Defensive freezing reaction: The effect of acute psychosocial stress on body movement and heart rate

Bachelor's Thesis in Medical Engineering

submitted
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Übersicht

Stress ist ein allgegenwärtiges Element des täglichen Lebens und seine Dysregulation durch chronische oder übermäßige Belastung kann die Gesundheit eines Individuums beeinträchtigen. In stressreichen Situationen aktiviert der Körper das sympathische Nervensystem (SNS) und die Hypothalamus-Hypophysen-Nebennieren-Achse (HPA-Achse), was zu einem erhöhten Cortisolspiegel und vermehrten Entzündungsmarkern führt. Neuere Forschungsergebnisse deuten darauf hin, dass Stress auch Körperhaltung und Bewegungen beeinflusst. Insbesondere bedrohungsbasierte Stressoren können ein "Freezing"-Verhalten beim Menschen auslösen. Wenn Menschen mit einer unmittelbaren, als bedrohlich empfundenen Situation konfrontiert sind, können sie instinktiv in einen Freeze-Zustand übergehen. Defensives Freezing ist durch zwei Hauptmerkmale gekennzeichnet: verminderte Körperbewegung und Bradykardie, eine verringerte Herzfrequenz. Wenn beide Bedingungen gleichzeitig auftreten, kann das Verhalten als defensives Freezing eingestuft werden.

Ziel dieser Studie war es, das Verständnis der menschlichen Stressreaktion durch die Analyse von Körperhaltung, Bewegung und Herzfrequenz während einer akuten Stresssituation zu vertiefen. Mit einem Motion-Capture-System und Elektrokardiogramm (EKG)-Überwachung wurden Daten von 41 Teilnehmenden erhoben, die sowohl dem Trier Social Stress Test (TSST) als auch seiner freundlicheren Variante, dem friendly Trier Social Stress Test (f-TSST), in zufälliger Reihenfolge an zwei aufeinanderfolgenden Tagen ausgesetzt wurden. Beide Tests boten Bedingungen, um Bewegungs- und EKG-Daten in stressauslösenden Situationen zu untersuchen.

Um das Freezing-Verhalten als Reaktion auf einen psychosozialen Stressor zu bewerten, wurden Bewegungsdaten zunächst analysiert, um statische Phasen oder Momente minimaler Bewegung zu identifizieren. Diese wurden im Anschluss mit EKG-Daten kombiniert, um Herzfrequenzänderungen während dieser Intervalle zu untersuchen. Durch diesen Ansatz konnte eine verfeinerte Definition für Freezing-Phasen im Kontext länger andauernder Stressereignisse entwickelt werden, die sich mit den in früheren Studien etablierten Kriterien deckt. In einer weiteren Analyse wurde die defensive Freezing-Reaktion in Bezug auf das Stressevent untersucht. Die Ergebnisse zeigten, dass die Teilnehmenden während der TSST-Bedingung eine ausgeprägtere Bradykardie und längere statische Phasen aufwiesen – typische Merkmale des defensiven Freezing-Verhaltens. Eine klare Korrelation zwischen der Häufigkeit der Freezing-Phasen und der Stressbedingung konnte jedoch nicht festgestellt werden. Diese Analyse bietet eine vielversprechende Grundlage für künftige Forschung, insbesondere für Studien mit größeren Datensätzen, um die Rolle des Freezing in der menschlichen Stressreaktion und seine gesundheitlichen Implikationen weiter zu erforschen, sowie die Faktoren zu identifizieren, welche die defensive Freezing-Reaktion beeinflussen könnten.

Abstract

Stress is an ever-present element of daily life, and its dysregulation through chronic or excessive levels can significantly impact individual health. In stressful situations, the body quickly activates the sympathetic nervous system (SNS) and the hypothalamic-pituitar-adrenal (HPA)-axis, leading to increased cortisol levels and heightened inflammatory markers. Emerging research shows that stress also influences body posture and movement. Notably, threat-based triggers can lead to a "freezing" behavior in humans. When faced with an immediate and threatening situations, humans may instinctively enter a freeze state. Defensive freezing is characterized by two primary markers: reduced body motion and bradycardia, meaning a slowed heart rate. When both conditions are present, the behavior can be classified as defensive freezing.

This study aimed to deepen the understanding within the human stress response by analyzing body posture, movement, and heart rate during an acute stress situation. Using a motion capture system and electrocardiogram (ECG) monitoring, data were collected from 41 participants who were exposed to both TSST and its friendly variant, the f-TSST in a randomized order over two consecutive days. Both tests provided conditions for examining motion and ECG data during stress-inducing situations.

To assess freezing behavior in response to a psychosocial stressor, motion data were first analyzed to identify static periods, or moments of minimal movement, which were then paired with ECG data to examine heart rate changes during these intervals. Through this process, a refined definition for freezing periods to prolonged stress events was developed, aligning with criteria established in previous research. In further analysis the defensive freezing reaction was examined in regard to the stress event. Results indicated that during the TSST condition, participants exhibited stronger bradycardia and longer static periods - characteristics of defensive freezing behavior. However, a clear correlation between the frequency of freezing periods and the stress condition could not be established. This analysis provides a promising foundation for future research, especially studies with larger datasets, to further explore the role of freezing in human stress response and its potential implications for health and underlying factors which could influence the defensive freezing reaction.

Contents

Chapter 1

Introduction

The human stress response is a complex and adaptive mechanism, to help individuals respond effectively to threats and challenges. This intricate system is activated during perceived danger or stress, mobilizing both physical and psychological resources to manage the situation and ultimately promote survival [Bur21]. Although stress is an unavoidable part of daily life, excessive or chronic stress can disrupt these adaptive mechanisms, leading to a variety of health problems. Stress dysregulation, triggered by prolonged or intense stress, can adversely impact both physical and mental well-being, and has been implicated in various health conditions [McE98].

When a threat is perceived, the body initiates a series of physiological responses, primarily via activation of the HPA axis and the SNS. Central to the stress response, the HPA axis triggers the release of cortisol, a hormone that manages energy resources, regulates immune function, and enhances alertness. Simultaneously, the SNS releases catecholamines, such as adrenaline, that increase heart rate, blood pressure, and blood flow to muscles, thus preparing the body for a ”fight or flight” response [Eve19]. However, sustained activation of these systems and elevated cortisol levels have been linked to health risks, including immune suppression, cardiovascular disease, metabolic disorders, and mental health issues such as anxiety and depression [Ock95]. Chronic SNS activation, in particular, strains the cardiovascular system, contributing to hypertension and increasing the risk of heart disease [AJ14].

Recent research has also highlighted the importance of the freezing response as a third mode of reacting to psychosocial stressors, particularly intense or inescapable ones [Roe10]. This reaction is thought to serve an evolutionary purpose, enhancing survival by minimizing movement and thus reducing the likelihood of detection in threatening situations [Bla86]. Physiologically, freezing involves a marked reduction in movement and a pronounced slowing of heart rate, known as bradycardia, and is driven by parasympathetic nervous system (PNS) activity. Originally

identified in animal studies in response to physical threats, freezing behavior is now also observed in humans under psychosocial stress [Hag14a]. Analyzing the freezing response in this context offers unique insights into human stress physiology beyond traditional ”fight or flight” responses. By examining markers such as heart rate variability, bradycardia, and reductions in movement, researchers can gain a more nuanced understanding of individual differences in stress responses. These insights are valuable for advancing diagnostic tools and targeted interventions, especially for individuals highly sensitive to stress or susceptible to stress-related health conditions. Furthermore, examining freezing behavior may clarify distinctions between adaptive and maladaptive responses, contributing to preventative strategies for stress-induced health concerns.

In laboratory settings, psychosocial stress is often triggered using scenarios that involve social evaluation, such as public speaking or performance tasks under observation. The TSST, involving a public speaking component and a challenging arithmetic task, is widely regarded as the gold standard for inducing acute psychosocial stress [Kir93]. As a control, the friendly TSST (f-TSST) offers a stress-free condition to compare against stress-inducing scenarios [Wie13]. Understanding freezing behavior and its physiological markers within these experimental frameworks provides a valuable perspective on how stress responses manifest in both behavioral and physiological domains.

While studies confirm freezing responses to psychosocial stress, they often examine either movement or heart rate individually rather than as an integrated defensive response. This thesis aims to address this gap by systematically investigating the full freezing response with the reduced movement alongside bradycardia in psychosocial stress contexts without a set stimulus. The freezing response is investigated by a study focusing on movement and heart rate data recorded from 41 participants during acute stress. These subjects were exposed to both the TSST and f-TSST in a randomized order across two consecutive days, within the framework of the *EmpkinS collaborative research center* [Emp21]. Body posture and movement data were captured using a inertial measurement units (IMU)-based motion capture (MoCap) system, while heart rate was monitored via ECG. By identifying periods of minimal movement, referred to as static periods, and examining corresponding heart rate changes, this study aims to assess whether defensive freezing behavior occurs in response to psychosocial stress and to develop a definition for freezing within this context. Further analysis explores whether freezing behavior correlates with increased stress levels, thereby positioning freezing as a potential marker of stress intensity and resilience. This work aims to contribute to the broader understanding of the human stress response and to pave the way for future research on the physiological and psychological underpinnings of stress-related freezing behavior.

Chapter 2

Related Work

Body movement, a key nonverbal communication channel, plays a crucial role in conveying emotional states and intentions. Humans have a natural ability to interpret emotional cues through body posture, facial expressions, and movement patterns. While emotions such as happiness, anger, and anxiety are often easily identifiable through nonverbal signals, identifying the specific movement patterns associated with these emotions is still an area of ongoing research across various disciplines [Wal86; Gel15; Zac14]. A deeper understanding of body movement, especially in relation to stress and the defensive freezing response, has emerged through work in emotion recognition, stress responses, and the study of freezing in both animals and humans.

Emotion Recognition

Body movement is a powerful medium for emotion recognition, capable of conveying subtle emotional states and social intentions. Early work in emotion recognition primarily focused on facial expressions, notably through Ekman et al.'s Facial Action Coding System (FACS), which categorizes facial muscle movements into basic emotions like happiness, fear, and anger [Ekm78]. However, body movement and posture offer additional information about emotional states and intentions, prompting researchers to explore this modality more deeply. Modern applications can assess emotion recognition in humans for expressions like happiness, anger, and neutrality. An example for such a tool is *EmBody/EmFace*, which uses isolated cues from body movements, reduced to point-light displays (PLD), and cropped facial features. In these studies, participants could accurately differentiate emotions based solely on body movement or facial expressions [Lot22]. Both dynamic and static body expressions have also been shown to communicate basic emotions, such as fear, happiness, and sadness. For instance, Atkinson et al. used PLD to represent basic

movements, showing that even minimal body representations effectively convey emotion [Atk04]. Expanding on this, Dael et al. demonstrated that body posture can reflect not only emotions but also future intentions , revealing complex psychological states beyond basic emotions [Dae12]. This increasing body of research highlights that body movement is crucial for emotion recognition, providing insight into nuanced emotional and psychological states. For example a study by Van der Zee et al. examined whether body movement could distinguish liars from truth-tellers. Dividing participants into instructed "lying" and "truth-telling" groups, researchers tracked their movements using IMU-based motion capture systems. Analysis showed that participants instructed to lie exhibited more movement, likely due to fidgeting. A binary logistic regression based on these motion capture data achieved a 74.4 % accuracy rate in distinguishing lying from truth-telling [Zee19].

Detection of illnesses

Body movement analysis has proven valuable for detecting various psychological and physiological conditions. For instance, gait analysis has shown promising results in detecting high inflammation. Lasselin et al. examined gait patterns in patients with induced lipopolysaccharide (LPS), a compound known to elicit immunological and behavioral changes [Las18]. Compared to the placebo group, participants with injected LPS exhibited shorter, slower, and wider strides, reduced arm extension, limited knee flexion, and a downward-tilted head, indicating that movement patterns provide information on inflammation status [Las20]. Another study by Lasselin et al. explored whether facial cues can signal health status. Participants were divided into an LPS group and a placebo group, and facial photos were taken for evaluation. Observers identified certain features, such as pale lips or skin, as indicators of sickness, underscoring that subtle facial changes can reflect immune activation [Axe18]. Similarly, depression often manifests in distinctive movement patterns, such as slumped posture, slower gait, and reduced overall movement. Feldmann et al. found that depressed individuals displayed decreased gait velocity and poorer balance [Fel20]. Facial recognition technologies, like *AutoDep*, have also been effective in detecting and assessing depression severity through subtle facial cues with high accuracy [Tad21].

These studies suggest that both physiological conditions and complex psychological states, such as depression, may be inferred from specific movement patterns and facial cues. This presented research lays the groundwork for examining how other disruptions in movement and expression, such as those induced by stress, might reveal additional psychological phenomena.

Stress and Movement

The relationship between stress and movement has received growing attention, especially in the context of psychosocial stress. Stress, a common aspect of modern life, can influence posture and movement patterns. For instance, a study by Shahidi et al. explored the effects of increased workload-induced stress on workplace posture, finding that heightened stress led to a more pronounced forward head posture and increased muscle activity in the upper trapezius, while cervical extensors and flexors remained unaffected. This suggests that stress impacts not only internal functions but also visibly affects posture [Sha13]. In another study, Aigrain et al. examined body movement and facial features during a socially evaluated arithmetic task, which triggers a stress response [Dic04]. They evaluated body movement metrics - such as movement quantity, high-activity periods, posture changes, self-touching - alongside with facial features based on the FACS [Ekm78]. Using a support vector machine (SVM), the study achieved 77 % accuracy in stress detection, with facial indicators like brow movement, posture changes, and body activity levels serving as prominent stress markers [Aig15]. A recent study conducted in the *EmpkinS collaborative research center* by Richer et al. investigated body movement during the TSST, a widely applied psychosocial stress protocol. Findings showed that participants exhibited reduced movement under stress, suggesting that decreased movement may serve as an outward indicator of stress; the stress condition was classified with 80 % accuracy based on these findings [Ric22].

Freezing

Reduced movement is well-established as a part of the defensive freezing response studied in animals facing physical threats. Blanchard et al. first documented freezing as a behavior marked by reduced movement and bradycardia in response to predator threats [Bla86]. This “fight, flight or freeze” response enhances survival by minimizing detection in dangerous situations [Bra04].

Recently, there has been growing interest in human freezing responses, especially concerning both physical and psychosocial threats. Hagenaars et al. compared animal and human freezing, concluding that animal studies provide valuable insights into human freezing behavior [Hag14a]. In one study, participants showed significantly reduced body sway when watching emotionally charged video scenes, such as car accidents, compared to calm scenes, suggesting that freezing can be triggered by psychosocial stressors as well as physical threats [Hag14b]. Roelofs et al. further explored this by examining body sway in 50 female participants viewing images of emotionally charged faces. Reduced body sway was noted in response to angry faces, which were interpreted as

social threats, and this correlated with heart rate deceleration (bradycardia), indicating a freezing response triggered by social cues [Roe10]. These findings support the idea that freezing is not solely a response to physical threats but also to social stressors. A likely explanation for freezing is that it serves as a preparatory state before engaging in action. While traditionally viewed as a passive fear response, Roelofs and Dyan propose that freezing may be an active preparatory state, with parasympathetic activation preceding the sympathetic ”fight or flight” response [Roe22]. There is also interest in understanding freezing in individuals with mental health differences. Hashemi et al. examined police recruits and a comparable group of healthy participants in a rapid-decision-shooting task to induce freezing. Those with lower hair cortisol concentrations and trait anxiety - both markers of long-term stress coping - showed higher rates of postural freezing, indicating a link between the HPA axis activity and the freezing response [Has21]. Although research has primarily been focused on freezing responses to physical threats, psychosocial stressors like public speaking, social evaluation, and relationship conflicts increasingly show that social stressors can evoke freezing responses. For instance, the TSST reliably induces psychosocial stress, where participants display reduced movement under evaluative stress [Abe22]. This was further investigated by Richer et al. revealing that periods of no movement were significantly longer and more frequent during the stress condition compared to the control condition. Incorporating these periods alongside other features, their analysis led to a machine learning pipeline capable of classifying the stress condition from the control condition with an accuracy of 73.4 ± 7.7 % [Ric24b].

Chapter 3

Methods

3.1 Data Acquisition

This thesis was written with one main dataset [Ric24a]. The dataset was acquired in a study conducted by the Machine Learning and Data Analytics Lab in 2022. The participants performed the TSST and the f-TSST on two consecutive days in randomized order.

3.1.1 Study Population

The used dataset for this thesis consisted of 41 total participants (18 female, 23 male). An overview with each condition order can be found in Table 3.1.

	Gender		
Condition order	**Female**	**Male**	Total
TSST first	10	12	22
f-TSST first	8	11	19
Total	18	23	41

Table 3.1: Gender and condition order overview

Participants were recruited for the study through flyers and advertisements. To be eligible, individuals had to be between 18 and 50 years old, possess proficient German language skills, and be in good health. This led to the exclusion of individuals with chronic diseases, Body Mass Index (BMI) above 30, or any mental health conditions. Additionally, participants were required to refrain from using medication or drugs, and they should not have prior knowledge of stress test

protocols. Master's psychology students were excluded from participation, as their curriculum likely familiarized them with the TSST, which could bias the results. Before enrolling in the study, potential participants were required to complete a digital screening questionnaire to ensure they met all the inclusion criteria. Upon completing the study, participants were offered a choice of compensation: either 50 Euros or 5 *Versuchspersonenstunden* (for psychology students).

Due to technical issues encountered in the data acquisition during the (friendly) Tier Social Stress Test ((f-)TSST) not all data could be utilized for evaluating the defensive freezing response. For some participants, the ECG data was not successfully recorded ($n = 2$), for some participants the MoCap data showed errors ($n = 2$) leading to their exclusion from the data analysis. As a result, this led to a revised distribution of the remaining participants included in the study shown in Table 3.2.

	Gender		
Condition order	**Female**	**Male**	Total
TSST first	8	12	20
f-TSST first	8	9	17
Total	16	21	37

Table 3.2: Final gender and condition order overview

3.1.2 Acute Stress Induction

To induce a state of acute psychosocial stress, the TSST was employed, which is widely regarded as the gold standard for eliciting a stress response in research settings [Kir93]. As a control condition, the f-TSST was used with an added math part. Although structurally similar to the TSST, the f-TSST is designed to avoid triggering a stress response in participants [Wie13]. The overview for the used protocol of both the TSST and f-TSST can be found in Figure 3.1.

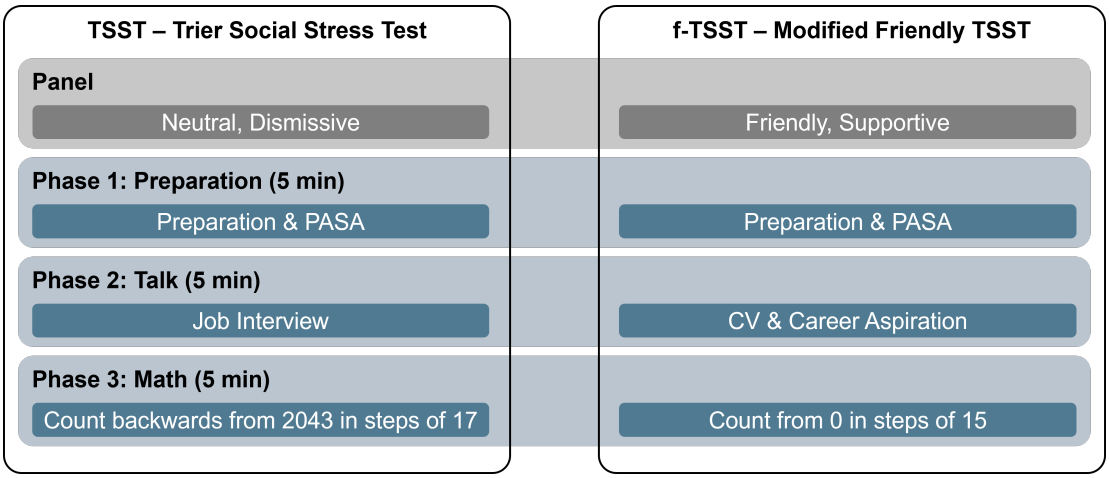

Figure 3.1: Protocol for both TSST and f-TSST

TSST

The TSST was conducted in front of a panel consisting of two people, one female and one male. The member of the panel with the opposite gender to the participant took on the role of the active interviewer, while both were dressed in white lab coats to maintain a professional and neutral appearance. The panel was instructed to remain neutral throughout the entire procedure, refraining from any noticeable reactions or feedback in response to the participant's behavior. If the participant paused during their speech, the panel was required to observe a 20 second period of silence before responding or prompting further interaction. In general the panel should talk as little as possible, while the participant had the majority of the share of speech.

The TSST protocol was structured into three distinct phases, each lasting 5 minutes, for a total duration of 15 minutes. Upon entering the testing room, the participant was introduced to the scenario by the study leader. They were asked to imagine that the panel members were recruiters from their ideal company, and they were now undergoing a high-stakes job interview where they had to convince the panel that they were the perfect fit for the position in talking only about their personality. Following this briefing, participants were instructed to sit down and given time to prepare for the upcoming interview. After 3 minutes they were reminded to fill out a questionnaire to record situation-related cognitive evaluation, the primary appraisal secondary appraisal (PASA) (Phase 1). When the preparation time concluded, the active panel member invited the participant to stand and begin their presentation. During this phase, the participant had to present themselves and their qualifications to the panel (Phase 2). Once the interview portion was completed, the

participant was presented with an unexpected arithmetical task. They were instructed to count backwards from 2043 in steps of 17. If they made an error, they were required to restart from 2043 (Phase 3). After completing the arithmetical task, the TSST was concluded, and the participant was dismissed from the room.

f-TSST

In this study, the f-TSST also consisted of three 5 minute phases: a preparation phase, an interview phase, and a math phase. However, unlike the TSST, the panel did not wear lab coats and was supportive and friendly, with panel members allowed to ask questions and engage in conversation.

The procedure for the f-TSST began similarly to the TSST, with the study leader leading the participant to the room. There, the study leader introduced the panel members by their first names and instructed the participant to talk about their CV during the interview phase. After these instructions were given, the participant was asked to sit down, take time to prepare for the interview and fill out the PASA questionnaire (Phase 1). During the preparation phase, the active panel member temporarily left the room. Once the preparation phase was complete, the participant stood in front of the panel to present their CV. In contrast to the TSST, the panel was allowed to ask questions, and the talk phase was designed to resemble a relaxed, friendly conversation. The math phase in the f-TSST was also simplified compared to the TSST. Here, participants were asked to count upwards from 0 in increments of 15. If they made a mistake, they were simply corrected and asked to continue from the last correct number rather than restarting the task (Phase 3).

3.1.3 Procedure

Pre-Test Phase

Upon arriving at the lab, the participant was escorted to the preparation room by the study leader, where they were asked to complete a consent form and different questionnaires. The study leader provided a brief overview of the study procedure, and the first saliva sample was collected to measure the baseline cortisol level. After this initial collection S_0, participants were given 200 ml of grape juice, or glucose water in cases of fructose intolerance, to help stabilize the cortisol response [All17]. To assess the participants' cortisol levels throughout the study, a total of 8 saliva samples were collected at specific time points, as outlined in Table 3.3.

Relative time [min]	-40	-1	0-15	+16	+ 25	+35	+45	+60	+75
Saliva samples	S_0	S_1	(f-)TSST	S_2	S_3	S_4	S_5	S_6	S_7
Passive drooling sample *		P_0							

Table 3.3: Saliva samples relative to (f-)TSST start
*only for female participants on the second day

On the second day of the study, all female participants were instructed to provide a passive drooling sample P_0. This sample was used to assess progesterone levels in order to determine the participant's menstrual cycle phase, helping to control for the effects that different phases of the cycle might have on cortisol measurements in women [All17]. Following this, the study leader measured the participant's weight, BMI, and muscle mass index. Additional body measurements, such as height, arm length, and hip height, were also taken to calibrate the *Xsens MVN Awinda* MoCap system (Xsens, Enschede, Netherlands), a MoCap system based on 17 IMU sensors designed to track body movement. These sensors were then securely attached to the participants' respective body parts using Velcro straps, along with an ECG sensor (Portabiles GmbH, Erlangen, Germany) fastened by a chest strap. The specific placements of the Xsens sensors are shown more detailed in section 3.1.4 under the MoCap system section. Before the Xsens system could begin recording movement data, it had to be calibrated by performing an N-Pose and Movement sequence, as defined by the Xsens MVN system manual [BV19]. Just before the (f-)TSST began, the second saliva sample S_1 was collected.

(f-)TSST

Approximately 40 minutes after the preparation phase began, participants were brought to the room where the (f-)TSST was conducted. The entire procedure for both tests is described in section 3.1.2. Throughout the process, both MoCap and ECG data were continuously collected. After the preparation phase of the (f-)TSST, video recordings of the participant's face and full body were also initiated. A *Sony SRG-300H RGB* camera (Minato, Japan) captured facial footage, while a *Microsoft Azure Kinect RGB-D* camera (Redmond, WA) recorded the participant's full body movements. To ensure accurate synchronization of the different data during the analysis phase, the panel used a smartphone application to track different phases of the (f-)TSST.

Post-Test Phase

After completing the (f-)TSST, participants were guided back to the preparation room, where additional saliva samples (S_2 to S_7) were collected. Participants were also asked to complete further questionnaires. To gather the remaining saliva samples S_2 to S_7, participants had to wait for an additional time to collect the samples at their respective time as reference in Table 3.3. At the conclusion of the session, participants were either reminded about their next test session scheduled for the following day (Day 1), or they were given a short debriefing, provided with information about the study, and asked to sign a non-disclosure agreement to ensure the integrity of the study's procedures (Day 2).

3.1.4 Measurements

Motion Capture Data

The motion capture data is acquired by a *Xsens MVN* software at a sampling rate of 60 Hz. After recording, the data is saved as a native Xsens file format (.mvn). The recordings were cropped to only include the (f-)TSST and then exported to MVN open XML format (.mvnx). These files include frame-wise information about all 23 body segments and 22 joints, as well as data of 17 IMU sensors, center of mass, and foot contacts. An overview of the different segments and their respective body parts group can be found in Table 3.4, additionally the placement of these segments can be found in Figure 3.2.

Body part group	**Body Parts**
Trunk	Pelvis, Spine (L3, L5, T8, T12), Neck
Upper Body	(L/R) Shoulder, (L/R) Upper Arm, (L/R) Fore Arm, (L/R) Hand
Lower Body	(L/R) Upper Leg, (L/R) Lower Leg, (L/R) Foot, (L/R) Toe
Total Body	All segments

Table 3.4: Xsens segments with group definitions. (L/R) both left and right side

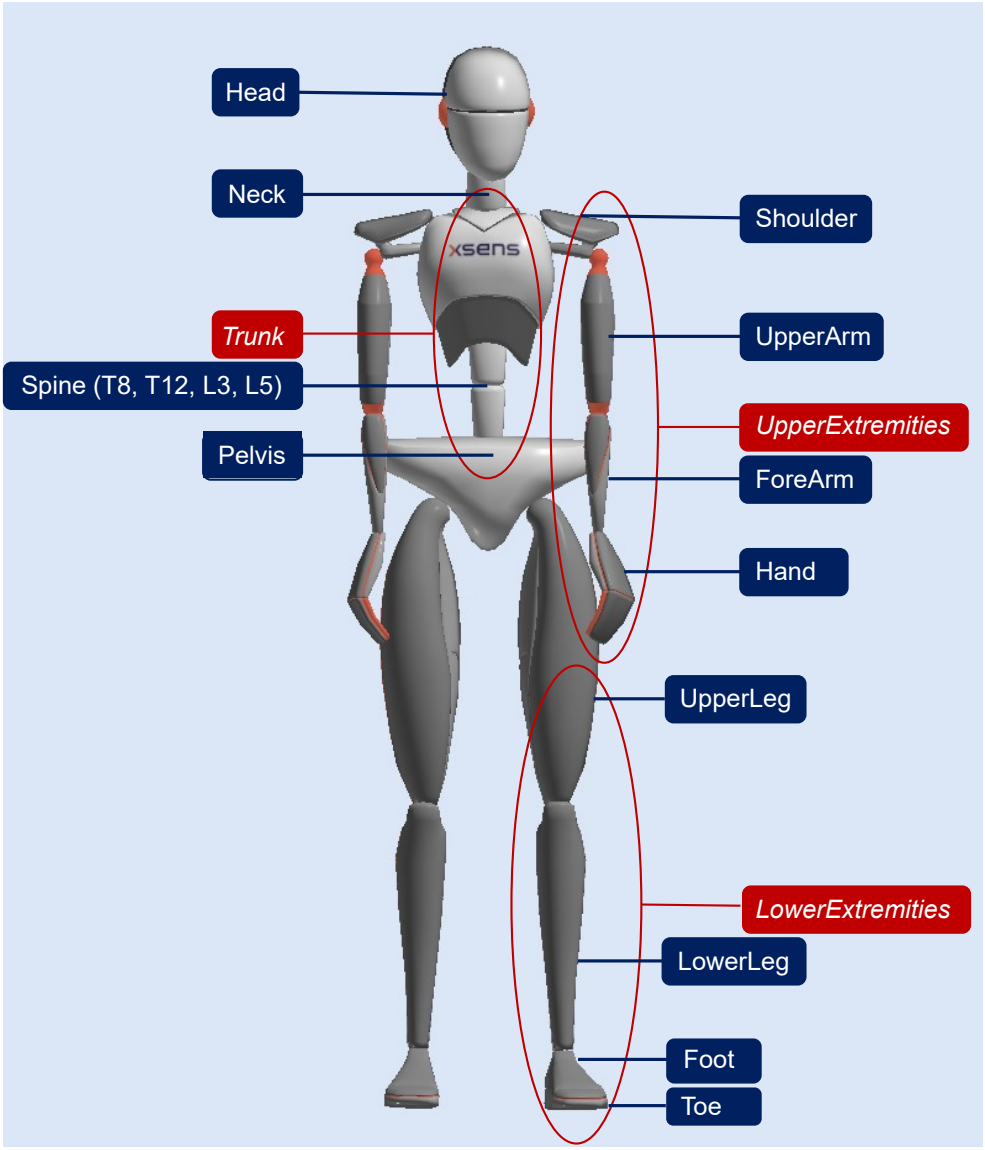

Figure 3.2: Xsens body part definition. Body parts are labeled in blue, body part groups are labeled in red.

ECG Data

The ECG data were recorded by using a wearable sensor node (Portabiles GmbH, Erlangen, Germany) attached to a chest strap. The sensor node records a 1-channel ECG according to Lead I of Einthoven's Triangle and logs data onto the internal storage with a sampling frequency of 256 Hz for subsequent data processing on a computer.

Endocrinological data

During each study day in total 8 saliva samples as seen in Table 3.3 were collected per participant to identify the cortisol levels and therefore the activation of the HPA axis as a response to the stress event. Two saliva samples were taken before the (f-)TSST as a baseline indicator and the other ones were taken periodically after the test as described in Table 3.3. The samples were collected with *Salivettes* (Sarstedt AG & Co. KG, Numbrecht, Germany), a swab collection device. After the collection, the samples were kept at room temperature until the end of the day and afterwards stored at –18 °C in a freezer until the cortisol concentrations were examined in a laboratory as described in previous work by Richer et al. [Ric21].

Based on the saliva samples, there were three different parameters derived to quantify the activation of the HPA axis: The maximum cortisol increase Δc_{max}, the area under the curve with respect to ground AUC_g and the area under the curve with respect to increase AUC_i.

The maximum cortisol increase is defined as the difference of the highest cortisol sample value after the (f-)TSST (S_2 to S_7) compared to the baseline before the (f-)TSST S_1 stated in Equation 3.1.

$$\Delta c_{\text{max}} = \max(S_i) - S_1, \quad \forall i \in [2, 7] \tag{3.1}$$

The area under the curve was computed to assess the total amount of released cortisol after the start of the (f-)TSST. The AUC_g is computed according to the following Equation 3.2 using the trapezoidal rule, where S_i is the cortisol value at time point t_i and Δt_i is the time difference between two consecutive samples S_i and S_{i+1} in minutes.

$$AUC_g = \sum_{i=1}^{6} \frac{(S_{i+1} + S_i)}{2} \cdot \Delta t_i \tag{3.2}$$

Additionally, the area under the curve with respect to increase AUC_i was computed. Here the area under the curve is computed in the same way as stated in equation 3.2, but the value of the baseline sample S_1 is subtracted to only gain the value of the cortisol increase in response to the (f-)TSST. The AUC_i is defined in Equation 3.3.

$$AUC_i = \left(\sum_{i=1}^{6} \frac{(S_{i+1} + S_i)}{2} \cdot \Delta t_i \right) - (6 \cdot S_1) \tag{3.3}$$

3.2 Movement and Heart Rate Analysis

3.2.1 Static Periods

In this study, static periods are defined as intervals during which participants exhibit minimal movement, as measured across the full set of MoCap data of each subject and condition. Only segments of the (f-)TSST were examined where participants were positioned in front of the panel, specifically focusing on the speaking and math portions of the (f-)TSST. This approach yielded approximately 10-minute time frames for each subject and condition to identify static periods. These periods are calculated based on detected reductions in movement intensity. Static periods were defined as the definition stated by Abel et al. [Abe22]. The information about the body movement was obtained by the different Xsens sensors in the *vel* and *gyr* channels. The signal was analyzed in 0.5 s windows with an overlap of 50 % and subsequently the variance (Var_w) of the L2 Norm of the 3-dimensional (3D) vectors within the window was computed ($\|\mathbf{x}_t\|_2$). When the variance of a window was below a certain threshold θ_{SP} it was classified as static and consecutive static windows were marked as longer static periods. The θ for the *vel* channel was set to $1 \times 10^4\,(\text{m/s})^2$ while the threshold of the *gyr* was set to $5\,(\text{deg/s})^2$. These thresholds were determined by previous test data which was obtained with simulated static periods and led to successful results in previous work with this dataset [Abe22]. To determine the whole body freezing reaction only static periods which did not only include single body parts, but the total body as defined in Table 3.4 on the *gyr* channel were used in this thesis. Using this definition the start and the end time of each static period was obtained and later used to examine the heart rate in these time windows.

$$\text{Var}_w\left(\|\mathbf{x}_t\|_2\right) = \frac{1}{T}\sum_{t=1}^{T}\left(\|\mathbf{x}_t\|_2 - \frac{1}{T}\sum_{t=1}^{T}\|\mathbf{x}_t\|_2\right)^2$$

$$\text{if} \quad \text{Var}_w\left(\|\mathbf{x}_t\|_2\right) < \theta_{\text{SP}}, \quad \text{then window is static}$$

This analysis focused on examining heart rate changes over time within defined static periods. To ensure that these static periods provide meaningful insights into heart rate variability, only those lasting at least 3 seconds were included in the analysis. Given that an average human heart rate ranges between 60 to 100 bpm, heartbeats occur approximately every 0.5 to 1 second [Cou94]. Static periods shorter than 3 seconds, being less than the duration of multiple heartbeats, would not yield a reliable picture of heart rate variability. Thus, only longer static periods were chosen to ensure accurate detection of heart rate changes.

The heart rate was derived from the ECG signal, as detailed in section 3.1.4. Firstly the signal was cleaned and filtered to prepare it for future evaluation. A key feature of the ECG signal is the R-peak, which corresponds to the depolarization of the ventricles and is the most prominent feature of the ECG waveform. The R-peak marks a clear point of distinction for each heartbeat, making it a reliable marker for analyzing cardiac activity. Due to its sharp and consistent nature, the R-peak is frequently used in heart rate analysis, as it allows for accurate detection of each cardiac cycle. Once all R-peaks were identified in the ECG signal, the detected R-peaks were corrected by imputing removed outliers with linear interpolation. The heart rate was then calculated by measuring the time interval between two consecutive R-peaks, referred to as the RR-interval. Following formula was used to calculate the heart rate in beats per minute (bpm):

$$\text{Heart Rate (bpm)} = \frac{60}{\text{RR-interval (in seconds)}}$$

This formula converts the RR-interval, measured in seconds, into heart rate by determining how many beats occur in a 60-second period. This method provides a continuous measure of heart rate over time, allowing for precise tracking of heart rate changes during the study. For the heart rate evaluation of the ECG signal the *biopsykit* package, developed by Richer et al. [Ric21] was used. Within the same time frames as described in 3.2.1, heart rate was also analyzed and afterwards synchronized with body movement data. This combined plotting allowed for a more comprehensive view of the relationship between heart rate and body movement.

3.2.2 Freezing Periods

For the defensive freezing reaction, it is especially important to look at the heart rate over time during the extracted static periods. In other studies, a clear stressor is present and therefore the heart rate can easily be compared to the baseline before the stress event and therefore a drop in heart rate can be detected [Roe10; Roe17]. In this study there was not a punctual stressor, but a stress event over time, the TSST. So, detecting freezing had to be adapted. Since freezing is defined by the absence of body movement accompanied by bradycardia, static periods were used as the basis for identifying freezing events. The specific time frames for analyzing heart rate were defined as immediately before, during, and after the static periods. If a static period also showed a decrease in heart rate, it was classified as a freezing period. In this thesis, the definition of freezing behavior was derived from findings in previous studies. To determine whether a specific static period could be characterized as freezing, a baseline heart rate was established. This baseline was calculated as the mean heart rate during the 2 seconds immediately preceding the onset of

the static period. This approach ensures that the baseline reflects the participant's heart rate just before the moment of stillness, providing a clear reference point for assessing changes during the static period itself. This is marked in the Figure 3.3 as the baseline in the darker shade of blue.

Building on previous studies that identified a critical time window for observing heart rate reductions in response to stressors between 5 and 6 seconds after stimulus onset [Voo22], this thesis applied a similar framework. Rather than focusing on external stimuli as the examined time window, the analysis centered on the moments following the beginning of a static period. This decision was based on the assumption that the freezing response would manifest within a similar time frame following a reduction in movement. To accommodate variance for different subjects the area of interest was extended to the interval between 4 and 7 seconds after the start of the static period as seen in Figure 3.3 in light blue.

To classify a static period as freezing, the mean heart rate during that period was compared against the pre-established baseline. A significant drop in heart rate between the baseline and the heart rate observed in the defined area of interest was used as a key indicator of freezing. Specifically, a decrease of at least 3 bpm was established, as this more substantial drop was more likely to indicate genuine bradycardia associated with freezing. If these criteria were met, the entire static period was classified as freezing.

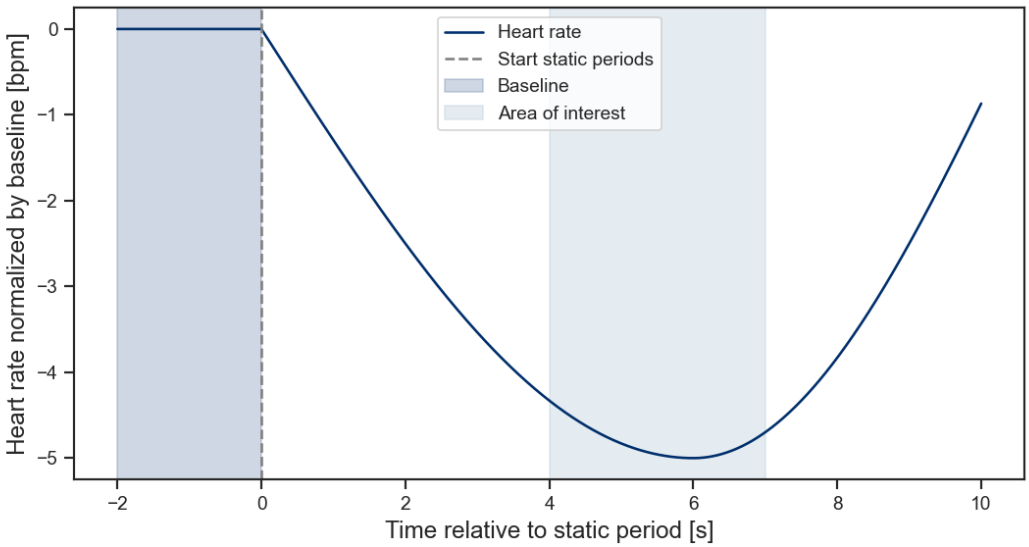

Figure 3.3: Example heart rate progression for freezing

This method of classification was designed to ensure that only periods showing a clear physiological response - rather than random fluctuations - were considered instances of freezing. By setting a defined threshold for heart rate reduction and applying it consistently across participants, this thesis aimed to provide a robust and reliable identification of freezing periods as stated in Table 3.5.

Baseline	**Area of interest**	**Change in Heart Rate**
-2 < t < 0	4 < t < 7	< -3 bpm

Table 3.5: Definition of freezing with the start of the static period being t_0 = 0 s

3.2.3 Statistics

To analyze the dataset, the distribution was initially assessed using the Shapiro-Wilk test to determine if it followed a normal distribution, which guided the choice of the appropriate statistical analysis. Each participant in the dataset experienced both a stress condition, the TSST, and a control condition, the f-TSST, enabling paired comparison. Given that the dataset did not meet parametric assumptions, the Wilcoxon signed-rank test was used as a non-parametric alternative to the paired t-test. Here the TSST and f-TSST served as within-subject variables.

The statistical analyses were conducted with the *biopsykit* package, developed by Richer et al. [Ric21], which utilizes Python's *pingouin* library for robust statistical computations. The significance threshold was set at $\alpha = 0.05$.

Chapter 4

Results and Discussion

While there is evidence that freezing occurs in response to psychosocial stress detailed in section 2, few studies have systematically examined the full defensive freezing response - characterized by both reduced body movement and bradycardia - in psychosocial contexts without a set stimulus. Research has primarily focused on either movement or heart rate, but not both in combination. This gap highlights the need for a comprehensive investigation of the freezing response in psychosocial stress scenarios over time, which this thesis aims to address.

4.1 Endocrine Response

The cortisol progression shown in Figure 4.1 illustrates a twofold increase in cortisol levels during the TSST. Specifically cortisol levels more than doubled, rising over 100 % from the baseline sample S_1 to the peak sample S_3 during the TSST. In contrast, the f-TSST condition showed only a modest increase of approximately 10 %, resulting in a notably lower cortisol response. This trend aligns with previous literature [Kir93; Wie13], which also reports smaller responses for control conditions. However, to maximize the contrast between conditions, the ideal control condition would produce no cortisol response at all. Thus, the TSST will be referred to as the stress condition, while the f-TSST functions as the control condition.

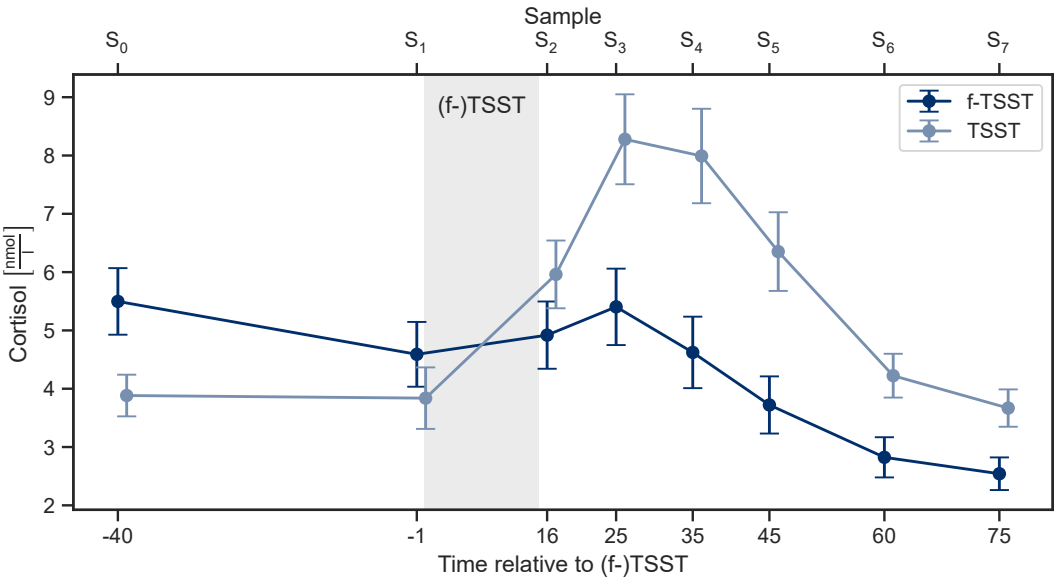

Figure 4.1: Cortisol response of all participants for (f-)TSST with mean ± standard error (SE)

Among the parameters used to quantify cortisol responses, the maximum cortisol increase $\Delta c_{\max}$ has proven to be most effective in prior analysis with this dataset [Abe22]. Consequently, this parameter is selected here to quantify cortisol responses. Using the 2.5 $\frac{\text{nmol}}{\text{l}}$ threshold for maximum cortisol increase $\Delta c_{\max}$, as suggested by Wiemers et al., classifies each subject and condition as either a stress response or not. The distribution of cortisol responses and conditions, shown in Table 4.1, suggests that the conditions do not perfectly align with cortisol responses. For analysis of freezing in combination with stress response, a differentiation will be made between study conditions (TSST and f-TSST) and the 2.5 $\frac{\text{nmol}}{\text{l}}$ threshold for $\Delta c_{\max}$ will identify responders who showed a maximum cortisol increase $\Delta c_{\max}$ bigger than 2.5 $\frac{\text{nmol}}{\text{l}}$ and non-responders who exhibited a maximum cortisol increase lower than the determined threshold. This approach allows differentiation not only between stress and control conditions but also between participants exhibiting a stress response and those who did not. For $n = 2$ participants and for $n = 1$ during the f-TSST condition, no evaluation of the saliva samples was possible, hence they were excluded regarding the cortisol analysis. This resulted in a new distribution visible in Table 4.1.

	TSST	**f-TSST**	**Total**
Cortisol Response	21	5	26
No Cortisol Response	14	29	43

Table 4.1: Distribution of the of the classification of cortisol response for (f-)TSST

4.2 Body Movement and Heart Rate

To initiate the analysis, static periods were identified for each participant and condition, following the criteria outlined in the previous chapter 3.2. Additionally the heart rate, extracted from the ECG signal as outlined in section 3.2, was simultaneously analyzed.

In Figure 4.2, the heart rate for a single subject during the TSST condition is plotted, with static periods marked based on the definition from section 3.2. Within this time frame, heart rate for this subject varied between 74.2 and 104.5 bpm. Although heart rate variability was noticeable, no distinct pattern emerged at first glance. The light blue shading indicates static periods, which varied considerably in duration, though most were longer in duration. For this individual, static periods clustered more in the second half of the time frame, aligning with the math section of the TSST.

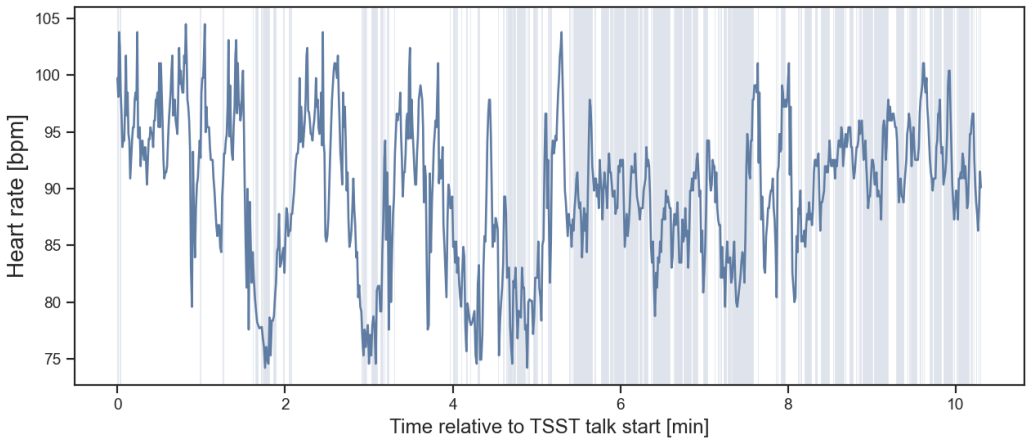

Figure 4.2: Exemplary heart rate of one subject during the TSST condition with all static periods marked

Figure 4.3 illustrates the heart rate progression for the same subject in the control condition, allowing comparison with Figure 4.2 (TSST condition). Heart rate fluctuates similarly in both conditions, ranging between 75.3 and 111.3 bpm during the f-TSST, and no distinct differences emerge when viewing the heart rate progression alone between the stress and control conditions.

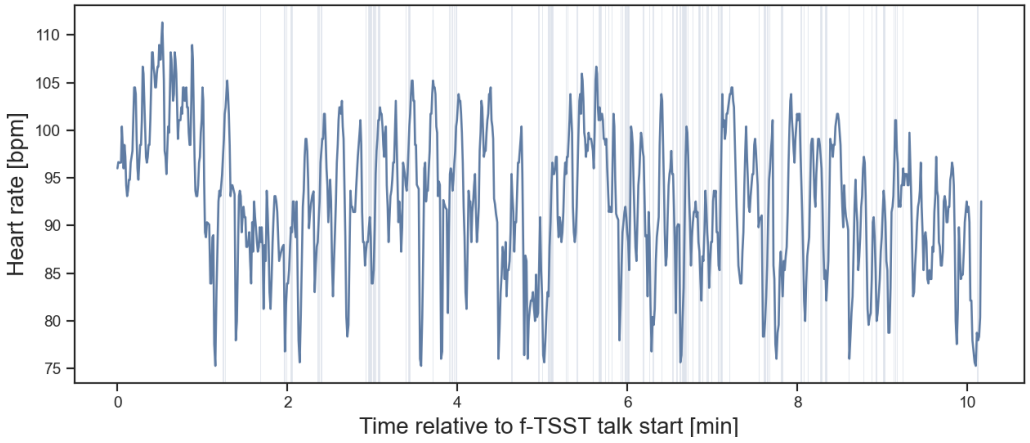

Figure 4.3: Exemplary heart rate of one subject during the f-TSST condition with all static periods marked

However, differences in static periods between conditions are evident. In the f-TSST, fewer static periods are detected, and those present are notably shorter, resulting in an overall reduction in the duration and frequency of static periods over time. This distinction in static periods suggests a divergence in bodily response patterns between the stress and control conditions, potentially offering insight into the impact of stress on movement behavior as suggested by Abel et al. [Abe22].

As stated in section 3.2.1, only static periods longer than 3 seconds were included, resulting in a considerable reduction in the number of static periods available for analysis. Figure 4.4 shows an example of static periods marked over time for one participant, with a minimum duration of 5 seconds.

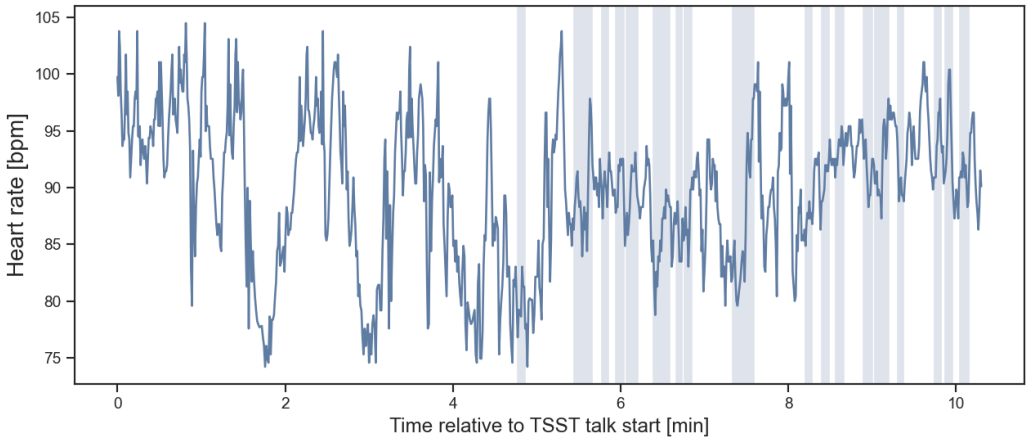

Figure 4.4: Exemplary heart rate of one subject during the TSST condition with min 5 Sec static periods marked

The f-TSST condition, which involved shorter and fewer static periods overall, contributed fewer suitable static periods for the analysis due to the lack of continuous stillness across the time frame, as noted in prior work [Ric24b]. For the provided example subject during the f-TSST condition, no static periods over 5 seconds could be detected. This difference further underlines the observed distinctions in body movement patterns between the stress and control conditions.

To gain a better understanding of the available data worked with, Table 4.2 shows the number of static periods under certain time constraints for the minimum duration of the static periods.

	Min 3 Sec	**Min 5 Sec**	**Min 7 Sec**
TSST	429	193	102
f-TSST	182	58	24
Total	611	251	126

Table 4.2: Number of static periods with different time constraints

4.3 Static Periods and Heart Rate

At first all the longer static periods with a minimum duration of 3, 5 and 7 seconds were identified in the dataset as detailed in Table 4.2. The longer the static periods, the smaller the available dataset was, so there had to be found a good compromise between having a meaningful dataset and being able to focus on longer time frames. The main duration of static periods worked with in this thesis are the ones with a minimum of 5 sec, as they provide enough data to make a meaningful conclusion and align with the considerations about the change in heart rate.

An approach to analyze the dataset over time was to plot the heart rate of each static period on top of each other and compare how the heart rate behave altogether before and after the start of the static period and to see whether a conclusive trend is visible. In Figure 4.5, the progression of the heart rate across all static periods of at least 5 seconds in duration was plotted and averaged, resulting in a dataset of 251 static periods from both the TSST and f-TSST conditions. To enhance comparability, heart rate data was normalized against a baseline calculated as the mean heart rate over the 2 seconds leading up to the start of each static period. This baseline mean was then subtracted from all data points to focus on relative heart rate changes within each period. All further plots which show the normalized heart rate were adapted this way to enhance overall comparability.

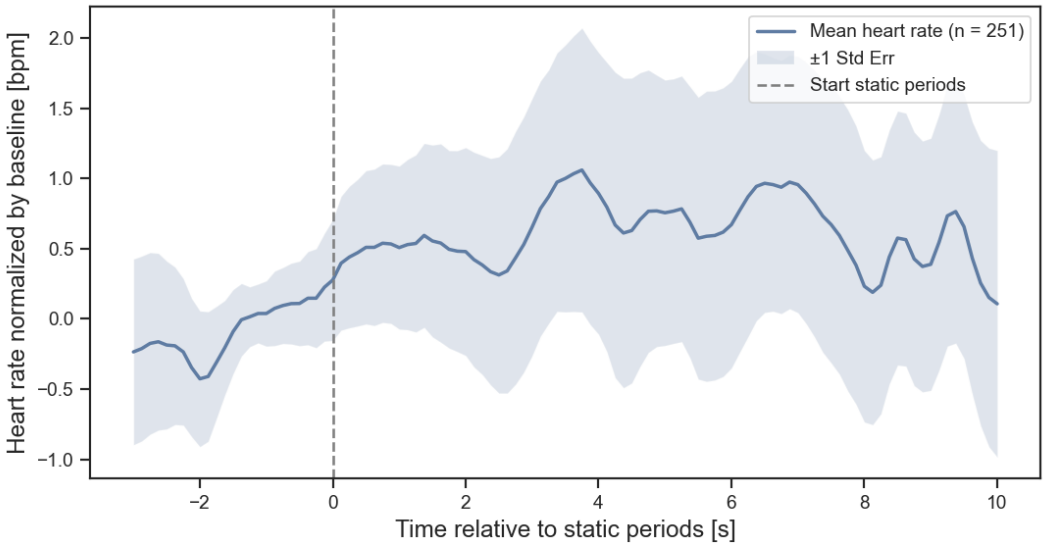

Figure 4.5: Mean heart rate of all 5 sec static periods normalized by baseline

The heart rate progression revealed no clear trend of decrease after the onset of static period. Instead, a gradual increase was observed, though with huge variability and no distinct pattern. The large SE further limited the possibility of drawing any definitive conclusions or establishing clear trends in heart rate response during static periods. Looking at the distribution of static periods in Figure 4.2, it is evident that these periods often appear in rapid succession, occasionally interspersed with short intervals of detected movement. In cases where static periods occur in close sequence, the boundary between active and static states is less clear, as these periods often blend into each other. Since static periods were defined based on a threshold of body movement, minor fluctuations slightly above this threshold were marked as active, potentially compromising the accuracy of distinguishing between active and static phases. This threshold-based approach complicates the detection of transitions into truly static phases, which are necessary for reliably identifying a freezing response. To address this, an ”active baseline” was introduced, excluding any static periods that occurred immediately following another static period. This baseline was designed to act as a marker for significant shifts in body movement, offering a clearer starting point for potential freezing episodes.

Consequently, both a minimum duration and an active baseline were applied as constraints for selecting static periods. In this refined subset, only static periods lasting at least 5 seconds were included, and any periods that were preceded by another static period within 2 seconds before their start were excluded. This resulted in a subset, which will be referred to as the *Baseline 2*

subset. This adjustment reduced the number of static periods available for analysis but provided a more promising dataset for detecting freezing. The updated distribution of these refined static periods is presented in Table 4.4, offering a clearer framework for analyzing heart rate changes linked to distinct static episodes.

Figure 4.6 displays the static periods selected for analysis for the example participant. In contrast to Figure 4.2, which includes all detected static periods, this refined dataset reflects a substantial reduction in data. This reduction was necessary to improve the quality of analysis and to provide a more reliable basis for investigating the presence of freezing episodes within the dataset. By applying stricter selection criteria, non-representative short and sporadic static periods were filtered out, allowing for a clearer assessment of whether freezing behavior can be consistently observed in the remaining, more stable static periods.

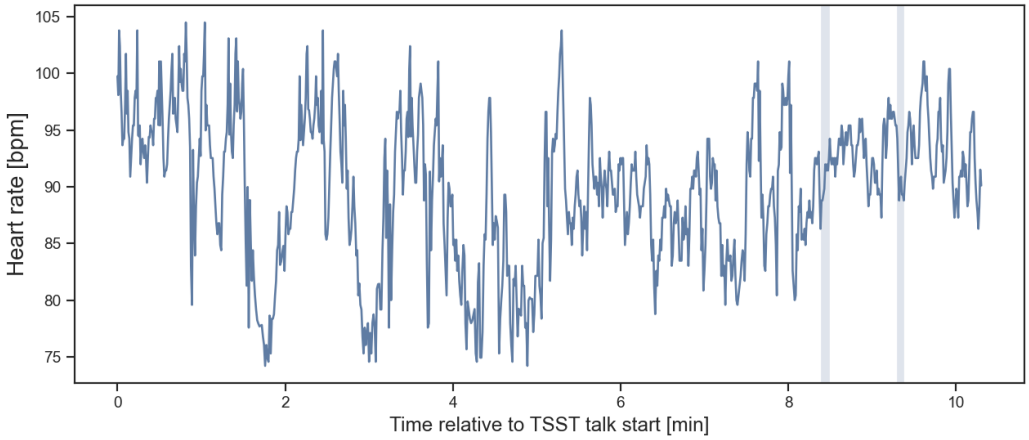

Figure 4.6: Exemplary heart rate of one subject during the TSST condition with all static periods in *Baseline 2* marked

The newly established subset, referred to as *Baseline 2*, consists of 36 static periods derived from all participants during the TSST condition and 9 static periods from the f-TSST condition. Although the total number of static periods is considerably smaller than in previous analysis, data from this refined selection appears to yield promising results. Unlike the findings displayed in Figure 4.5, which lacked distinct patterns, the dataset *Baseline 2* suggests clearer trends and behaviors that may better indicate the presence of freezing responses as seen in Figure 4.7. This indicates that the stringent selection criteria applied have enhanced the dataset's quality, allowing for a more nuanced understanding of the dynamics at play during these static periods.

In comparison to the subset labeled *Min 5 Seconds*, which includes all static periods lasting at least 5 seconds, a noticeable decrease in heart rate is observed immediately after the start of the static period in the *Baseline 2* analysis in Figure 4.7. Following some minor fluctuations in heart rate, a clear decline of approximately 2.5 bpm is evident around the 4 second mark. This reduction persists for roughly 2 seconds, after which the heart rate begins to rise slowly back towards the levels recorded prior to the onset of the static period. This pattern suggests that the heart rate reduction typically associated with freezing responses may not manifest until approximately 4 seconds into the static period and may last until the 6 seconds mark. Additionally, the 2 second baseline measured before the commencement of the static periods appears more stable in this subset compared to the baseline observed in Figure 4.5. This stability may lend support to the idea of introducing an active baseline, which serves as a more reliable indicator of physiological responses. The time frame during which bradycardia occurs aligns with findings reported by Roelofs et al. [Roe22; Voo22], reinforcing the notion that a defined period of reduced heart rate is characteristic of freezing behavior.

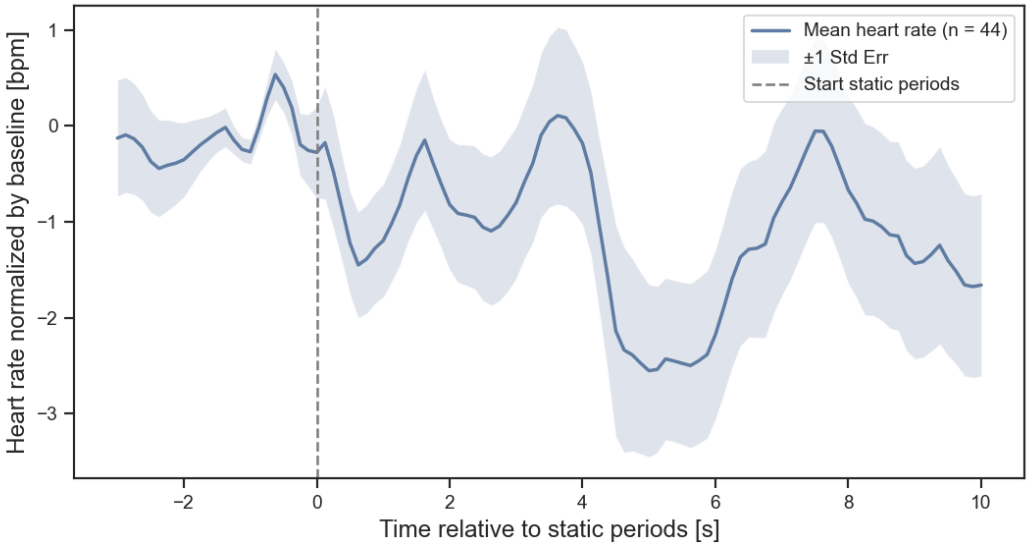

Figure 4.7: Heart rate progression of static periods within the *Baseline 2* subset with marked beginning of static periods

While these results appear promising, a significant limitation of the current definition is that only about 17.5 % of the 5 second static periods were included, resulting in a small fraction of overall static periods being analyzed. As shown in Table 4.4, reducing the active baseline requirement before the start of the static periods to just 1 second considerably increases the number

of eligible periods, effectively doubling available data for analysis. Consequently, the final subset used for the majority of subsequent figures is the one containing a minimum duration of 5 seconds for static periods, along with a baseline of at least 1 second. This subset is now referred to as *Baseline 1* and its definition is stated in Table 4.3. The used adjustment allows for a more robust analysis while still maintaining a focus on detecting potential freezing responses within the dataset.

	Baseline 1	**Baseline 2**
Active baseline	> 1 second	> 2 second

Table 4.3: Definition of *Baseline 1* and *Baseline 2* subsets. Active baseline prior to start of static period

		All static periods	**Baseline 1**	**Baseline 2**
Min 3 Sec	TSST	429	182	67
	f-TSST	182	69	32
	Total	611	251	99
Min 5 Sec	TSST	193	73	35
	f-TSST	58	21	9
	Total	251	94	44
Min 7 Sec	TSST	102	35	19
	f-TSST	24	8	4
	Total	126	43	23

Table 4.4: Number of static periods with different time constraints and active baseline

4.4 Freezing Periods

After thoroughly analyzing the dataset and examining the heart rate dynamics, a definition for freezing during acute psychosocial stress events has been established, as detailed in section 3.2. Utilizing this refined definition, the dataset was scrutinized for the defined freezing periods. Ultimately, a total of 30 distinct freezing periods were identified within the *Baseline 1* subset while 19 freezing periods could be detected for the *Baseline 2* subset. To further understand the freezing reaction, the heart rate reduction associated with each of these identified freezing periods was quantified in Figure 4.8 for the *Baseline 1* subset and in Figure 4.9 for the *Baseline 2* subset. This analysis aims to explore the characteristics and patterns of heart rate changes that

accompany freezing behavior in response to psychosocial stress, providing deeper insights into the physiological responses that occur during such events.

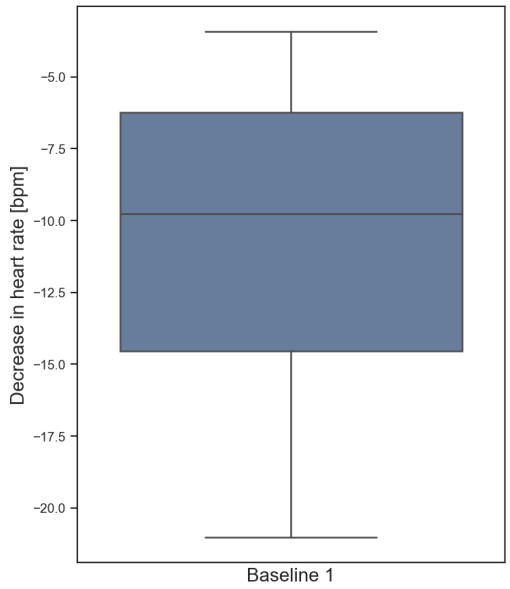

Figure 4.8: Freezing bradycardia in *Baseline 1* subset

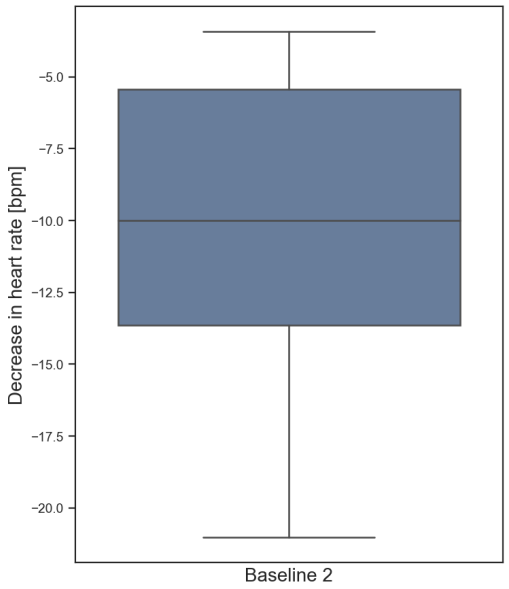

Figure 4.9: Freezing bradycardia in *Baseline 2* subset

The boxplots reveal a range of bradycardia intensities, with the median heart rate reduction consistently hovering around -10 bpm. When comparing the ratio of freezing to static periods across the two subsets, notable differences emerge as seen in Table 4.5. In the *Baseline 1* subset, the freezing ratio resulted in 31.9 %, whereas in the *Baseline 2* subset, this ratio increases to 43.2 %.

	Min 5 Seconds	*Baseline 1*	*Baseline 2*
Freezing periods	64	30	19
Static periods	251	94	44
Ratio	25.5 %	31.9 %	43.2 %

Table 4.5: Freezing ratio across different subsets

This increase suggests a more pronounced freezing effect when the periods preceding the static intervals are definitively characterized as active. It implies that the effects of any prior

static periods, which could have diminished the freezing response, are less likely to interfere in the *Baseline 2* conditions. This indicates that the introduction of an active baseline provides a good constraint for further analysis of the defensive freezing reaction. Though, similar to the introduction of minimum duration, the 2 second baseline leads to a huge loss in available data. This verifies the considerations made in section 4.3 and approves the focus on the *Baseline 1* subset in the further analysis. However, no significant difference in the intensity of bradycardia was observed between the two subsets, indicating that while the freezing response may be seem to occur more likely, the physiological impact on heart rate remains relatively consistent across both subsets. If the heart rate is examined more closely over time, a similar progression to the static plot in Figure 4.7 is visible in Figure 4.10.

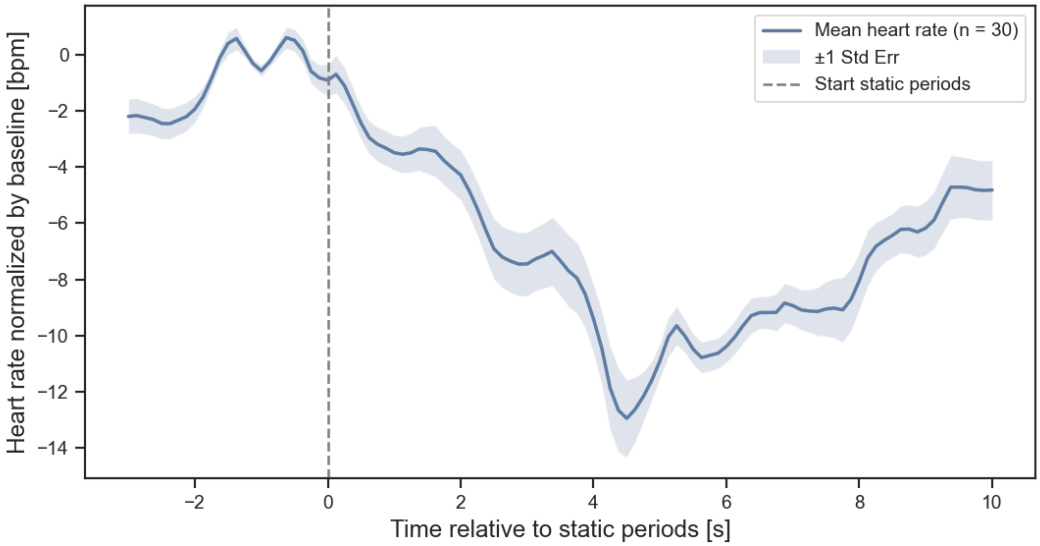

Figure 4.10: Freezing periods with heart rate over time in *Baseline 1* subset

Following the onset of the static period, characterized by a reduction in movement, the heart rate exhibited a consistent decrease for the first 4 seconds. The lowest recorded heart rate marked a notable decrease of 12.9 bpm compared to the active baseline described in section 3.2.2. Subsequently, the heart rate began to rise gradually. However, even after 10 seconds, it remained approximately 4 bpm below the established baseline. These findings align closely with existing research on the freezing response, further validating the observed patterns of bradycardia associated with the cessation of movement during acute psychosocial stress events.

4.5 Defensive Freezing Reaction as a Stress indicator

For stress research it is of great benefit to discover parameters apart from saliva samples and questionnaires to find out more about the stress state of the patient and gain a more holistic understanding of the human stress response. Since it was already established that during the stress condition participants showed a higher amount of static periods [Ric24b]. This approach can be advanced to analyze how the stress response and freezing correlates.

Table 4.6 show the distribution of static periods during the (f-)TSST. If the mere number of freezing periods is compared it is also visible that during the TSST there were more absolute freezing periods. If the ratio of freezing periods is compared in each condition a 30.1 % ratio for the TSST and a 38.1 % ratio for the f-TSST is found if the *Baseline 1* subset is analyze, making freezing periods compared to the overall static periods slightly more prevalent during the control condition.

	TSST	**f-TSST**
Static periods	73	21
Freezing periods	22	8
Percentage	30.1 %	38.1 %

Table 4.6: Ratio and distribution of freezing periods for the (f-)TSST

When examining the normalized heart rate during the freezing periods, a similar trend emerges in both the stress (TSST) and control (f-TSST) conditions as seen in Figure 4.11. In both scenarios, the heart rate decreases for approximately 4 seconds following the onset of the static period, after which a gradual increase is observed. However, notable differences exist between the two conditions. During the TSST, the heart rate experiences a more pronounced decline, reaching a minimum of -14.8 bpm below the baseline. In contrast, the lowest heart rate recorded during the f-TSST is at the -10.0 bpm mark, occurring around the 5 second mark. Overall, the heart rate progression in the control condition remains consistently higher than that in the stress condition, suggesting that the bradycardia response is more pronounced during the TSST. This observation highlights the differential impact of acute psychosocial stress on heart rate dynamics during freezing periods.

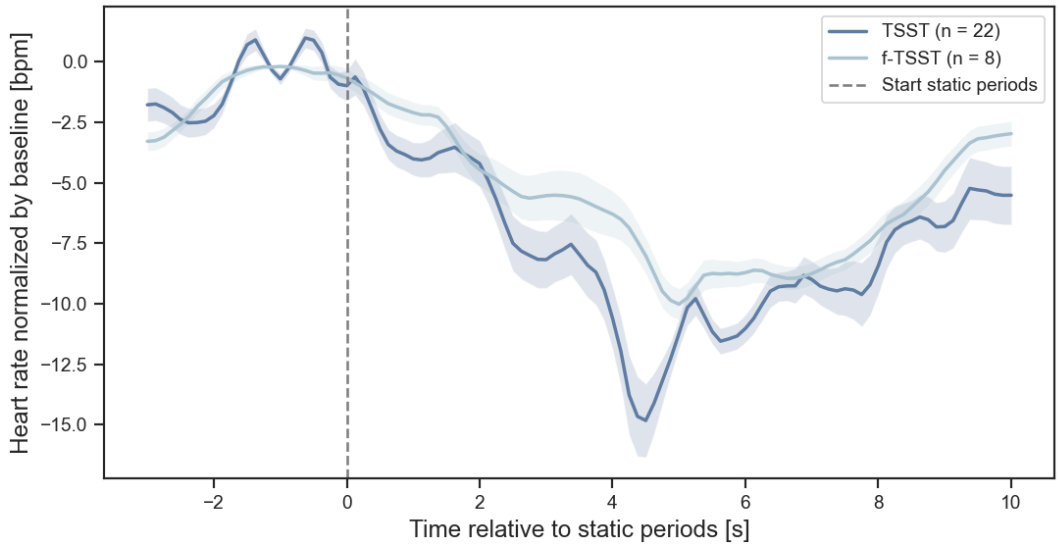

Figure 4.11: Mean heart rate progression during freezing periods for (f-)TSST ± SE

The findings presented in section 4.1 indicate that simply differentiating between control and stress conditions does not adequately account for variations in cortisol response. To provide a more nuanced analysis, the data will also be evaluated based on the presence or absence of a cortisol response. This distinction will be made using a threshold of 2.5 $\frac{\text{nmol}}{\text{l}}$ for the maximum cortisol increase Δc_{max}, as established by Wiemers et al. This approach aims to identify whether participants exhibit a significant cortisol response during the stress condition, thereby offering deeper insights into the relationship between cortisol levels and the physiological reactions observed during the experiments. The distribution for the freezing and static periods regarding the cortisol response can be found in Table 4.7

	Cortisol response	**No cortisol response**
Static periods	58	30
Freezing periods	17	12
Ratio	29.3 %	40.0 %

Table 4.7: Ratio and distribution of freezing and static periods regarding the cortisol response

Overall, the *Baseline 1* subset contains only 58 static periods in the where a cortisol response was observed, which translates to a 29.3 % ratio of freezing periods. In contrast, the ratio of freezing periods in settings where no cortisol response was detected is considerably higher at

40.0 %. This disparity suggests that the presence of a cortisol response may be linked to a slightly reduced incidence of observable freezing periods. This could highlight a potentially important relationship between physiological stress responses and the manifestation of freezing behavior. This trend was already explored in the pure distinction between the TSST and the f-TSST and seems reinforced analyzing the cortisol response. Figure 4.12 shows the heart rate progression for freezing periods regarding the cortisol response.

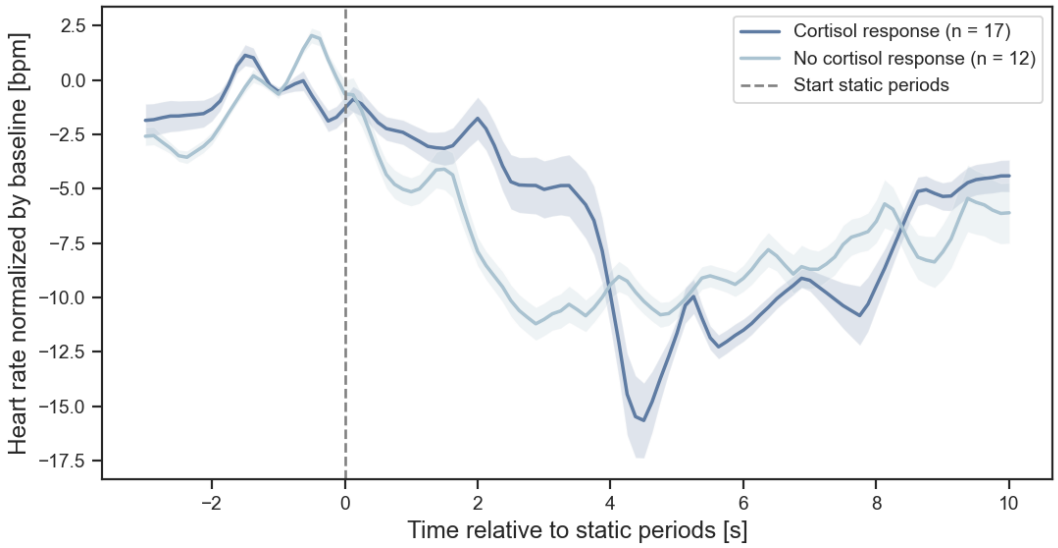

Figure 4.12: Mean heart rate progression during freezing periods for cortisol vs. no cortisol response ± SE

The progression of heart rate during freezing periods associated with a cortisol response demonstrates a slow decrease in the beginning up until approximately 3 seconds into the static period, reaching its nadir shortly after 4 seconds after a rapid decline at -15.7 bpm compared to the baseline. Following this low point, the heart rate accelerates rapidly before gradually returning to baseline levels. In contrast, freezing periods without a cortisol response reach their lowest point around 3 seconds after the start of the static period at -11.2 bpm before also rising back towards the baseline.

Figure 4.13 combines both the test condition as well as the cortisol response. Each condition is divided into cortisol responders and non-responders. The most pronounced decline in heart rate progression occurs during freezing periods that coincide with a cortisol response, reaching their respective lowest points at -16.6 bpm. At the onset of the static period, the heart rate progression for the TSST condition gradually decreases until approximately 4 seconds. Following this, a rapid

and sharp decline is observed, with the nadir occurring shortly after the 4 second mark. In contrast, for the freezing period detected within the f-TSST condition, the heart rate starts relatively low at the beginning of the static period, then rises quickly, only to experience a sudden drop at the 4 second mark. This decline continues until the heart rate reaches its lowest point approximately 5 seconds into the freezing period. Both conditions demonstrate a steep decline, with their lowest points occurring between 4 to 5 seconds after the freezing period begins. Freezing periods without a cortisol response, whether during the TSST or f-TSST, exhibit a more uniform progression. In these cases, the lowest point is -13.4 bpm for the TSST and -10.6 bpm for the f-TSST. These conditions show a gradual decline starting with the onset of the static period, reaching their lowest points around the 3 second mark, after which the heart rate slowly returns toward baseline.

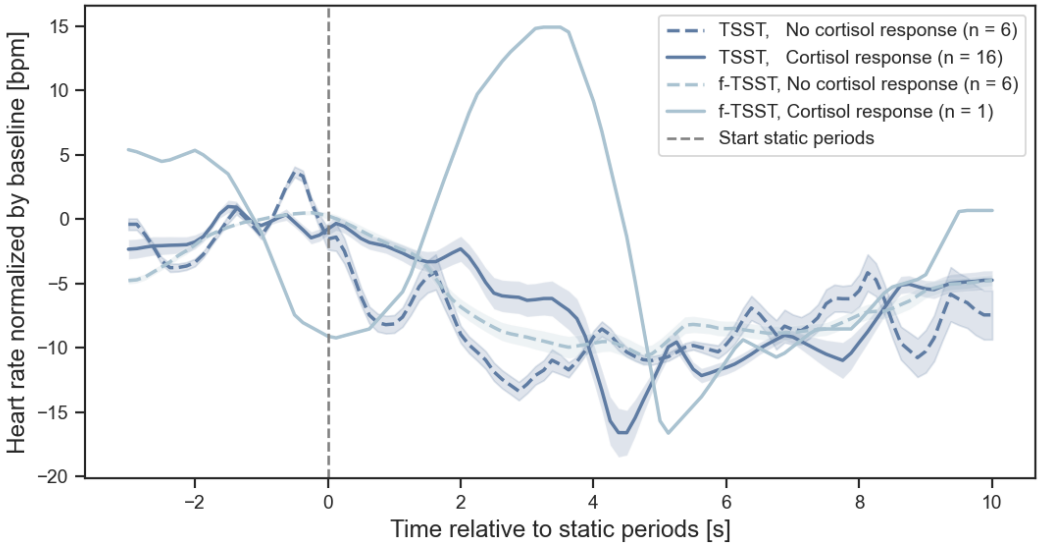

Figure 4.13: Mean heart rate progression during freezing periods for all conditions ± SE

These findings align with the trends observed in Figure 4.11 and Figure 4.12, suggesting that the presence of a stressor and a corresponding cortisol response amplify freezing-related bradycardia. Notably, the timing of the lowest points differs across conditions: in cases with a cortisol response the lowest point occurs later, typically after a brief but rapid fall.

The other parameter by which the freezing reaction can be identified is the duration of the bodily freezing reaction, which is symbolized by the static periods. Figure 4.14 illustrates the duration of freezing periods across different conditions. Notably, the data reveals that the duration of low movement phases during the TSST is longer (7.2 s $\pm$ 3.3 s) than those observed in the f-TSST (5.9 s $\pm$ 1.9 s). This finding aligns with the earlier analysis conducted by Richer et al., which highlighted longer overarching static periods during the TSST. The current analysis reinforces this conclusion by confirming that freezing periods exhibit a similar pattern, further emphasizing the pronounced impact of stress on movement duration during the TSST.

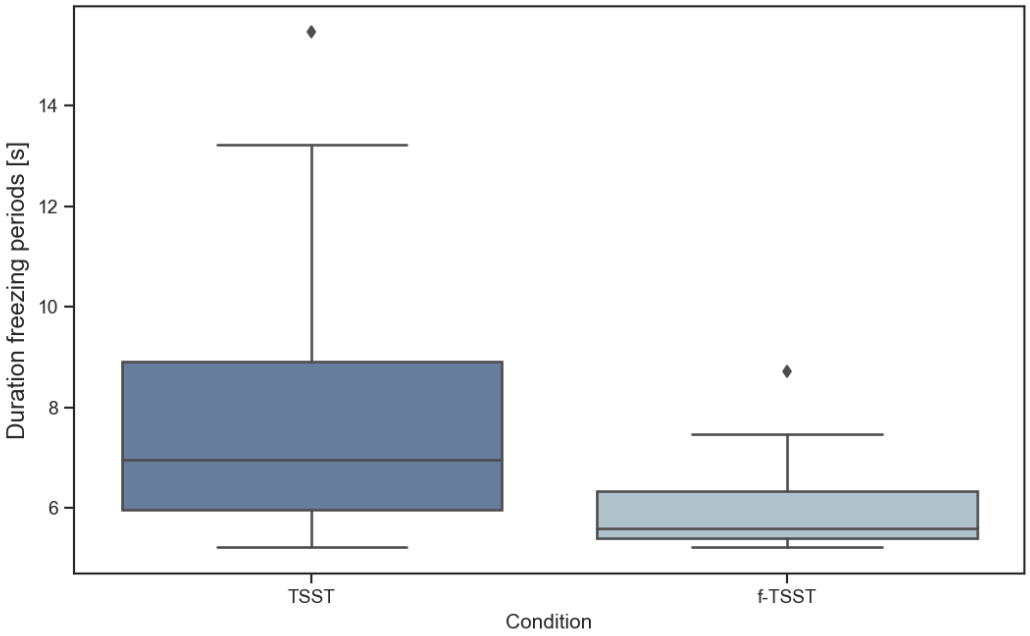

Figure 4.14: Duration of freezing periods by condition

Analyzing the key components of the defensive freezing reaction showed that both the bradycardia and the reduction in body movement were more pronounced for the stress condition than compared to the f-TSST. While the progression of the heart rate had a similar shape, the overall reduction in heart rate was more visible, both during the TSST as well as looking at the mere cortisol response (Figure 4.11 and Figure 4.12). Additionally, the duration of the static periods which exhibit freezing were longer during the TSST condition. Therefore it can be concluded that the intensity of the freezing periods seems to be more pronounced during the stress condition. On the other hand the overall ratio of freezing periods seems to be more prominent during the control

condition (Table 4.6) and when no cortisol response is measured (Table 4.7). This may indicate that freezing can even be a healthy reaction when experiencing stressful situations and may not only be associated with stress.

For the statistical analysis the mean bradycardia, the freezing ratio and the overall occurrence of freezing periods were compared for each participant during the stress and control condition. All examinations were conducted on the basis of the static and freezing periods found in the *Baseline 1* subset in the context of the overall occurrence of freezing periods.

Mean Bradycardia

Figure 4.15 shows the mean bradycardia for each subject and condition and puts it in relation within each participant. For the mean bradycardia, all static periods within a subject and conditions were examined and compared between the baseline and the area of interest, using the same time frames as stated in the definition for freezing (Table 3.5 and Figure 3.3). The baseline was then subtracted from the mean heart rate of the area of interest. If more than one static period occurred, the mean of all differences between the baseline and the area of interest was taken.

The heart rate changes during the TSST condition were less variable compared to the f-TSST condition, wheras the control condition showed huge variation in both increase and decrease. The mean heart rate change in the TSST was 0.7 bpm with a standard deviation (SD) of 7.3 bpm, while the f-TSST had a mean heart rate change of 1.1 bpm and a SD of 8.7 bpm. This makes the overall heart rate change slightly smaller for the stress condition as also seen for the freezing periods reflected in Figure 4.11. The lines connecting individual subjects were marked in yellow if the heart rate change increased for the TSST compared to the f-TSST, red if an decrease was detected. These direct connections showed a lack of a consistent directional change across all participants. Some subjects showed greater heart rate increases in the f-TSST, while others experienced higher changes in the TSST condition.

In this analysis, the Wilcoxon signed-rank test yielded a *p-value* of 0.7164, indicating no statistically significant difference in mean heart rate change during static periods between the TSST and f-TSST conditions. This high *p-value* suggests that any observed differences could be due to random chance rather than a systematic effect of the stress condition. Therefore, the null hypothesis - that there is no difference in heart rate change between the stress and control conditions - could not be rejected.

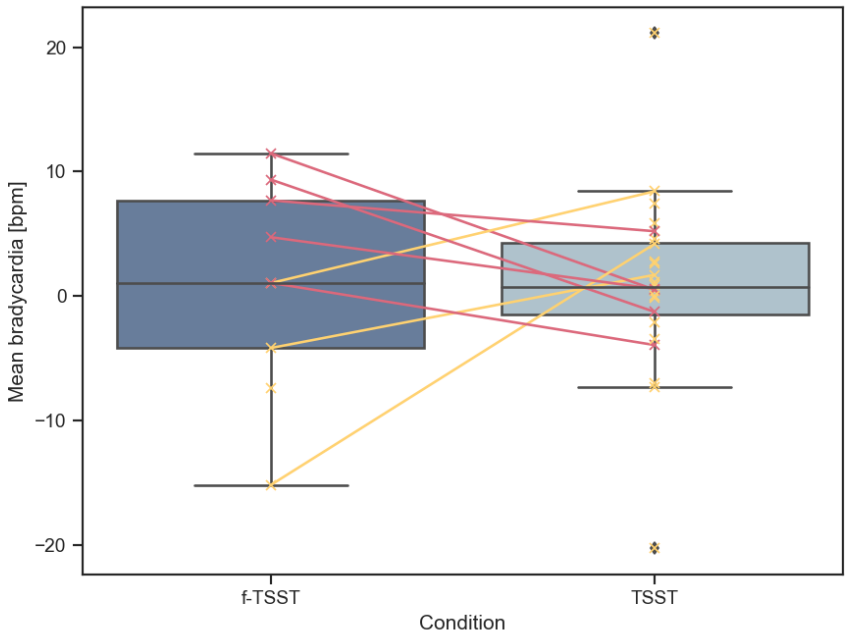

Figure 4.15: Paired boxplot for mean bradycardia for (f-)TSST with connected subjects

To address limitations in this approach, it is important to note that only subjects with static periods meeting the criteria of the *Baseline 1* subset were included in this analysis. This subset restriction led to a reduced sample size, as few participants exhibited qualifying static periods for both the stress condition (TSST) and the control condition (f-TSST). This limited overlap reduced the overall statistical power of the analysis, thereby impacting the generalizability of the findings.

Freezing Ratio

Another parameter compared between conditions for each subject was the freezing ratio, defined as the fraction of freezing periods relative to the total number of static periods observed for each subject in each condition. When no static period was detected for a given subject and condition, the freezing ratio was set to 0.

The boxplot shown in Figure 4.16 for the TSST condition reveals a wider distribution of freezing ratios, with a higher median compared to the f-TSST condition. In contrast, the f-TSST condition exhibits a much narrower range, with many participants showing a freezing ratio close to zero. This difference suggests that the freezing ratio tends to be higher and more variable in the TSST condition, supporting the hypothesis that the stress condition may trigger more frequent

or pronounced freezing behaviors. The difference in subjects is marked in yellow for an increase from the TSST compared to the f-TSST, whereas the lines marked in red indicate a decrease.

However, limitations in detecting static periods within the *Baseline 1* subset, especially in the f-TSST condition, may limit the effectiveness of the freezing ratio as a meaningful parameter for differentiating between stress and control conditions. Assigning a freezing ratio of 0 to participants without detected static periods may skew the results, potentially contributing to the low overall freezing ratio observed for the f-TSST condition. This issue underscores the challenge of accurately capturing stress-related differences when static periods are sparse or inconsistently distributed across conditions.

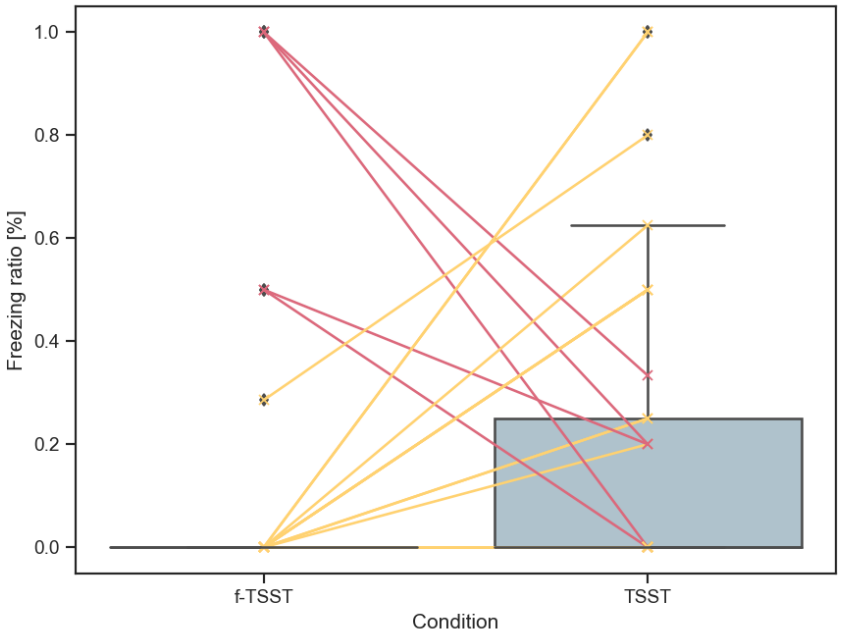

Figure 4.16: Paired boxplot for freezing ratio for (f-)TSST with connected subjects

The Wilcoxon signed-rank test for this parameter yielded a *p-value* of 0.2755, indicating no statistically significant difference in freezing ratio between the TSST and f-TSST conditions. This non-significance suggests that the freezing ratio may lack sensitivity as a reliable indicator of stress-induced responses.

Absolute number of Freezing Periods

Figure 4.17 analyzed the overall number of freezing periods for each subject and condition. The definition used to identify a freezing period is described in Table 3.5 which was applied to the *Baseline 1* subset. Altogether this plot suggests that the TSST condition generally elicited more freezing periods than the f-TSST, but the response was not uniform across participants. Just as for the other two parameters do the red lines indicate a decrease for the TSST condition compared to the f-TSST, in contrast the yellow lines display an increase.

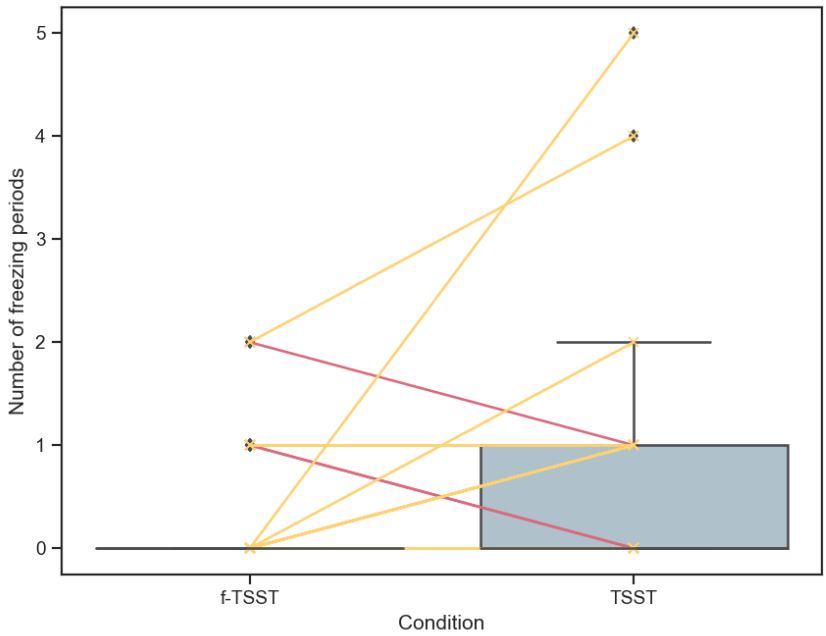

Figure 4.17: Paired boxplot for number of freezing periods for (f-)TSST with connected subjects

The statistical analysis of the absolute number of freezing periods per subject across both conditions resulted in a *p-value* of 0.0065, indicating a statistically significant difference between the TSST and f-TSST conditions. This finding suggests that participants experienced a significantly higher number of freezing periods in the stress condition compared to the control condition.

It is important to note that the overall number of static periods was much higher in the stress condition than in the control condition. This suggests that the increased number of freezing periods observed in the TSST condition may be partly due to the higher occurrence of static periods overall, which could contribute to the likelihood of freezing episodes.

4.6 General Discussions and Limitations

This examination of freezing within the context of acute psychosocial stress suggests that analyzing the freezing behavior can lead to further insight into the underlying condition of the stress test and even the cortisol response.

Through this analysis, a working definition for freezing periods during acute psychosocial stress was developed (Table 3.5). In synchronizing heart rate data with the static periods a relationship between body movement and heart rate during these stressful moments was detected. Upon analyzing the heart rate over time, a consistent pattern emerged: specific static periods were associated with a clear reduction in heart rate, typically beginning about 4 seconds after the onset of immobility. This observation aligns with findings from previous studies, suggesting that bradycardia associated with freezing only becomes apparent after a brief delay following the onset of specific stressors [Voo22; Roe22].

Identifying the relationship between freezing behavior and stress responses presented several challenges, particularly when contrasting the stress condition with the control condition. Analysis of freezing periods and their associated physiological characteristics revealed that freezing-related bradycardia appeared to be more pronounced during the stress condition (Figure 4.11). This suggests that the physiological markers of freezing - particularly the reduction in heart rate - may be amplified under stress. Interestingly, even when the cortisol response was examined alongside heart rate data, it became evident that heart rate reductions were more pronounced in individuals who exhibited a stress response, as indicated by elevated cortisol levels. This connection suggests that the physiological state associated with stress not only influences freezing behaviors but also intensifies the associated bradycardia (Figure 4.12).

However, identifying the relationship between freezing and stress proved to be more challenging. The absolute occurrence of freezing periods was higher for the stress condition and can therefore link the stress response with higher incidences of freezing. But for the f-TSST condition freezing seemed to occur more likely (Table 4.5) considering the freezing to static ratio overall static periods and for each subject and condition. This was affirmed if the cortisol response was examined (Table 4.7), indicating that freezing can even appear as a healthy mechanism in not stressful situations. Further parameters including the mean bradycardia and the freezing ratio could not show a statistical significance and therefore not adequately indicate a difference between the stress and the control condition. The only parameter showing significance was the absolute occurrence of freezing periods, which were more prominent during the TSST.

All these results suggest that freezing can be more prominent and overall appear more often during the stress condition. But not every static period automatically translates into a freezing

period and can therefore not be used interchangeably. Regarding the freezing to static periods ratio between the conditions, it even showed that during the f-TSST or in subjects with no cortisol response, a static period translating into a freezing periods seemed to be more likely compared to the stress condition. This suggests that freezing does not only happen as an adaptive response to stress, but seems to be a healthy mechanism even present if no endocrine stress response can be measured.

In looking between the relation of static periods and freezing periods, during the control condition, significantly fewer static periods were identified, which limited the number of periods available for analysis as potential freezing periods. This scarcity of static periods during the control condition made it difficult to draw firm conclusions whether freezing occurred more frequently during the stress condition. This limitation highlights a key challenge in studies of psychosocial stress: the variability in individual responses to stressors like the TSST can result in inconsistent data, complicating attempts to standardize measurements such as freezing frequency. This variability made statistical comparisons between subjects more complex and less straightforward than in tightly controlled laboratory settings.

One of the primary challenges encountered in this study was the strict criteria imposed for defining static periods suitable for analysis. The prolonged nature of the stressor introduced variability in how participants responded over time, complicating the process of extracting standardized data for static and freezing periods. Static periods often overlapped, occurred in short sequences, or lacked a clear starting point, making it difficult to precisely identify the onset of freezing episodes. Therefore strict constraints were imposed for defining static periods suitable for analysis. These criteria included an active baseline of at least 1 second prior to the static period and the minimum duration of 5 seconds, which reduced the overall number of periods available for analysis (Table 4.4). Although this stricter definition ensured greater precision in identifying genuine freezing episodes, it also led to a relatively small sample size of static periods across the study. Despite the clear distinction of freezing within these periods, the small number of samples makes it difficult to generalize these findings to broader populations. Future studies with larger sample sizes and more flexible criteria may yield more robust results.

Overall, the findings of this thesis contribute to the growing body of research on defensive freezing, offering valuable insights into how this response manifests in a more naturalistic setting. The results highlight the importance of examining both physiological and behavioral responses to stress and suggest that future research could benefit from further refining the methods used to detect freezing in less controlled environments.

Chapter 5

Conclusion and outlook

This thesis laid the groundwork for examining defensive freezing behavior during acute stress from a new perspective, focusing on identifying physiological and behavioral markers associated with this response. Specifically, the TSST was employed to trigger the stress response, while the f-TSST served as a control condition. This choice of methodology allowed for a direct comparison of physiological and behavioral responses in varying stress contexts.

By carefully analyzing static periods during the stress event, valuable insights were gained into the intricacies of what occurs during these moments of immobility, enhancing our understanding of the freezing phenomenon in the context of psychosocial stress. A comprehensive examination of heart rate patterns before, during, and after the static periods revealed significant findings. The ability to define and identify freezing periods during the (f-)TSST was a crucial outcome of this analysis. The results suggest that acute stress can lead to more intense freezing episodes, characterized by more pronounced bradycardia and a longer duration of the static periods. This indicates that the physiological response associated with freezing may be more robust during stressful situations, reinforcing the notion that defensive freezing serves an adaptive function in response to perceived threats.

Additionally, the data suggest that freezing behavior tends to occur more frequently under no-stress conditions compared to the static periods observed during these conditions. However, it is important to note that no clear conclusions could be drawn regarding the ratio of freezing periods to static periods overall, given the relatively small dataset available in this study.

The limitations imposed by the sample size necessitate caution in interpreting the findings. A larger dataset would be highly valuable for further exploration of these results. With a more substantial pool of freezing periods observed under both stress and control conditions, it would be possible to derive more specific and robust conclusions regarding the dynamics of freezing behavior.

Furthermore, such an expanded dataset could enable researchers to perform more sophisticated statistical analyses, enhancing the reliability and validity of the findings. The overall problem within the structure of this study is that the number of static periods is not equally distributed between the stress and the control condition and therefore also the number of freezing periods can vary immensely depending on the underlining condition, making it hard to compare these conditions and further even the subjects themselves. The lack of a clearly defined trigger for the occurrence of freezing periods, as seen in other studies, makes it more challenging to identify distinct patterns.

In addition to increasing the sample size, incorporating other factors such as gender, age, personality traits, and questionnaire data could provide a more nuanced understanding of defensive freezing behavior during acute psychosocial stress. Individual differences can play a critical role in how people respond to stress, and understanding these variations could help tailor interventions aimed at managing stress responses. Exploring these additional variables could also reveal important insights into the psychological and physiological mechanisms underlying freezing behavior.

Moreover, the existing literature supports the theory of a ”freeze-for-action” response, which posits that after a period of freezing, both heart rate and body movement increase as individuals prepare to react to the stressor [Gla16]. This concept underscores the adaptive nature of freezing. It serves not only as a means of temporary immobility but also as a precursor to subsequent action. The exploration of this response, particularly the transition from freezing to action, presents an interesting avenue for future research. Investigating the factors that facilitate this transition could enhance our understanding of the functional role of freezing in stress responses and its implications for survival.

Overall, this thesis contributes to the growing body of research on defensive freezing behavior, offering valuable insights into its manifestation in a more naturalistic setting. By exploring freezing in the context of psychosocial stress, this work paves the way for future studies aimed at highlighting the mechanisms of freezing and its role in stress responses. As the understanding of these processes deepens, it may lead to more effective strategies for managing stress-related disorders and improving mental health outcomes. Further research in this area could have significant implications not only for psychology and neuroscience but also for clinical practices focused on stress management and intervention. In conclusion, while this thesis provides a foundational understanding of defensive freezing behavior during acute psychosocial stress, it also highlights the need for continued exploration in this field. The complexity of human responses to stress necessitates ongoing research to unravel the intricate interplay between physiological and psychological factors, ensuring

a comprehensive understanding of defensive mechanisms like freezing. As future studies build upon these findings, they will contribute to a more holistic understanding of how individuals navigate the challenges posed by stress and the adaptive responses that arise in the face of adversity.

Appendix A

Additional Figures

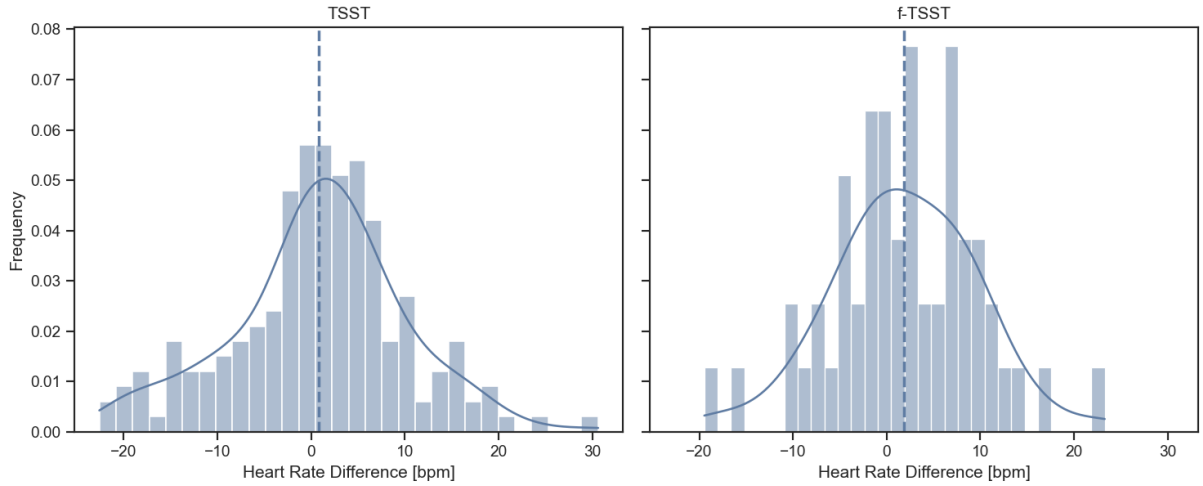

Figure A.1: Differences between baseline and area of interest in heart rate for all 5 sec Static Periods and their relative appearance

Appendix B

Additional Tables

Subject	Condition	Static Periods	Freezing	Maximum cortisol increase [$\frac{\text{nmol}}{\text{l}}$]
VP 04	f-TSST	0	0	1.456
VP 04	TSST	4	1	2.598
VP 05	f-TSST	0	0	5.411
VP 05	TSST	2	1	7.000
VP 06	f-TSST	0	0	6.454
VP 06	TSST	0	0	11.916
VP 07	f-TSST	0	0	-1.245
VP 07	TSST	5	1	6.587
VP 08	f-TSST	0	0	-0.826
VP 08	TSST	0	0	2.411
VP 09	f-TSST	0	0	-0.089
VP 09	TSST	2	0	1.692
VP 10	f-TSST	0	0	0.844
VP 10	TSST	0	0	1.522
VP 11	f-TSST	0	0	-1.245
VP 11	TSST	4	1	7.235
VP 12	f-TSST	0	0	-1.410
VP 12	TSST	1	0	-0.850
VP 13	f-TSST	0	0	-0.132
VP 13	TSST	2	0	0.773
VP 14	f-TSST	0	0	1.749

VP 14	TSST	0	0	13.936
VP 15	f-TSST	1	1	3.993
VP 15	TSST	0	0	8.084
VP 16	f-TSST	0	0	-3.477
VP 16	TSST	0	0	-2.178
VP 17	f-TSST	4	2	-1.035
VP 17	TSST	5	2	7.280
VP 18	f-TSST	1	0	-*
VP 18	TSST	4	0	5.207
VP 19	f-TSST	2	1	-
VP 19	TSST	3	0	-
VP 20	f-TSST	0	0	-
VP 20	TSST	0	0	-
VP 21	f-TSST	0	0	2.330
VP 21	TSST	2	1	11.041
VP 22	f-TSST	0	0	0.557
VP 22	TSST	1	1	3.766
VP 23	f-TSST	0	0	-0.723
VP 23	TSST	5	0	8.053
VP 24	f-TSST	0	0	0.335
VP 24	TSST	0	0	1.185
VP 25	f-TSST	1	0	-1.768
VP 25	TSST	2	0	7.065
VP 26	f-TSST	1	0	0.789
VP 26	TSST	5	2	3.137
VP 27	f-TSST	0	0	2.340
VP 27	TSST	0	0	1.336
VP 28	f-TSST	0	0	2.315
VP 28	TSST	0	0	9.756
VP 29	f-TSST	0	0	-0.881
VP 29	TSST	8	4	7.114
VP 30	f-TSST	0	0	0.011
VP 30	TSST	0	0	0.408
VP 32	f-TSST	1	1	-0.647

VP 32	TSST	3	1	7.396
VP 33	f-TSST	0	0	0.943
VP 33	TSST	1	0	18.922
VP 34	f-TSST	0	0	-0.79
VP 34	TSST	0	0	0.383
VP 35	f-TSST	0	0	2.355
VP 35	TSST	2	1	-0.062
VP 36	f-TSST	0	0	4.235
VP 36	TSST	0	0	3.677
VP 37	f-TSST	7	2	-0.591
VP 37	TSST	5	4	0.210
VP 38	f-TSST	1	0	3.029
VP 38	TSST	4	2	8.982
VP 39	f-TSST	0	0	-0.949
VP 39	TSST	1	1	0.166
VP 40	f-TSST	2	0	-1.065
VP 40	TSST	1	0	4.510
VP 41	f-TSST	0	0	-1.032
VP 41	TSST	1	0	-0.927
Total amount		94	30	*no evaluation possible

Table B.1: Subject and condition overview for *Baseline 1* subset

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Appendix C

Acronyms

3D 3-dimensional

bpm beats per minute

BMI Body Mass Index

ECG electrocardiogram

FACS Facial Action Coding System

f-TSST friendly Trier Social Stress Test

(f-)TSST (friendly) Tier Social Stress Test

HPA hypothalamic-pituitar-adrenal

IMU inertial measurement units

LPS lipopolysaccharide

MoCap motion capture

.mvn native Xsens file format

.mvnx MVN open XML format

PASA primary appraisal secondary appraisal

PLD point-light displays

PNS parasympathetic nervous system

SD standard deviation

SE standard error

SNS sympathetic nervous system

SVM support vector machine

TSST Trier Social Stress Test