	1	89.3	0.18
Group * Phase * Sex	4.90	1	90.5	0.03	5.01	1	89.3	0.03
Preparation to Speech								
Group	0.09	1	98.7	0.76	1.59	1	100.9	0.21
Phase	0.15	1	91.5	0.70	2.30	1	93.6	0.13
Sex	0.29	1	98.7	0.59	0.04	1	100.9	0.83
Group * Phase	2.62	1	91.5	0.11	3.91	1	93.6	0.05
Group * Sex	1.19	1	98.7	0.28	0.00	1	100.9	0.97
Phase * Sex	0.63	1	91.5	0.43	0.35	1	93.6	0.56
Group * Phase * Sex	1.36	1	91.5	0.25	2.07	1	93.6	0.15

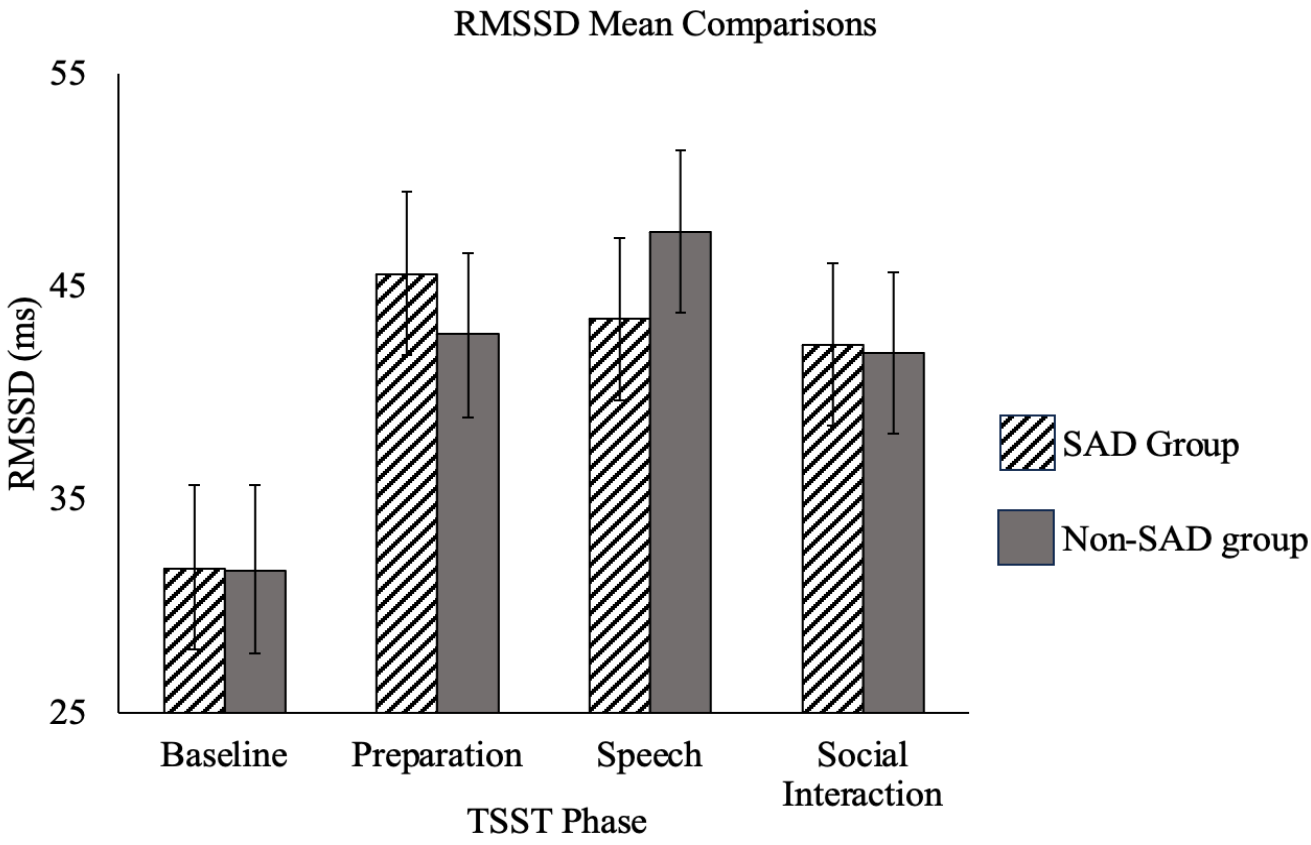
Speech to Social Interaction								
Group	0.24	1	109	0.63	2.05	1	110	0.16
Phase	9.02	1	100	0.01	5.69	1	103	0.02
Sex	0.40	1	109	0.53	0.01	1	110	0.93
Group * Phase	3.42	1	100	0.07	4.13	1	103	0.05
Group * Sex	1.67	1	109	0.20	0.35	1	110	0.55
Phase * Sex	1.04	1	100	0.31	1.55	1	103	0.22
Group * Phase * Sex	1.79	1	100	0.18	8.85	1	103	<.01

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; *F* = F-value; Num df = numerator degrees of freedom; Den df = denominator degrees of freedom; *p* = two-tailed p-value. Non-SAD *n* = 59. SAD *n* = 94

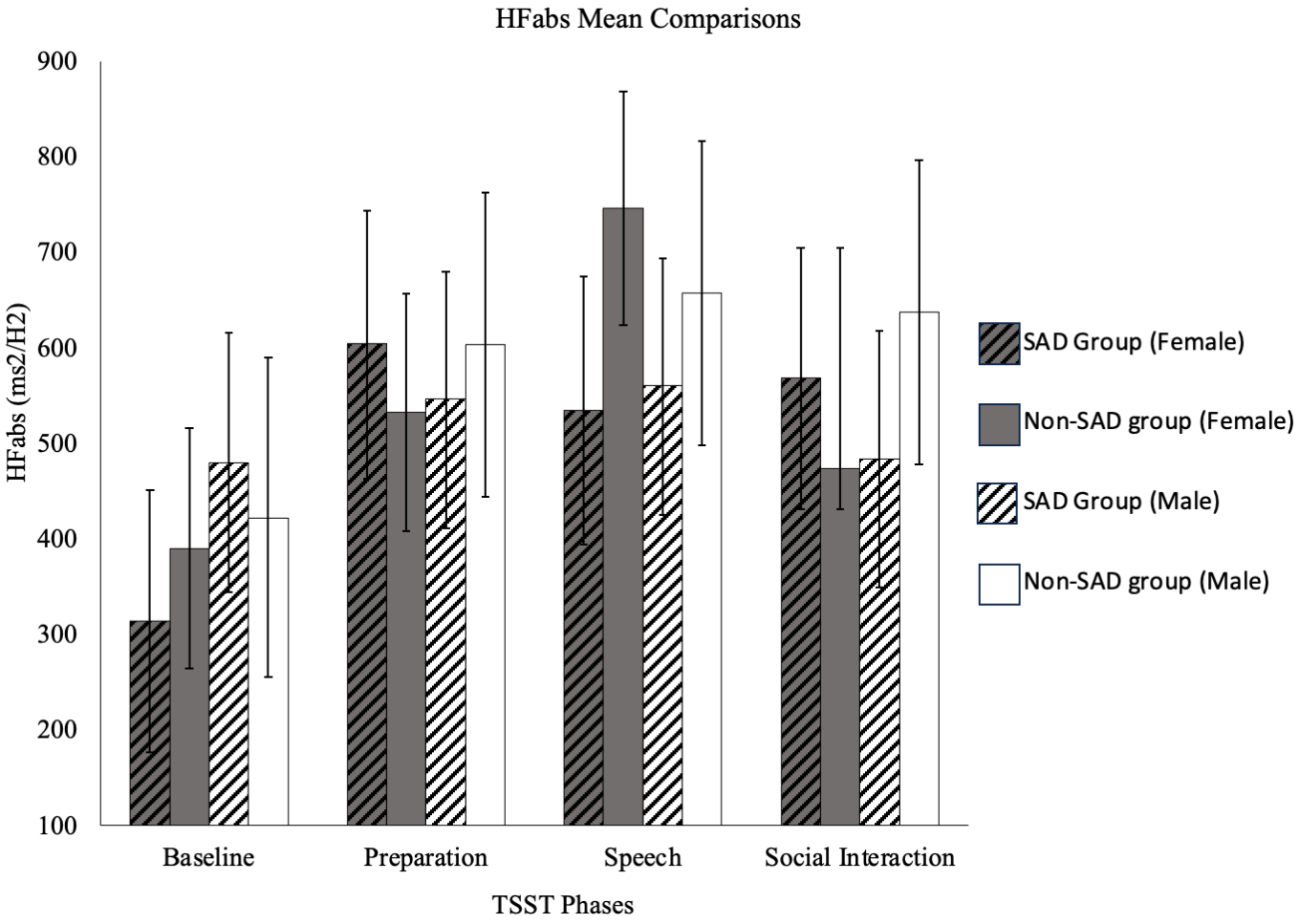
Figure 3

Mean Comparisons with 95% confidence intervals between the SAD and Non-SAD Participants (Female and Male) during Four Phases of the TSST

a) RMSSD



b) HFabs


HFabs Mean Comparisons
900
800
700
600
500
400
300
200
100
HFabs (ms2/H2)
Baseline
Preparation
Speech
Social Interaction
TSST Phases
SAD Group (Female)
Non-SAD group (Female)
SAD Group (Male)
Non-SAD group (Male)

3.6 Hypothesis six: Autoregressive Models Comparing SAD vs. non-SAD Groups

3.6.1 RMSSD

The autoregressive models only controlled for age, as sex was not significant for any phase. Consistent with study one, we ran an autoregressive model regressing each phase on the preceding two phases (AR2), which provided an excellent fit on the CFI and SRMR, and an adequate fit on the TLI, although the RMSEA remained somewhat high (see Table 11). Modification indices did not identify additional substantive sources of model strain.

Results for within-group comparisons showed that all autoregressive pathways for the post-treatment SAD group were significantly different from zero (*ps* < .05), except for between preparation and social interaction (β = .12, 95%CI = -0.12, 0.31, *p* =.32; see Table 14). The autoregressive pathways for the non-SAD sample were also all significant (*ps* < .05), except for between the preparation and speech phase (β = .07, 95%CI = -0.16, 0.49, *p* =.68; see Table 14). No autoregressive parameters significantly differed between-groups (*ps* >.05, see Table 14).

3.6.2 HFabs

The AR2 model provided an excellent fit on CFI, TLI, SRMR, and RMSEA (see Table 11). Results for within-group comparisons showed that all autoregressive pathways for the post-treatment SAD group were significantly different from zero (*ps* ≤ .05; see Table 14). The autoregressive parameters for the post-treatment non-SAD group were significantly different from zero (*ps* ≤ .05; see Table 14), except for between the baseline and preparation phase (*p* = .64) and the preparation to social interaction phase (*p* = .10). No autoregressive parameters significantly differed between-groups (*ps* >.05; see Table 14).

Table 14

Autoregressive Model 2 – Within- and Between-groups

	Post-Treatment SAD			Non-SAD			Group Comparisons		
HRV	β	[95% CI]	*p*	β	[95% CI]	*p*	β	[95% CI]	*p*
RMSSD									
Baseline → Preparation	0.49	[0.16, 0.86]	.01	0.94	[0.53, 1.49]	<.001	-0.45	[-1.10, 0.12]	.15
Baseline → Speech	0.37	[0.12, 0.73]	.01	0.65	[0.03, 1.02]	.01	-0.28	[-0.75, 0.39]	.32
Preparation → Speech	0.32	[0.05, 0.56]	.01	0.07	[-0.16, 0.49]	.68	0.25	[-0.30, 0.58]	.25
Preparation → Social Interaction	0.12	[-012, 0.31]	.32	0.32	[0.14, 0.50]	<.001	-0.22	[-0.50, 0.06]	.13
Speech → Social Interaction	0.73	[0.52, 0.94]	<.001	0.52	[0.20, 0.71]	<.001	0.21	[-0.07, 0.53]	.17
HFabs									
Baseline → Preparation	0.76	[0.49, 1.06]	<.001	0.16	[-0.64, 0.78]	.64	0.02	[-0.59, 0.40]	.94
Baseline → Speech	0.36	[0.02, 0.63]	.02	-0.67	[-1.10, -0.39]	<.001	-0.08	[-0.68, 0.50]	.78
Preparation → Speech	0.32	[0.09, 0.56]	.01	0.52	[0.24, 0.87]	.46	0.14	[-0.32, 0.50]	.51
Preparation → Social Interaction	0.24	[0.03, 0.41]	.02	0.32	[-0.07, 0.69]	.10	-0.12	[-0.51, 0.30]	.60
Speech → Social Interaction	0.39	[0.21, 0.58]	<.001	0.33	[-<.001, 0.62]	.03	<.001	[-0.29, 0.36]	.99

Note. RMSSD = root mean square of successive differences between heartbeats; HFabs = Absolute value of high frequency; β = beta estimate; CI = confidence intervals; *p* = two-tailed p-value. Non-SAD *n* = 59. SAD *n* = 94.

4. Discussion

The aim of the present study was to investigate HRV in SAD by examining differences in HRV across four phases of the TSST (baseline, preparation, speech task and social interaction) between (a) the sample with SAD at pre- versus post-treatment and (b) the sample with SAD at post-treatment versus a non-SAD sample. These comparisons are important for understanding the processes of psychophysiological change but also for potentially identifying signals that might represent risk factors for relapse if differences remain. Our first hypothesis was that individuals with SAD at pre-treatment would have less trait biological capacity to respond to stress compared to individuals with SAD at post-treatment. This hypothesis was not supported, as there were no significant baseline (trait HRV) differences between the pre- and post-treatment SAD groups on either measure of HRV. Our second hypothesis was that individuals with SAD will be less physiologically flexible when under social stress at pre-treatment, which would be reflected in smaller mean changes in state HRV when anticipating and experiencing social stress compared to post-treatment. This hypothesis was also not supported; there was no time by phase interaction that would have indicated that participants with SAD responded differently across the phases at post-treatment compared to pre-treatment.

Our third hypothesis was that individuals with SAD will be less flexible in their HRV across contexts at pre-treatment, and if this is the case, then HRV will be more persistent across the TSST phases at pre-treatment compared to post-treatment. This hypothesis was partially supported, specifically for RMSSD, where there was a significant between-group difference between the baseline and preparation phases, indicating a reduction in HRV persistence at post-treatment compared to pre-treatment. This indicates a potential increase in flexibility and a more adaptive response to anticipatory anxiety during the preparation phase for individuals with SAD post-treatment. However, there were no significant between-group differences for HFabs. When looking within-groups, all autoregressive pathways were significant for individuals with SAD at pre-treatment, and all pathways were also

significant at post-treatment except for between the preparation and social interaction phases for RMSSD and baseline and speech task phases for HFabs. This indicates that individuals with SAD exhibit persistence in HRV across most phases at both time points; however, they may be becoming more flexible (less persistent) at post-treatment. However, there were no significant between-group differences, so we cannot put too much weight on this.

Our fourth hypothesis was that individuals with SAD at post-treatment will have less trait biological capacity to respond to stress than individuals without SAD. This hypothesis was not supported, similar to pre-treatment (study one), there were no significant baseline (trait HRV) differences between the SAD and non-SAD groups on either measure of HRV, and no main effects of group (SAD versus non-SAD) in any of the linear mixed models. Our fifth hypothesis was that individuals with SAD at post-treatment will be less physiologically flexible when under social stress, and if this were the case, we would expect to observe smaller mean changes in state HRV when anticipating and experiencing social stress compared to individuals without SAD. This would indicate that, on average, individuals without SAD are able to adapt better to stressful situations as they are more physiologically flexible in comparison to the SAD group. This hypothesis was not supported as there were no significant group by phase interactions, indicating the groups did not significantly differ in the degree to which they physiologically responded to each phase on either HRV measure. Interestingly, at pre-treatment there was a significant group by phase interaction, which indicated, specifically in the preparation phase, that the SAD group, particularly females, significantly increased more than the non-SAD group and males with SAD. In contrast, at post-treatment this interaction was not significant, indicating that the SAD group at post-treatment and non-SAD groups no longer differed in changes in HRV across phases. Thus, the SAD group's pattern of responding is more similar to the non-SAD group at post-treatment. This may still indicate a treatment effect.

Our sixth hypothesis was that individuals with SAD at post-treatment will be less flexible in their HRV across contexts, and if this is the case, HRV will be more persistent across the TSST phases in individuals with versus without SAD. This hypothesis was not supported as there were no significant between-group differences of perseverance between any phase on either HRV index. However, the AR models showed evidence that the groups differed with respect to the 'within-group persistence' of HRV, specifically, the SAD group demonstrated more persistence (i.e., less flexibility) across the phases except for between the preparation and social interaction task. In comparison, the non-SAD group demonstrated less persistence (i.e., better emotion regulation) between some phases. Although these findings indicate that the non-SAD group may be more physiologically flexible, the lack of between-group differences means that we cannot draw definitive conclusions that the level of persistence differed between the groups.

In comparison to study one, this present study found a significant between-group difference in the AR model for RMSSD between the baseline and preparation phases, indicating a reduction in HRV (better flexibility) for the SAD group pre- to post-treatment. Additionally, this study revealed a significant main effect of time for both HRV indices, indicating that HRV changed significantly from pre-treatment to post-treatment (overall reduction) regardless of the specific phase. However, the overall lack of between-group differences (for AR models and linear mixed models) for both HRV indices between the SAD versus non-SAD groups is consistent with study one, which found no significant between-group differences in both HRV indices between individuals with and without SAD at pre-treatment. Additionally, the between-group results from this study are consistent with Klumbies et al.'s (2014) study, which found no between-group differences in RMSSD between people with and without social phobia at rest or during social stress. The results of the present study seem to be inconsistent with a study by Alvares et al. (2013), which found reduced HRV levels in those with compared to without SAD.

Sex significantly influenced the pattern of effect for HFabs, where females in the SAD group showed higher HRV during the preparation phase and increased their need to regulate (during anticipation) to a greater degree than the non-SAD group and males with SAD. There was also no evidence that this pattern changed between pre- and post-treatment. These findings could be explained by gender differences in cognitive processes, like rumination and repetitive negative thinking styles. Females are more likely to negatively ruminate and remain self-focused in response to distress (Nolen-Hoeksema & Jackson, 2001). This may play a part in the observed pattern of findings, as females engage in rumination concerning their impending stressors. This rumination, as suggested by the Response Styles Theory, may lead to an escalation in anticipatory anxiety, which is driven by the memory of previous failures and social situations (Mellings & Alden, 2000). The Response Styles Theory posits that women are more likely to exhibit a higher propensity for worry and rumination than men, which aligns with the gender-specific HRV patterns observed in this study (John & Whisman, 2013). Thus, this theory may offer a credible explanation for the increased HRV observed during the preparation phase for females with SAD. These insights may be valuable for exploring more tailored interventions that account for gender-specific aspects of SAD and offer a more nuanced understanding of the mechanisms of SAD.

The results of this study may provide valuable insights into the potential influence of CBT on HRV in individuals with SAD. The findings comparing the SAD group pre- to post-treatment, specifically the significant between-group difference for RMSSD in the AR model between the baseline and preparation phases, indicating a reduction in HRV persistence at post-treatment compared to pre-treatment, suggests a possible treatment effect. Additionally, when comparing the SAD group pre- to post-treatment, the significant main effect of time found for both HRV indices may suggest the influence of CBT; however, because there was no SAD control group who did not receive treatment, we cannot attribute these changes to CBT. There is a possibility that these findings are due to a practice effect,

where completing the TSST a second time may be less stressful for participants, therefore, resulting in less activated HRV.

The mean HRV results of this study demonstrate that at post-treatment, individuals with SAD started to demonstrate similar patterns of HRV to the non-SAD participants, especially in respect to earlier phases (in both HRV indices). The shift towards more normalised HRV may imply that CBT had a positive impact on the vagal tone, which may have led to better physiological responses. Additionally, in comparison to pre-treatment (study one), the two-way group by phase interaction was not significant for both HRV indices, which could suggest a treatment effect. These changes align with the Polyvagal Theory (Porges, 2003) and the Process Model of Emotion Regulation (Gross, 2015), which suggest that interventions targeting emotion regulation, cognitive reappraisal and social engagement can lead to better physiological responses to social stress. The results from this study may also be explained by the GUTS, which suggests that when an individual identifies a safe environment, vagal outflow increases, leading to a more emotionally regulated state (Brosschot et al., 2018). An explanation for the post-treatment SAD group showing similar mean HRV to the non-SAD group may be that CBT assisted individuals with SAD in identifying a safe environment, leading to more control of maladaptive autonomic processes (Brosschot et al., 2018; Porges, 2003). However, similar to the pre-treatment (study one) findings, the between-group results for the SAD versus non-SAD group show some inconsistencies with the Polyvagal Theory (Porges, 2003) and the GUTS (Brosschot et al., 2018), which suggest that disorders like SAD are associated with reduced HRV levels (PNS activation). While the two groups continued to show substantial differences in social anxiety symptomatology, the lack of significant between-group differences in HRV for the SAD group challenges the association between anxiety and reduced parasympathetic influence on HRV as proposed by Porges' (2003) model.

The results of the current study have some clinical implications. Specifically, the findings from the comparison of individuals with SAD at pre- and post-treatment and the more normalised patterns of

HRV observed in individuals with SAD compared to without indicate the potential value of interventions like CBT that focus on emotion regulation and physiological arousal. However, the overall lack of between-group differences for the AR models between individuals with versus without SAD and between HFabs at pre- versus post-treatment may indicate the need for more tailored and adjunctive interventions that are more effective at modulating HRV responses and producing longer-term outcomes. For example, biofeedback and mindfulness, together with CBT, may result in reduced symptoms of anxiety and greater flexibility and adaptability in response to social stress (Lehrer et al., 2020).

This study had several strengths, such as it being one of the first studies to examine the impact of CBT on HRV patterns across the four phases of the TSST in individuals with and without SAD. However, there are limitations that need to be considered. The post-treatment data only reflect short-term effects of CBT (one-month post-treatment), so longer follow-up periods could investigate more sustained evidence of physiological changes. As noted in Study 1, future research should also consider other variables that may impact or moderate HRV (physical activity, caffeine, smoking, alcohol, sleep quality; Alvares et al., 2013; Tiwari et al., 2021). Future studies could also examine the potential benefits of adjunctive interventions targeting psychophysiological responses alongside CBT. Additionally, higher-powered research with larger samples may detect more between-group differences. The lack of a SAD control group that did not receive an intervention also reduced our capacity to attribute changes to the intervention rather than the passage of time or practice effects (i.e., the TSST being less stressful due to greater familiarity rather than treatment effects at post-treatment). There were also a substantial number of comparisons in this study, and although adjustments were made to reduce family-wise Type I error rates, findings must be considered preliminary until they are replicated.

The results from this study highlight the potential positive effects of CBT and could indicate that adjunctive interventions may lead to better psychophysiological responses/outcomes for individuals

with SAD. This study found a significant between-group difference for RMSSD between the baseline and preparation phase, indicating an overall reduction in HRV persistence for the SAD group post-treatment compared to pre-treatment. Additionally, there was a main effect of time found in the linear mixed models for both HRV indices, demonstrating a significant change of HRV (reduction) from pre- to post-treatment regardless of a specific phase. These findings may highlight a potential treatment effect of CBT, where the post-treatment SAD group seem to be more physiologically flexible, although further research and replications are required. Overall, the SAD group's mean HRV pattern was more similar to the non-SAD group at post-treatment. For both HRV measures, the two-way group by phase interaction was not significant, indicating the groups did not significantly differ in the degree to which they physiologically responded to each phase on either HRV measure. This could suggest a potential treatment effect of CBT as it may indicate that post-treatment individuals with SAD responded similarly to the TSST compared to the non-SAD individuals. However, as there were no significant between-group differences between the SAD and non-SAD groups, the differing patterns seen in the 'within-group' AR models and the mean changes should be regarded with caution. More research is required to replicate these findings in larger samples to develop a more thorough understanding of the complex relationship between HRV, SAD, CBT and social stress. Future research should also examine how evidence-supported treatments long-term influence HRV indices.

Chapter 4: General Discussion

The aim of Study 1 was to investigate HRV in SAD by examining differences in HRV between individuals with and without SAD and across baseline, preparation (anticipation), speech task and social interaction phases of the Trier Social Stress Test. Results from Study 1 (pre-treatment) found that for both HRV indices, the SAD group increased their need to regulate their emotions (peak HRV) during the preparation (i.e., anticipation) phase, particularly among females, whereas HRV only peaked for the non-

SAD group during the social-evaluative context. Between some of the phases, no significant persistence of HRV was observed for the non-SAD group, whereas the SAD group demonstrated significant persistence across all phases. Although these within-group patterns were evident, there were no significant between-group differences. These findings provide novel evidence of similarities and differences in HRV between individuals with and without SAD while anticipating and encountering social-evaluative contexts.

The aim of study 2 was to investigate differences in HRV across the four phases of the TSST (baseline, preparation, speech task and social interaction) between (a) the sample with SAD at pre- versus post-treatment and (b) the sample with SAD at post-treatment versus a non-SAD sample. This helped to understand the effects of CBT on HRV patterns and the processes of psychophysiological change. Results from Study 2 (post-treatment) found that HRV changed (reduction) significantly from pre- to post-treatment for individuals with SAD. Individuals with SAD at post-treatment showed more adaptive responses to stress compared pre-treatment. The findings from the AR models showed a significant between-group difference, specifically a reduction in HRV (RMSSD) persistence at post-treatment compared to pre-treatment, indicating greater flexibility. The within-group results from the AR models also revealed more flexibility in the post-treatment SAD group compared to pre-treatment. Additionally, the SAD group started to demonstrate more similar patterns of HRV to the non-SAD participants, especially with respect to earlier phases (in both HRV indices). The within-group results from the AR models revealed that compared to the SAD group, the non-SAD group was more physiologically flexible between the preparation and speech phases with respect to changes in mean HRV level and persistence. However, there was a lack of significant between-group differences; thus, reliable conclusions regarding group differences cannot be drawn.

Specifically at pre-treatment, the non-significant differences between the groups on objective measures of physiological responding may be explained by cognitive-behavioural theories. These models

indicate that individuals who are socially anxious tend to have situation-specific thoughts and negatively biased perceptions of themselves and how others view them. Awareness of physiological symptoms of anxious arousal influences these mental representations (Clark & Wells, 1995; Heimberg et al., 2010; Rapee & Heimberg, 1997). Thus, a more significant between-group difference that maintains SAD may be subjective hypervigilance and hypersensitivity to internal physiological cues rather than objective psychophysiological responding. Challenging this hyperfocus to physiological symptoms (self-perceived significance to sweating, blushing etc.) is commonly used in evidence-based cognitive behavioural treatments to reduce the perceived cost of internal physiological reactions. Thus, it is interesting that individuals with SAD at post-treatment started to show more flexibility and similarities to the non-SAD group, indicating that CBT may have influenced a more adaptive pattern of responding, aligning the physiological responses of individuals with SAD more closely with those without SAD. Thus, strategies that reduce hypervigilance or hypersensitivity to physical cues in individuals with SAD may be significantly important in reducing the maintenance of SAD.

Together these studies provide a clearer picture of patterns of psychophysiological responding in individuals with SAD at pre- and post-treatment and versus individuals without SAD in anticipation of and within social-evaluative contexts. Study 2 indicated the potential value of interventions like CBT that focus on emotion regulation and physiological arousal. Our findings of similarities between people with and without SAD may help to challenge perceptions of critical differences in psychophysiology between these groups. The lack of between-group differences between the SAD versus non-SAD groups, particularly in study 2, may indicate the need for more tailored and adjunctive interventions, which may be beneficial in further modulating HRV responses and producing longer-term outcomes.

Research that focuses on physiology with the aim of diminishing the severity of anxiety and stress symptoms has yielded promising results. A study by Goessl et al. (2017) investigated the impact of HRV biofeedback on symptoms of anxiety and stress across different populations (those with speech

and performance anxiety, trait anxiety and general stress). Biofeedback increases control over vagal tone (PNS) activity and may result in increased HRV (better adaptability) in response to social-evaluative stress (Lehrer et al., 2020). Goessl et al.'s (2017) study revealed a large and significant within-group effect of HRV biofeedback on symptoms of anxiety (Hedges' g = .81). These results indicate that treatments like biofeedback can lead to notable enhancements in self-reported symptom severity of anxiety and stress. This underscores the idea that addressing physiological parameters of anxiety and stress may impact self-reported symptom severity, further highlighting the intricate relationship between cognitions and physiological responding. Thus, the results from study 2 may highlight the need for adjunctive interventions that target physiological responding, particularly during the anticipation of social-evaluative contexts, which may enhance longer-term outcomes from existing evidence-based treatments.

The studies reported in this thesis show several strengths. These studies are the first to examine psychophysiological responding across four phases of the TSST in both the SAD and non-SAD groups and the impact of CBT on HRV patterns. For the first time, study 1 allowed us to understand how HRV changes from rest to anticipation of social stress (anticipatory processing) and from anticipation through to direct exposure to social stressors. Study 2 extended Study 1 by increasing our understanding of the effects of CBT on HRV patterns and the processes of psychophysiological change.

Both studies also demonstrated important limitations that need to be considered for future research directions. First, both studies highlighted the need for higher-powered research to capture absolute differences between groups and differences in the profiles of HRV reactivity. Future studies should consider the impact of other factors that are known to influence HRV like physical activity, caffeine, smoking, alcohol, and sleep quality (Alvares et al., 2013; Tiwari et al., 2021), leading to a more complete understanding of physiological processes underlying SAD. Given the gender-specific differences observed, future research could investigate the relationship between sex, HRV and response

styles (repetitive negative thinking) when anticipating a stressor. Additionally, study 2 only reflects the short-term effects of CBT, thus, future research should investigate longer-follow up periods. This may provide more reliable insights into psychophysiological changes and potential signals that might represent risk factors for relapse. Future research could investigate more tailored and adjunctive interventions (e.g., mindfulness and biofeedback) targeting psychophysiological responses alongside CBT to see whether there are benefits in further modulating HRV responses and greater longer-term outcomes for individuals with SAD.

In conclusion, the results from pre-treatment (study 1) found that the SAD group showed higher HRV at the preparation phase (context sensitivity) may be due to females with SAD being less able to distinguish between 'safe' (preparation) and 'unsafe' (the actual social-evaluative context) situations (Porges, 2003; Brosschot et al., 2018). These findings are broadly consistent with cognitive behavioural and psychophysiological models and suggest that targeting emotion regulation in anticipation of social stressors may be critical for treating SAD. The post-treatment results revealed a significant change in HRV (reduction) from pre- to post-treatment and that individuals with SAD are becoming more physiologically flexible following treatment. Overall, the SAD group's mean HRV pattern seemed to be more like the non-SAD group post-treatment. However, there were no significant between-group results found at pre-treatment or post-treatment between these groups and therefore, the differing patterns seen in the 'within-group' AR models and the mean changes should be regarded with caution. This thesis expands the work of previous literature examining physiology in SAD, further extending our knowledge of how HRV differentiates individuals with and without SAD. The project also exposes important gaps in research, such as a detailed understanding of the complex interplay between HRV, SAD, CBT, sex, and social stress. It is clear that more research is required to replicate these findings in larger samples and develop a more comprehensive understanding of this complex relationship.

References

Aderka, I., Hofmann, S., Nickerson, A., Hermesh, H., Gilboa-Schechtman, E., & Marom, S. (2012). Functional impairment in social anxiety disorder. *Journal of Anxiety Disorders*, *26*(3), 393-400. https://doi.org/10.1016/j.janxdis.2012.01.003

Allen, A., Kennedy, P., Dockray, S., Cryan, J., Dinan, T., & Clarke, G. (2017). The Trier Social Stress Test: Principles and practice. *Neurobiology of Stress*, *6*, 113-126. https://doi.org/10.1016/j.ynstr.2016.11.001

Alvares, G., Quintana, D., Kemp, A., Van Zwieten, A., Balleine, B., Hickie, I., & Guastella, A. (2013). Reduced Heart Rate Variability in Social Anxiety Disorder: Associations with Gender and Symptom Severity. *PLoS One*, *8*(7), e70468. https://doi.org/10.1371/journal.pone.0070468

American Psychiatric Association. (2013). Diagnostic and statistical manual of mental disorders. Washington, DC: American Psychiatric Association.

Andrews, G., Bell, C., Boyce, P., Gale, C., Lampe, L., Marwat, O., Rapee, R., & Wilkins, G. (2018). Royal Australian and New Zealand College of Psychiatrists Clinical Practice Guidelines for the treatment of panic disorder, social anxiety disorder and generalised anxiety disorder. *Australian & New Zealand Journal of Psychiatry*, *52*(12), 1109–1172. https://doi.org/10.1177/0004867418799453

Bandelow, B., Michaelis, S., & Wedekind, D. (2017). Treatment of anxiety disorders. *Dialogues in Clinical Neuroscience*, *19*(2), 93-107. https://doi.org/10.31887/dcns.2017.19.2/bbandelow

Brosschot, J. F., Verkuil, B., & Thayer, J. F. (2018). Generalized unsafety theory of stress: Unsafe environments and conditions, and the default stress response. *International Journal of Environmental Research and Public Health*, 15, 464. https://doi.org/10.3390/ijerph15030464

Bulley, A., Miloyan, B., Brilot, B., Gullo, M. J., & Suddendorf, T. (2016). An evolutionary perspective on the co-occurrence of social anxiety disorder and alcohol use disorder. *Journal of Affective Disorders*, *196*, 62–70. https://doi.org/10.1016/j.jad.2016.02.028

Cheah, C., Lavery, C., Johnson, A., Clarke, P., & McEvoy, P. (2022). *Changes and Persistence in Heart Rate Variability Before and During Social Stress: A Comparison of Individuals With and Without Social Anxiety Disorder.* [Manuscript submitted for publication]. Master of Research (Psychology), Curtin University.

Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. G. Heimberg, M. R. Liebowitz, D. A. Hope, & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment, and treatment* (pp. 69–93). The Guilford Press.

Cline, Graysen. Nonparametric Statistical Methods Using R. United Kingdom, EDTECH, 2019

De Castella, K., Goldin, P., Jazaieri, H., Heimberg, R. G., Dweck, C. S., & Gross, J. J. (2014). Emotion beliefs and cognitive behavioural therapy for Social Anxiety Disorder. *Cognitive Behaviour Therapy*, *44*(2), 128–141. https://doi.org/10.1080/16506073.2014.974665

Egizio, V., Jennings, J., Christie, I., Sheu, L., Matthews, K., & Gianaros, P. (2008). Cardiac vagal activity during psychological stress varies with social functioning in older women. *Psychophysiology*, *45*(6), 1046-1054. https://doi.org/10.1111/j.1469- 8986.2008.00698.x

Falkenström, F., Solomonov, N., & Rubel, J. A. (2022). How to model and interpret cross-lagged effects in psychotherapy mechanisms of change research: A comparison of multilevel and structural equation models. *Journal of Consulting and Clinical Psychology*, *90*(5), 446–458. https://doi.org/10.1037/ccp0000727

Faul, F., Erdfelder, E., Lang, A.-G., & Buchner, A. (2007). G*Power 3: A flexible statistical power analysis program for the social, behavioral, and biomedical sciences. *Behavior Research Methods*, 39(2), 175-191. http://www.gpower.hhu.de/fileadmin/redaktion/Fakultaeten/MathematischNaturwissenschaftliche_Fakultaet/Psychologie/AAP/gpower/GPower3-BRMPaper.pdf

First, M. B., Williams, J. B. W., Karg, R. S., & Spitzer, R. L. (2016). *Structured Clinical Interview for DSM-5 Disorders, Clinician Version (SCID-5-CV).* American Psychiatric Association.

George, D., & Mallery, P. (2003). SPSS for Windows step by step: A simple guide and reference. 11.0 update (4th ed.). Boston, MA: Allyn & Bacon.

Goessl, V. C., Curtiss, J. E, & Hofmann, S. E. (2017. The effect of heart rate variability biofeedback training on stress and anxiety: A meta-analysis. Psychological Medicine, 15, 2578-2586. https://doi.org/10.1017/S0033291717001003.

Gross, J. J. (2015). The extended process model of emotion regulation: Elaborations, applications, and Future Directions. *Psychological Inquiry*, *26*(1), 130–137. https://doi.org/10.1080/1047840x.2015.989751

Gullett, N., Zajkowska, Z., Walsh, A., Harper, R., & Mondelli, V. (2023). Heart rate variability (HRV) as a way to understand associations between the autonomic nervous system (ANS) and affective states: A critical review of the literature. *International Journal of Psychophysiology*, *192*, 35–42. https://doi.org/10.1016/j.ijpsycho.2023.08.001

Heimberg, R., Brozovich, F., & Rapee, R. (2010). A Cognitive Behavioral Model of Social Anxiety Disorder. *Social Anxiety*, 395-422. https://doi.org/10.1016/b978-0-12-375096-9.00015-8

Held, J., Vîslă, A., Wolfer, C., Messerli-Bürgy, N., & Flückiger, C. (2021). Heart rate variability change during a stressful cognitive task in individuals with anxiety and control participants. *BMC Psychology*, *9*(1). https://doi.org/10.1186/s40359-021-00551-4

Higham, D. J., & Higham, N. J. (2016). MATLAB guide (Vol. 150). Siam

Hofmann, S. (2007). Cognitive factors that maintain social anxiety disorder: A comprehensive model and its treatment implications. *Cognitive Behaviour Therapy*, *36*(4), 193-209. https://doi.org/10.1080/16506070701421313

Hu, L., & Bentler, P. (1999). Cutoff criteria for fit indexes in covariance structure analysis: Conventional criteria versus new alternatives. *Structural Equation Modelling: A Multidisciplinary Journal*, *6*(1), 1-55. https://doi.org/10.1080/10705519909540118

Hyett, M., Bank, S., Lipp, O., Erceg-Hurn, D., Alvares, G., & Maclaine, E. et al. (2018). Attenuated Psychophysiological Reactivity following Single-Session Group Imagery Rescripting versus Verbal Restructuring in Social Anxiety Disorder: Results from a Randomized Controlled Trial. *Psychotherapy and Psychosomatics*, *87*(6), 340-349. https://doi.org/10.1159/000493897

Johnson, D. P., & Whisman, M. A. (2013). Gender differences in rumination: A meta-analysis. *Personality and Individual Differences*, *55*(4), 367–374. https://doi.org/10.1016/j.paid.2013.03.019

Kaufmann, T., Sutterlin, S., Schulz, S. M., & Vogele, C. (2011). ARTIIFACT: A tool for heart rate artifact processing and heart rate variability analysis. *Behaviour Research Methods*, 4, 1161-1170. https://doi.org/10.3758/s13428-011-0107-7

Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey

Replication. *Archives of General Psychiatry*, 62(6), 593–602. https://doi.org/10.1001/archpsyc.62.6.593

Kimhy, D., Crowley, O., McKinley, P., Burg, M., Lachman, M., & Tun, P. et al. (2013). The association of cardiac vagal control and executive functioning – Findings from the MIDUS study. *Journal of Psychiatric Research*, *47*(5), 628-635. https://doi.org/10.1016/j.jpsychires.2013.01.018

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The 'tier social stress test' – a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology*, 28, 76-81. https://doi.org/10.1159/000119004

Klumbies, E., Braeuer, D., Hoyer, J., & Kirschbaum, C. (2014). The reaction to social stress in social phobia: Discordance between physiological and subjective parameters. *PLoS One*, 9. https://doi.org/10.1371/journal.pone.0105670

Kristal-Boneh, E., Raifel, M., Froom, P., & Ribak, J. (1995). Heart rate variability in health and disease. *Scandinavian Journal of Work, Environment and Health*, *21*(2), 85-95. https://doi.org/10.5271/sjweh.15

Kushki, A., Drumm, E., Pla Mobarak, M., Tanel, N., Dupuis, A., Chau, T., & Anagnostou, E. (2013). Investigating the autonomic nervous system response to anxiety in children with autism spectrum disorders. *PLoS One*, *8*(4), e59730. https://doi.org/10.1371/journal.pone.0059730

Laborde, S., Mosley, E., & Thayer, J. F. (2017). Heart rate variability and cardiac vagal tone in psychophysiological research – Recommendations for experiment planning, data analysis, and data reporting. *Frontiers in Psychology*, *08*. https://doi.org/10.3389/fpsyg.2017.00213

Lavery, C. (2022). *Does heart rate variability better differentiate individuals with and without an anxiety disorder at rest or during stress?* [Manuscript submitted for publication]. Master of Research (Psychology), Curtin University.

Lehrer, P., Kaur, K., Sharma, A., Shah, K., Huseby, R., & Bhavsar, J. et al. (2020). Heart rate variability biofeedback improves emotional and physical health and performance: A systematic review and meta-analysis. *Applied Psychophysiology and Biofeedback*, *45*(3), 109-129. https://doi.org/10.1007/s10484-020-09466-z

Leigh, E., & Clark, D. (2018). Understanding social anxiety disorder in adolescents and improving treatment outcomes: Applying the cognitive model of Clark and Wells (1995). *Clinical Child and Family Psychology Review*, *21*(3), 388-414. https://doi.org/10.1007/s10567-018-0258-5

Li, C. (2013). Little's test of missing completely at random. The State Journal, 13, 795-809. https://doi.org/10.1177/1536867X1301300407

Mattick, R. P., & Clarke, C. (1998). Development and validation of measures of social phobia scrutiny fear and social interaction anxiety. *Behaviour Research and Therapy*, 36, 455-470. https://doi.org/10.1016/S0005-7967(97)10031-6

McEvoy, P. M., Hyett, M. P., Bank, S. R., Erceg-Hurn, D. M., Johnson, A. R., Kyron, M. J., Saulsman, L. M., Moulds, M. L., Grisham, J. R., Holmes, E. A., Moscovitch, D. A., Lipp, O. V., Campbell, B. N. C., & Rapee, R. M. (2022b). Imagery-enhanced v. verbally-based group cognitive behaviour therapy for social anxiety disorder: a randomised clinical trial. *Psychological Medicine*, 1-10. https://doi.org/10.1017/S0033291720003001

McEvoy, P. M., Moulds, M. L., Grisham, J. R., Holmes, E. A., Moscovitch, D. A., Hendrie, D., Saulsman, L. M., Lipp, O. V., Kane, R. T., Rapee, R. M., Hyett, M. P., & Erceg-Hurn, D. M. (2022a). Assessing the

efficacy of imagery-enhanced cognitive behavioural group therapy for social anxiety disorder: study protocol for a randomized controlled trial. *Contemporary Clinical Trials*, 60, 34-41. https://doi.org/0.1016/j.cct.2017.06.010

McEvoy, P. M., Watson, H., Watkins, E. R., & Nathan, P. (2013). The relationship between worry, rumination, and comorbidity: Evidence for repetitive negative thinking as a transdiagnostic construct. *Journal of Affective Disorders*, *151*(1), 313–320. https://doi.org/10.1016/j.jad.2013.06.014

McLean, C. P., Asnaani, A., Litz, B. T., & Hofmann, S. G. (2011). Gender differences in anxiety disorders: Prevalence, course of illness, comorbidity and burden of illness. *Journal of Psychiatric Research*, *45*(8), 1027–1035. https://doi.org/10.1016/j.jpsychires.2011.03.006

Mellings, T. M. B., & Alden, L. E. (2000). Cognitive processes in social anxiety: The effects of self-focus, rumination and anticipatory processing. *Behaviour Research and Therapy*, *38*(3), 243–257. https://doi.org/10.1016/s0005-7967(99)00040-6

Mulcahy, J., Larsson, D., Garfinkel, S., & Critchley, H. (2019). Heart rate variability as a biomarker in health and affective disorders: A perspective on neuroimaging studies. *Neuroimage*, *202*, 1-11. https://doi.org/10.1016/j.neuroimage.2019.116072

Muthén, L. K., & Muthén, B. O. (1998-2015). *Mplus user's guide* (7th ed.). Los Angeles, CA: Author.

National Institute for Health and Care Excellence. (2013). Social anxiety disorder: *Recognition, assessment and treatment*. https://www.nice.org.uk/guidance/cg159/resources/social- anxiety-disorder-recognition-assessment-and-treatment-pdf-35109639699397

Nolen-Hoeksema, S., & Jackson, B. (2001). Mediators of the gender difference in rumination. *Psychology of Women Quarterly*, *25*(1), 37–47. https://doi.org/10.1111/1471-6402.00005

Perna, G., Riva, A., Defillo, A., Sangiorgio, E., Nobile, M., & Caldirola, D. (2020). Heart rate variability: Can it serve as a marker of mental health resilience? *Journal of Affective Disorders*, *263*, 754-761. https://doi.org/10.1016/j.jad.2019.10.017

Pham, T., Lau, Z. J., Chen, S. H., & Makowski, D. (2021). Heart rate variability in psychology: A review of HRV Indices and an analysis tutorial. *Sensors*, *21*(12), 3998. https://doi.org/10.3390/s21123998

Porges, S. W. (2003). Social Engagement and Attachment. *Annals of the New York Academy of Sciences*, 1008, 31–47. https://doi.org/10.1196/annals.1301.004

Quintana, D. S., Alvares, G. A., & Heathers, J. A. J. (2016). Guidelines for reporting articles on psychiatry and heart rate variability (GRAPH): Recommendations to advance research communication. Translational Psychiatry, 6. https://doi.org/10.1038/tp.2016.73

Rapee, R. M., Gaston, J. E., & Abbott, M. J. (2009). Testing the efficacy of theoretically derived improvements in the treatment of social phobia. *Journal of Consulting and Clinical Psychology*, 77(2), 317–327. https://doi.org/10. 1037/a0014800.

Rapee, R., & Heimberg, R. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, *35*(8), 741-756. https://doi.org/10.1016/s0005-7967(97)00022-3

Reyes del Paso, G., Langewitz, W., Mulder, L., van Roon, A., & Duschek, S. (2013). The utility of low frequency heart rate variability as an index of sympathetic cardiac tone: A review with emphasis on

a reanalysis of previous studies. *Psychophysiology*, *50*(5), 477-487. https://doi.org/10.1111/psyp.12027

Segerstrom, S. C., & Nes, L. S. (2007). Heart rate variability reflects self-regulatory strength, effort, and fatigue. *Psychological Science*, *18*(3), 275–281. https://doi.org/10.1111/j.1467-9280.2007.01888.x

Shaffer & Ginsberg, 2017. An overview of heart rate variability metrics and norms. Frontiers in Public Health, 5, 258. https://doi.org/10.3389/fpubh.2017.00258

Tabachnick, B. G., & Fidel, L. S. (2013). Using multivariate statistics: Pearson new international edition (6th ed.). Pearson Education Limited. https://ebookcentral.proquest.com/lib/curtin/detail.action?docID=5175291.

Thayer, J. F., & Lane, R. D. (2000). A model of neurovisceral integration in emotion regulation and dysregulation. *Journal of Affective Disorders*, *61*(3), 201–216. https://doi.org/10.1016/s0165-0327(00)00338-4

The Jamovi Project (2022). *Jamovi* (Version 2.3) [Computer Software]. Retrieved from https://www.jamovi.org

Tiwari, R., Kumar, R., Malik, S., Raj, T., & Kumar, P. (2021). Analysis of heart rate variability and implication of different factors on heart rate variability. *Current Cardiology Reviews*, *17*(5). https://doi.org/10.2174/1573403x16999201231203854

Tolin, D. F., Lee, E., Levy, H. C., Das, A., Mammo, L., Katz, B. W., & Diefenbach, G. J. (2021). Psychophysiological assessment of stress reactivity and recovery in anxiety disorders. *Journal of Anxiety Disorders*, *82*, 1-9. https://doi.org/10.1016/j.janxdis.2021.102426

Vreijling, S. R., Troudart, Y., & Brosschot, J. F. (2020). Reduced heart rate variability in patients with medically unexplained physical symptoms: A meta-analysis of HF-HRV and RMSSD. *Psychosomatic Medicine*, *83*(1), 2–15. https://doi.org/10.1097/psy.0000000000000874

Wells, A., Fisher, P., Myers, S., Wheatley, J., Patel, T., & Brewin, C. R. (2009). Metacognitive therapy in recurrent and persistent depression: A multiple-baseline study of a new treatment. *Cognitive Therapy and Research*, *33*(3), 291–300. https://doi.org/10.1007/s10608-007-9178-2

Wong, Q., & Rapee, R. (2016). The aetiology and maintenance of social anxiety disorder: A synthesis of complementary theoretical models and formulation of a new integrated model. *Journal of Affective Disorders*, *203*, 84-100. https://doi.org/10.1016/j.jad.2016.05.069

Wu, Y., Gu, R., Yang, Q., & Luo, Y. (2019). How do amusement, anger and fear influence heart rate and heart rate variability? *Frontiers in Neuroscience*, *13*. https://doi.org/10.3389/fnins.2019.01131

Appendices

Appendix A

3.2 Hypothesis Two: Mean Comparisons

3.2.1 RMSSD

Between the baseline and speech task phases there was a significant main effect of phase, $F(1,123) = 65.58$, $p < .001$, however, no significant two-way or three-way interactions. Between the baseline and social interaction task phases there was a significant main effect of phase, $F(1,129) = 126.43$, $p < .001$, however, no significant two-way or three-way interactions. Between the preparation and social interaction task phases there was a significant main effect of phase, $F(1,123) = 9.65$, $p < .01$, a significant group by phase interaction, $F(1,123) = 4.62$, $p = .03$, a significant group by sex interaction, $F(140) = 4.17$, $p = .04$, and a significant group by phase by sex interaction, $F(1,123) = 4.50$, $p = .04$. Between these phases for the females, there was a significant main effect of phase, $F(1,67.6) = 8.49$, $p = .01$, a significant main effect of group, $F(1,76.8) = 5.71$, $p = .02$, and a significant group by phase interaction, $F(1,67.6) = 9.14$, $p < .01$, whereby females with SAD showed a reduction, $t(127) = 4.76$, $p < .001$, $\Delta = 10.79$ while the females without SAD showed an increase, $t(127) = -0.08$, $p = 1.00$, $\Delta = -.19$. For males, there was a non-significant main effect of phase, $F(1,55.6) = 2.72$, $p = .11$, a non-significant main effect of group, $F(1,63.4) = .45$, $p = .51$, and a non-significant group by phase interaction, $F(1,55.6) = .2.15$, $p = .99$.

3.1.2 HFabs

Between the baseline and speech task phases there was a significant main effect of phase, $F(1,128) = 39.84$, $p < .001$, however, no significant two-way or three-way interactions. Between the baseline and social interaction task phases there was a significant main effect of phase, $F(1,125) = 11.68$, $p < .001$, and a significant main effect of sex, $F(1,146) = 4.27$, $p = .04$, however, no significant two-

way or three-way interactions. Between the preparation and social interaction task phases there was no significant main effects, two-way or three-way interactions (p >.05).

Appendix B

3.5 Hypothesis Five: Mean Comparisons across TSST phases across Post-treatment SAD vs. non-SAD Groups

3.5.1 RMSSD

Additionally, the post-hoc comparisons revealed that there was a significant increase between baseline and speech task phases, $t(306) = -10.15$, $p < .001$, $\Delta = -13.76$, a significant increase between baseline and the social interaction task phases, $t(306) = -7.70$, $p < .001$, $\Delta = -10.32$, and a non-significant reduction between the preparation and social interaction task phases, $t(304) = 1.58$, $p = .23$, $\Delta = 2.08$.

3.5.2 HFabs 3

Between the baseline and speech task phases there was a significant main effect of phase, $F(1,95.1) = 34.30$, $p < .001$, however, no significant two-way or three-way interactions. Between the baseline and social interaction task phases there was a significant main effect of phase, $F(1,100) = 17.13$, $p < .001$, and a significant group by phase by sex interaction, $F(1,100) = 5.50$, $p = .02$. Between these phases for the females, there was a significant main effect of phase, $F(1,55.5) = 16.33$, $p < .001$, no significant main effect of group, $F(1,59.9) = .07$, $p = .79$ and no significant group by phase interaction, $F(1,55.5) = 1.86$, $p = .18$. For the males, there was no significant main effect of phase, $F(1,44.2) = 4.13$, $p = .05$, no significant main effect of group, $F(1,48.4) = .29$, $p = .60$, and no significant group by phase interaction, $F(1,44.2) = 3.88$, $p = .06$. Between the preparation and speech task phases there was no significant main effects, two-way or three-way interactions ($p > .05$).

Supplementary Materials

Material 1

Demographics

1. Please enter your age in years (e.g., 22):

2. Please indicate which gender you identify as:

   - ☐ Male
   - ☐ Female
   - ☐ Non-binary – please specify:

Material 2

Medication Questionnaire

What medication did you take during the last 3 months?

With medication we mean all drugs that were prescribed and medication that may be bought at the pharmacy. See below for an example.

What is the medication name?	***How much did you take per time?***	***How many times did you take this per day?***	***How many days during the past 3 months did you take the medication?***
Example 1 Furosemide (diuretic)	*Example* 40 mg	*Example* 1 time	*Example* 26 days (2 times a week; 90 days)

Material 3

Social Interaction Anxiety Scale (SIAS)

Social Interaction Anxiety Scale (SIAS)

Instructions: This questionnaire asks you to think about social situations. For each question, please circle a number to

Social Interaction Anxiety Scale (SIAS)

Instructions: This questionnaire asks you to think about social situations. For each question, please circle a number to indicate the degree to which you feel the statement is characteristic of you. The rating scale is as follows:

0	1	2	3	4
Not at all characteristic of me	Slightly characteristic of me	Moderately characteristic of me	Very characteristic of me	Extremely characteristic of me

1.	I get nervous if I have to speak with someone in authority (eg teacher, boss)	0	1	2	3	4
2.	I have difficulty making eye contact with others.	0	1	2	3	4
3.	I become tense if I have to talk about myself or my feelings.	0	1	2	3	4
4.	I find it difficult mixing comfortably with the people I work with.	0	1	2	3	4
5.	I find it easy to make friends of my own age.	0	1	2	3	4
6.	I tense up if I meet an acquaintance in the street.	0	1	2	3	4
7.	When mixing socially, I'm uncomfortable.	0	1	2	3	4
8.	I feel tense if I am alone with just one person..	0	1	2	3	4
9.	I am at ease meeting people at parties, etc.	0	1	2	3	4
10.	I have difficulty talking with other people.	0	1	2	3	4
11.	I find it easy to think of thing to talk about.	0	1	2	3	4
12.	I worry about expressing myself in case I appear awkward.	0	1	2	3	4
13.	I find it difficult to disagree with another's point of view.	0	1	2	3	4
14.	I have difficulty talking to attractive persons of the opposite sex.	0	1	2	3	4
15.	I find myself worrying that I won't know what to say in social situations.	0	1	2	3	4
16.	I am nervous mixing with people I don't know very well.	0	1	2	3	4
17.	I feel I'll say something embarrassing when talking.	0	1	2	3	4
18.	When mixing in a group, I find myself worrying I will be ignored.	0	1	2	3	4
19.	I am tense mixing in a group.	0	1	2	3	4
20.	I am unsure whether to greet someone I know only slightly.	0	1	2	3	4

Material 4

Social Phobia Scale

Social Phobia Scale (SPS)

Instructions: For each question, please circle a number to indicate the degree to which you feel the statement is characteristic of you. The rating scale is as follows:

0	1	2	3	4
Not at all characteristic of me	Slightly characteristic of me	Moderately characteristic of me	Very characteristic of me	Extremely characteristic of me

1.	I become anxious if I have to write in front of other people.	0	1	2	3	4
2.	I become self-conscious when using public toilets.	0	1	2	3	4
3.	I can suddenly become aware of my own voice and of others listening to me.	0	1	2	3	4
4.	I get nervous that people are staring at me as I walk down the street.	0	1	2	3	4
5.	I fear I may blush when I am with others.	0	1	2	3	4
6.	I feel self-conscious if I have to enter a room where others are already seated.	0	1	2	3	4
7.	I worry about shaking or trembling when I'm watched by other people.	0	1	2	3	4
8.	I would get tense if I had to sit facing other people on a bus or a train.	0	1	2	3	4
9.	I get panicky that others might see me faint or be sick or ill.	0	1	2	3	4
10.	I would find it difficult to drink something if in a group of people.	0	1	2	3	4
11.	It would make me feel self-conscious to eat in front of a stranger at a restaurant.	0	1	2	3	4
12.	I am worried people will think my behaviour is odd.	0	1	2	3	4
13.	I would get tense if I had to carry a tray across a crowded cafeteria.	0	1	2	3	4
14.	I worry I'll lose control of myself in front of other people.	0	1	2	3	4
15.	I worry I might do something to attract the attention of other people.	0	1	2	3	4
16.	When in an elevator, I am tense if people look at me.	0	1	2	3	4
17.	I can feel conspicuous standing in a line.	0	1	2	3	4
18.	I can get tense when I speak in front of other people.	0	1	2	3	4
19.	I worry my head will shake or nod in front of others.	0	1	2	3	4
20.	I feel awkward and tense if I know people are watching me.	0	1	2	3	4

Material 5

SONA Study Advertisement

Study Status	**Visible to participants :** Approved **Inactive study :** Does not appear on list of available studies
Duration	90 minutes
Points	6 Points
Abstract	This study will examine social stress and its impact on physiology and factors related to social anxiety
Description	This study is recruiting individuals who do not get overly anxious in social situations like having a conversation, meeting unfamiliar people, or speaking in front of others. This project is investigating how people with minimal or no social anxiety react to mild social stress. Specifically, this study is interested in the impact of social stress on physical sensations (e.g., increased heart rate) and behaviour. The results of this study will improve our understanding on why some people have more noticeable physical sensations and anxious behaviours than others in social settings, particularly compared to those with social anxiety disorder, which is characterised by fear and avoidance of social situations. The data we collect in this project will be compared to existing data from a project our team conducted that examined the effectiveness of different psychological treatments for social anxiety disorder. For this study, we plan to recruit approximately 60 participants older than 18 years with minimal or no social anxiety.
Eligibility Requirements	18+ years of age

Material 6

Trier Social Stress Test Protocol

Researcher introduces task:

"For the next part we will ask you to attach a number of recording devices to measure your heart rate and skin moisture levels, before, during and after a speech task. We will measure your heart rate and skin moisture levels for 5 minutes at rest before the task. During the speech task itself you will be required to discuss your personal strengths and weaknesses. Finally, following the speech task I will ask you some more questions."

Heart rate (electrocardiogram) electrode placement:

"These electrodes will be used to record your heart rate before, during, and after the task we are about to perform. Place one on the inner side of your left hip bone, and one under each collar bone." Researcher provides participant with some privacy at this point, then guides participant to attach the electrode leads.

Attach electrocardiogram leads:

Researcher to show participant the leads and highlight that the leads will be attached to the electrodes to allow heart rate information to be sent to the recording equipment. Describe how the clip works. Clip each lead to each electrode (BLACK ← UPPER LEFT COLLAR BONE; WHITE ← UPPER RIGHT COLLAR BONE; RED ← LOWER LEFT HIP). Ask participant to be seated (set-up such that experimenter will be seated between table/equipment and door, with participant on opposite side).

Skin sweat response (electrodermal activity) set-up:

Once participant is seated, researcher places two electrodes on participant's non-dominant hand, and attaches leads. Check physiology recording is accurate before starting experiment. If not, make adjustments to set-up (i.e., check placement of electrodes on participant) and then begin.

Video camera set-up

Once participant is comfortably seated, set-up video camera on tripod and then begin the test.

Social stress test - baseline acquisition (5 mins):

Explain to participant: "We will now measure your heart rate and skin response for 5 minutes while you are at rest. Whilst we are recording I will leave the room. Do you have any questions before we start?" Press start on acquisition file (and record on camera) and record for 5 minutes. Leave the room during this period and return after 5 minutes is up. Press stop on the acquisition file and save. Close file.

Social stress test - preparation phase (3-minute preparation):

Read verbatim to participant: "Your task in this experiment is the following: please imagine that you have applied for a job and have been invited for an interview. In contrast to a real interview, however, you are supposed to give a talk, in which you are to convince me, in three minutes, why you think that you would be the best candidate for this position. Please note that you will be recorded by a camera and a microphone for subsequent voice and behavioural analysis. You should try to leave the best possible impression, and assume the role of the applicant for the duration of the talk as best as you can. Following your talk, you will be given a task, which will be explained to you at that point. You may take some notes now for three minutes, which you must not use during your talk. Do you have any questions?"

Provide participant with notepad and pen. The researcher then leaves while the participant remains in room where they have three minutes to prepare for the talk. The participant is allowed to take notes but must not use these notes during their speech in front of the researcher.

Return to room after 3 minute preparation period (stop physiology recording and set-up interview recording file).

Social stress test - free speech (3-minute speech):

- Re-start physiology recording, and then ask participant to begin speech.
- If the participant talks for ***longer*** than 3 minutes, then interrupt and press stop on recording.
- If participant stops ***before*** 3 minutes, wait for about 20s and then say: "You still have time."
- Only after a pause of more than twenty seconds prior to the end of the three minute period are questions asked (e.g., "*Tell me more about that particular strength?*").

- If participant does not continue, press stop on recording. □ Save file and exit.

Social stress test - interaction (5 minutes):

For the following it is important to treat it like a natural interaction/conversation.

➢ What do your family/friends especially appreciate about you?

○ [Researcher's standard contribution to the conversation: "If I were to say one thing that my family likes about me, I'd probably say that I am kind…and probably loyal too."]

➢ What do you appreciate about your friends?

○ [Researcher's standard contribution to the conversation: "If I were to say one thing that I appreciate about my friends, is that I have one friend in particular who would always be there for me no matter what."] ➢ What do you appreciate about colleagues?

○ [Researcher's standard contribution to the conversation: "I really like sharing ideas about new work projects, but also I see them outside of work sometimes, so some of them are like friends as well."]

➢ Please complete the following sentence: I am the best at/in…" ○ [Researcher's standard contribution to the conversation: "I find this a hard one to answer, but I think I am quite empathic and I try to understand where other people are coming from."]

- If participant gets stuck on any question, say, for example "Can you tell me more about that …" Continue for up to 5 minutes.
- After 5 minutes, press stop on recording. □ Save file and exit.