# MASTERARBEIT | MASTER'S THESIS

Titel | Title

Exploring the Impact of Chronic Fatigue and Social Support on Heart Rate Variability in Chronically Stressed Women

verfasst von | submitted by

Nicole Heyer BSc

angestrebter akademischer Grad | in partial fulfilment of the requirements for the degree of

Master of Science (MSc)

Wien | Vienna, 2025

| | |
|---|---|
| Studienkennzahl lt. Studienblatt \| Degree programme code as it appears on the student record sheet: | UA 066 840 |
| Studienrichtung lt. Studienblatt \| Degree programme as it appears on the student record sheet: | Masterstudium Psychologie |
| Betreut von \| Supervisor: | Univ.-Prof. Dr. Urs Markus Nater |
| Mitbetreut von \| Co-Supervisor: | Nida Ali BSc (Hons) MSc PhD |

**Contents**

**Abstract**

Chronic stress significantly impacts autonomic nervous system (ANS) function, particularly among women, contributing to adverse health outcomes. Heart rate variability (HRV), a sensitive marker of ANS-regulation, reflects the body's capacity to adapt to stress and recover. This thesis investigates the influence of chronic fatigue and perceived social support on HRV reactivity to and recovery after acute stress in chronically stressed women, addressing three research questions: (1) whether the level of chronic stress predicts HRV reactivity and recovery to acute stress, (2) whether chronic fatigue moderates the relationship between chronic stress and HRV reactivity/recovery to acute stress, and (3) whether perceived social support moderates the relationship between chronic stress and HRV reactivity/recovery to acute stress, such that higher support leads to increased HRV reactivity and recovery. A cross-sectional design was employed with 31 German-speaking women aged 20-65. HRV reactivity was measured during the Trier Social Stress Test and HRV recovery during a subsequent rest phase, with SDNN as the HRV index. Women with higher levels of chronic stress exhibited significantly lower HRV reactivity and recovery during and after acute stress compared to women with lower levels of chronic stress, indicating impaired autonomic flexibility. Chronic fatigue did not significantly moderate these relationships. While perceived social support did not significantly buffer the effects of chronic stress on HRV outcomes, trends suggested its potential role in improving HRV recovery. These findings emphasize the detrimental impact of chronic stress on autonomic regulation in women and highlight the importance of interventions to improve autonomic balance.

*Keywords:* chronic stress, heart rate variability, chronic fatigue, perceived social support, Trier Social Stress Test

**Exploring the Impact of Chronic Fatigue and Social Support on Heart Rate Variability in Chronically Stressed Women**

Chronic stress has become a pervasive health challenge in contemporary society, impacting individuals across all demographic groups. It arises from prolonged exposure to stressors that activate the stress response system, triggering a cascade of physiological, psychological, and behavioral changes. Defined as a maladaptive state of persistent activation of the *autonomic nervous system* (ANS), particularly the sympathetic branch, chronic stress leads to disruptions in cardiovascular regulation, metabolic processes, and immune functioning (Kim et al., 2018; Móra et al., 2022). Over time, these disruptions contribute to a range of adverse health outcomes, from cardiovascular diseases to mental health disorders, underscoring the critical need to understand the mechanisms underlying chronic stress.

*Heart rate variability* (HRV), which measures variations in the time interval between heartbeats, serves as a sensitive and non-invasive biomarker of autonomic regulation. High HRV reflects a well-functioning balance between the sympathetic ("fight-or-flight") and parasympathetic ("rest-and-digest") branches of the ANS, enabling effective adaptation to internal and external demands. Conversely, low HRV indicates autonomic dysregulation, often associated with chronic stress and reduced resilience to future stressors (Brugnera et al., 2018; Kim et al., 2018). Although different situations tend to trigger distinct stress response patterns, individuals can also exhibit varying stress responses to the same situation. This tendency to exhibit a particular pattern of stress responses across a variety of stressors is referred to as “response stereotypy” (Lacey & Lacey, 1958), influencing both reactivity to stress and the capacity for recovery (Schneiderman et al., 2004). Stress research has consistently demonstrated that acute stress, such as that induced by the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), reliably decreases HRV as the body mobilizes resources to confront immediate challenges. In healthy individuals, HRV recovers following the stressor, signaling a return to baseline autonomic function. However, in chronically stressed populations, HRV reactivity (the response to stress) and recovery (the return to baseline) are often impaired, suggesting a diminished capacity to adapt to stress (Da Estrela et al., 2020; Jiryis et al., 2022). These interindividual differences in stress response patterns are shaped by psychosocial factors, and HRV emerges as a particularly sensitive biomarker for capturing the variability in autonomic regulation influenced by these factors.

This thesis investigates the interaction between chronic stress and HRV patterns, focusing on two key psychosocial factors: chronic fatigue and perceived social support. *Chronic fatigue*, characterized by persistent exhaustion not alleviated by rest, is closely associated with autonomic dysfunction and heightened stress sensitivity (Escorihuela et al., 2020; Meeus et al., 2013). Women are disproportionately affected by chronic fatigue, which often exacerbates the physiological and

psychological burdens of chronic stress, making it a critical variable in this study. Conversely, *perceived social support*, defined as an individual's belief in the availability of interpersonal support, has been identified as a protective factor that buffers the effects of stress on autonomic function. High levels of perceived social support are associated with improved HRV, enhanced recovery from stress, and overall health benefits, offering a promising avenue for resilience-building interventions (Cohen & Wills, 1985; Kao et al., 2013).

Women are particularly vulnerable to the effects of chronic stress, consistently reporting higher stress levels and lower HRV compared to men. Biological factors such as hormonal fluctuations combined with sociocultural stressors like caregiving responsibilities and workplace pressures contribute to their heightened susceptibility (Almeida, 2005; Lampert et al., 2016). Moreover, gender-sensitive frameworks, such as the "tend-and-befriend" model (Taylor et al., 2000), suggest that women's stress responses are uniquely influenced by their social environment, further underscoring the importance of examining perceived social support in this context. By focusing on chronically stressed women, this thesis aims to address a critical gap in the literature, providing insights into the gender-specific mechanisms underlying stress resilience.

The present study seeks to explore how chronic fatigue and perceived social support moderate HRV reactivity and recovery in chronically stressed women. By analyzing these interactions, the research aims to identify modifiable factors that could inform tailored interventions to improve health outcomes. Specifically, this thesis addresses three key questions: (1) Do women with lower levels and higher levels of chronic stress differ in HRV reactivity and recovery in response to acute stress? (2) Does chronic fatigue moderate the relationship between the level of chronic stress and HRV reactivity and recovery to acute stress by blunting or exaggerating HRV reactivity and recovery in chronically fatigued individuals? (3) Can perceived social support buffer the adverse effects of chronic stress on HRV with women with higher levels of perceived social support displaying increased HRV reactivity and recovery to acute stress? In doing so, this thesis aims to contribute to a deeper understanding of the physiological and psychosocial dynamics of chronic stress, with implications for developing effective resilience-building strategies for vulnerable populations.

## 1. Theoretical Background

### 1.1 Stress and The Nervous System

To fully understand how chronic stress impacts HRV and specifically affects women, a deeper exploration of the nervous system is essential. The following section will delve into the autonomic nervous system (ANS), the primary mediator of the physiological responses to stress. This section lays the foundation for understanding HRV as a marker of autonomic function and stress resilience. By

examining the structure and function of the ANS, we can better comprehend how (chronic) stress influences bodily functions and impacts health outcomes. This knowledge is crucial for developing targeted interventions that effectively address the autonomic dysregulation associated with chronic stress.

### *1.1.1 The Autonomic Nervous System*

The autonomic nervous system (ANS) is part of the *peripheral nervous system* and critical in regulating involuntary physiological processes, including those of the cardiovascular system, respiratory rate, digestion, and more. It consists of two branches: the *sympathetic nervous system* (SNS) and the *parasympathetic nervous system* (PNS) (Anisman et al., 2018; Meeus et al., 2013; Thayer et al., 2009). The SNS is often characterized as the system responsible for the "fight-or-flight" response, which rapidly mobilizes the body's resources to confront or flee from threats. This response includes an increase in heart rate, a rise in blood pressure, and a surge in energy-providing blood sugar levels – physiological changes that are crucial during acute stress situations but can be detrimental when continuously activated (Móra et al., 2022; Weissman & Mendes, 2021). In contrast, the PNS is known as the "rest and digest" system, which predominates during peaceful, non-stressful conditions and helps to conserve energy by slowing the heart rate, decreasing blood pressure, and stimulating digestive activities. It plays a vital role in bringing the body back to a state of calm and balance after the resolution of a stressor (Jiryis et al., 2022; Phu et al., 2019; Thayer et al., 2009). This dynamic interplay between the SNS and PNS, working to maintain homeostasis and counterbalance one another, allows the body to respond appropriately to stressful events (Kim et al., 2018; Laborde et al., 2018; Weissman & Mendes, 2021). These systems were traditionally modeled as antagonistic: when environmental triggers, such as emotionally arousing events, cause *sympathetic* activity to dominate, an increase in heart rate can be observed. In other cases, the *parasympathetic* nervous system may be overly active, causing heart rate to become inordinarily low (Anisman et al., 2018).

However, the myriad challenges and stressors of our environment require flexible modulation of ANS activity for different contexts (Weissman & Mendes, 2021). The *Autonomic Space Model* (ASM; Berntson et al., 1991) is a conceptual framework developed to describe these complex interactions between the sympathetic and parasympathetic nervous systems. As previously stated, these systems were traditionally thought to function in a reciprocal manner – when one is activated, the other is suppressed. The ASM proposes a more nuanced understanding, suggesting that these systems can operate independently and sometimes concurrently, depending on the specific psychophysiological demands placed on an individual (Berntson et al., 1991). According to the ASM, autonomic responses are not limited to simple increases or decreases in sympathetic or parasympathetic activity but can involve diverse patterns of activation across both systems. This

model introduces the concept of *autonomic space*, which is defined by the level of sympathetic activation plotted against parasympathetic activation. Within this space, physiological states can be mapped to show varying degrees of sympathetic and parasympathetic activity, illustrating that certain situations may require simultaneous activation or inhibition of both systems (Berntson et al., 1991). For example, increased PNS activity may therefore be associated with a decrease, increase, or even no change in SNS activity (Shaffer & Ginsberg, 2017). This framework is particularly useful in stress research, as it allows for a more precise characterization of the ANS responses to stressors. For instance, an individual experiencing a stressor might not only show an increase in sympathetic activity (typical fight-or-flight response) but could also display changes in parasympathetic activity that do not conform to traditional models of reciprocal inhibition. Such patterns might indicate a more complex, adaptive response that involves maintaining some level of parasympathetic tone even during sympathetic activation, potentially aiding quicker recovery once the stressor is removed (Berntson et al., 1991).

Heart rate variability (HRV), which will be covered in more detail in the next section, emerges as a valuable metric in this framework, as it reflects autonomic balance and is considered a robust indicator of an individual's ability to adapt to stress (Brugnera et al., 2018; Mora et al., 2022). A healthy autonomic system quickly alternates between sympathetic dominance during stressful periods and parasympathetic recovery following stress. However, in situations of chronic stress, this balance may be disrupted, leading to a predominance of sympathetic activity that can negatively impact long-term health (Kim et al., 2018; Laborde et al., 2018). Chronic activation of the SNS, without adequate recovery through the PNS, leads to what is known as *autonomic dysfunction*, a state that is often found in individuals experiencing prolonged stress exposure. Such a condition not only diminishes the body's capacity to handle stress but also increases the risk for a variety of diseases, including cardiovascular disorders, metabolic syndrome, and mental health issues (Lampert et al., 2016; McEwen, 1998; Meeus et al., 2013).

An in-depth understanding of the ANS not only elucidates the physiological mechanisms underlying stress reactivity and recovery but also informs clinical and psychological practices designed to optimize health and resilience in individuals facing chronic stress. This knowledge is especially pertinent for populations that are particularly susceptible to stress, such as women, who exhibit distinct autonomic responses to chronic stressors (Almeida, 2005; Lampert et al., 2016). Furthermore, the Autonomic Space Model has significant implications for understanding individual differences in stress reactivity and recovery, even within women as a population. By using this model, researchers can identify unique signatures of autonomic regulation that may predict how individuals cope with stress. These signatures help in distinguishing between adaptive and maladaptive stress responses, providing insights into why some individuals are more resilient to stress than others

(Bonne et al., 2004). The Autonomic Space Model enriches our understanding of the ANS by depicting a more intricate picture of how autonomic functions can co-regulate and adapt to environmental demands. It underscores the complexity of physiological responses and highlights the importance of considering individual stress responses in the assessment of stress-related conditions. Building on this framework, the first aim of this thesis is to examine whether women with higher and lower levels of chronic stress differ in their stress response patterns. Furthermore, this thesis investigates whether chronic fatigue and perceived social support respectively moderate these stress responses, as measured by heart rate variability.

### *1.1.2 Heart Rate Variability*

Heart rate variability (HRV) is a well-established biomarker of ANS activity, particularly during laboratory stress tasks (Brugnera et al., 2018; Mora et al., 2022). HRV refers to the physiological phenomenon of variation in the time interval between heartbeats, influenced by the autonomic nervous system (Bamert & Inauen, 2022; Manser et al., 2021). HRV is recognized as a very sensitive indicator of ANS function and an integral measure in psychophysiological research. It is the most commonly used, fastest, and a non-invasive method, using basic cardiac measurements (e.g. an *electrocardiogram* (ECG)) (Meeus et al., 2013). It reflects the body's ability to adapt to stress and maintain homeostasis through the activities of the sympathetic and parasympathetic branches (Mora et al., 2022). By representing cardiac autonomic function, specifically parasympathetic modulation, HRV reflects the autonomic nervous system's sensitivity to psychophysiological stressors. As suggested by the *cardiovascular reactivity hypothesis* (Obrist, 1982), tracking cardiovascular responses to these types of stressors can offer valuable insights into individual patterns of psychophysiological stress responses (Manser et al., 2021).

The heart is dually innervated by the autonomic nervous system such that relative increases in sympathetic activity are associated with heart rate increases and relative increases in parasympathetic activity are associated with heart rate decreases. Thus, relative sympathetic increases cause the time between heart beats (the *interbeat interval*) to become shorter and relative parasympathetic increases cause the interbeat interval to become longer (Thayer et al., 2011). High HRV indicates a greater variability in the interbeat interval and reflects a more flexible autonomic nervous system, where both the sympathetic (which shortens the interval) and parasympathetic (which lengthens the interval) systems can respond dynamically to environmental demands. High HRV typically is associated with good health, indicative of a strong ability to engage in effective stress reactivity and recovery. Conversely, low HRV indicates more consistent, less variable interbeat intervals. It is associated with poor stress adaptation and a predictor of poor cardiovascular health

(Halko & Sääksvuori, 2017). Low HRV is also associated with both pathophysiological and psychopathological risk factors (Thayer et al., 2009).

There are two main methods for measuring HRV: *time-domain* and *frequency-domain measurements* (Brugnera et al., 2018). Frequency-domain parameters break down the heart rate signal into its component frequencies and measure their relative power. Time-domain variables involve statistical analyses of interbeat intervals (also known as *RR intervals* as they represent the time between two consecutive R-waves in the ECG), assessing how much individual cardiac cycle lengths vary around the mean. Shorter or more consistent RR intervals typically reflect higher sympathetic activity (e.g., during stress), while longer and more variable intervals indicate parasympathetic activity, associated with relaxation and recovery (Escorihuela et al., 2020; Meeus et al., 2013). Within time-domain indices, the *standard deviation of normal-to-normal (NN) intervals* (SDNN) is commonly used as a measure of total variability in heart rate. In contrast to RR intervals, NN intervals exclude irregular heartbeats that could distort the data for a more accurate reflection of autonomic activity (Escorihuela et al., 2020).

The impact of physical and psychological stress on HRV, though recognized as a sensitive physiological measure, remains an underexplored area in stress research. While distinct types of stressors elicit unique cardiovascular responses, how specific HRV indices shift in response to stress interventions is still not fully understood (Mora et al., 2022). This research is essential because HRV is not fixed; despite its genetic component, HRV levels are adaptable and can be modified (Thayer et al., 2009). Individual differences in HRV represent a potentially adaptable basis for various processes related to self-regulation, resilience, and overall health. For those affected by chronic stress, improving HRV through physical exercise, improving sleep quality or interventions such as mindefulness-based stress reduction or biofeedback training could foster better stress adaptation and resilience (Earnest et al., 2011; Rådmark et al., 2019; Yang et al., 2021). Understanding how HRV changes under stress opens pathways for developing interventions to improve autonomic flexibility, offering meaningful health benefits for vulnerable populations. There are three main theoretical accounts that explain the links between autonomic function and self-regulation: the Polyvagal Theory (Porges, 2007), the Neurovisceral Integration Model (Thayer et al., 2009) and the Vagal Tank Theory (Laborde et al., 2018).

Developed by Stephen Porges, *Polyvagal Theory* emphasizes the role of the vagus nerve in social behaviors and stress responses. The theory emphasizes the role of the vagus nerve, the principal component of the parasympathetic nervous system, which is critical in controlling heart rate, breathing, and digestive processes (Porges, 2007). The Polyvagal Theory introduces the concept of a “vagal brake”, which the body applies to slow heart rate and promote calm and restorative states, facilitating social communication, bonding, and overall emotional regulation (Porges, 2007). Polyvagal

Theory has been influential in explaining how disturbances in autonomic function may contribute to psychiatric conditions and suggests that enhancing vagal tone could improve emotional resilience and social engagement (Porges, 2007).

The *Neurovisceral Integration Model* (Thayer et al., 2009) expands on the autonomic nervous system's role by linking HRV to a broader neural network involved in threat appraisal, emotional regulation, and executive functioning. This model suggests that HRV is associated with structures of the neural network including the amygdala which are important in perceptions or appraisals of threat and safety, and stress (Thayer et al., 2011). The model postulates that cardiac vagal control, as reflected by HRV, is associated with positive outcomes regarding executive functions, emotion, and health, as well as a better self-regulation of the organism (Laborde et al., 2018).

In this model, the autonomic system's processes are ideally balanced, enabling the body to respond flexibly to a variety of stimuli. However, when this balance is lost, one process can dominate, making the system rigid and less adaptable to typical changes in the environment. For physiological regulation, especially in heart function, a balanced system reflects good health, as it allows the heart to adjust smoothly to physical and environmental demands. In contrast, when the system becomes "locked in" a particular mode, it becomes dysregulated. This is why a healthy heart shows natural variability in heart rate (high HRV), whereas a heart under stress or illness often lacks this variability, responding with a more rigid pattern (Thayer et al., 2011).

While both Polyvagal Theory and the Neurovisceral Integration Model provide insights into autonomic regulation and stress adaptation, they do not offer a practical framework for assessing real-time changes in vagal tone. *Vagal Tank Theory*, proposed by Laborde et al. (2018), fills this gap by conceptualizing vagal tone as a supply of "vagal energy" that can be depleted or replenished in response to stress. This model describes three phases of stress response – rest, reactivity, and recovery – which together reflect the dynamic use of vagal resources under stress:

1. Rest: At rest, the body should exhibit high vagal activity, indicating a full "vagal tank" and a state of calm.

2. Reactivity: During stress exposure, the body consumes this "vagal energy", temporarily reducing vagal activity and increasing sympathetic arousal to deal with the immediate challenge.

3. Recovery: After the stressor is removed, the rate of recovery depends on the remaining vagal resources. Quick recovery is a sign of a healthy stress response system and indicates sufficient residual vagal tone (Laborde et al., 2018).

At rest, higher *vagally mediated HRV* (vmHRV) is associated with better self-regulation, which includes processes like executive functioning and stress coping, enabling goal-directed behavior across various situations (Thayer et al., 2009). As such, higher vmHRV at rest is considered adaptive and predictive of more effective stress reactivity and recovery (Bamert & Inauen, 2022). During reactivity, changes in HRV depend on the metabolic demands of the situation. In tasks requiring executive functions without physical exertion, such as psychosocial stress tasks like the Trier Social Stress Test, both an increase or a smaller decrease in vmHRV can be adaptive responses to the challenge (Laborde et al., 2018). At recovery, rapid restoration of resting vmHRV levels is considered adaptive, reflecting having sufficient resources for recovery and readiness for future stressors. An exception arises when vmHRV increases during stress, which may indicate a training effect that contributes to higher resting HRV over time (Laborde et al., 2018). Empirical studies support these patterns, demonstrating the theory's validity (Bamert & Inauen, 2022). Although SDNN reflects total HRV rather than isolating vagal-mediated activity, it also provides useful insights into autonomic regulation across these phases. At rest, high SDNN typically reflects parasympathetic dominance in healthy individuals. During reactivity, SDNN generally decreases due to reduced parasympathetic activity and increased sympathetic dominance, mirroring vmHRV patterns. At recovery, SDNN is expected to increase, reflecting the return of parasympathetic activity and restoration of autonomic balance.

The Vagal Tank Theory helps in assessing the efficiency of stress management strategies by observing how quickly and effectively a person's "vagal tank" is replenished following stress exposure. This provides a practical approach to evaluating individual differences in stress resilience and the effectiveness of interventions aimed at enhancing autonomic flexibility (Laborde et al., 2018). This model's emphasis on rest, reactivity, and recovery phases aligns with the objectives of this thesis, which seeks to understand stress response patterns and resilience in chronically stressed women during these phases. Based on Vagal Tank Theory, this thesis aims to assess the efficiency of autonomic adaptation during reactivity and recovery and identify factors that may enhance or hinder stress adaptation, particularly among vulnerable groups, such as women suffering from chronic stress.

### 1.2 Chronic Stress

*Chronic stress* is characterized by an enduring presence of stressors that elicit prolonged activation of the stress response system (Kim et al., 2018; Móra et al., 2022). Shonkoff et al. (2009) categorized stress into three types. The first, *positive stress*, involves moderate stress responses that are brief, well-regulated, and resolve appropriately. The second, *tolerable stress*, encompasses potentially harmful stress responses that are mitigated by adequate support systems and can even

strengthen resilience to future challenges. The third, *toxic stress*, refers to harmful stress responses, where internal and external coping resources are insufficient (Juster & Misiak, 2023). Thus, unlike acute stress, which is typically short-lived and can be beneficial by preparing the body to face immediate challenges, chronic stress occurs when stressors persist over an extended period, leading to detrimental effects on health.

Chronic stress primarily affects the body through the sustained activation of the SNS and the *hypothalamic-pituitary-adrenal (HPA) axis*. This persistent activation results in the continuous release of stress hormones such as cortisol and adrenaline, which, over time, can lead to a myriad of health problems including cardiovascular disease, immune dysfunction, anxiety and depression (McEwen, 1998; Halko & Sääksvuori, 2017). Chronic stress disrupts HRV patterns and is associated with reduced overall HRV, indicating a diminished autonomic nervous system flexibility and poor cardiovascular health (Lampert et al., 2016; Halko & Sääksvuori, 2017). The mechanisms through which chronic stress exerts its effects involve both direct and indirect pathways. Directly, the overactivation of the SNS and HPA axis alters physiological functions, leading to elevated blood pressure, increased heart rate, and reduced HRV (Lampert et al., 2016; Halko & Sääksvuori, 2017). Indirectly, chronic stress influences behavior, prompting unhealthy habits such as poor diet, physical inactivity, and substance abuse, which further degrade health (Almeida, 2005; Lampert, 2016).

The Neurovisceral Integration Model posits that stressors are interpreted based on perceptions of threat and safety, with HRV serving as both a functional and structural marker of these processes (Thayer et al., 2011). Evolutionarily, the default response to uncertainty, novelty, or perceived threats is a sympathoexcitatory state, commonly referred to as the "fight-or-flight" response. This response, rooted in a phenomenon known as the *negativity bias*, prioritizes negative information over positive to maximize survival by erring on the side of caution (Thayer et al., 2009, 2011). While adaptive in evolutionary contexts, this heightened sensitivity to threat becomes maladaptive in modern daily life, where continual threat perception is linked to dysregulation in hippocampal circuits, endocrine and autonomic systems, and declines in cognitive and overall health (McEwen, 2007; Thayer et al., 2011). When this stress response is involuntarily prolonged in the absence of actual threats, it transforms into a taxing and maladaptive condition often referred to as *hyperarousal*. Hyperarousal is characterized by autonomic hyper-responsivity, with individuals displaying elevated baseline heart rate and exaggerated stress reactivity (Bonne et al., 2004).

Chronic stress fosters a maladaptive cycle in which stress reactivity becomes progressively heightened, reducing an individual’s ability to effectively manage new stressors. This diminished capacity is explained by allostatic load models, which describe the cumulative burden of chronic stress as leading to significant physiological wear and tear on the body (McEwen, 1998). *Allostasis*, the process by which the HPA axis and ANS regulate adaptation to stress, becomes maladaptive

when these systems are persistently overactive or fail to deactivate properly. This maladaptation, the multi-systemic strain attributable to chronic stress (e.g., toxic stress), is referred to *allostatic load* (Juster & Misiak, 2023; McEwen, 1998). While allostatic load models focus on the cumulative physiological strain from chronic stress, Vagal Tank Theory (Laborde et al., 2018) provides a dynamic perspective on how autonomic resources are depleted and replenished in response to stress. Together, these models provide a comprehensive framework to understand how chronic stress can lead to autonomic dysregulation and diminished resilience.

Allostatic load arises when stress-response systems are either overworked or fail to respond adequately, leading to compensatory overactivation in other systems and contributing to the development of pathophysiology (McEwen, 1998). Allostatic load can manifest in three primary patterns of physiological dysregulation: frequent activation due to repeated stress (Type 1), prolonged activity without proper shutdown (Type 2), or insufficient activation in response to a challenge (Type 3). These imbalances highlight how systems that typically facilitate adaptation can, when dysregulated, exacerbate stress-related health risks by either failing to shut down after a stressor or underreacting, thereby creating vulnerabilities in other physiological systems. However, research has shown that chronically stressed individuals do not necessarily show exaggerated responses to threat as much as they show threat responses to neutral or harmless stimuli (Thayer et al., 2011), indicating that this demographic may exhibit stress responses beyond the three proposed types in the original allostatic load model. Despite it not yet being common practice to integrate HRV measurements into allostatic load models, their potential to provide valuable insights into autonomic dysregulation associated with chronic stress is increasingly recognized; high allostatic load has been linked to decreased HRV (Viljoen & Claassen, 2017) and elevated HRV despite chronic stress has been proposed to foster adaptive behavior and contribute to lower levels of allostatic load (Jiryis et al., 2022).

### *1.2.1 Chronic Stress and HRV*

As described in the Vagal Tank Theory, stress responses can be understood through three temporal phases: rest, reactivity, and recovery. These phases provide a framework for interpreting autonomic dynamics under stress. Examining how individuals react to and recover from stressful events is crucial to understanding how stress becomes harmful; however, research on HRV reactivity and recovery patterns in chronically stressed individuals remains limited (Bamert & Inauen, 2022). Table 1 consolidates theoretical predictions and empirical findings, summarizing expected SDNN patterns across stress phases in healthy and chronically stressed individuals. At rest, healthy individuals typically exhibit high SDNN, reflecting balanced autonomic regulation dominated by parasympathetic activity. In contrast, chronically stressed individuals are likely to have lower SDNN in

the rest phase due to reduced parasympathetic tone and sustained sympathetic activity. This lower resting SDNN indicates impaired vagal tone and autonomic rigidity, consistent with an inability to fully engage the parasympathetic "rest-and-digest" state (type 2 allostatic load). During the reactivity phase, healthy individuals are expected to show a decrease in SDNN, reflecting vagal withdrawal and sympathetic activation for an adaptive stress response. In chronically stressed individuals, SDNN may show blunted reactivity (a smaller decrease) due to chronically elevated baseline sympathetic activity, leaving little capacity for further increase. Alternatively, SDNN may exhibit exaggerated reactivity (a larger decrease) due to a hyperactive sympathetic response to stressors. Blunted reactivity reflects autonomic inflexibility and diminished adaptive capacity (type 3 allostatic load), while exaggerated reactivity aligns with heightened stress sensitivity and poor autonomic control, as described by the hyperarousal concept. In the recovery phase, SDNN in healthy individuals typically increases as parasympathetic activity restores autonomic balance, replenishing the vagal tank. However, in chronically stressed individuals, recovery is often delayed or incomplete, with SDNN remaining low or showing minimal improvement after the stressor. This impaired recovery indicates persistent autonomic dysfunction and highlights chronic stress's detrimental effect on autonomic flexibility and resilience.

**Table 1**

*Summary of SDNN Expectations for Chronic Stress*

| Phase | Healthy Individuals | Chronically Stressed Individuals |
|---|---|---|
| Rest | Higher SDNN (autonomic balance) | Lower SDNN (reduced vagal tone, SNS dominance) |
| Reactivity | Moderate SDNN (adaptive response) | Lower SDNN due to blunted or exaggerated stress responses (autonomic inflexibility) |
| Recovery | Higher SDNN (gradual increase reflecting autonomic balance restoration) | Lower SDNN due to delayed or incomplete increase (prolonged dysregulation) |

*Note:* SDNN = Standard Deviation of Normal-to-Normal Intervals.

To summarize, in the context of Vagal Tank Theory, chronically stressed individuals are expected to display a partially "empty tank" at rest, reflected in low SDNN due to reduced parasympathetic tone. During stress, they may exhibit a limited capacity to further deplete or adapt, resulting in either blunted reactivity (minimal change in SDNN due to chronically heightened sympathetic activity) or excessive depletion (exaggerated SDNN reduction from hyperactive sympathetic responses). In the recovery phase, their SDNN often remains low or shows minimal improvement, indicating delayed or insufficient parasympathetic reactivation and persistent

autonomic dysfunction. These patterns highlight the impaired autonomic flexibility and resilience associated with chronic stress (Da Estrela et al., 2020; Escorihuela et al., 2020; Lutin et al., 2022).

Although the connection between chronic stress and reduced HRV is well-established (Lampert et al., 2016; Halko & Sääksvuori, 2017), findings on HRV reactivity and recovery in chronically stressed individuals remain inconsistent. While most studies support the theory of exaggerated stress responses in this population (Bonne et al., 2004; Schubert et al., 2008), some align with type 2 allostatic load models, where stress responses remain chronically active rather than inhibited (Held et al., 2021; Kim et al., 2018). In contrast, blunted stress reactivity has been observed in individuals with chronic stress and co-occurring depressive symptoms (Lutin et al., 2022). To date, little research has focused specifically on HRV reactivity and recovery patterns in high chronically stressed versus low chronically stressed individuals in response to psychosocial stress tasks. This thesis seeks to address this gap by analyzing potential intergroup differences in HRV stress reactivity and recovery to acute stress. For the purposes of this study, the working assumption is that chronically stressed individuals will exhibit consistently lower SDNN across all three phases – rest, reactivity, and recovery – compared to the non-chronically stressed sample. As emerging evidence highlights, women represent a particularly vulnerable subgroup to the effects of chronic stress. The next section delves deeper into this gender-sensitive perspective.

### *1.2.2 Chronic Stress in Women*

Women have been identified as particularly vulnerable to the effects of chronic stress, exhibiting higher stress levels and lower HRV compared to men (Almeida, 2005; Brugnera et al., 2018; Lampert et al., 2016). This gender-specific susceptibility stems from a combination of biological, psychological, and social factors, including hormonal variations, societal roles, and differing coping mechanisms (Dalla et al., 2005; Lampert et al., 2016). Reduced HRV in women is associated with adverse health outcomes such as increased cardiovascular risk and impaired emotional regulation (Halko & Sääksvuori, 2017). Still, gender differences in the study of physiological stress responses are often erroneously overlooked (Adjei et al, 2018).

Notably, mental stress produces distinct patterns of autonomic regulation in men and women. For example, mental stress has been shown to increase low-frequency (LF) HRV power in men, reflecting heightened sympathetic dominance, while decreasing it in women. Similarly, while high-frequency (HF) HRV power decreases significantly in men under stress, indicative of reduced parasympathetic activity, it has been observed to increase in women, suggesting greater parasympathetic engagement. These findings align with an observed increase in signal variability in women's HRV during stress, contrasting with the reduced variability seen in men (Adjei et al., 2018). Together, these findings underscore the importance of accounting for gender differences in

autonomic responses to stress, as women's physiological strategies may involve increased HRV complexity and parasympathetic activity, potentially linked to adaptive coping mechanisms.

Women are disproportionately burdened by caregiving responsibilities, financial stress, and work-related stressors, which are critical contributors to their higher allostatic load. These stressors often stem from societal roles and expectations, making women particularly vulnerable to cumulative stress (Swinkels et al., 2017). Chronic stress in women is compounded by the intersection of biological factors and socio-cultural expectations, which amplify their vulnerability to adverse outcomes. Despite these vulnerabilities, evidence suggests that women may exhibit unique adaptive responses to acute stress following chronic stress exposure, such as enhanced serotonergic activity and active coping behaviors (Dalla et al., 2005). These adaptive responses offer insight into resilience mechanisms specific to women, which merit further exploration.

Traditional models of stress, such as Cannon's (1932) fight-or-flight paradigm, have historically shaped our understanding of stress responses. However, this model may be inadequate in capturing gender-specific patterns of behavior. Taylor et al. (2000) proposed the "tend-and-befriend" framework as a gender-sensitive alternative, emphasizing biobehavioral responses rooted in caregiving and social affiliation. This theory suggests that women, under stress, are more likely to engage in nurturing behaviors (tending) and seek out social support (befriending), with oxytocin playing a central role in modulating these responses. Unlike the fight-or-flight response, which prioritizes self-preservation, the tend-and-befriend mechanism fosters social bonding and cooperation, which may serve as an adaptive strategy in managing stress (Taylor et al., 2000; Turton & Campbell, 2005).

Emerging evidence underscores sex differences in biobehavioral stress responses, particularly in the role of oxytocin and the modulation of the HPA axis and sympathetic nervous system activity (Hamilton & Russo, 2006; Keyes et al., 2006). These differences may contribute to women's heightened vulnerability to stressors related to family, work, and social relationships, which align with the tend-and-befriend model (Maciejewski et al., 2001; Taylor et al., 2002). Social context plays a critical role, with certain stressors, such as the loss of social support, disproportionately affecting women (Keyes et al., 2006; Taylor et al., 2002).

This nuanced interplay between stressor type and gender highlights the importance of considering psychosocial factors when studying stress-related outcomes. Gender differences in stress coping mechanisms are evident. Women often engage in emotion-focused coping (e.g., seeking social support) compared to men, who may use problem-focused strategies. However, the effectiveness of these coping styles in mitigating physiological stress remains mixed (Gallo et al., 2010).

While women may display higher allostatic load, certain protective psychosocial factors, such as strong social support networks, can buffer the adverse effects of chronic stress (Gallo et al., 2010).

Conversely, vital exhaustion and fatigue have been implicated as risk factors for negative cardiovascular events and chronic stress, especially in women (Jain & Mills, 2007; Rimes et al., 2017). Interestingly, despite a strong tendency to seek social support, some women under chronic stress report increased irritability and verbal aggression, behaviors seemingly counterproductive to receiving support (Turton & Campbell, 2005). These findings suggest complex interindividual variations in stress responses, influenced by behavioral and contextual factors that deserve further investigation.

Given the heightened vulnerability of women to the physiological effects of chronic stress, understanding the psychosocial factors that exacerbate or buffer these effects is critical. There are a myriad of factors that compose interindividual variance in stress responses. This thesis will take a deeper look at two psychosocial variables that have been identified as particularly relevant to women (Keyes et al., 2006). Chronic fatigue, a significant risk factor, and perceived social support, a potential protective factor, are explored as key determinants of vulnerability and resilience in the context of chronic stress.

### 1.3 Psychosocial Risk and Protective Factors

Psychosocial factors play a crucial role in influencing an individual's response to stress, impacting both physiological and psychological health. These factors can act as either risk factors, exacerbating the effects of stress, or protective factors, mitigating these effects and enhancing resilience.

#### *1.3.1 Chronic Fatigue*

*Chronic fatigue* (CF) is a significant psychosocial risk factor of unknown aetiology that often co-occurs with chronic stress, contributing to a cycle of stress, impaired recovery, and diminished resilience (Escorihuela et al., 2020). Characterized by extreme, persistent tiredness not relieved by rest, CF is linked with various physiological changes, including disrupted sleep patterns, hormonal imbalances, and impaired immune function. Importantly, CF is associated with altered ANS functioning, evidenced by decreased HRV, reduced parasympathetic activity, and heightened sympathetic dominance (Rimes et al., 2017; Strahler et al., 2016).

Under acute conditions, fatigue serves as an adaptive mechanism, signaling the body to rest and recover from overextension. However, when fatigue becomes chronic (lasting six months or longer) it transitions into a maladaptive state, adversely affecting physical, mental, and social functioning (Klimas et al., 2012). While severe cases of persistent fatigue are classified as *chronic fatigue syndrome* (CFS), many individuals with prolonged fatigue fall outside the diagnostic criteria for CFS and experience a less severe but still debilitating condition known simply as chronic fatigue

(Leven et al., 2009). Women are disproportionately affected, being two to four times more likely than men to experience CF (Kocalevent et al., 2011; *OASH - Office On Women's Health,* 2024).

CF is closely linked to autonomic dysfunction, with symptoms often including exaggerated stress reactivity or a blunted response (Bosquet et al., 2008). Chronically fatigued women often display sympathetic hyperactivation or parasympathetic dysfunction, resulting in an inability to effectively respond to stressors and perpetuating fatigue symptoms (Ali et al., 2024; Meeus et al., 2013).

While there is evidence linking CF to dysfunctional stress responses (Ali et al., 2024; Kocalevent et al., 2011), there are some discrepancies in the literature about the behavior of the HRV variables in CF individuals (Escorihuela et al., 2020). People with CF have been described as having "stress intolerance", characterized by vulnerability to chronic stress, difficulty coping with acute stress, and slower recovery following stress exposure (Rimes et al., 2017). This aligns with research associating CF with lower baseline HRV, with fatigue symptoms often negatively correlating with all HRV indices (Escorihuela et al., 2020). However, some studies found this association only for some HRV measurement variables or only under certain conditions (Boneva et al., 2007; Kocalevent et al., 2011; Yataco et al., 1997). For example, in one study, CF patients' HRV was reduced only during sleep (Meeus et al., 2013). These findings highlight the need for further investigation into the relationship between CF, HRV, and stress responses (Kocalevent et al., 2011).

Importantly, while most studies frame chronic stress as a risk factor for fatigue, where prolonged stress depletes physiological and psychological resources, leading to exhaustion, there is value in exploring the reverse relationship. In this reversed model, chronic fatigue may also act as a precursor to chronic stress. CF-associated ANS dysfunction, such as reduced HRV and sympathetic dominance, might be a driver of heightened stress sensitivity and prolonged recovery, suggesting that CF directly primes the body for maladaptive stress responses and consequently may shape the physiological response trajectory during stress phases. Specifically, CF may influence how HRV changes during reactivity (e.g., a less pronounced decrease or an exaggerated drop) and recovery (e.g., delayed return to baseline). This model implies that intervening at the fatigue stage, such as improving sleep or addressing ANS imbalances, could prevent the onset or worsening of chronic stress. Since women are more likely to experience CF, modeling CF as a risk factor may partially explain their greater susceptibility to chronic stress. Furthermore, this perspective underscores the importance of addressing societal and structural stressors, such as caregiving burdens and work-life imbalance, that disproportionately affect women.

Given the heightened prevalence of CF in women and its connection to autonomic dysfunction, this thesis aims to investigate whether chronic fatigue moderates stress reactivity and recovery in chronically stressed women, as measured by HRV SDNN. By examining this interaction,

the study seeks to clarify how CF impacts resilience and recovery in the context of chronic stress. Finally, while CF increases vulnerability to stress, protective factors such as perceived social support may counterbalance these effects. The next section explores the potential moderating role of perceived social support in stress reactivity and recovery among chronically stressed women.

### *1.3.2 Perceived Social Support*

Perceived social support emerges as a key protective factor within the realm of psychosocial influences on health (Kothgassner et al., 2018; Taylor, 2012). Defined as an individual's belief in the availability of supportive resources, perceived social support reflects a sense of security and satisfaction with one's social environment and plays a critical role in stress management and overall well-being (Cohen & Wills, 1985; Cummins, 1988). While it is widely agreed that social support benefits individuals, there is ongoing debate regarding the mechanisms through which it exerts these effects.

The *main effect model* posits that social support directly enhances health, irrespective of stress levels by fostering mental well-being, physical health, adherence to medical treatments, and a sense of stability and self-worth. Regular positive social interactions, for instance, can elevate mood, reduce blood pressure, and strengthen the immune system, promoting overall well-being (Cohen & Wills, 1985; Laquidara & Lincoln, 2023). A generalized beneficial effect may occur because being integrated into a social network provides regular positive experiences, predictability, and a sense of belonging, while also helping individuals avoid negative experiences such as economic or legal problems (Cohen & Wills, 1985).

On the other hand, the *stress-buffering hypothesis* suggests that social support's primary benefits emerge during periods of heightened stress (Cohen & Wills, 1985; Laquidara & Lincoln, 2023). In this model, social support provides emotional comfort, practical assistance, and advice that can alter the perception of stressors, making them appear less threatening or more manageable. This buffering effect has been linked to physiological benefits, including a lower heart rate, reduced cortisol levels, and improved HRV, reflecting balanced autonomic function during stress (Cohen & Wills, 1985; Kao et al., 2013; Laquidara & Lincoln, 2023).

The distinction between perceived support and *received support* is critical for understanding the nuances of the stress-buffering hypothesis. Perceived support refers to an individual’s belief in the availability of supportive interpersonal resources, while received support denotes the actual provision of assistance during stressful times (Cummins, 1988; Kothgassner et al., 2018). Perceived support has been associated with increased coping competence, while received support may play a role when personal coping efforts are insufficient (Wethington & Kessler, 1986, as cited in Cohen & Wills, 1985). In the context of chronic stress, perceived support is especially relevant. Unlike received

support, which depends on external availability and situational demands, perceived support reflects an individual's enduring sense of connection and security. By fostering greater parasympathetic activity and reducing sympathetic dominance, perceived support has the potential to buffer the physiological dysregulation associated with chronic stress (Cohen & Wills, 1985; Ditzen & Heinrichs, 2014).

Social support can also be categorized into *emotional support*, which meets psychological needs through empathy, encouragement, and understanding, and *instrumental support*, which involves practical help or tangible resources (Cohen & Wills, 1985; Ditzen & Heinrichs, 2014). Emotional support in particular has been linked to enhanced HRV and other markers of autonomic balance during stress, aligning closely with the stress-buffering hypothesis (Hartley & Coffee, 2019; Kothgassner et al., 2018).

Perceived social support reflects an enduring expectation of support availability, making it relatively stable over time and thus a trait (Cohen & Wills, 1985; Kothgassner et al., 2018), that can act as a protective factor in the context of autonomic stress responses. Perceived availability of support has been shown to buffer the negative effects of high stress, suggesting that higher levels of perceived support may foster functional adaptation to heightened stress (Hartley & Coffee, 2019). For instance, participants receiving social support prior to exposure to the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), showed improved cardiovascular stress reactivity (Kothgassner et al., 2018).

As the tend-and-befriend theory (Taylor et al., 2000) suggests, social support plays a significant role as a gender-sensitive coping mechanism. Women may be particularly inclined to seek and benefit from social support as part of their adaptive coping strategies under stress, leveraging oxytocin-mediated pathways to dampen stress reactivity and promote recovery, which could mitigate the adverse physiological effects of chronic stress and fatigue (Keyes et al., 2006). Testing social support as a moderator provides a framework to explore how this coping mechanism translates into measurable HRV outcomes during stress phases.

As its final research question this thesis posits that perceived social support moderates HRV reactivity and recovery when controlling for chronic fatigue. By focusing on perceived social support, the study aligns with the stress-buffering hypothesis and seeks to illuminate how social connections may foster resilience in chronically stressed women. The next section outlines the specific research questions and hypotheses driving this investigation, with a particular emphasis on the interplay between chronic fatigue, social support, and HRV outcomes.

## 2. Research Questions and Hypotheses

The preceding discussion highlights the impact of chronic stress on HRV stress response patterns as well as the dual role of psychosocial factors in shaping these stress responses: chronic

fatigue amplifies autonomic dysregulation, while perceived social support may buffer its effects. Building on these insights, this study explores the following research questions and hypotheses.

Research Question 1 (RQ1): Does the level of chronic stress explain interindividual differences in patterns of HRV reactivity and recovery?

Hypothesis 1.1 (H1.1): Women with higher levels of chronic stress will show significantly lower HRV *reactivity* to acute stress compared to women with lower levels of chronic stress.

Hypothesis 1.2 (H1.2): Women with higher levels of chronic stress will show significantly lower HRV *recovery* after acute stress compared to women with lower levels of chronic stress.

These hypotheses are based on the premise that chronic stress, by persistently activating the sympathetic nervous system, impairs the body's ability to both react and recover flexibly to and from stressors, thereby reducing HRV as a measure of this impaired autonomic function.

Research Question 2 (RQ2): Does the concomitance of chronic fatigue alter (increase or decrease) HRV reactivity and recovery in women with chronic stress?

Hypothesis 2.1 (H2.1): Chronic fatigue moderates the relationship between the level of chronic stress and HRV *reactivity* to acute stress. Higher levels of chronic fatigue will be associated with higher or lower HRV reactivity in the context of chronic stress.

Hypothesis 2.2 (H2.2): Chronic fatigue moderates the relationship between the level of chronic stress and HRV *recovery* after acute stress. Higher levels of chronic fatigue will be associated with higher or lower HRV recovery in the context of chronic stress.

These hypotheses acknowledge that chronic fatigue may exaggerate or blunt the effects of chronic stress on the autonomic nervous system, complicating the body's response patterns to stress.

Research Question 3 (RQ3): When controlling for chronic fatigue, do higher scores in perceived social support lead to increased HRV reactivity and recovery in individuals with chronic stress?

Hypothesis 3.1 (H3.1): Perceived social support moderates the relationship between chronic stress and HRV *reactivity* to acute stress when controlling for chronic fatigue. High levels of perceived social support will be associated with higher HRV reactivity to acute stress in the context of chronic stress.

Hypothesis 3.2 (H3.2): Perceived social support moderates the relationship between chronic stress and HRV *recovery* after acute stress when controlling for chronic fatigue. High levels of perceived social support will be associated with higher HRV recovery after acute stress in the context of chronic stress.

These hypotheses propose that social support can provide a buffering effect against the negative impacts of chronic stress on the autonomic nervous system. By controlling for chronic fatigue, the study aims to isolate the effect of social support, ensuring any observed buffering effect is not confounded by the influence of fatigue.

These research questions and hypotheses aim to dissect the multifaceted influences of psychosocial factors on the physiological processes underlying stress responses. By disentangling the influences of chronic fatigue and social support on HRV, this research seeks to identify modifiable factors that could inform interventions for enhancing resilience and well-being in chronically stressed women and to contribute valuable insights into the mechanisms by which chronic stress, chronic fatigue, and social support interact to affect HRV.

## 3. Method

### 3.1 Participants

The study included $N$ = 31 female, German-speaking participants aged 20-65 years ($M$ = 40.2, $SD$ = 14) who met specific inclusion criteria. These criteria were: not being pregnant or breastfeeding, having a BMI < 30, not taking medications or undergoing medical treatments with neuroendocrine, autonomic, or immunological effects, and not using psychotropic drugs (or having discontinued them at least 14 days before the study). Participants also needed to have a regular menstrual cycle, and their chronic fatigue symptoms could not be attributed to an underlying somatic disease. Further criteria excluded those with a severe depressive episode or recurrent depression, an addiction disorder within the last two years, an eating disorder within the last five years, bipolar disorder, psychosis, or social phobia.

Personal data were collected from participants during the study. All data were handled in strict compliance with medical confidentiality and legal data protection regulations. Only information necessary for achieving the study's objectives was collected, including participants' first and last names, date of birth, address, medical findings, prescribed medications, and questionnaire responses. The scientific data were stored and analyzed in a pseudonymized format (i.e., without direct links to participants' names) and were accessible only to study-related professionals in a coded form for scientific evaluation.

**3.2 Study Design and Methods**

The study was conducted as an empirical cross-sectional investigation, spanning the period between May 2013 and March 2018 at the Clinical Biopsychology Laboratory at Philipps University Marburg. The study procedure was standardized to ensure consistent conditions across all participants, involving a phone interview, a screening, and a controlled laboratory setting to simulate a stress-inducing situation. Participants were compensated with up to 35€ for their time and 50€ to cover travel expenses.

On the day of the session, participants arrived at the laboratory at 11:30 a.m., where, at 12:00 p.m., an ECG heart rate monitor was fitted around their chest to track heart rate throughout the session. Since HRV can be affected by numerous factors including the intake of specific substances like alcohol, nicotine, and caffeine, the rate of breathing or changes in body posture (Bamert & Inauen, 2022), participants were advised to avoid behaviors that might impact physiological measurements prior to the laboratory session: they were to avoid strenuous physical activity for two days before the laboratory session, refrain from sports on the day before testing, and abstain from caffeine and alcohol on the evening prior. Participants were moved around in rolling office chairs to avoid movement artifacts in the data.

To induce a standardized stress response, the Trier Social Stress Test (TSST; Kirschbaum et al., 1993) was administered. The TSST is a widely used method in psychological research for inducing and measuring stress in a controlled setting. It is designed to elicit a psychological and physiological stress response by placing participants in a socially evaluative situation. During the TSST, participants undergo a series of tasks that involve social judgment and performance pressure, which are known triggers for stress (Kudielka et al., 2007): The TSST procedure begins with a preparation phase, where participants enter the test room and are given around 5 minutes to prepare a mock job interview or a short speech on a specific topic. They are informed that they will soon have to deliver this speech in front of a panel of judges, which builds a sense of anticipation and mild anxiety, effectively priming them for the tasks that follow. Next, in the speech task, participants present their speech to an evaluating panel of two or three individuals. The panel remains neutral and provides minimal feedback, maintaining a serious demeanor throughout. This lack of response adds a layer of social evaluative threat, as participants often interpret the panel's neutrality as judgment or disapproval, enhancing the psychological pressure. This speech task lasts five minutes. Following the speech, participants immediately proceed to the mental arithmetic task, where they are asked to complete a challenging calculation, such as counting backward in steps of 13 from a large number. Any mistake requires the participant to restart the calculation, adding to the stress of needing to perform accurately under scrutiny. This task continues for another five minutes, cumulatively creating a socially evaluative and cognitively demanding situation designed to reliably trigger stress responses.

Throughout the session, which lasted approximately 3.5 hours, participants completed several questionnaires to capture relevant contextual information, among which three are relevant for the purposes of this thesis. Before the lab session, participants completed a questionnaire on perceived social support. During the lab session, two questionnaires were administered, assessing chronic stress and chronic fatigue levels. Following the TSST, participants were given a recovery period, allowing for measurement of physiological recovery. This procedure enabled a comprehensive analysis of participants' baseline HRV, reactivity to the TSST stressor, and subsequent recovery.

### 3.3 Measured Variables/Assessment Instruments

#### *3.3.1 Heart Rate Variability*

Heart Rate Variability (HRV) was measured using an ambulatory ECG monitoring system (Equivital™ Life Monitoring Sensor System, Hidalgo Ltd.). The standard deviation of normal-to-normal intervals (SDNN) as the most typically studied HRV time domain measure in the context of stress (Jiryis et al., 2022) was calculated from recordings. HRV reactivity (dependent continuous variable) was analyzed by examining the mean HRV SDNN during the 10min TSST stress period. HRV recovery (dependent continuous variable) was operationalized as the mean HRV SDNN from the first 15min of the recovery period.

#### *3.3.2 Chronic Stress*

Chronic Stress (independent categorical variable) was measured by the *Short Screening Scale for Chronic Stress* (SSCS; Schulz et al., 2004), which derives from the Trier Inventory for Chronic Stress (TICS; Schulz et al., 2004). The SSCS was administered electronically via *Unipark* (ww3.unipark.de) during laboratory testing. It assesses five interrelated factors of chronic stress (*Chronic Worry*, *Work Overload*, *Excessive Demands from Work*, *Lack of Social Recognition*, and *Social Overload*) that are derived from 12 items rated on a five-point rating scale (labeled as: "*never*" (0), "*rarely*" (1), "*sometimes*" (2), "*frequently*" (3), "*always*" (4)). Participants rate the occurrence of specific situations with a recall period of the previous three months to assess their degree of self-perceived chronic stress. An example item is "times when I worry a lot and cannot stop". A total score is calculated by summing all item scores, with scores ranging from 0 to 48; higher values indicate greater stress. The TICS-SSCS sum-score of the 12 items was linearly transformed into standard T-values ($M$ = 50, $SD$ = 10). Based on a cutoff score of $T$ = 61 ("above average"), participants with a total SSCS score of ≥ 25 were classified as experiencing "high chronic stress" while those with scores < 25 were classified as "low chronic stress".

Confirmatory factor analysis was conducted to evaluate the factor structure of the SSCS. A five-factor model representing the hypothesized subscales was tested using maximum likelihood estimation. The model demonstrated an acceptable fit ($\chi^2$ (45) = 81.3, $p < .001$, *RMSEA* = .180, 95% CI [0.115, 0.241], *SRMR* = .086, *CFI* = .788, *TLI* = .688) All factor loadings were statistically significant ($p < .05$), ranging from .67 to .91, indicating adequate construct validity of the SSCS. The SSCS demonstrated good internal consistency in the current sample, with a Cronbach's Alpha of .86 (95% CI [.79, .93]). Corrected item-total correlations ranged from .43 to .77, indicating satisfactory item contributions to the overall scale. Removal of any individual item did not significantly improve the reliability of the scale, confirming the robustness of the SSCS for assessing chronic stress.

### *3.3.3 Chronic Fatigue*

Chronic Fatigue (independent continuous variable) was measured by the German version of the *Multidimensional Fatigue Inventory* (MFI; Smets, 1995), also administered electronically via Unipark during laboratory testing. This instrument consists of 20 self-report items and encompasses five dimensions (*General Fatigue, Physical Fatigue, Mental Fatigue, Reduced Motivation* and *Reduced Activity*). Participants rate how accurately certain statements about fatigue reflect their experiences on a scale from 1 ("*yes, that is true*") to 5 ("*no, that is not true*"). Several positively worded items are reverse scored. Higher overall scores indicate higher levels of fatigue (Shahid et al., 2011). Participants are categorized as fatigued when their MFI score exceeds 16. An example item is "I tire easily". The instrument demonstrates strong internal consistency ($\alpha = .84$). The validity of the MFI was previously established, indicating that the five-factor model is supported, and through evidence of construct and convergent validity (Smets, 1995).

A confirmatory factor analysis was conducted to evaluate the five-factor structure of the MFI in the current sample. The model demonstrated poor fit ($\chi^2(160) = 366$, $p < .001$, *RMSEA* = .227, 95% CI [0.196, 0.258], *SRMR* = .156, *CFI* = .472, *TLI* = .373). Despite the poor overall model fit, most items loaded significantly onto their respective factors, though some showed weaker loadings. These results likely reflect sample-specific variations but do not undermine the instrument's established validity. The MFI demonstrated excellent internal consistency in the current sample ($\alpha = .91$, 95% CI [.86, .95]). Corrected item-total correlations ranged from .38 to .78, indicating strong item contributions to the overall scale reliability. Removal of any individual item did not significantly improve the overall Cronbach's Alpha, confirming the robustness of the scale.

### *3.3.4 Perceived Social Support*

Perceived Social Support (independent continuous variable) was measured by the perceived support subscale of the *Berlin Social Support Scales* (BSSS; Schulz & Schwarzer, 2003), which

participants completed in a paper-and-pencil format prior to the laboratory testing. Participants rate their agreement with the statements on a four-point scale (*strongly disagree* (1), *somewhat disagree* (2), *somewhat agree* (3) and *strongly agree* (4)). The perceived support subscale comprises eight items on two scales (*emotional support* and *instrumental support*), reflecting participants' subjective perceptions of the availability and quality of their support network. An example item of the BSSS perceived support subscale is "There is always someone there for me when I need comforting".

Confirmatory factor analysis and reliability analysis were conducted on the study sample. The hypothesized two-factor structure was tested using maximum likelihood estimation and demonstrated acceptable fit ($\chi^2$ (19) = 26.6, $p$ = .115, *RMSEA* = 0.108, 95% CI [.000, .198], $p$ = .177, *SRMR* = .055, *CFI* = .958, *TLI* = .938). All factor loadings were statistically significant ($p$ < .001), ranging from .586 to .918 for Factor 1 (emotional support) and .659 to .886 for Factor 2 (instrumental support), supporting the construct validity of the scale. Reliability analysis showed good internal consistency for both subscales. The emotional support subscale demonstrated a Cronbach's Alpha of $\alpha$ = .85 (95% CI [0.77, 0.92]) with corrected item-total correlations ranging from 0.65 to 0.82, and no improvement in reliability if items were dropped. The instrumental support subscale showed similar reliability ($\alpha$ = .86, 95% CI [0.79, 0.93]), with corrected item-total correlations ranging from .74 to .87. These results, consistent with those reported by Schulz & Schwarzer (2003), confirm the internal consistency and construct validity of the perceived social support scale within the current sample.

### 3.4 Data Processing and Analysis

#### *3.4.1 Data Processing*

To process the data, batch processing in *Vivonetics VivoSense* HRV software was used to extract ECG and RR intervals from raw Equivital recordings for the baseline, TSST, and recovery periods. These periods were divided into non-overlapping 5-minute segments. The HRV data were then analyzed using *Kubios* HRV software, with an ECG sampling rate of 256Hz (the standard setting for Equivital devices). Since physiological recordings can be affected by motion artifacts and poor sensor attachment, Kubios settings were configured with Automatic Noise Detection set to "Medium" and Beat Correction set to "Automatic Correction", using 5-minute sample lengths to align with the previously defined segments. SDNN values (in ms) were extracted.

Occasionally, Kubios displayed an error message indicating "HRV data quality is very low, automatic artifact correction cannot be performed". In such cases, the beat correction rate was manually reviewed. Data were excluded from analysis if the correction rate exceeded 5%, as such recordings were deemed unreliable. This procedure was applied consistently across all participants and time periods of interest, including baseline, TSST, and recovery intervals.

To assess reactivity, two 5min segments from the TSST period were analyzed. For recovery, the first three 5min segments of the recovery phase were considered. Out of 90 initially recruited participants, 56 were excluded because either no HRV data were recorded or the HRV data during both the TSST and recovery periods were of insufficient quality, with beat correction rates exceeding 5% for all five 5-minute segments. This data loss stemmed from challenges commonly associated with HRV data collection, including poor signal quality caused by improper sensor attachment, or interference from excessive motion despite precautionary measures. This left 34 participants in the final analysis, of whom 29 had utilizable data for both the TSST reactivity and recovery periods. Four participants had low-quality data for both TSST segments, and one participant's recovery data were excluded due to consistently poor data quality across all three segments. SSCS questionnaire data necessary for group assignment to low and high chronic stress groups was missing for four people, leaving 30 women in the final analysis.

Given these limitations, HRV reactivity and recovery were operationalized as the average SDNN during the respective periods, rather than calculating SDNN differences from baseline to the stressor (for reactivity) or from the stressor to the subsequent resting phase (for recovery), as is commonly done in the literature (Hamilton & Alloy, 2016). This approach allowed for the inclusion of more participants, even when reliable data were missing for some periods. In cases where only one of the TSST segments or one or two of the recovery segments had acceptable data, the average SDNN was calculated from the available high-quality data.

### *3.4.2 Statistical Analysis*

Analyses were carried out using Jamovi version 2.3.26. The level of significance was defined in accord with the probability of error as $\alpha = .05$. Therefore, results with $p \leq .05$ were defined as significant and results with $p \leq .01$ as highly significant while testing the hypotheses. Outlier inspection was conducted for all variables using boxplots and density plots. The assumption of normality for the dependent variables, HRV reactivity and HRV recovery, was assessed using the Shapiro-Wilk test and Q-Q plots.

**3.4.2.1 Research Question 1.** Research Question 1 examined whether the level of chronic stress explains interindividual differences in patterns of HRV reactivity and recovery. It was hypothesized that women in the high chronic stress group (SSCS score ≥ 25) would exhibit significantly lower HRV stress *reactivity* compared to women in the low chronic stress group (SSCS score < 25) (H1.1). Similarly, women in the high chronic stress group were expected to show significantly lower HRV *recovery* compared to women in the low chronic stress group (H1.2).

Linear regression analysis was conducted to test these hypotheses, with chronic stress (low/high categorical variable) as the independent variable and HRV reactivity (H1.1) and HRV recovery (H1.2) as the dependent variables. A significant negative relationship between high chronic stress and HRV outcomes would support the theory that chronic stress impairs physiological stress responses and recovery by reducing HRV.

**3.4.2.2 Research Question 2.** Research Question 2 aimed to understand if chronic fatigue acts as a moderator in that it exaggerates (heightens) or blunts (diminishes) the effects of chronic stress on HRV reactivity (H2.1) and recovery (H2.2) in women. H2.1 (Chronic fatigue moderates the relationship between the level of chronic stress and HRV *reactivity*. High levels of chronic fatigue will be associated with higher or lower HRV reactivity in the context of chronic stress) and H2.2 (Chronic fatigue moderates the relationship between the level of chronic stress and HRV *recovery.* High levels of chronic fatigue will be associated with higher or lower HRV recovery in the context of chronic stress) were analyzed using a linear regression analysis with an interaction term.

**3.4.2.3 Research Question 3.** Lastly, Research Question 3 proposed perceived social support as another moderator, potentially buffering the effects of chronic stress on HRV reactivity (H3.1) and recovery (H3.2). H3.1 (Perceived social support moderates the relationship between chronic stress and HRV *reactivity* when controlled for chronic fatigue. High levels of perceived social support will be associated with higher HRV reactivity in the context of chronic stress) and H3.2 (Perceived social support moderates the relationship between chronic stress and HRV *recovery* when controlled for chronic fatigue. High levels of perceived social support will be associated with higher HRV recovery in the context of chronic stress) will be analyzed by adding an interaction term to the previously established linear regression model. Additionally, chronic fatigue is controlled for so as to be able to attribute the resulting effect clearly to social support. These hypotheses propose that social support can provide a buffering effect against the negative impacts of chronic stress on the autonomic nervous system. By controlling for chronic fatigue, the study aims to isolate the effect of social support, ensuring any observed buffering effect is not confounded by the influence of fatigue.

## 4. Results

### 4.1 Descriptive Statistics

Participants were divided into two groups based on their chronic stress levels (SSCS score): high chronic stress and low chronic stress. Among the 30 participants (four missing data points), 63.3% (n = 19) reported high chronic stress and 36.7% (n = 11) reported low chronic stress. Tables 2 and 3 summarize descriptive findings for independent (chronic fatigue, perceived social support) and

dependent variables (HRV reactivity and HRV recovery) for the whole sample. Table 4 depicts descriptive findings by participants' level of chronic stress.

**Table 2**

*Descriptive Statistics for Independent Variables*

| Scale (Number of Items) | *N* | *M* | *SD* | *Min* | *Max* | *Mdn* |
|---|---|---|---|---|---|---|
| MFI (20) | 31 | 12 | 2.79 | 6 | 16.4 | 12 |
| BSSS (12) | 34 | 3.16 | .58 | 2.13 | 4 | 3.06 |

*Note:* M = Mean, SD = Standard Deviation, Mdn = Median.

**Table 3**

*Descriptive Statistics for Dependent Variables*

| Variable | *N* | *M* | *SD* | *Min* | *Max* | *Mdn* |
|---|---|---|---|---|---|---|
| HRV Reactivity (ms) | 30 | 37.3 | 12.7 | 9.05 | 60.9 | 36.5 |
| HRV Recovery (ms) | 33 | 45.1 | 19.3 | 15.2 | 84.4 | 44.4 |

*Note:* HRV = Heart Rate Variability.

**Table 4**

*Descriptive Statistics by Chronic Stress Level*

| Variable | *M* | | *SD* | | *Mdn* | |
|---|---|---|---|---|---|---|
| | low CS | high CS | low CS | high CS | low CS | high CS |
| MFI | 9.89 | 13.2 | 2.63 | 2.13 | | |
| BSSS | 3.52 | 2.97 | .37 | .57 | | |
| HRV Reactivity (ms) | 43.2 | 33.2 | 11.2 | 13.1 | 43.7 | 27.4 |
| HRV Recovery (ms) | 57.5 | 38.5 | 17.6 | 17.9 | 53.8 | 30.7 |

*Note:* CS = Chronic Stress.

### 4.2 Inferential Statistics

No extreme outliers were detected for HRV reactivity, HRV recovery, perceived social support, or chronic fatigue. The distributions were deemed suitable for parametric analyses, with perceived social support showing slight skewness toward higher values. For chronic fatigue, one mild outlier below the 1.5xIQR threshold was identified, but it was deemed valid and retained for analysis. Additionally, multivariate outlier detection using Mahalanobis distance was conducted in R for the

same variables. No outliers were identified, as all Mahalanobis distances fell below the Chi-square critical value of 18.47 at $p < .001$.

The Shapiro-Wilk results indicated no significant deviations from normality for HRV reactivity ($W = .981$, $p = .848$) or HRV recovery ($W = .961$, $p = .284$). Additionally, Q-Q plots confirmed that the distributions were approximately normal. For all analyses, the same reference level was used for chronic stress, with low chronic stress serving as the baseline comparison group.

#### *4.2.1 RQ1: Chronic Stress and HRV Reactivity and Recovery*

A simple linear regression analysis was conducted to examine whether chronic stress (low vs. high) predicts HRV reactivity. Chronic stress significantly predicted HRV reactivity ($R^2 = .145$, adjusted $R^2 = .112$, $F(1, 26) = 4.40$, $p = .046$) indicating that chronic stress explains 11.2% of the variance in HRV reactivity. Participants with higher levels of chronic stress had significantly lower HRV reactivity compared to those with lower levels of chronic stress ($b = -10.1$, $\beta = -.765$, $t(26) = -2.10$, $p = .046$, 95% CI [-20, -.21]). Cohen's f² was calculated as $f^2 = .17$, indicating a moderate effect size. The Shapiro-Wilk test indicated that residuals were approximately normally distributed ($W = 0.94$, $p = .108$), which was supported by the Q-Q plot. Residual plots demonstrated no violations of linearity or homoscedasticity, confirming the validity of the model.

Similarly, linear regression was conducted to examine whether chronic stress predicts HRV recovery. The model accounted for a significant portion of the variance in HRV recovery ($R^2 = .221$, adjusted $R^2 = .193$, $F(1, 28) = 7.92$, $p = .009$). The results indicated a significant effect of chronic stress. Participants with higher chronic stress exhibited significantly lower HRV recovery scores ($b = -19$, $\beta = -.958$, $t(28) = -2.81$, $p = .009$, 95% CI [-32.8,-5.17]) compared to participants with lower chronic stress. Cohen's f² was calculated as $f^2 = .28$, reflecting a moderate effect size. The Shapiro-Wilk test showed no significant deviation from normality ($W = .945$, $p = .121$). Visual inspection of the Q-Q plot and residuals supported these findings, with no evidence of violations of homoscedasticity or linearity.

Levene's test could not be performed due to sample size limitations. However, a comparison of group standard deviations indicated similar variance across groups for both HRV reactivity and recovery (see Table 4), supporting the assumption of homogeneity.

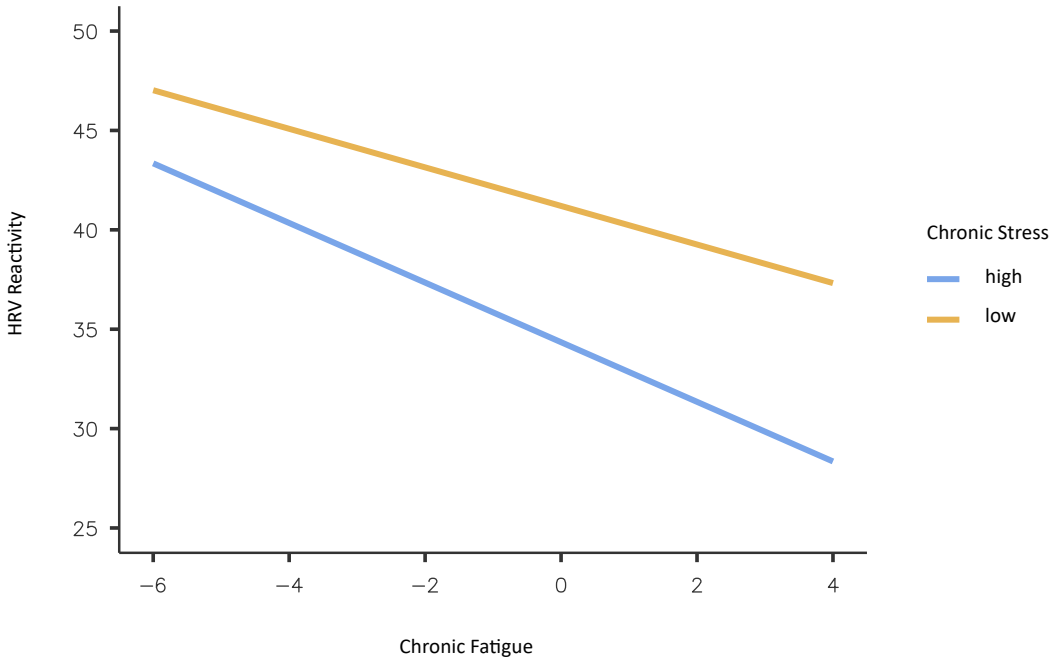
#### *4.2.2 RQ2: Chronic Fatigue as a Moderator of the Chronic Stress - HRV Relationship*

To evaluate whether chronic fatigue moderates the relationship between chronic stress and HRV reactivity, a linear regression analysis was conducted with an interaction term between chronic stress (low vs. high) and chronic fatigue. Chronic fatigue and chronic stress were included as

predictors, along with their interaction term, to predict HRV reactivity. The overall model was not statistically significant ($R^2$ = .19, adjusted $R^2$ = .089, $F(3, 24) = 1.88$, $p$ = .16), indicating that the predictors did not explain a significant amount of variance in HRV reactivity. Chronic stress ($b$ = -6.855, β = -.506, $t(24) = -1.153$, $p$ = .26) and chronic fatigue ($b$ = -.97, β = -.196, $t(24)$= -.642, p = .527) did not significantly predict HRV reactivity individually. The interaction term between chronic fatigue and chronic stress was also non-significant ($b$ = -.529, β = -.107, $t(24) = -.244$, $p$ = .809). Cohen's f² was calculated as $f^2 = .235$, indicating a moderate effect size for the overall model. Variance inflation factors were below the recommended cutoff of 10 for all predictors ($VIF_{chronic\ stress}$ = 1.49, $VIF_{chronic\ fatigue}$ = 2.75, $VIF_{Interaction}$ = 2.07), indicating no severe multicollinearity concerns after centering the chronic fatigue variable by subtracting the sample mean from participants' individual scores. The Shapiro-Wilk test indicated no significant deviation from normality ($W$ = .943, $p$ = .134), and the Q-Q plot indicated approximate normality of residuals. Residual plots suggested no major violations of linearity or homoscedasticity assumptions. Figure 1 illustrates the interaction between chronic stress, chronic fatigue, and HRV reactivity.

**Figure 1**

*Interaction Effect of Chronic Stress and Fatigue on HRV Reactivity*



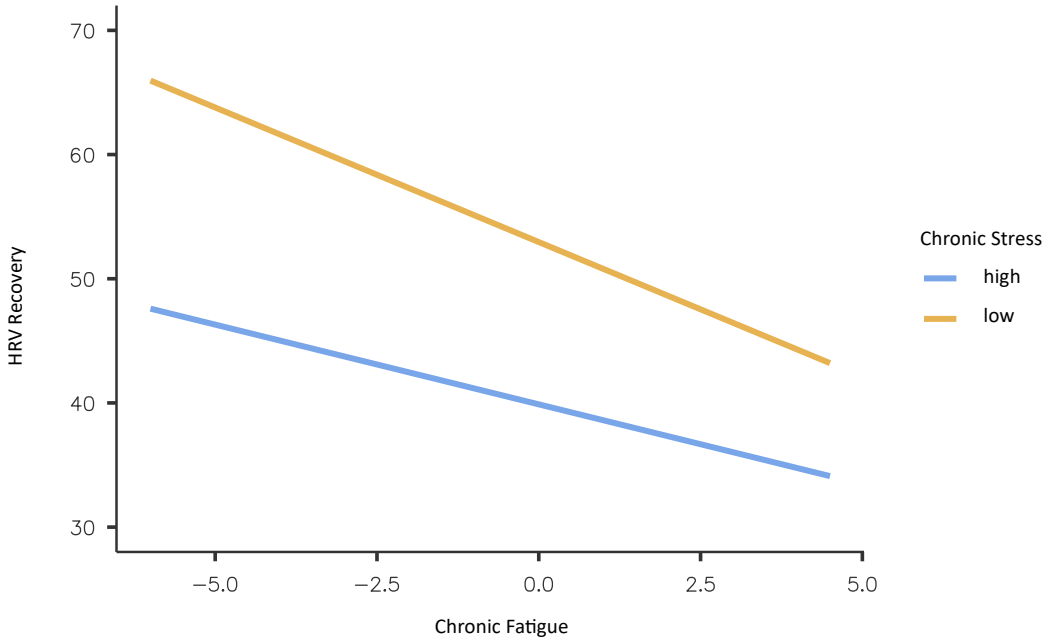

Similarly, chronic stress, chronic fatigue, and their interaction term were included as predictors of HRV recovery. The overall model explained 17.5% of the variance in HRV recovery ($R^2$ = .26, adjusted $R^2$ = .175, $F(3, 26) = 3.05$, $p$ = .046). Chronic stress ($b$ = -13.07, β = -.663, $t(26) = -1.539$, $p$ = .136), chronic fatigue ($b$ = -2.167, β = −.299, $t(26) = -1.001$, $p$ = .326) as well as the interaction term

between chronic stress and chronic fatigue ($b$ = 0.883, β = .122, $t(26)$ = .297, $p$ = .769) did not significantly predict HRV recovery. Effect size was large ($f^2 = .35$).

Variance inflation factors were well below the critical threshold of 10 for all predictors ($VIF_{chronic\ stress}$ = 1.55, $VIF_{chronic\ fatigue}$ = 3.14, $VIF_{Interaction}$ = 2.34), indicating no concerns about multicollinearity. Residuals met the assumptions of linearity and homoscedasticity. The Shapiro-Wilk test for normality indicated a significant deviation from normality ($W$ = .912, $p$ = .017). However, visual inspection of the Q-Q plot suggested approximate normality. For certainty, a bootstrapping analysis with 1000 samples was conducted supporting previous results in that chronic stress ($p$ = .271), chronic fatigue ($p$ = .301) and their interaction ($p$ = .411) did not significantly predict HRV reactivity or HRV recovery. The 95% confidence intervals for all coefficients included zero, showing no indication for significant direct or interactive effects of chronic stress and chronic fatigue on HRV recovery. Figure 2 depicts the interaction between chronic stress, chronic fatigue, and HRV recovery.

**Figure 2**

*Interaction Effect of Chronic Stress and Fatigue on HRV Recovery*



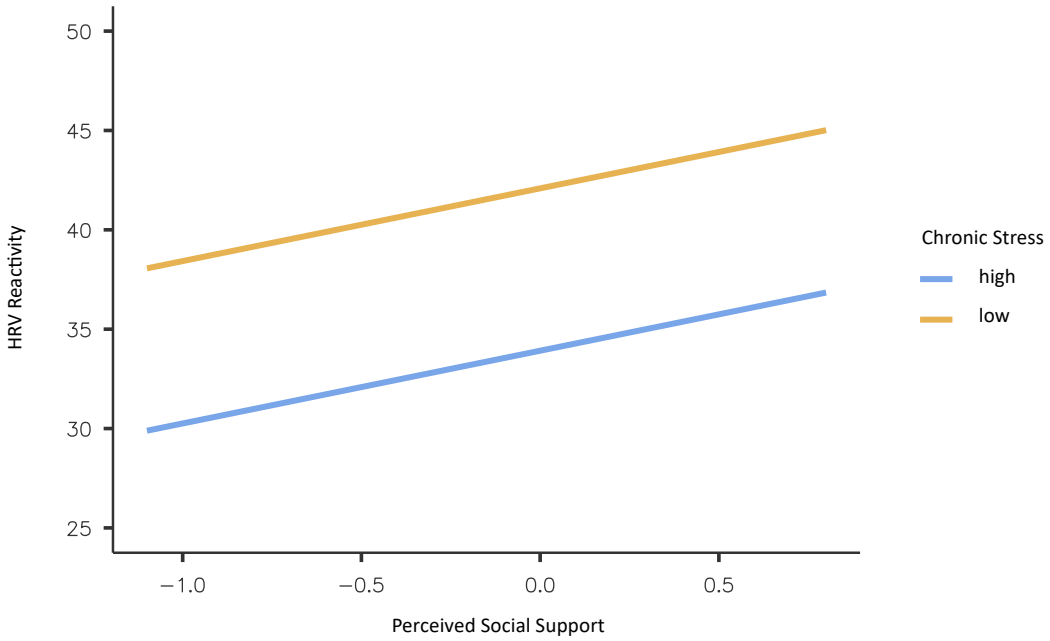

### *4.2.3 RQ3: Social Support as a Moderator of the Chronic Stress - HRV Relationship*

A linear regression analysis was conducted to examine whether perceived social support moderates the relationship between chronic stress and HRV reactivity. The model accounted for 8.5% of the variance in HRV reactivity ($R^2$ =.187, adjusted $R^2$ = .085), though the overall regression was not

statistically significant ($F(3, 24) = 1.84$, $p = .167$). Neither chronic stress ($b = -5.63$, $\beta = .46$, $t(24) = -.89$, $p = .383$), perceived social support ($b = 11.43$, $\beta = .503$, $t(24) = 1.049$, $p = .305$), nor their interaction ($b = -9.52$, $\beta = -.419$, $t(24) = .-789$, $p = .438$) were significant predictors of HRV reactivity, with a moderate effect size (Cohen's $f^2 = .23$). Assumption testing supported the appropriateness of the regression model. After centering, the VIF values for chronic stress, perceived social support, and their interaction term were low (VIF values from 1.69 to 6.8), indicating no concerns with multicollinearity. Additionally, the residuals were approximately normally distributed, as confirmed by the Shapiro-Wilk test ($W = .972$, $p = .628$) and the Q-Q plot. Figure 3 visualizes the interaction between chronic stress, perceived social support, and HRV reactivity.

**Figure 3**

*Interaction Effect of Chronic Stress and Perceived Social Support on HRV Reactivity*

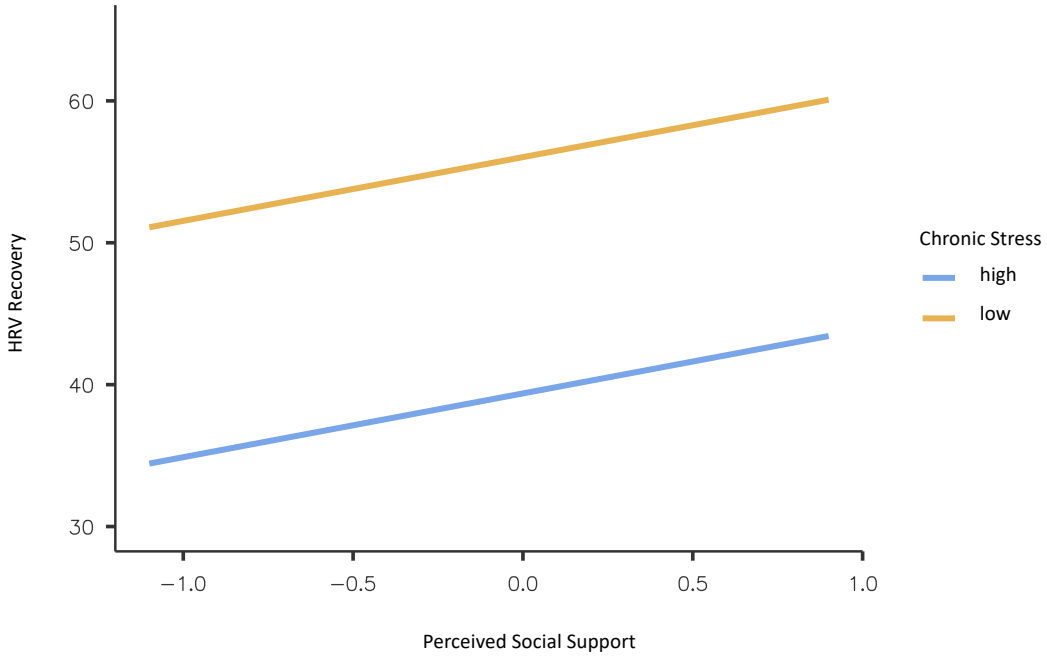



A regression analysis tested whether perceived social support moderates the relationship between chronic stress and HRV recovery. The model explained 18.1% of the variance in HRV recovery ($R^2 = .266$, adjusted $R^2 = .181$) and was statistically significant ($F(3, 26) = 3.13$, $p = .043$). Chronic stress was a significant predictor ($b = -21.54$, $\beta = -1.056$, $t(26) = 2.42$, $p = .023$), with individuals experiencing higher levels of chronic stress showing lower HRV recovery. However, perceived social support ($b = -10.5$, $\beta = -.295$, $t(26) = -.675$, $p = .505$) and its interaction with chronic stress ($b = 18.42$, $\beta = .519$, $t(26) = -1.07$, $p = .294$) were not significant predictors. Cohen's $f^2$ was calculated as $f^2 = .362$, suggesting a large effect. Assumption testing confirmed the appropriateness of the regression model. VIFs were below the acceptable threshold ($VIF_{chronic\ stress} = 1.72$,

$VIF_{social\ support}$ = 6.77, $VIF_{Interaction}$ = 5.51), indicating no multicollinearity concerns. Additionally, the residuals were approximately normally distributed ($W$ = .981, $p$ = .857), as supported by the Shapiro-Wilk test and the Q-Q plot. Figure 4 depicts the interaction between chronic stress, perceived social support, and HRV recovery.

**Figure 4**

*Interaction Effect of Chronic Stress and Perceived Social Support on HRV Recovery*




Chronic fatigue was excluded from the models testing H3.1 and H3.2 after preliminary moderation analyses (RQ2) demonstrated that it did not significantly predict HRV reactivity or recovery, nor did it moderate the relationship between chronic stress and HRV outcomes. Including chronic fatigue in the model did not improve overall model fit, as indicated by non-significant changes in $R^2$ and lack of meaningful contributions to explained variance.

### 4.3 Post-Hoc Analyses

Two post-hoc analyses were conducted to further explore the study's findings and address potential methodological considerations. First, HRV reactivity and recovery were re-operationalized as HRV SDNN difference scores between phases, which, as noted previously, is standard practice in most of the literature (Hamilton & Alloy, 2016). This approach aimed to ensure alignment with common measurement techniques and to examine whether alternative operationalization would yield different results. Second, a post-hoc power analysis was conducted to assess whether the study

was adequately powered to detect significant effects in the regression models. Given the relatively small sample size, this analysis was crucial for evaluating the robustness of non-significant findings and determining whether observed effects might have been limited by statistical power.

For the re-operationalization of HRV, HRV reactivity was defined as the difference between baseline HRV SDNN and HRV SDNN during the stressor period, and HRV recovery was defined as the difference between HRV SDNN during the stressor period and HRV SDNN during the recovery phase.

Means and standard deviations for HRV SDNN reactivity and recovery differences showed variability between high and low chronic stress groups. However, the observed differences did not achieve statistical significance in the models. Table 5 summarizes descriptive statistics for HRV SDNN reactivity and recovery differences for high and low chronic stress groups.

**Table 5**

*Descriptive Statistics for HRV SDNN Reactivity and Recovery Differences by Chronic Stress Level*

| **Variable** | ***N*** | | ***M*** | | ***SD*** | | ***Mdn*** | |
|---|---|---|---|---|---|---|---|---|
| | **low CS** | **high CS** | **low CS** | **high CS** | **low CS** | **high CS** | **low CS** | **high CS** |
| HRV Reactivity | 10 | 13 | 6.29 | 3.57 | 20.7 | 13.8 | -.12 | 5.35 |
| HRV Recovery | 11 | 16 | 14.3 | 6.67 | 17.9 | 11.9 | 7.22 | 5.06 |

Linear regression models were employed to examine the impact of chronic stress on these difference scores. Table 6 depicts regression results analyzing whether the level of chronic stress predicts HRV SDNN reactivity and recovery (RQ1) when operationalized as differences in SDNN between the baseline and stressor period (HRV reactivity difference), and stressor and the consecutive rest period (HRV recovery difference). For HRV reactivity, the model explained a negligible amount of variance, with no significant effect of chronic stress group. Similarly, the model for HRV recovery was not statistically significant, with no significant effect of chronic stress group. The Shapiro-Wilk test indicated that the residuals for both models deviated significantly from normality, suggesting potential issues with model assumptions. Q-Q plots also visually confirmed deviations from normality in residual distributions.

**Table 6**

*Regression Results for RQ1 using HRV SDNN Reactivity and Recovery Difference Scores*

| Variable | $R^2$ | Adjusted $R^2$ | *F (df)* | *b* | *t* | *p* | Shapiro-Wilk *p* |
|---|---|---|---|---|---|---|---|
| HRV Reactivity | .00677 | .007 | .14 (1, 26) | 2.72 | .378 | .709 | .031 |
| HRV Recovery | .0662 | .029 | 1.78 (1,28) | 7.61 | 1.33 | .195 | .014 |

Further, the moderation effects of chronic fatigue on the relationship between chronic stress and HRV SDNN reactivity and recovery difference scores were examined. For HRV reactivity, the overall model was not significant ($R^2$ = .014, adjusted $R^2$ = .137, $F(3, 19)$ = .092, $p$ = .964). Neither chronic stress ($b$ = 4.18, $p$ = .662), chronic fatigue ($b$ = .932, $p$ = .723), nor their interaction ($b$ = −.656, $p$ = .850) predicted HRV reactivity difference scores. However, residuals were approximately normally distributed (Shapiro-Wilk W = .924, p = .083). For HRV recovery, the model was not significant ($R^2$ = .105, adjusted $R^2$ = -.012, $F(3, 23)$ = .896, $p$ = .458). Chronic stress ($b$ = 5.95, $p$ = .407), chronic fatigue ($b$ = 1.48, $p$ = .47), and their interaction ($b$ = -2.68, $p$ = .331) failed to significantly predict HRV recovery difference scores. Notably, the adjusted $R^2$ value is negative and residuals were not normally distributed ($W$ = .886, $p$ = .007), again limiting the interpretability of the results.

In the moderation analyses involving perceived social support, there was a significant interaction effect for HRV reactivity but not for HRV recovery. For HRV reactivity, the interaction between chronic stress and perceived social support was significant ($p$ = .022), explaining 13.7% of variance ($R^2$ = .255, adjusted $R^2$ = .137, $F(3, 19)$ = 2.17, $p$ = .125). Residuals were normally distributed ($W$ = .972, $p$ = .730). For HRV recovery, the interaction was not significant ($p$ = .067), but chronic stress itself was a significant predictor ($b$ = 15.04, $p$ = .046). The overall model was not significant ($R^2$ = .196, adjusted $R^2$ = .092, $F(3, 23)$ = 1.87, $p$ = .162). Residuals were approximately normally distributed ($W$ = .935, $p$ = .09).

To assess whether the study was sufficiently powered to detect effects in the regression models for RQ1 (H1.1 & H1.2), a post-hoc power analysis was conducted using G*Power 3.1 and based on the original operationalization of HRV reactivity and recovery as mean HRV SDNN during stressor and subsequent rest periods respectively. The analysis was performed for a linear multiple regression model with one predictor, with a total sample size of $N$ = 31, an alpha level of α = .05, and using an observed effect size of $f^2$ = .17 for H1.1. The results indicated a non-centrality parameter of λ = 5.27, a critical F-value of 4.18, and a power (1−β) of .60. This suggests that the study had a 60% chance of detecting a true effect, given the specified parameters. Although this power is below the commonly recommended threshold of 0.80 (Cohen, 1988), it reflects a moderate power level given the sample size and observed effect size. For H1.2, the regression model similarly used one predictor with an observed effect size of $f^2$ = .28, with results indicating that the study had 81% power to detect the

observed effect size for H1.1 (λ = 8.68, Critical *F* = 4.18, 1-β = .81), meeting the conventional threshold for adequate statistical power (≥ .80). For RQ2, the regression model included three predictors, with an observed effect size of $f^2$ = .235 for H2.1. The achieved power for H2.1 is 54% (λ = 7.29, Critical *F* = 2.96, 1-β = .54), which is below the conventional threshold of 0.80. The achieved power for H2.2 is 73% ($f^2$ = .235, λ = 10.85, Critical *F* = 2.96, 1-β = .73), which approaches but does not fully meet the conventional threshold of 0.80. This indicates moderate power to detect the observed effect size in the regression model with three predictors. For RQ3, the regression model included three predictors, with an observed effect size of $f^2$ = .23 for H3.1 and $f^2$ = .362 for H3.2, a total sample size of *N* = 31, and an alpha level of α = .05. The achieved power for H3.1 was 53% (λ = 7.13, Critical *F* = 2.96, 1-β = .53), while H3.2 had a 75% chance of detecting a true effect (λ = 11.22, Critical *F* = 2.96, 1-β = .75), indicating low to moderate power for RQ3.

## 5. Discussion

The primary aim of this thesis was to investigate the relationship between chronic stress and heart rate variability (HRV) in women, with a particular focus on how chronic fatigue and perceived social support might influence this relationship. Chronic stress has long been known to cause significant physiological and psychological strain, which this study has further elucidated by demonstrating its negative impact on autonomic regulation as evidenced by decreased HRV (Brugnera et al., 2018; Kim et al., 2018).

Hypothesis 1.1 posited that women with higher levels of chronic stress would exhibit lower HRV reactivity to acute stress compared to those with lower stress levels. The results confirmed this hypothesis, indicating that chronic stress significantly impairs the autonomic flexibility necessary for an adequate response to immediate stressors. This impairment is characterized by an overactivation of the sympathetic nervous system and a suppression of the parasympathetic response, consistent with the maladaptive ANS response described in the Neurovisceral Integration Model (Thayer et al., 2009). These findings underscore the potential for chronic stress to disrupt the body's natural stress response, rendering it less capable of managing acute stress effectively.

Hypothesis 1.2 suggested that women with higher levels of chronic stress would show lower HRV recovery following acute stress exposure. The findings supported this hypothesis, demonstrating that chronic stress is associated with reduced parasympathetic rebound after stressors are removed, indicative of a sustained sympathetic activation that hinders the body's ability to return to baseline autonomic functioning. This prolonged dysregulation is important in that it can lead to increased allostatic load, heightening the risk for various health complications including cardiovascular diseases and mental health disorders (McEwen, 1998).

Hypotheses 2.1 and 2.2 regarding the moderating effects of chronic fatigue on HRV reactivity and recovery were not supported. This suggests that chronic fatigue, within the context of this study, does not significantly exacerbate the autonomic dysregulation associated with chronic stress. However, the descriptive data and non-significant trends observed in post-hoc analyses imply that chronic fatigue may still play a complex role in influencing the body's stress response, warranting further investigation. Visual inspection of trends revealed that as fatigue increases, HRV reactivity decreases for both high and low stress levels. The graph for high chronic stress starts higher but decreases at a rate that suggests a stronger decline in HRV reactivity with increased fatigue compared to the graph for low chronic stress. This suggests that in individuals with high chronic stress, increases in chronic fatigue may lead to a greater reduction in HRV reactivity than in those with low chronic stress, pointing to chronic stress amplifying the negative effects of fatigue on the physiological response measured by HRV. Thus, despite non-significant results, chronic stress may still act as a moderator in the relationship between fatigue and HRV. Hypotheses 3.1 and 3.2 explored the moderating role of perceived social support on HRV reactivity and recovery but also did not find significant effects.

In this study, fatigue was modeled as a risk factor of chronic stress, rather than the other way around. Though the moderation analysis was non-significant, future studies might further explore fatigue-first pathways, examining how fatigue alters physiological, psychological, and social systems to create conditions conducive to chronic stress. Further research may reveal subpopulations (e.g., women, individuals with underlying health conditions) where this fatigue-first model is relevant. Fatigue-focused interventions might prioritize improving ANS functioning, such as vagal tone enhancement through HRV biofeedback, relaxation techniques, or exercise. These approaches could help break the cycle before chronic stress becomes entrenched.

This study's conclusions are constrained by its sample size and the methodological choices necessitated by data limitations. Specifically, the operationalization of HRV reactivity and recovery as mean values during stress phases, rather than differences between phases, was driven by the need to maximize statistical power with a small sample. Small sample sizes were problematic in estimating effect sizes, likely having contributed to wide confidence intervals and limited power, potentially obscuring true effects.

The G*Power analysis for RQ2 and RQ3 suggests that the study was underpowered to detect the observed effect size for the model with three predictors. Consequently, the non-significant results in this analysis may have been influenced by insufficient statistical power, reducing confidence in the findings. Overall low statistical power to detect the observed effect size in the regression models with three predictors (main effects and interaction) was observed, especially for analyses including HRV reactivity. The study was therefore at substantial risk of a Type II error.

The non-significant findings for post-hoc analyses with both HRV reactivity and recovery difference scores may be partially attributable to the violation of normality assumptions. The deviations from normality in the residuals highlight the need for caution in interpreting these results, supporting the need for the alternate operationalization in this sample. Given the small sample size and the potential sensitivity of these models to outliers and non-normal distributions, future research should aim to replicate these findings with larger samples. Moreover, exploring the incorporation of additional HRV metrics such as vmHRV, RMSSD, or frequency-domain measures like LF/HF metrics could provide deeper insights into the nuances of autonomic function under chronic stress.

Another limitation lies in the study's controlled laboratory environment. Although stress reactivity and recovery processes are well-documented within laboratory settings, their applicability to real-life situations remains uncertain (Bamert & Inauen, 2022). Observing stress in natural, day-to-day contexts would allow for a better understanding of how multiple stressors accumulate over time and hinder recovery following stressful events. This accumulation highlights the temporal dynamics of stress as they occur in everyday life (Smyth et al., 2017). Future research could leverage advancements in HRV tracking technology (Li et al., 2023), employing smart wearable devices such as smartwatches to monitor HRV in more ecologically valid, everyday/real-world settings.

Despite these limitations, the present study has enhanced understanding of the relationship between chronic stress and HRV stress response patterns in women as well as shed light on two potential risk and protective factors relevant to this dynamic, raising a variety of intriguing questions and potential directions for future research. The findings underline the need for interventions designed to manage or mitigate chronic stress, particularly in populations at high risk of chronic stress exposure. Stress management programs that include techniques such as mindfulness, relaxation training, and therapeutic exercises have been shown to enhance autonomic regulation (Lehrer et al., 2020), thereby improving HRV reactivity and recovery. Moreover, considering the specific vulnerabilities of women to stress-related autonomic dysregulation, gender-sensitive approaches in health interventions could be particularly beneficial. Such approaches should consider the unique stressors faced by women, such as dual demands of work and family life and social pressures, which may contribute to their stress profiles.

## 6. References

Adjei, T., Xue, J., & Mandic, D. P. (2018). The Female Heart: Sex Differences in the Dynamics of ECG in Response to Stress. *Frontiers in Physiology*, *9*. https://doi.org/10.3389/fphys.2018.01616

Ali, N., Strahler, J., & Nater, U. M. (2024). The effects of chronic fatigue and chronic stress on alterations in immune cell responses to acute psychosocial stress. *Brain Behavior and Immunity*, *123*, 707–716. https://doi.org/10.1016/j.bbi.2024.10.013

Almeida, D. M. (2005). Resilience and Vulnerability to Daily Stressors Assessed via Diary Methods. *Current Directions in Psychological Science*, *14*(2), 64–68. https://doi.org/10.1111/j.0963-7214.2005.00336.x

Anisman, H., Hayley, S., & Kusnecov, A. (2018). Multiple Pathways Linked to Mental Health and Illness. In *Elsevier eBooks* (pp. 1–36). https://doi.org/10.1016/b978-0-12-811351-6.00001-2

Bamert, M., & Inauen, J. (2022). Physiological stress reactivity and recovery: Some laboratory results transfer to daily life. *Frontiers in Psychology*, *13*. https://doi.org/10.3389/fpsyg.2022.943065

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review*, *98*(4), 459–487. https://doi.org/10.1037/0033-295x.98.4.459

Boneva, R. S., Decker, M. J., Maloney, E. M., Lin, J., Jones, J. F., Helgason, H. G., Heim, C. M., Rye, D. B., & Reeves, W. C. (2007). Higher heart rate and reduced heart rate variability persist during sleep in chronic fatigue syndrome: A population-based study. *Autonomic Neuroscience*, *137*(1–2), 94–101. https://doi.org/10.1016/j.autneu.2007.08.002

Bonne, O., Grillon, C., Vythilingam, M., Neumeister, A., & Charney, D. S. (2004). Adaptive and maladaptive psychobiological responses to severe psychological stress: implications for the discovery of novel pharmacotherapy. *Neuroscience & Biobehavioral Reviews*, *28*(1), 65–94. https://doi.org/10.1016/j.neubiorev.2003.12.001

Bosquet, L., Merkari, S., Arvisais, D., & Aubert, A. E. (2008). Is heart rate a convenient tool to monitor over-reaching? A systematic review of the literature. *British Journal of Sports Medicine*, *42*(9), 709–714. https://doi.org/10.1136/bjsm.2007.042200

Brugnera, A., Zarbo, C., Tarvainen, M. P., Marchettini, P., Adorni, R., & Compare, A. (2018). Heart rate variability during acute psychosocial stress: A randomized cross-over trial of verbal and non-verbal laboratory stressors. *International Journal of Psychophysiology*, *127*, 17–25. https://doi.org/10.1016/j.ijpsycho.2018.02.016

Cannon, W. B. (1932). *The Wisdom of the Body*.

Cohen, S., & Wills, T. A. (1985). Stress, social support, and the buffering hypothesis. *Psychological Bulletin*, *98*(2), 310–357. https://doi.org/10.1037/0033-2909.98.2.310

Cummins, R. C. (1988). Perceptions of social support, receipt of supportive behaviors, and locus of control as moderators of the effects of chronic stress. *American Journal of Community Psychology*, *16*(5), 685–700. https://doi.org/10.1007/bf00930021

Da Estrela, C., McGrath, J., Booij, L., & Gouin, J. (2020). Heart Rate Variability, Sleep Quality, and Depression in the Context of Chronic Stress. *Annals of Behavioral Medicine*, *55*(2), 155–164. https://doi.org/10.1093/abm/kaaa039

Dalla, C., Antoniou, K., Drossopoulou, G., Xagoraris, M., Kokras, N., Sfikakis, A., & Papadopoulou-Daifoti, Z. (2005). Chronic mild stress impact: Are females more vulnerable? *Neuroscience*, *135*(3), 703–714. https://doi.org/10.1016/j.neuroscience.2005.06.068

Ditzen, B., & Heinrichs, M. (2014). Psychobiology of social support: The social dimension of stress buffering. *Restorative Neurology and Neuroscience*, *32*(1), 149–162. https://doi.org/10.3233/rnn-139008

Earnest, C. P., Blair, S. N., & Church, T. S. (2011). Heart rate variability and exercise in aging women. *Journal of Women S Health*, *21*(3), 334–339. https://doi.org/10.1089/jwh.2011.2932

Escorihuela, R. M., Capdevila, L., Castro, J. R., Zaragozà, M. C., Maurel, S., Alegre, J., & Castro-Marrero, J. (2020). Reduced heart rate variability predicts fatigue severity in individuals with chronic fatigue syndrome/myalgic encephalomyelitis. *Journal of Translational Medicine*, *18*(1). https://doi.org/10.1186/s12967-019-02184-z

Gallo, L. C., Jiménez, J. A., Shivpuri, S., De Los Monteros, K. E., & Mills, P. J. (2010). Domains of Chronic Stress, Lifestyle Factors, and Allostatic Load in Middle-Aged Mexican-American Women. *Annals of Behavioral Medicine*, *41*(1), 21–31. https://doi.org/10.1007/s12160-010-9233-1

Halko, M., & Sääksvuori, L. (2017). Competitive behavior, stress, and gender. *Journal of Economic Behavior & Organization*, *141*, 96–109. https://doi.org/10.1016/j.jebo.2017.06.014

Hamilton, J. A., & Russo, N. F. (2006). Women and Depression. In *Cambridge University Press eBooks* (pp. 479–522). https://doi.org/10.1017/cbo9780511841262.021

Hamilton, J. L., & Alloy, L. B. (2016). Atypical reactivity of heart rate variability to stress and depression across development: Systematic review of the literature and directions for future research. *Clinical Psychology Review*, *50*, 67–79. https://doi.org/10.1016/j.cpr.2016.09.003

Hartley, C., & Coffee, P. (2019). Perceived and Received dimensional support: Main and Stress-Buffering effects on dimensions of burnout. *Frontiers in Psychology*, *10*. https://doi.org/10.3389/fpsyg.2019.01724

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

Jain, S., & Mills, P. (2007). Cytokines, Chronic Stress, and Fatigue. In *Elsevier eBooks* (pp. 698–704). https://doi.org/10.1016/b978-012373947-6.00727-3

Jiryis, T., Magal, N., Fructher, E., Hertz, U., & Admon, R. (2022). Resting-state heart rate variability (HRV) mediates the association between perceived chronic stress and ambiguity avoidance. *Scientific Reports*, *12*(1). https://doi.org/10.1038/s41598-022-22584-4

Juster, R., & Misiak, B. (2023). Advancing the allostatic load model: From theory to therapy. *Psychoneuroendocrinology*, *154*, 106289. https://doi.org/10.1016/j.psyneuen.2023.106289

Kao, C., Tseng, L., Lin, W., & Cheng, S. (2013). Association of Psychosocial Factors and Heart Rate Variability in Heart Failure Patients. *Western Journal of Nursing Research*, *36*(6), 769–787. https://doi.org/10.1177/0193945913505922

Keyes, C. L. M., Keyes, C. L. M., Carter, R., Keyes, C. L. M., Wilhelm, K., Kessler, R. C., Korszun, A., Somerset, W., Derose, L. M., Penninx, B. W. J. H., Girgus, J. S., Widiger, T. A., Klein, L. C., Whisman, M. A., Goodman, S. H., Marecek, J., Lennon, M. C., Jackson, P. B., Penza, K. M., . . . Hamilton, J. A. (2006). Women and Depression. In *Cambridge University Press eBooks*. https://doi.org/10.1017/cbo9780511841262

Kim, H., Cheon, E., Bai, D., Lee, Y. H., & Koo, B. (2018). Stress and Heart Rate Variability: A Meta-Analysis and Review of the Literature. *Psychiatry Investigation*, *15*(3), 235–245. https://doi.org/10.30773/pi.2017.08.17

Kirschbaum, C., Pirke, K., & Hellhammer, D. H. (1993). The 'Trier Social Stress Test' – A Tool for Investigating Psychobiological Stress Responses in a Laboratory Setting. *Neuropsychobiology*, *28*(1–2), 76–81. https://doi.org/10.1159/000119004

Klimas, N. G., Broderick, G., & Fletcher, M. A. (2012). Biomarkers for chronic fatigue. *Brain Behavior and Immunity*, *26*(8), 1202–1210. https://doi.org/10.1016/j.bbi.2012.06.006

Kocalevent, R. D., Hinz, A., Brähler, E., & Klapp, B. F. (2011). Determinants of fatigue and stress. *BMC Research Notes*, *4*(1). https://doi.org/10.1186/1756-0500-4-238

Kothgassner, O. D., Goreis, A., Kafka, J. X., Kaufmann, M., Atteneder, K., Beutl, L., Hennig-Fast, K., Hlavacs, H., & Felnhofer, A. (2018). Virtual social support buffers stress response: An experimental comparison of real-life and virtual support prior to a social stressor. *Journal of Behavior Therapy and Experimental Psychiatry*, *63*, 57–65. https://doi.org/10.1016/j.jbtep.2018.11.003

Kudielka, B., Wüst, S., Kirschbaum, C., & Hellhammer, D. (2007). Trier Social Stress test. In *Elsevier eBooks* (pp. 776–781). https://doi.org/10.1016/b978-012373947-6.00681-4

Laborde, S., Mosley, E., & Mertgen, A. (2018). Vagal Tank Theory: The Three Rs of Cardiac Vagal Control Functioning – Resting, Reactivity, and Recovery. *Frontiers in Neuroscience*, *12*. https://doi.org/10.3389/fnins.2018.00458

Lacey, J. I., & Lacey, B. C. (1958). Verification and Extension of the Principle of Autonomic Response-Stereotypy. *The American Journal of Psychology*, *71*(1), 50. https://doi.org/10.2307/1419197

Lampert, R., Tuit, K., Hong, K., Donovan, T., Lee, F., & Sinha, R. (2016). Cumulative stress and autonomic dysregulation in a community sample. *Stress*, *19*(3), 269–279. https://doi.org/10.1080/10253890.2016.1174847

Laquidara, J. R., & Lincoln, S. H. (2023). Testing two theories of social support benefits and implications for individuals with psychotic-like experiences. *Schizophrenia Research*, *264*, 87–89. https://doi.org/10.1016/j.schres.2023.12.008

Lehrer, P., Kaur, K., Sharma, A., Shah, K., Huseby, R., Bhavsar, J., Sgobba, P., & Zhang, Y. (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

Leven, M. V., Zielhuis, G. A., Van Der Meer, J. W., Verbeek, A. L., & Bleijenberg, G. (2009). Fatigue and chronic fatigue syndrome-like complaints in the general population. *European Journal of Public Health*, *20*(3), 251–257. https://doi.org/10.1093/eurpub/ckp113

Li, K., Cardoso, C., Moctezuma-Ramirez, A., Elgalad, A., & Perin, E. (2023). Heart Rate Variability Measurement through a Smart Wearable Device: Another Breakthrough for Personal Health Monitoring? *International Journal of Environmental Research and Public Health*, *20*(24), 7146. https://doi.org/10.3390/ijerph20247146

Lutin, E., Schiweck, C., Cornelis, J., De Raedt, W., Reif, A., Vrieze, E., Claes, S., & Van Hoof, C. (2022). The cumulative effect of chronic stress and depressive symptoms affects heart rate in a working population. *Frontiers in Psychiatry*, *13*. https://doi.org/10.3389/fpsyt.2022.1022298

Maciejewski, P. K., Prigerson, H. G., & Mazure, C. M. (2001). Sex differences in event-related risk for major depression. *Psychological Medicine*, *31*(4), 593–604. https://doi.org/10.1017/s0033291701003877

Manser, P., Thalmann, M., Adcock, M., Knols, R. H., & De Bruin, E. D. (2021). Can Reactivity of Heart Rate Variability Be a Potential Biomarker and Monitoring Tool to Promote Healthy Aging? A Systematic Review With Meta-Analyses. *Frontiers in Physiology*, *12*. https://doi.org/10.3389/fphys.2021.686129

McEwen, B. S. (1998). Protective and Damaging Effects of Stress Mediators. *New England Journal of Medicine*, *338*(3), 171–179. https://doi.org/10.1056/nejm199801153380307

McEwen, B. S. (2007). Physiology and Neurobiology of Stress and Adaptation: Central Role of the Brain. *Physiological Reviews*, *87*(3), 873–904. https://doi.org/10.1152/physrev.00041.2006

Meeus, M., Goubert, D., De Backer, F., Struyf, F., Hermans, L., Coppieters, I., De Wandele, I., Da Silva, H., & Calders, P. (2013). Heart rate variability in patients with fibromyalgia and patients with

chronic fatigue syndrome: A systematic review. *Seminars in Arthritis and Rheumatism*, *43*(2), 279–287. https://doi.org/10.1016/j.semarthrit.2013.03.004

Móra, Á., Komka, Z., Végh, J., Farkas, I., Kocsisné, G. S., Bosnyák, E., Szmodis, M., Ligetvári, R., Csöndör, É., Almási, G., Oláh, A., Kemper, H. C. G., Tóth, M., & Ács, P. (2022). Comparison of the Cardiovascular Effects of Extreme Psychological and Physical Stress Tests in Male Soccer Players. *International Journal of Environmental Research and Public Health*, *19*(2), 715. https://doi.org/10.3390/ijerph19020715

*OASH | Office on Women's Health*. (2024, September 10). Office on Women's Health. https://www.womenshealth.gov/a-z-topics/chronic-fatigue-syndrome

Obrist, P. A. (1982). Cardiovascular Psychophysiology: A Perspective. *Psychophysiology*, *19*(3), 357–360. https://doi.org/10.1111/j.1469-8986.1982.tb02569.x

Phu, T., Senehi, N., & Watamura, S. (2019). Early life stress and development. In *Elsevier eBooks* (pp. 487–494). https://doi.org/10.1016/b978-0-12-809324-5.23650-x

Porges, S. W. (2006). The polyvagal perspective. *Biological Psychology*, *74*(2), 116–143. https://doi.org/10.1016/j.biopsycho.2006.06.009

Rådmark, L., Sidorchuk, A., Osika, W., & Niemi, M. (2019). A Systematic Review and Meta-Analysis of the Impact of Mindfulness based interventions on heart rate variability and Inflammatory Markers. *Journal of Clinical Medicine*, *8*(10), 1638. https://doi.org/10.3390/jcm8101638

Rimes, K. A., Lievesley, K., & Chalder, T. (2017). Stress vulnerability in adolescents with chronic fatigue syndrome: experimental study investigating heart rate variability and skin conductance responses. *Journal of Child Psychology and Psychiatry*, *58*(7), 851–858. https://doi.org/10.1111/jcpp.12711

Schneiderman, N., Ironson, G., & Siegel, S. D. (2004). Stress and Health: Psychological, Behavioral, and Biological Determinants. *Annual Review of Clinical Psychology*, *1*(1), 607–628. https://doi.org/10.1146/annurev.clinpsy.1.102803.144141

Schubert, C., Lambertz, M., Nelesen, R., Bardwell, W., Choi, J., & Dimsdale, J. (2008). Effects of stress on heart rate complexity—A comparison between short-term and chronic stress. *Biological Psychology*, *80*(3), 325–332. https://doi.org/10.1016/j.biopsycho.2008.11.005

Schultz, P., Schlotz, W., & Becker, P. (2004). *Trierer Inventar zum chronischen Stress (TICS)*. Hogrefe. https://www.testzentrale.de/shop/trierer-inventar-zum-chronischen-stress.html

Schulz, U. & Schwarzer, R. (2003). Soziale Unterstützung bei der Krankheitsbewältigung: Die Berliner Social Support Skalen (BSSS). Diagnostica, 49(2), 73–82. https://doi.org/10.1026/0012-1924.49.2.73

Shaffer, F., & Ginsberg, J. P. (2017). An Overview of Heart Rate Variability Metrics and Norms. *Frontiers in Public Health*, *5*. https://doi.org/10.3389/fpubh.2017.00258

Shahid, A., Wilkinson, K., Marcu, S., & Shapiro, C. M. (2011). Multidimensional Fatigue Inventory (MFI). *Springer eBooks* (pp. 241–243). https://doi.org/10.1007/978-1-4419-9893-4_57

Shonkoff, J. P., Boyce, W. T., & McEwen, B. S. (2009). Neuroscience, Molecular Biology, and the Childhood Roots of Health Disparities. *JAMA*, *301*(21), 2252. https://doi.org/10.1001/jama.2009.754

Smets, E., Garssen, B., Bonke, B., & De Haes, J. (1995). The multidimensional Fatigue Inventory (MFI) psychometric qualities of an instrument to assess fatigue. *Journal of Psychosomatic Research*, *39*(3), 315–325. https://doi.org/10.1016/0022-3999(94)00125-o

Smyth, J. M., Sliwinski, M. J., Zawadzki, M. J., Scott, S. B., Conroy, D. E., Lanza, S. T., Marcusson-Clavertz, D., Kim, J., Stawski, R. S., Stoney, C. M., Buxton, O. M., Sciamanna, C. N., Green, P. M., & Almeida, D. M. (2017). Everyday stress response targets in the science of behavior change. *Behaviour Research and Therapy*, *101*, 20–29. https://doi.org/10.1016/j.brat.2017.09.009

Strahler, J., Skoluda, N., Rohleder, N., & Nater, U. M. (2016). Dysregulated stress signal sensitivity and inflammatory disinhibition as a pathophysiological mechanism of stress-related chronic fatigue. *Neuroscience & Biobehavioral Reviews*, *68*, 298–318. https://doi.org/10.1016/j.neubiorev.2016.05.008

Swinkels, J., Van Tilburg, T., Verbakel, E., & Van Groenou, M. B. (2017). OUP accepted manuscript. *The Journals of Gerontology Series B*. https://doi.org/10.1093/geronb/gbx036

Taylor, S. E. (2012). Social Support: a review. *Oxford University Press eBooks*, 190–214. https://doi.org/10.1093/oxfordhb/9780195342819.013.0009

Taylor, S. E., Klein, L. C., Lewis, B. P., Gruenewald, T. L., Gurung, R. a. R., & Updegraff, J. A. (2000). Biobehavioral responses to stress in females: Tend-and-befriend, not fight-or-flight. *Psychological Review*, *107*(3), 411–429. https://doi.org/10.1037/0033-295x.107.3.411

Thayer, J. F., Åhs, F., Fredrikson, M., Sollers, J. J., & Wager, T. D. (2011). A meta-analysis of heart rate variability and neuroimaging studies: Implications for heart rate variability as a marker of stress and health. *Neuroscience & Biobehavioral Reviews*, *36*(2), 747–756. https://doi.org/10.1016/j.neubiorev.2011.11.009

Thayer, J. F., Hansen, A. L., Saus-Rose, E., & Johnsen, B. H. (2009). Heart Rate Variability, Prefrontal Neural Function, and Cognitive Performance: The Neurovisceral Integration Perspective on Self-regulation, Adaptation, and Health. *Annals of Behavioral Medicine*, *37*(2), 141–153. https://doi.org/10.1007/s12160-009-9101-z

Turton, S., & Campbell, C. (2005). Tend and Befriend Versus Fight or Flight: Gender Differences in Behavioral Response to Stress Among University Students. *Journal of Applied Biobehavioral Research*, *10*(4), 209–232. https://doi.org/10.1111/j.1751-9861.2005.tb00013.x

Viljoen, M., & Claassen, N. (2017). Allostatic load and heart rate variability as health risk indicators. *African Health Sciences*, *17*(2), 428. https://doi.org/10.4314/ahs.v17i2.17

Weissman, D. G., & Mendes, W. B. (2021). Correlation of sympathetic and parasympathetic nervous system activity during rest and acute stress tasks. *International Journal of Psychophysiology*, *162*, 60–68. https://doi.org/10.1016/j.ijpsycho.2021.01.015

Wethington, E., & Kessler, R. C. (1986). Perceived support, received support, and adjustment to stressful life events. *Journal of Health and Social Behavior*, *27*(1), 78. https://doi.org/10.2307/2136504

Yang, H., Koh, E., & Kang, Y. (2021). Susceptibility of women to cardiovascular disease and the prevention potential of Mind–Body intervention by changes in neural circuits and cardiovascular physiology. *Biomolecules*, *11*(5), 708. https://doi.org/10.3390/biom11050708

Yataco, A., Talo, H., Rowe, P., Kass, D. A., Berger, R. D., & Calkins, H. (1997). Comparison of heart rate variability in patients with chronic fatigue syndrome and controls. *Clinical Autonomic Research*, *7*(6), 293–297. https://doi.org/10.1007/bf02267720

## 7. Tables and Figures

## 8. Abbreviations

| | |
|---|---|
| ANS | Autonomic Nervous Sytem |
| ASM | Autonomic Space Model (Berntson et al., 1991) |
| BSSS | Berlin Social Support Scales (Schulz & Schwarzer, 2003) |
| CF | Chronic Fatigue |
| CFS | Chronic Fatigue Syndrome |
| CS | Chronic Stress |
| ECG | Electrocardiogramm |
| HF | High Frequency |
| HPA axis | Hypothalamic-Pituitary-Adrenal Axis |
| HRV | Heart Rate Variability |
| LF | Low Frequency |
| MFI | Multidimensional Fatigue Inventory (Smets, 1995) |
| PNS | Parasympathetic Nervous System |
| RMSSD | Root Mean Square of Successive Differences |
| SDNN | Standard Deviation of Normal-to-Normal Intervals |
| SNS | Sympathetic Nervous System |
| SSCS | Short Screening Scale for Chronic Stress (Schulz et al., 2004) |
| TSST | Trier Social Stress Test (Kirschbaum et al., 1993) |
| vmHRV | Vagally Mediated HRV |

# 9. Appendix

## 9.1 Abstract

Chronic stress significantly impacts autonomic nervous system (ANS) function, particularly among women, contributing to adverse health outcomes. Heart rate variability (HRV), a sensitive marker of ANS-regulation, reflects the body's capacity to adapt to stress and recover. This thesis investigates the influence of chronic fatigue and perceived social support on HRV reactivity to and recovery after acute stress in chronically stressed women, addressing three research questions: (1) whether the level of chronic stress predicts HRV reactivity and recovery to acute stress, (2) whether chronic fatigue moderates the relationship between chronic stress and HRV reactivity/recovery to acute stress, and (3) whether perceived social support moderates the relationship between chronic stress and HRV reactivity/recovery to acute stress, such that higher support leads to increased HRV reactivity and recovery. A cross-sectional design was employed with 31 German-speaking women aged 20-65. HRV reactivity was measured during the Trier Social Stress Test and HRV recovery during a subsequent rest phase, with SDNN as the HRV index. Women with higher levels of chronic stress exhibited significantly lower HRV reactivity and recovery during and after acute stress compared to women with lower levels of chronic stress, indicating impaired autonomic flexibility. Chronic fatigue did not significantly moderate these relationships. While perceived social support did not significantly buffer the effects of chronic stress on HRV outcomes, trends suggested its potential role in improving HRV recovery. These findings emphasize the detrimental impact of chronic stress on autonomic regulation in women and highlight the importance of interventions to improve autonomic balance.

*Keywords:* chronic stress, heart rate variability, chronic fatigue, perceived social support, Trier Social Stress Test

**9.2 Abstract (Deutsch)**

Chronischer Stress beeinträchtigt die Funktion des autonomen Nervensystems (ANS) erheblich und führt zu negativen gesundheitlichen Folgen, insbesondere bei Frauen. Die Herzratenvariabilität (HRV), ein sensibler Marker der ANS-Regulation, spiegelt die Anpassungs- und Erholungsfähigkeit des Körpers auf Stress wider. Diese Masterarbeit untersucht den Einfluss von chronischer Erschöpfung und wahrgenommener sozialer Unterstützung auf die HRV-Reaktivität auf und -Erholung nach akutem Stress bei chronisch gestressten Frauen. Dabei wurden drei Forschungsfragen addressiert: (1) Sagt das chronische Stresslevel die HRV-Reaktivität und -Erholung nach akutem Stress vorher? (2) Moderiert chronische Erschöpfung die Beziehung zwischen chronischem Stress und der HRV-Reaktivität/-Erholung nach akutem Stress? (3) Moderiert wahrgenommene soziale Unterstützung die Beziehung zwischen chronischem Stress und der HRV-Reaktivität/-Erholung nach akutem Stress moderiert, sodass höhere Unterstützung zu einer gesteigerten HRV-Reaktivität und -Erholung führt? Es wurde eine Querschnittsstudie mit 31 deutschsprachigen Frauen im Alter von 20 bis 65 Jahren durchgeführt. Die HRV-Reaktivität wurde während des Trier Social Stress Tests gemessen, die HRV-Erholung in der anschließenden Ruhephase, wobei SDNN als HRV-Index diente. Frauen mit hohem chronischem Stress wiesen signifikant geringere HRV-Reaktivität und -Erholung während und nach akutem Stress auf als Frauen mit niedrigerem chronischem Stressniveau, was auf eine eingeschränkte autonome Flexibilität hinweist. Chronische Erschöpfung moderierte diese Zusammenhänge nicht signifikant. Während wahrgenommene soziale Unterstützung die Auswirkungen von chronischem Stress auf die HRV-Ergebnisse nicht signifikant abpufferte, deuteten deskriptive Trends auf eine potenzielle Rolle bei der Verbesserung der HRV-Erholung hin. Diese Ergebnisse betonen die negativen Auswirkungen von chronischem Stress auf die autonome Regulation bei Frauen und die Bedeutung von Interventionen zur Verbesserung des autonomen Gleichgewichts.

*Schlüsselwörter*: chronischer Stress, Herzratenvariabilität, chronische Erschöpfung, wahrgenommene soziale Unterstützung, Trier Social Stress Test