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**„Trait Anxiety and Heart Rate Variability:
The effect of trait anxiety on the change in heart rate variability after an acute
stressor and its mediation by retrospective stress appraisal“**

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Introduction

Stress, and especially chronic stress, are common phenomena in our modern society. The physical as well as mental health issues associated with them range from cardiovascular diseases (Steptoe & Kivimäki, 2012) to burnout (Maslach et al., 2001), depression (Yang et al., 2015), and even an increased risk for cancer (Reiche et al., 2004). For many years, researchers investigated how and why stress emerges and what could be done to prepare for, or even prevent, stressful situations and the accompanying stress response and its unpleasant consequences.

One of the most acknowledged and researched models of stress is the transactional model of stress and coping proposed by Lazarus and further developed throughout the years by Lazarus and colleagues (Lazarus & Folkman, 1984). It defines stress as a transactional concept that only emerges if the person concerned appraises the situation as relevant and threatening to their own well-being and evaluates their available resources to cope with the situation as insufficient. These so-called appraisal processes are crucial in understanding how to maintain both mental and physical health, as they have been shown to influence the likelihood of developing stress-related health issues by affecting the amount of stress an individual experiences (e.g., Gomes et al., 2017; Kerr et al., 2020).

Anxiety is an emotion that is caused by intrusive thoughts and manifests in physiological and psychological responses similar to the human stress response. These symptoms include, for example, elevated blood pressure or an increase in heart rate (Xi, 2020). In contrast to stress, which is not an emotion but the result of transactional appraisal processes that include the current situation and available coping resources (Lazarus & Folkman, 1984), anxiety is caused by concern about potential future harm even when there is no actual threat (Lovallo, 2016). Its natural function as an emotion was an evolutionary advantage since the very beginning of humankind, increasing our ancestors' chances of survival via physiological signals (Crocq, 2015). Due to its relevance for our everyday lives and its common occurrence, it has been researched intensively throughout the years. Anxiety disorders are among the most prevalent mental disorders worldwide, and recent developments around the globe are further increasing their prevalence (World Health Organization, 2022). But it is not only clinically relevant anxiety levels that influence human cognition, perception, and health. *Trait anxiety*, which can be understood as

an individual's tendency to perceive situations as threatening or fear-inducing, is a personality disposition that is innate to every human being. It can be expressed on a continuous scale from 0 to 100, and every individual differs in the extent to which they score on the scale (Gaudry et al., 1975). It has been indicated to increase the likelihood of the emergence of stress-related diseases such as burnout (Gomes et al., 2017) and therefore needs to be further investigated for us to be able to prevent possible harm to the mental as well as physical health of individuals without clinically relevant anxiety disorders who score highly on trait anxiety.

Anxiety

Anxiety is an emotional state that is innate to every healthy human being. Experiencing and enduring anxiety as well as learning to cope with it are among the basic skills every child must acquire during its development, and even adults still encounter anxiety-inducing situations on a regular basis. Since anxiety seems to be so common, it might not be surprising that anxiety disorders are the most prevalent mental disorders worldwide. Before the COVID-19 pandemic, an estimated 298 million people had some form of anxiety disorder. During its first year alone, the global pandemic worsened the overall numbers and increased the number of people affected by it by 25%, reaching a new all-time high estimate of 374 million affected people (World Health Organization, 2022). But what is anxiety as such, and why do we experience it?

The word Anxiety derives from the Latin word *angor*, which can be translated as constriction. A relationship between anxiety and narrowness (of the mind) can be found throughout many languages and even in biblical texts (Crocq, 2015). In his analysis of the use of the anxiety concept in recent literature, Xi (2020) subsumed the definition as an unpleasant emotion caused by intrusive thoughts, which manifests through physical and psychological responses, while Lovallo (2016, p. 72) describes anxiety as “(...) *a sense of apprehension and the anticipation of harm even in the absence of threat.*” In the Diagnostic and Statistical Manual of Mental Disorders (DSM-5), anxiety is simply defined as the anticipation of a future threat (American Psychiatric Association, 2013).

Throughout the evolution of the human species, anxiety provided adaptive value that promoted the individual's survival. Although anxiety still functions as a regular emotional response and has its use in modern society, a distinction between normal adaptive anxiety in everyday life and pathological anxiety must be made. Contrary to the belief that anxiety, like schizophrenia, was hardly known as an illness before the 19th century, there are indications that anxiety was already known to ancient philosophers and physicians (Crocq, 2015). In a collection of Greek medical texts called *The Hippocratic Corpus*, a case of phobia involving a man named Nicanor is described and labelled as a medical disorder (Smith, 1994).

Since then, clinically relevant anxiety has become a well-studied topic, but it is not only pathological anxiety that influences many aspects of our everyday lives. The amount of empirical research on anxiety got a huge upswing during the early 1950s due to the publication of a scale for the measurement of anxiety as a trait (Taylor, 1953). Based on this scale, an enormous amount of research has been conducted on the influence of anxiety on topics like learning, memory, perception, and performance, concluding anxiety to be either a drive or a source of cognitive interference (Spielberger, 1966).

In more recent concepts, researchers distinguish between two different kinds of anxiety, namely, state anxiety and trait anxiety. State anxiety can be described as transitory unpleasant feelings of apprehension, tension, nervousness, or worry, and it includes the momentary expression of anxiety (Spielberger, 1985). This definition coincides with the subsumed definition found by Xi (2020) and the DSM-5.

Trait anxiety, on the other hand, can be understood as the personality disposition that describes a person's tendency to perceive situations as threatening (Gaudry et al., 1975) and react with increased state anxiety (Morales, 2012). Individuals who are high in trait anxiety seem to be particularly susceptible to threats to their self-esteem in interpersonal situations (Spielberger et al., 1970) and tend to anticipate negative outcomes more easily in objectively stressful situations (Rapee & Heimberg, 1997; Sarason & Sarason, 1990). Furthermore, the higher propensity for anxiety following this trait seems to make individuals more vulnerable to burnout, especially in a competitive environment (Gomes et al., 2017).

Stress

Because of our increasingly fast paced society and lifestyles, it seems like many people experience the feeling of being stressed occasionally, or even on a regular basis. But although we use the term *stress* frequently to describe demanding situations in our everyday lives it has not been for long that we began to understand what stress really is and how it emerges.

The first mention of the term *stress* dates back to the 14th century. Back then, it was used to describe hardship, straits, adversity, or affliction (Lumsden, 1981). Over time, stress was introduced to the physical sciences, where it was made systematic within the early 19th century, once again using the concept of stress stemming from the influence of external forces on an internal counterforce. This basic concept prevailed and throughout the 19th century got conceived as a basis of possible health influence factor by the medical field (Hinkle, 1974).

By 1936, the endocrinologist Hans Selye was using the term stress in a technical way, which can be seen as the basis of more recent psychological stress theories. He described it as a set of bodily defences against any form of noxious stimulus, which also includes psychological threats. He called this reaction the *General Adaptation Syndrome*. By his definition, stress itself was not the environmental demand on the individual (which he called a “stressor”), but an adaptive universal physiological response pattern of the human body (Selye, 1946). This can be interpreted as the existence of an underlying general adaptational response to all types of stressors, with additional response patterns that are specifically characteristic to the type of occurring stimulus (Lovallo, 2016).

At this point, it is important to clearly communicate the differences between, and specific characteristics of, physical and psychological stressors. While it might be easy to determine a physical stressor as a direct threat to our well-being, such as heightened physical demands during sports or work, toxic substances, diseases, or being exposed to extreme temperatures, one might not be able to identify psychological stressors just as easily. A psychological stressor proposes a challenge to our safety just like a physical threat, but not because of actual physical danger. Instead, our thoughts, perceptions, expectations, and interpretations of the situation may evoke major discomfort. An important characteristic of psychological stressors is that they may not have a clear on-and-offset. They account for most of the tension that people would

refer to as stress during everyday life. Since they don't have a clear beginning and end, people tend to drag psychological stressors with them for a longer period of time. Hereby it needs to be noted that a full separation of the two types of stressors is rarely possible. Because we are usually fully conscious, our awareness of the situation comes with a prospect of possible future consequences, which is often accompanied by anxiety and psychological stress (Lovallo, 2016).

The assumption of this adaptational dynamic reaction of the body implicates the importance of aspects such as the available resources to cope with the current situation, the costs of the long-term usage of coping mechanisms, such as disease and distress, as well as the beneficial outcomes of overcoming adversity. With his theory of stress, Selye emphasized the dynamic relationship between the organism and the environment and therefore settled for a more sophisticated definition of stress than theories solely based on the processes within the individual. With his work, Hans Selye sparked interest in the topic, and although he was unable to fully clarify the underlying processes of his stress model, the influence of Selye's work on further future stress research and the resulting stress theories is undeniable (Lazarus & Folkman, 1984).

Although Selye's theory had a concept of stress in mind, there was ambiguity about the definition of the term stress. Hence, criticism arose about the applicability of the stress concept since the term seemed to be used as an explanation for any kind of altered psychophysiological state. This resulted in a reduction of the heuristic value of the concept due to the broad scope and descriptive nature of the term at that time (Ader, 1980).

Following Selye's work, a wide range of theories tried to explain the concept of stress via various definitions. A short overview of the major categories and some examples and critique on each of the definitions are going to be described below.

Stress as a stimulus

In accordance with the then-dominant traditional thought of human beings as mainly reactive to stimulation, the most common definition of stress within the field of psychology has been the concept of stress as a stimulus. A stressful stimulus has simply been described as any kind of event that affects the individual. These stimuli

are not limited to external sources but also include internal conditions such as drive stimuli (e.g., hunger or sex) (Lazarus & Folkman, 1984).

As to the definition of stress stimuli, whether internal or environmental, Lazarus and Cohen (1977) categorized three types of possible stressors. First, major changes within the environment; these kinds of events are usually disastrous and include natural disasters, man-made catastrophes, and atrocities such as war, imprisonment, and torture, as well as forced relocation. The duration of the stressor ranges from prolonged exposure (e.g., imprisonment) to brief experiences (e.g., earthquakes). It is important to note that the aftermath of a brief disaster can have long-lasting consequences for the individuals involved.

Second, to qualify as a major change, it is not necessary that many individuals be affected. In fact, the number of people affected does not crucially alter the potential for such threatening events to be disturbing. On a much smaller scale, events that are outside of an individual's control can result in similar disturbances among the people affected (Lazarus & Folkman, 1984; Lovallo, 2016). These events include the death of a loved one (Bowlby, 1961), a life-threatening illness (Hackett & Weisman, 1964), or the unexpected loss of one's job (Kasl & Cobb, 1970).

Third, Lazarus and Cohen (1977) acknowledged the influence of daily, less dramatic stressful experiences within the category of "daily hassles". These include negative events that occur on a much more regular basis, such as feeling lonely, having an argument with a loved one, or feeling under pressure from handling too many responsibilities.

Further taxonomies, such as the one by Elliot and Eisdorfer (1982), tried to consider formal properties of the affecting situations, such as the predictability of the event or the difference between acute and chronic demands of a stressor. They categorized four major types of stressors, namely: 1. *acute and time-limited stressors*, such as awaiting a surgery; 2. *stressor sequences*, which can be understood as a series of events that occur over an extended period because of an initiating event (e.g., job loss, divorce, etc.); 3. *chronic intermittent stressors*, which may occur on a regular basis (e.g., once a day, once a week), such as conflict-filled discussions with a relative or a colleague; and 4. *chronic stressors*, which include permanent disabilities or chronic work stress.

Although the taxonomies proposed by Elliott and Eisdorfer (1982) as well as Lazarus and Cohen (1977) provide a theoretical framework for differentiating stressful events and their influence on the individual, they fail to consider that these events are only to be regarded as stressful due to the general normative response. There may be differences in the way individuals respond to the stressors based on the examples given above. Due to the vast variance in personality traits and experiences within the human population, individual responses might differ significantly from the average reaction, and therefore a theoretical framework that relies on the general normative response cannot reliably provide valid evaluations for a chosen individual. Furthermore, as soon as the response pattern of the people affected must be considered, the definition of stress as a pure stimulus-bound concept can no longer be supported (Lazarus & Folkman, 1984).

Stress as a response

Another major group of stress researchers defines stress as a pure response to a range of stimuli. This category includes, for example, the abovementioned theory of Hans Selye and was most commonly used in biology and medicine in terms of an organism reacting to stress or being under stress and therefore disturbing the established bodily homeostasis. But already within this definition of stress as a response lies its major problem. If one assumes that stress must be defined as a sole response, there is no systematic way of prospectively identifying what kind of stimuli can be considered stressors, and therefore there can never be a proper way of identifying methods to prevent stress-inducing situations and their consequences. One might be able to deduce the situations in which most individuals commonly experience stress, but this does not have to reflect the person of interest's reaction, resulting in the same problem of insufficient explanatory value for individual people of interest as the abovementioned stimulus definitions (Lazarus & Folkman, 1984).

Another major point of criticism is the understanding of the stress response as a disturbance of bodily homeostasis. Since all aspects of living somehow interact with the individual and therefore either produce disturbance or decrease it, which is the definition of influencing homeostasis, it is impossible to distinguish stressful events from any other situation in everyday life. Stress could then only be identified if the degree of disturbance is extreme. Furthermore, the physiological measurements that

indicate the stress response, such as a rise in heart rate, cannot be considered exclusive to stress. For example, even a minor exercise like jogging or hiking produces similar increases in heart rate, while the individual might not only show no psychological indications of stress whatsoever but might even feel relaxed or at ease. This applies to the general stimulus-response approaches, which in turn pose a circular flaw in their logic. Once the stress response is dependent on the presented stimulus, one can no longer speak of a simple response definition. Furthermore, as soon as an individual's response must be considered to identify a stimulus as a stressor, it contradicts the very definition of a stimulus-based stress concept. Therefore, neither pure stimulus nor pure response frameworks can sufficiently define stress (Lazarus & Folkman, 1984).

Stress as a relational concept

As mentioned above, the simple concept of an external cause or stimulus being the reason for stress or illness was not sufficient to explain the complex nature of stress. A pathogen, or a stressor can only cause the respective illness, or stress, if the individual is susceptible to it which indicates that the individual's characteristics are just as important as the external noxious stimuli itself. The emergence of stress can therefore only be explained through the complex and fluctuating person-environment relationship. The multicausal nature of the stress concept makes it necessary to investigate the contribution of potential covariables such as personality traits, vulnerability, or available coping mechanisms to fully understand their possible mediating role within the stress-illness relationship. Based upon this knowledge, Lazarus and Folkman identified psychological stress as “(...) *a particular relationship between the person and the environment that is appraised by the person as taxing or exceeding his or her resources and endangering his or her well-being*” (Lazarus & Folkman, 1984, S. 40).

Cognitive appraisal

The concept of appraisal has always been seen as a necessary tool to account for the significant interindividual and group differences in the stress reaction. While the importance of the appraisal concept has been implied as well as explicitly stated

throughout the field of psychological stress research for many years (e.g., Grinker & Spiegel, 1945; Barber & Coules, 1959), the first attempt at a systematic approach was made by Magda Arnold in 1960. She described appraisal as an immediate and indeliberate estimation of a situation's relevance or possible harm to the individual (Arnold, 1960).

Despite Arnold's early attempt at defining appraisal as a cognitive concept, most of the stress research conducted during that time focused on non-cognitive theoretical models such as drive reinforcement and general arousal. These models reflect the abovementioned stimulus and response theories in the sense that they typically regard stress as a state of disequilibrium that leads to general arousal. The general arousal concept, which states that when one physiological stress indicator rises, other possible indicators rise as well, has been proven wrong many times (e.g., Lazarus et al., 1962).

Therefore, Lazarus and Folkman (1984) advocated for a cognitive appraisal approach to stress, in which they defined cognitive appraisal as a continuous process of categorizing daily situations and their various aspects with respect to their significance for the individual's well-being. In line with earlier accounts of appraisal theory, a distinction between primary and secondary appraisal is made as the two main stages of appraisal in the context of stress.

Primary appraisal

Primary appraisal is concerned with the relevance of the current situation for the individual. Depending on the implication, or lack thereof, for the person, three kinds of categories of primary appraisal can be distinguished, namely, *irrelevant*, *benign-positive*, and *stressful* (Lazarus & Folkman, 1984). The classification of the situation is based on the individuals' beliefs and commitments, which define the boundaries of what might be threatening and to what extent. An illustrative example would be an accident leading to a broken arm, affecting one's everyday life, and resulting in unpleasant or painful feelings. While the event and its consequences may be appraised as threatening for most people, they would only pose a temporary burden with no long-term consequences if a full recovery could be achieved. If, on the other hand, a professional athlete breaks his arm, he cannot keep up his training schedule, let alone compete in important events. A lot more would be on the line since his career

might end if he is unable to fully recover swiftly, and he might lose out on his only chance to keep his lifestyle, therefore posing an existential threat and leading to an increased sense of threat for the individual. By considering the beliefs and commitments of the individual, it is possible to account for the cognitive differences between individuals and their emotional and stress reactions to one and the same event (Lovallo, 2016).

If the occurring situation has no bearing on the person's well-being it is deemed *irrelevant*. This type of event demands no personal investment or commitment, and nothing is to be lost or gained from it. The process of identifying and distinguishing irrelevant cues from relevant ones has been of major importance from an evolutionary perspective since mobilization for action is only desirable if it is necessary (Lazarus & Folkman, 1984).

A *benign-positive* appraisal occurs if the expected outcome of the situation is deemed positive for the individual. As a result, any consequence that preserves or improves well-being, even if it only promises to do so, is included. Benign-positive appraisals are typically characterized by positive emotions such as joy, happiness, or peacefulness (Lazarus & Folkman, 1984).

The final appraisal category is called *stress* appraisals, and it includes the three subcategories *harm/loss*, *threat*, and *challenge*. If harm/loss appraisals occur, some form of damage to the individual has already been sustained. Hereby included are, for example, injuries with long-lasting consequences, damage to one's self-esteem, or the loss of a loved one (Lazarus & Folkman, 1984).

A threat appraisal concerns harms or losses that have not yet happened but are anticipated. It is worth noting that a harm/loss appraisal may still be accompanied by threat, since an already occurred injury always proposes negative implications for the individual's future. Threat appraisal differs from harm/loss mainly by the fact that it allows the individual to prepare for possible loss through anticipatory coping. A human being can plan into the future to limit the impact of an expected incoming harm or loss (Lazarus & Folkman, 1984).

The third kind of stress appraisal is called challenge. A challenge appraisal shares many features with a threat appraisal due to its mobilization of coping efforts. Contrary to threat, challenge appraisals do not focus on the prevention of harm/loss

but on the potential for gain or growth that lies within the encounter. Therefore, a challenge has important implications for adaptation, as challenging situations are usually experienced as pleasurable, whereas a threat is characterized by negative emotions such as fear, anxiety, or anger. This provides an advantage in the face of adversity since a challenged individual is more likely to have a high morale, quality of functioning and overall somatic health due to the overall positive connotation of their circumstances when compared to people who experience threat. Although the emotional nature of the two categories might make it seem as if they were opposite extremes on a single continuum, they are not necessarily mutually exclusive and can even occur simultaneously. A situation might show potential for further future development but at the same time pose a threat to one's well-being or self-esteem, such as a promotion to a better job with a higher salary but a broader scope of responsibility (Lazarus & Folkman, 1984).

Secondary appraisal

Whenever an individual encounters a situation that demands further action to overcome the circumstances, whether it be a threat or a challenge, an additional form of appraisal occurs. Secondary appraisal, which can be understood as the evaluation of available resources and how they might be implemented, is a crucial tool for the successful management of every stressful encounter. Additionally, the individual also estimates the likelihood of a given strategy to accomplish its goal and resolve the situation, which has been labelled outcome expectancy, as well as the likelihood that the person can apply the strategy effectively, also known as efficacy expectation (Bandura, 1982).

Furthermore, Lazarus, and Folkman (1984) attribute to secondary appraisal the evaluation and consideration of possible consequences of the considered strategies. Together primary appraisal of the situational demands and secondary appraisal of the available coping resources form a complex interdependent relationship whose interaction determines the amount of stress and the quality as well as the intensity of the emotional reaction of the individual.

Reappraisal

A third and final form of appraisal proposed by Lazarus and Folkman (1984) is the so-called reappraisal. As the name suggests, reappraisal acts as a re-evaluation of former appraisals due to new information about the occurring situation or available coping resources. Through reappraisal, an individual can alter the stress outcome stemming from the interaction of the primary and secondary appraisal of the situation. The emotional as well as stress-related reactions can either be further intensified, mitigated, or even reversed. For example, a situation that has been appraised as threatening at first glance might become challenging and therefore have a positive connotation due to new information about the manageability.

Although it may seem like appraisal is a conscious and rational process, Lazarus (1984) argued that, while there might be instances of conscious appraisal, unawareness of some or all aspects of the appraisal situation is possible. A threat appraisal, for example, can occur without the individual's awareness of the internal or environmental factors, values, and goals that contribute to the result.

Due to the implicit nature of the influence of appraisal on the emergence of stress during the early stages of stress research, a group around Lazarus and his colleagues initiated systematic research on the cognitive mediation of stress via appraisal. All the studies had their origins in a quasi-naturalistic stress-inducing experimental situation during which participants had to witness recordings of other people being mutilated in rituals, get into accidents in a woodworking shop, et cetera (Lazarus, 1966).

To further improve the understanding of the role of appraisal processes, four methods have been used to manipulate the conditions of the experiment. The appraisal itself was manipulated by influencing the interpretation of the filmed events either as painful, benign, via denial-like processes, or detached through incentivizing a distancing or intellectualizing view. Thereby, it was found that by influencing the appraisal before the recordings were presented, both physiological as well as subjective stress response levels could be altered (e.g., Folkins et al., 1968; Lazarus & Alfert, 1964).

Other studies manipulated the given anticipation time before the stress inducing stimuli were presented. By altering the waiting period before the confrontation with the

recordings, it was found that the duration influenced the stress response. It has been shown that slightly longer anticipation periods produce greater stress reactions than very short ones, but if the participants wait for at least three to five minutes, they can mitigate the stress response significantly. These findings were attributed to a reappraisal of the situation and further deep thought about the anticipated event (Folkins, 1970; Nomikos et al., 1968).

In a study conducted by Koriatic et al. (1972), the researchers tried to influence the emotional reaction of their participants by instructing them to either increase their emotional involvement while watching a video, which has been shown to induce stress in previous studies or decrease their involvement by detaching themselves emotionally. The researchers assessed physiological stress parameters such as heart rate and skin conductance in 115 male subjects, as well as a retrospective self-report of experienced distress. While no significant reduction in heart rate or skin conductance could be found, participants who detached themselves emotionally while watching the video reported lower subjective distress retrospectively.

Finally, research concerning the influence of personality factors or cognitive styles on appraisal through denial or intellectualization was conducted. These studies concluded that a reduction in stress response was possible if the habitually utilized coping strategy matched the influences given to manipulate the appraisal. For example, a participant inclined to use denial as a coping strategy experienced a much weaker stress response after being incentivized to appraise the situation with a denial-like process than a participant with an incongruent condition (Speisman et al., 1964). This extensive series of studies was the first systematic demonstration of the mediation of stress response levels via cognitive appraisal processes (Lazarus & Folkman, 1984).

The transactional model of stress and coping

To sum up the abovementioned information, the transactional model of stress and coping defines stress as a complex relational concept. The emergence of stress depends on various intraindividual as well as environmental factors such as personality traits of the individual, the commitment and relevance of the occurring situation for the individual, the available resources and coping mechanisms, the predictability and

controllability of the situation as well as the appraisal of the situation based on these factors. Cognitive appraisal, defined as a mostly unconscious cognitive process that intervenes between the encounter and the reaction, is utilized to evaluate the relevance of the situation. Depending on the outcome of the primary appraisal, which refers to the evaluation of the relevance and threat or growth potential of the situation, a secondary appraisal, which encompasses the available resources and coping mechanisms, occurs. After a given time span, the individual can utilize the information gathered in the recent moments to undergo possible reappraisal, which refers to a changed appraisal based upon new information. All these processes influence the following physiological as well as emotional stress responses (Lazarus & Folkman, 1984). As a result, it is indicated that the individual stress response is mediated by the result of cognitive appraisal, as has been shown in the abovementioned research.

Risks and consequences of stress

For many decades now, the influence of stress on physiological as well as mental health has been a major topic throughout a wide range of research fields. The impact of prolonged stress exposure has been indicated to be a severe risk factor for the development of cardiovascular disease (Steptoe & Kivimäki, 2012), burnout (Maslach et al., 2001), depression (Yang et al., 2015), and other diseases such as cancer (Reiche et al., 2004) by impairing the immune response. Under certain circumstances, chronic stress has even been linked to alterations in certain brain structures associated with cognition and behaviour (Lupien et al., 2009).

Therefore, the role of stress appraisal as a mediator variable of the effect of stress on physiological and mental health needs to be well understood to be able to prevent the health-related consequences of intense and chronic stress. Examples of research supporting the transactional model have been conducted by Gomes et al. (2017), who have been assessing the mediating effect of cognitive appraisal on the emergence of burnout in a stressful competitive environment, or in the research of Kerr et al. (2020), which indicated the importance of appraisal for the psychobiological stress response caused by work stress. Even in objectively stressful situations like these, cognitive appraisal has been indicated to influence the likelihood of further health issues in people who score highly in trait anxiety.

The physiology of the stress response

As has been shown above, a wide range of problematic consequences can be associated with stress. To be able to measure and influence these psychological and physiological changes induced by stress, one needs to understand the physiological stress response as well as the complex relationship between psychological stress and the human body.

Branching from the central nervous system (the brain and spine), a complex network of nerve strands traverses the entire human body. These networks can be divided into two major components: the somatic nervous system (SNS) and the autonomic nervous system (ANS). The SNS regulates the somatic and sensory communication with the environment and mainly contains bodily functions that are under conscious, voluntary control, like the skeletal muscles or the use of our sensory organs (Hoyer & Knappe, 2020). Its sensory nerves give the brain information about the position and motions of the limbs, and they allow us to be aware of where our limbs are and what they are doing (Lovallo, 2016). The ANS, on the other hand, is responsible for the regulation of the functions of our vital organs, like the heart or liver, and the glands within our body. Therefore, the ANS has a major influence on essential mechanisms like our respiration, circulation, reproduction, digestion, glandular secretion, and body temperature, and hence plays a significant role in the regulation of bodily homeostasis. As the name indicates, the bodily functions affected by the ANS are mainly outside of voluntary control. Although there is a difference between the function and anatomical location of the SNS and ANS throughout the human body, they share some close connections with the central nervous system, for example within the hypothalamic region (Hoyer & Knappe, 2020).

The ANS can be further divided into the sympathetic, parasympathetic, and enteric nervous systems. A stimulation of the sympathetic nervous system, as would be the case under stress, usually leads to increased levels of activation within the corresponding organ system (Hoyer & Knappe, 2020). During the encounter, the amygdala, a brain area responsible for the processing of emotions, sends a so-called distress signal to the hypothalamus, a region within the brain that functions as a command centre and is responsible for the communication with the rest of the body through the nervous system, triggering a fight or flight response to handle the event. By utilizing the ANS, a response from the adrenal glands is incited, leading to the

release of the amine hormones epinephrine, also known as adrenaline, and norepinephrine into the bloodstream (Harvard Health, 2020).

The adrenal medulla is the main source of epinephrine and norepinephrine within the human body. Norepinephrine is known as the primary neurotransmitter in sympathetic fibres, and it enhances the heart rate, contraction of smooth muscle and cardiac muscle, as well as blood pressure. Epinephrine has been found to act on many organ systems by stimulating specialized receptors and thereby reinforcing the actions of sympathetic nerves. Because of this, it is a central contributor to the fight-or-flight response during stressful situations. Additionally, epinephrine has been identified as an important factor for the general stress response as well as the emergence of diseases related to chronic stress (Lovallo, 2016).

Activity of the parasympathetic nervous system on the other hand is associated with bodily hibernation and the overall reduction of the level of activation within the body and it is predominantly active during digestion and reproduction (Hoyer & Knappe, 2020). The parasympathetic fibres utilize acetylcholine to regulate the smooth muscle fibres of the heart muscle and cardiac pacemakers (Lovallo, 2016). Although separating the sympathetic and parasympathetic nervous systems into activating and reducing system seems feasible, it is not as simple as that. While an antagonistic relationship between the two nervous systems applies for some organs, like the heart, lungs, or stomach, the salivary glands, for example, are stimulated by both the sympathetic as well as the parasympathetic nervous systems (Hoyer & Knappe, 2020).

The enteric nervous system, which is sometimes described as the real autonomous nervous system, functions widely independently from the ANS and SNS and governs the function of the gastrointestinal tract (Hoyer & Knappe, 2020). As indicated by its name, it is located entirely within or around organs of digestion. Although seen as a distinct part of the nervous system, it is to some degree responsive to the sympathetic and parasympathetic nervous systems (Lovallo, 2016).

As already indicated above, another major player in the physiological stress response is the endocrinological system. Early research on psychophysiological research has shown a specificity in the hormonal response to stressful and arousing conditions (Natelson et al., 1976). If a healthy individual gets exposed to an acute stressor, homeostasis is ensured by the carefully coordinated functioning of diverse glands that are located throughout the body. Prolonged stress exposure represents an

ongoing challenge to the bodily homeostasis, referred to as the allostatic load (McEwen & Stellar, 1993), which can result in over- or underproduction of certain hormones and, as a result, contributes to a wide range of diseases (McEwen, 1998). Amongst the most notable glands concerning the stress response are the hypothalamus, the pituitary gland, the adrenal cortex, as well as the cortical medulla. These four glands are part of an important system called the hypothalamic-pituitary-adrenocortical axis (HPAC). A malfunctioning of the HPAC has been associated with the emergence of many psychological disorders such as depression or post-traumatic stress disorder (Ehlert et al., 2001).

Hans Selye recognized the relevance of HPAC as an essential topic of biopsychological and medical stress research as early as the 1930s. Decades before the discovery of the hypothalamic control hormone corticotropin-releasing hormone (CRH) within sheep by Vale et al. (1981) or the breakdown of adrenocorticotrophic hormone (ACTH) as two of the major stress hormones, Selye deduced the importance of cortisol, a hormone produced within the adrenal cortex, for pathological alterations as a consequence of physical or chemical stressors (Hoyer & Knappe, 2020).

Simply put, these three hormones are intimately linked, and a chemical imbalance within the CRH-ACTH-Cortisol-axis has a significant impact on the human body's susceptibility to diseases and physical impairments. CRH gets released from neuroendocrine neurons within the hypothalamus and thereby stimulates the production and release of ACTH from the pituitary into the bloodstream. The circulating ACTH further incites the adrenal glands to synthesize corticosteroids like cortisol, which in turn influence a vast array of physiological processes throughout the human body (DeMorrow, 2018). A rise in cortisol can even be considered a definitive sign of stress, making cortisol a central indicator of the stress response. Nonetheless, it must be made clear that even though cortisol plays a major role in the stress response, it does not stimulate the stress response; it regulates it. The major drivers of the fight-or-flight response are the sympathetic nervous system and the epinephrine circulating in the bloodstream (Munck et al. 1984).

It is worth noting that cortisol is not exclusive to the stress response. During normal periods of function, it fulfils a permissive role by ensuring the normal functioning of other systems like the control of inflammation, water diuresis, sodium retention within the kidneys, and many more. A negative feedback loop ensures that cortisol output is

regulated in a healthy individual. The cortisol returns to the hypothalamus and the pituitary via circulation, which in turn reduces the release of other neuroendocrine substances like ACTH, causing a reduction of the cortisol release at the adrenal gland (Lovallo, 2016).

Amongst the major influential factors that can induce sustainable changes in the composition of this hormonal balance are psychological stressors, chronic pain, malnutrition, and overeating (Kirschbaum & Hellhammer, 1999). An elevated level of CRH, for example, increases the likelihood of depression or anxiety disorders (de Kloet et al., 2005), while alterations in the cortisol level contribute to coronary heart diseases as well as hypertension (McEwen, 1998).

The physiological stress response can be understood as an attempt to maintain homeostasis in the face of adversity. A simplistic version of this process would be described as a force (a stressor) putting tension on the bodily homeostasis by evoking a stress response to combat the threat. If the body is unable to overcome the situation, the consequence is a chronic response, which might result in harm or even death to the individual if unrelieved (Lovallo, 2016).

Measuring the impact of stress

This complex interaction of the ANS and the endocrinological system can be measured by looking at the changes within some of the vital organs, like the heart. It is necessary to mention that the heart can regulate its own function, like the heart rate or the blood pressure. The natural rate of contraction of our heart is given by the sinoatrial node, a group of specialized neurons that steadily produce a heart rate of 110 beats per minute due to internal organization. The better known 60 beats per minute during a resting state are caused by the influence of the ANS, more specifically the parasympathetic nervous system. Another example would be the regulation of blood pressure by the so-called *Frank-Starling mechanism*. This response regulates the force of the heart's contraction internally by contracting more effectively when stretched to a greater degree. Therefore, the heart can keep up with the flow demands placed on it by systemic circulation. Although, like every other vital organ, the heart can regulate its appropriate functioning on its own to adapt to slowly changing environmental demands, the influence of the ANS and endocrinological system is

needed to sustain in an inconsistent environment, which is everyday life (Hall & Hall, 2020).

Hereby, the signals from the autonomic sensory nerves and the endocrine feedback engage in a negative feedback loop which acts by detecting the state of the regarding organ and comparing it to a set point. If the function departs from the regular set point, countermeasures act and regulate the parameters back to normal. One could think of the negative feedback loop as if a thermostat shuts off a furnace when it detects that the room temperature has reached a certain level (Lovallo, 2016).

Stress has been identified as a major player concerning influences on bodily homeostasis, and it has been linked to physiological responses in the form of the HPAC and the ANS (Kerr et al., 2020), which reflect onto various changes in heart rate, blood pressure (Lovallo, 2016), or the variability of the heart rate (Fuller, 1992; Agorastos et al., 2019). By measuring the alterations of these representative parameters, it is possible to indicate the individual's stress response to an acute stressor.

Heart rate variability

The frequently researched *heart rate variability* (HRV) refers to the beat-to-beat variability between individual heart beats, called interbeat intervals. These complex oscillations of our heart can be described as chaotic in nature and allow our cardiovascular system to adjust to unexpected physiological challenges (Malik & Camm, 1995; Shaffer & Ginsberg, 2017). They are usually described by the standard deviation of the successive R-waves of the cardiac cycle (Dishman et al., 2000).

To be able to fully understand R-waves, a short overview of the heart's function is necessary. When deoxygenated blood returns to the heart, a pump mechanism transports the blood into the lungs, where it gets enriched with oxygen. The oxygenated blood then gets pumped out of the heart's ventricle and into the body. This sequential pumping process is controlled by electrical signals that are generated by the heart. An electrocardiogram (ECG) detects and measures these signals in the form of upward or positive and downward or negative deflections. In general, there are three components to the ventricular depolarization, which can be depicted by the ECG and are called the QRS complex. The first negative deflection on an ECG is called a Q-wave, while the first positive deflection is called an R-wave. If another negative deflection occurs after

the first one, it is called an S-wave. Since our pulse is generated by ventricular contraction, the distance between two of these QRS cycles, measured from one peak of the R-wave to the next, can be used to estimate our heart rate (Kusumoto & Bernath, 2012).

There have been findings indicating the influence of stress on HRV within healthy individuals (Sloan et al., 1994; Dishman et al., 2000), and even medically induced activations of the HPAC, therefore only simulating a stressful situation, showed a significant reduction in HRV in healthy individuals (Agorastos et al., 2019).

A reduction in HRV has been linked to a variety of health-related issues like cardiac arrhythmia and cardiac mortality (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996), increased proinflammatory cytokines (Thayer & Sternberg, 2006), as well as psychopathologies including depression, anxiety, and schizophrenia (Clamor et al., 2014). Further research indicates an inverse relationship between high levels of anxiety and heart rate variability in patients with clinically relevant anxiety disorders such as panic disorders (Friedman & Thayer, 1998), social anxiety, obsessive compulsive disorders (OCD), and generalized anxiety disorders (GAD) (Thayer & Friedman, 1996; Pittig, et al., 2013). This inverse relationship could also be found in otherwise healthy individuals (Fuller, 1992; Watkins et al., 1998). Ruiz-Padial and Thayer (2014) indicated that higher HRV allowed participants to process emotional stimuli more efficiently and recognize the corresponding threat or safety signals, while low HRV showed slower processing and recognition of threat or safety signals, which imposed a great risk on the individuals since they might not be able to react accordingly to an incoming threat. In a meta-analysis conducted by Kim et al. (2018), they compared 37 recent studies and confirmed that increased stress levels have been found to be reflected in reduced HRV, therefore further supporting the use of HRV for the objective assessment of psychological health and stress.

Knowledge gap and hypotheses

The literature presented above indicates that trait anxiety is a personality trait that is pronounced to a certain degree within every person. Throughout the years, it has been linked to a higher likelihood of developing stress-related diseases (e.g., Gomes et al., 2017) and anxiety, due to the individual's increased susceptibility to threats to their self-esteem (Spielberger et al., 1970) and a more regular negative state of mind in objectively stressful situations (Rapee & Heimberg, 1997; Sarason & Sarason, 1990). The physiological stress response has been extensively studied within the last century, and the consequences of frequent as well as chronic stress response patterns, ranging from acute health issues to long-term damages and even burnout, are well known. The indicated effect of high trait anxiety scores as a potential stress enhancer should therefore be measurable by assessing one of the most prevalent stress markers known, the variability of the heart rate.

This has already been done in a study by Fuller (1992), where he investigated the effect of trait anxiety levels on heart rate and HRV in a group of 45 female baccalaureate students in the context of a naturally occurring stressor (e.g., their graduate comprehensive examination). The participants were tested two weeks before their exam, on the day before the exam, and a week afterwards. He found that female participants who scored high on trait anxiety had an overall lower HRV than those who scored lower on trait anxiety. HRV values were estimated using the low frequency to high frequency (LF/HF) ratio, which represents the influence of the sympathetic and parasympathetic nervous systems on the heart rate. These findings were consistent throughout all three time points of measurement.

Another example would be the work of Watkins and colleagues done in 1998, which investigated the effect of trait anxiety, measured via the STAI-T scale, on the respiratory sinus arrhythmia (RSA) as well as baroreflex control of the heart rate (BRC), which represent the vagal control of the heart. Both variables contribute to the HF component of the LF/HF ratio and hence were estimated using the LF/HF ratio of the HRV. Within a sample of 49 men and 44 women ($N = 93$), the researchers found that trait anxiety scores were inversely related to the levels of BRC ($r = -.30, p < .005$) and RSA ($r = -.26, p < .05$). Furthermore, high trait anxiety was associated with significantly reduced vagal control of the heart when compared with low trait anxiety participants, as has been indicated by a 36% reduction in BRC ($p < .001$) and an 8% reduction in

RSA ($p < .05$), therefore concluding a reduced HRV in highly trait anxious individuals (Watkins et al., 1998).

However, the research on the topic does not only provide evidence in support of this hypothesis. In their study, Dishman et al. (2000) investigated the relationship between self-reported trait anxiety, emotional stress, and the parasympathetic nervous systems components of HRV and whether these relationships are independent of personality and cardiorespiratory fitness. Trait anxiety was assessed by the short form of the Taylor Manifest Anxiety Scale (TMAS), and HRV was estimated by the LF/HF ratio. Their findings report an inverse relationship between subjectively perceived stress and HRV; however, this relation as well as both variables were independent of age, gender, trait anxiety, and cardiorespiratory fitness levels.

A major critique has come up regarding the LF/HF parameter used by Fuller and Watkins. The parameter is based on the assumption that the LF component is generated by the sympathetic nervous system and the HF component reflects parasympathetic nervous system activity. In a healthy individual, the regulatory influence of the parasympathetic nervous system exceeds the influence of the sympathetic nervous system, resulting in a reduction of the heart rate to an average of 75 beats per minute and a reduced blood pressure. This imbalance in the influence of the sympathetic and parasympathetic nervous systems is called parasympathetic dominance (Shaffer et al., 2014). Due to the theoretical nature of the LF/HF ratio, this dominance should be reflected in the measure. The problem is that the LF component isn't purely produced by the sympathetic nervous system; up to half of it stems from the parasympathetic nervous system as well as other factors. Furthermore, a simple ratio does not consider the complex interactions of a non-linear and non-reciprocal process. Therefore, especially concerning the most commonly used 5-min measurement interval for HRV, the *root mean squared standard deviation* (RMSSD) has been suggested (Shaffer & Ginsberg, 2017).

All the research concerning the effects of trait anxiety on HRV provided in this section refers to the overall reduced tonic HRV of individuals high in trait anxiety. To my knowledge, this paper is the first to investigate the effect of trait anxiety on the change in HRV after an acute stressor.

Based upon the findings concerning the increased experienced anxiety within highly trait anxious individuals (Morales, 2012), the inverse relationship between trait

anxiety and HRV by Fuller (1992) and Watkins et al. (1998), further studies linking stress to a decrease in HRV (Sloan et al., 1994; Agorastos et al., 2019), as well as considering the most recent knowledge regarding the representativity of the RMSSD for HRV, I propose my first hypothesis:

H1: Trait anxiety increases the change in HRV measured by the RMSSD in response to an acute stressor

Since, according to Lazarus' transactional stress model, the human stress response is a product of our appraisal of the situation, an individual's tendency to perceive situations as threatening or harmful should be considered important within the equation of the human stress response. In his doctoral thesis, Abdullatif (2006) found a positive effect of trait anxiety on primary appraisal and a negative effect on secondary appraisal in healthy individuals, which in turn should result in an indirect effect of trait anxiety on the physiological stress response, such as the HRV.

While most research concerning appraisal processes has its focus on primary and secondary appraisal, it might be interesting to investigate the effects of retrospective stress appraisal, as has already been done by Korial et al. in 1972. Retrospective stress appraisal describes the process of asking the participants to rate their experienced stress level after the situation has occurred. Hereby, the primary and secondary appraisals have already concluded, and the participants should be able to clearly communicate the result of the appraisal processes, which is how much stress they experienced. By utilizing their retrospective assessment, it should be easier to get an estimate of the stress evoked by the acute stressor itself and not by anticipatory stress before the stressful situation occurred. Of course, this way, it is possible that a reappraisal process has already happened.

Although a recent study by Gaab et al. (2005) indicated no effect of retrospective stress appraisal on the cortisol response of the HPAC, which should reflect the human stress response, more research needs to be done to further investigate the possible relevance of this construct. To my knowledge there are no existing papers concerning the possible influence of trait anxiety on the retrospective appraisal as well as possible effects of said appraisal on HRV as a stress response marker. Therefore, based on the findings by Abdullatif (2006) I propose my second hypothesis as follows:

H2: Trait anxiety influences retrospective stress appraisal after an acute stressor

In addition to the inverse relationship between trait anxiety and HRV found by Fuller (1992) and Watkins et al. (1998), further research conducted by Abdullatif (2006) found an effect of trait anxiety on appraisal processes, which in turn have been shown to affect the physiological stress response. This coincided with the theory proposed by Lazarus (1966) and Lazarus and Folkman (1968). Furthermore, Gomes et al. (2017) found that cognitive appraisal functioned as a mediator of the effect of anxiety on the likelihood of developing burnout, a consequence of chronic stress. Therefore, a mediation of the effect of trait anxiety on the physiological stress response by stress appraisal can be assumed. Thus, my final hypothesis is as follows:

H3: Retrospective stress appraisal mediates the effect of trait anxiety on the change in HRV in response to an acute stressor

For a better overview the hypotheses are illustrated in *Figure 1*.

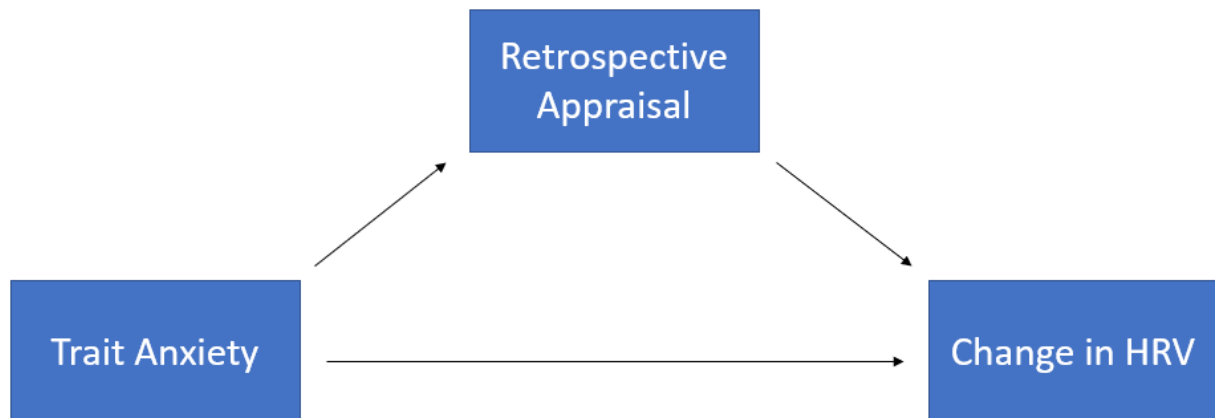


Figure 1. Illustration of the research model. Displayed are the elements of the research and how they are connected.

Methods

The MuSkiBa-Project

The study design of this thesis is part of an ongoing research project that examines a wide range of variables concerning the effects of music listening on skin barrier recovery in relation to stress. The main project has been initiated by the Music and Health Lab of the University of Vienna under the guidance of Prof. Dr. Urs Nater, and the respective data has been gathered since February 2020. In this experimental study, female participants aged 18-35 can participate in one of two studies. In both studies, the same wide range of variables will be assessed in all suitable applicants. However, the *MuSkiBa2 Project*, which will be the basis for my thesis, also actively induces stress via a social stressor, the so-called *Trier Social Stress Test* (TSST) (Kirschbaum et al., 1993), which is addressed in detail further below.

Recruitment and Sample

The participants were mostly recruited via postings on social media platforms such as Facebook. All interested participants had to contact the team via email and undergo a detailed telephone screening lasting approximately 20 minutes to ensure that none of the exclusion criteria were met. Due to available research by Kirschbaum et al. (1999) about the impact of gender and hormonal influences on the HPAC response to stress, certain precautions were taken. To minimize the known impact of hormones on the stress reaction, only female participants, who must not use hormonal contraception, were included. To further limit possible variations, data concerning the menstrual cycle of all applicants was gathered to ensure the test would take place during their follicular phase. Aside from the gender restriction for better comparability, participants had to be between the ages of 18 to 35 years old, physically, and mentally healthy, which also excluded over-, or underweight applicants measured using the BMI-Score derived from the provided information about their height and weight and they had to not smoke or drink alcohol on a regular basis. Due to the requirements of the MuSkiBa project, I had to exclude every applicant who studied music or some sort of instrument, worked in the music industry, or possessed absolute hearing, since they might react differently to the music presented to them as a part of the larger study.

Further, to prevent experience-based influences on the results, it was ensured that none of the participants had experience with the Trier Social Stress Test, which was used to induce stress. All participants received an expense allowance in the amount of 45€.

Due to the COVID-19 pandemic, there have been a wide range of difficulties concerning the feasibility and safety of in-person testing, leading to a decrease in the number of participants. On many occasions, appointments had to be cancelled due to possible COVID infections or missing vaccinations. The final sample consists of 41 participants, of whom 8 had to be excluded for various reasons. During the assessment of three individuals, the sensor used for recording the heart rate as well as heart rate variability was missing data, four participants did not fill out the questionnaire concerning their retrospective appraisal of the situation, and one specific participant had to be excluded as it became clear that she had informed herself about the TSST in advance to be prepared and score a better result, therefore rendering the data gathered useless. This leaves us with 33 participants for the calculation of the parameters of interest.

Materials and Measures

Acute stressor

For the acutely induced stress situation, I used the *Trier Social Stress Test* (TSST), which is a validated and reliable acute stressor that can be used under experimental conditions (Allen et al., 2017): It was originally developed by Kirschbaum et al. (1993) and is considered the gold standard and most employed paradigm in biopsychological stress research (Kudielka et al., 2007). It consists of a 5-minute-long interview-style presentation with 3 minutes of preparation time. The participants are told that the audience consists of two experts who are trained behavioural analysts and that the whole procedure will be recorded using a video recorder and a microphone presented on the table for later analysis purposes. The participants are told by the experimenter to apply for their dream job, while the two experts are taking notes whenever the participant asks a question or discontinues their presentation. They are instructed to not respond to or interact with the participant in any way unless the

participant does not continue with their presentation after a twenty-second break. Once the time is up, the participant is confronted with an unknown mental arithmetic test during which she needs to serially subtract 17 from the number 2043. Whenever an error occurs, one of the observers interrupts the participant and demands to start all over again from 2043. If no error occurs, the observers are instructed to remind the participant to look directly into the camera and speak clearly into the microphone to remind them of being recorded.

Mental arithmetic has been used as a prototype mental stressor since 1963. There are different versions of these tests, but they all place a demand on the working memory, which gets increasingly unpleasant after a short period of time (Brod, 1963). Mental arithmetic has been shown to produce a significant decrease in the vagal tone to the heart, which in turn increases sympathetically mediated cardiac activity, leading to significant increases in heart rate and contractility (Lovallo, 2016).

The TSST has been shown to elicit psychological stress on various measures (Childs et al., 2006; Rimmelé et al., 2017) and has therefore been chosen as the acute stressor in the overall study. Although the instructions given to the participant as well as the interview-style presentation are in German, no differences in validity are expected since the stress evoked should be independent of the language used.

Trait anxiety

To measure trait anxiety, the trait anxiety section of the *State-Trait-Angstinventar* (STAI) (Laux et al., 1981), translated from the original *State-Trait-Anxiety-Inventory* (Spielberger et al., 1970), consisting of 20 items, has been used. The full trait anxiety portion of the *State-Trait-Angstinventar* by Laux et al. (1981) is provided in the appendix. Both the state anxiety scale and the trait anxiety scale have a high level of internal consistency (Cronbach's $\alpha = .90$). Participants specify on a four-point Likert-scale how they usually feel, with answers ranging from “*Fast nie*” (almost never) to “*Fast immer*” (almost always). Higher overall scores indicate higher trait anxiety, with 7 of the 20 items being of negative polarity. Due to the nature of the possible answers given, the obtainable points range from 20 to 80. The questionnaire must be completed online at home before the laboratory appointment takes place.

Retrospective stress appraisal

Retrospective stress appraisal was assessed using a Visual Analog Scale (VAS) (see Appendix) immediately following the acute stressor by having participants rate the TSST-procedure on a scale of 0 to 100, with a higher score indicating more subjective stress. The German wording was “*Die Situation war stressig für mich*” and it represents the result of the finished primary appraisal of the situation as well as the secondary appraisal of whether the available resources were sufficient to cope with the stressful situation. This item, which originated with the TSST-VAS, was chosen due to a lack of research on the role of retrospective appraisal in the matter. Although no reliability or validity scores are available for this scale, I decided to use it since it is part of the standardized measurements taken as part of the TSST.

Stress response and heart rate variability

Finally, the HRV has been measured via a chest strap by *Movisens (GmbH)*. The *Move 4* is a physical activity sensor specifically developed for mobile monitoring of physiological activity and has been designed and tailored for use in research applications. I decided to measure the change in HRV using the root mean squared standard deviation (RMSSD) of the R-R intervals. The RMSSD reflects the beat-to-beat variance in the heart rate and is the primary time-domain measure to estimate the changes in HRV. While many studies used the so-called low frequency to high frequency (LF/HF) ratio to measure the changes of the HRV, there have been uncertainties about the influence of the respiration rate on the HF index (Shaffer & Ginsberg, 2017). Therefore, I decided to use the RMSSD as my preferred index. Hereby I looked upon the difference between the HRV during the baseline measurement and the respective HRV value during the peak stress of every participant. This seemed feasible since some participants might not experience the most stress after the TSST is over but rather at the beginning or during a specific part of the test. By utilizing the continuous recording of the chest strap, we were able to use the first five minutes of the measurement as a baseline value and identify each participant's personal peak stress value by identifying the time window with the lowest HRV. A lower HRV indicates higher psychological stress and is therefore expected. The HRV data has been cut into eleven four-minute (240s) windows, which are crucial

points during the overall test protocol ranging from the baseline to the end of the overall MuSkiBa 2 project.

Further explorative analyses

Due to the complex nature of the physiological stress response and the many influences of multiple processes on the heart rate and its variability, I decided to do some alternative explorative analyses in case of a non-significant main effect using the proposed methods above. First, I planned on analysing possible differences within the HRV at baseline that might affect the change in HRV from baseline to peak stress. Furthermore, I decided to use the data gathered on perceived subjective stress by asking the participants directly how stressed they are feeling now (*“Wie sehr fühlen sie sich gestresst?”*) via visual analogue scales (VAS) during the experiment to investigate another more direct measurement of stress that might be more suitable to show the proposed correlation between trait anxiety and the stress response. The VAS is provided in the appendix. I decided to investigate this relationship using a linear regression analysis. The STAI-T score was used as the independent variable and the subjective stress measured via the VAS as the dependent variable. The three timepoints used for these calculations are at baseline before (VAS1), immediately preceding (VAS2), and directly after the TSST (VAS3). These three items provide insight into the subjective stress of each participant at that specific moment.

Statistical Analyses

The analyses of the hypotheses in question have been conducted using IBM SPSS 27. To investigate the paths of the mediation model, simple linear regression has been used. The mediation analysis is based on the *PROCESS* tool by Andrew Hayes (2013).

Results

The final sample consisted of 33 female participants aged 18 to 33, with a mean age of 24,12 ($SD = 3,18$). On average, the participants scored 37,18 ($SD = 9,49$) points on the STAI-T scale, with a range of 20-56. At the baseline (before the start of the TSST), the participants showed an average RMSSD of 47,46 ($SD = 27,05$), ranging from 14,32 to 147,47. The average reduction in HRV from the baseline value of each participant to their lowest HRV value, indicating their peak stress, was 26,44 ($SD = 20,63$).

As can be seen in *Figure 2*, visual inspection suggests that the expected reduction in heart rate variability over the course of the TSST (T1, baseline, to T5, after TSST), indicating the physiological stress response, could be found.

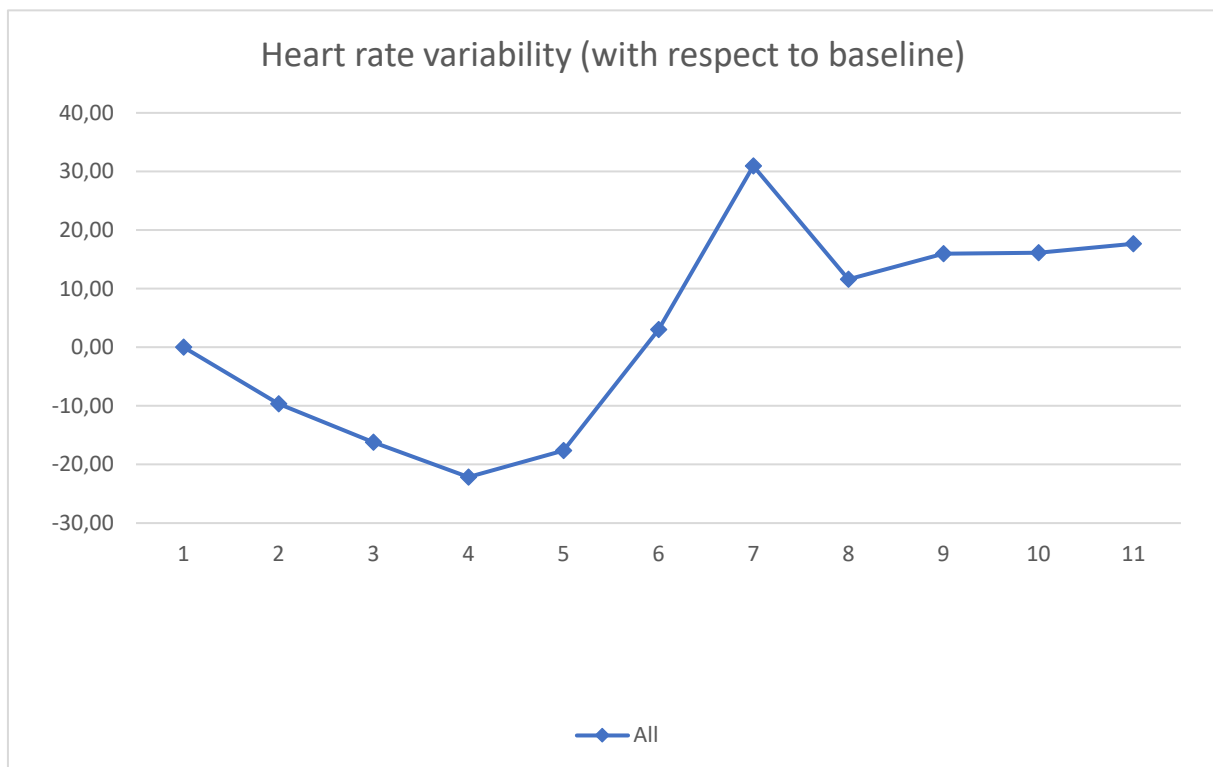


Figure 2. Change in HRV with respect to baseline (using RMSSD)

First, to ensure the requirements for a mediation analysis are fulfilled, I analysed the main effect of the model using a linear regression analysis with the participants' STAI-T scores as the independent variable and the change in HRV from the baseline to each participant's personal peak stress value, which is indicated by the lowest HRV score as the dependent variable.

The result of the simple linear regression indicated no explanation of the variance of the change in HRV with respect to baseline with $R^2 = 0.00$ and the model was not significant, $F(1,31) = 0.00$, $p = .943$. As a result, it could not be shown that trait anxiety predicted the change in HRV ($\beta_1 = -.03$, $p = .943$).

To illustrate the results of the linear regression analysis investigating the effect of trait anxiety on change in HRV I created a scatterplot (see *Figure 3*) using the variables STAI-T Scores and the change in HRV between the baseline and peak stress.

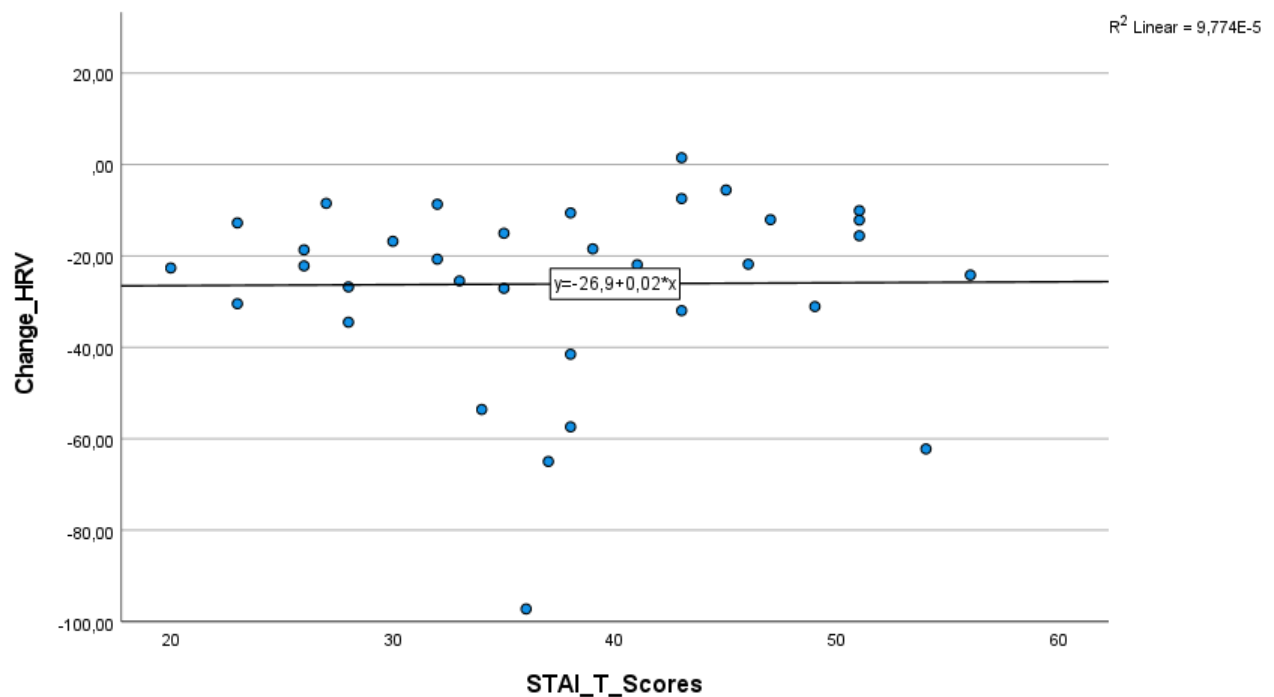


Figure 3. Scatterplot of STAI-T scores and changes in HRV.

To investigate the second hypothesis, I analysed the effect of the STAI-T score on the retrospective stress appraisal. The simple linear regression analysis utilizing STAI-T scores as the independent variable and retrospective stress appraisal as the dependent variable showed no significance for the model proposed, $F(1,31) =$

0.07, $p = .796$, with $R^2 = 0.00$. Trait anxiety scores did not predict the retrospective stress appraisal ($\beta_1 = .13$, $p = .796$).

For illustrative purposes, another scatterplot (see *Figure 4*) was created using STAI-T scores and retrospective appraisal measured via TSST_VAS.

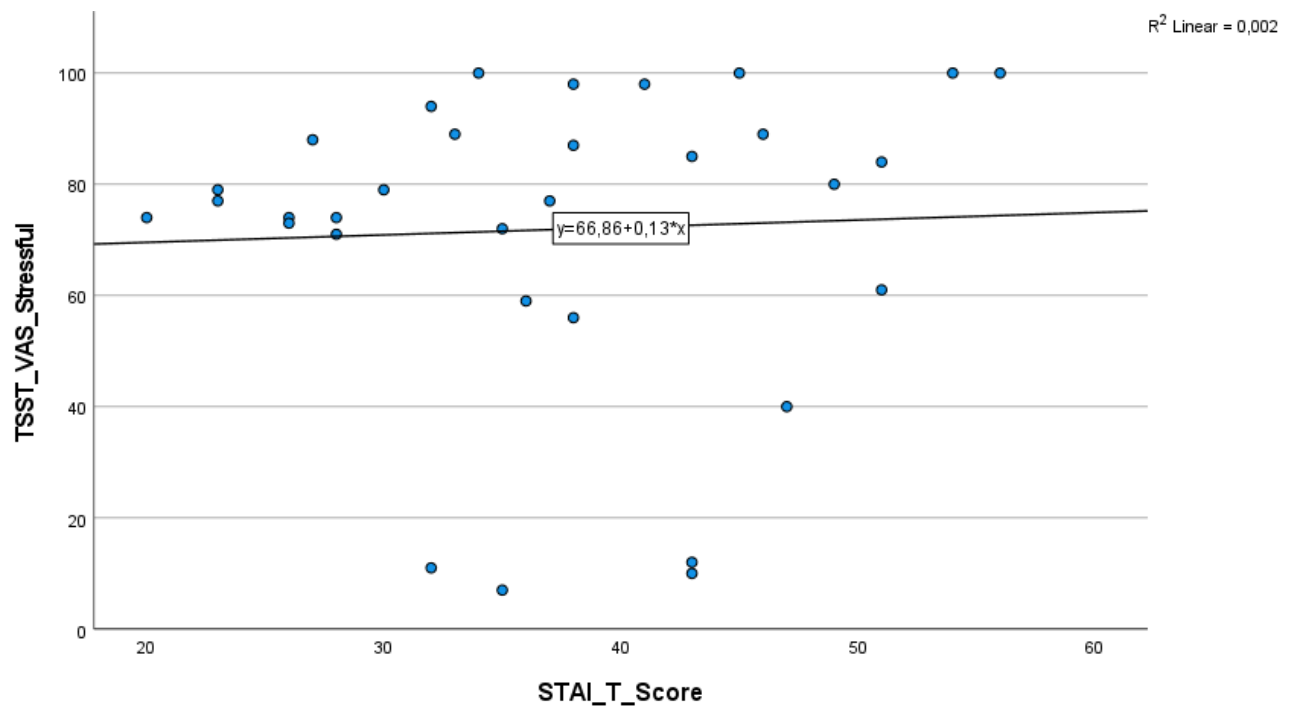


Figure 4. Scatterplot of STAI-T scores and retrospective stress appraisal.

Because the direct effects among the variables were not significant, the requirements for a mediation were not met. As a result, no further analysis concerning the proposed mediation of the effect of trait anxiety on the change in HRV by retrospective appraisal was conducted.

Since existing differences at the baseline HRV measure may have affected the resulting change scores from baseline to peak stress, further analysis investigating the effect of STAI-T scores on HRV at baseline was conducted. This is part of the proposed explorative analyses mentioned earlier. Another simple linear regression using STAI-T scores as the independent variable and the HRV at baseline as the dependent variable was done to examine a possible effect of trait anxiety scores on the HRV at baseline.

The according simple linear regression analysis depicts the non-correlation between the two parameters, $F(1,31) = 0.00$, $p = 1.00$, with $R^2 = 0.00$. STAI-T scores did not predict HRV at baseline ($\beta_1 = .00$, $p = 1.00$)

As can be seen in the scatterplot in Figure 5 no trend could be found within the data.

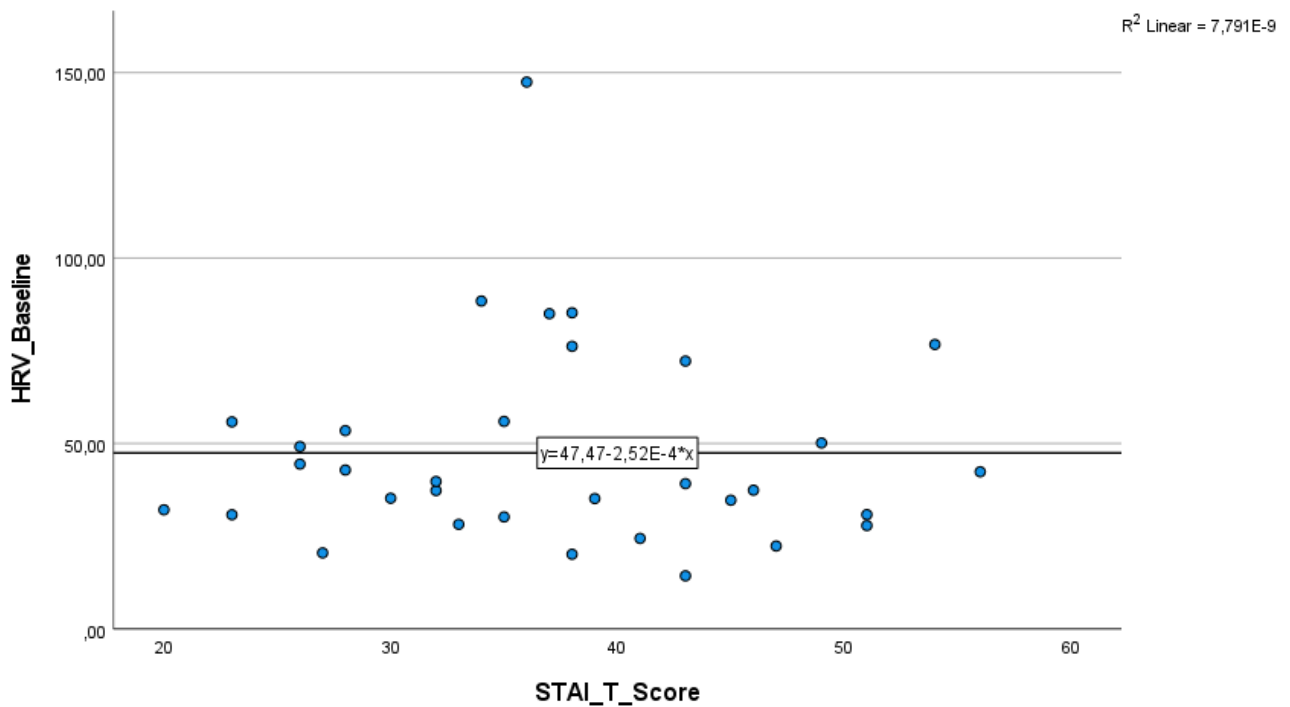


Figure 5. Scatterplot of STAI-T scores and HRV at baseline.

As has been proposed during the methods part of my thesis, due to the complex interactions of the mechanisms influencing the heart, it is possible that HRV might not be the most suitable parameter for representing the stress experienced by the participants. Therefore, a further analysis of the effect of the STAI-T score on perceived stress, utilized by VAS scales, has been conducted. Hereby, a slightly larger sample (36 participants) was used, since participants previously excluded due to technical difficulties with the HRV measurement could be included.

The simple linear regression indicated that baseline perceived stress was predicted by the STAI-T score $F(1,34) = 3.26$, $p = .080$ with $R^2 = 0.09$. Therefore, showing a positive trend of trait anxiety scores on the subjective stress at baseline ($\beta_1 = .44$, $p = .080$).

The scatterplot depicted in *Figure 6* illustrates the possible trend given by the data.

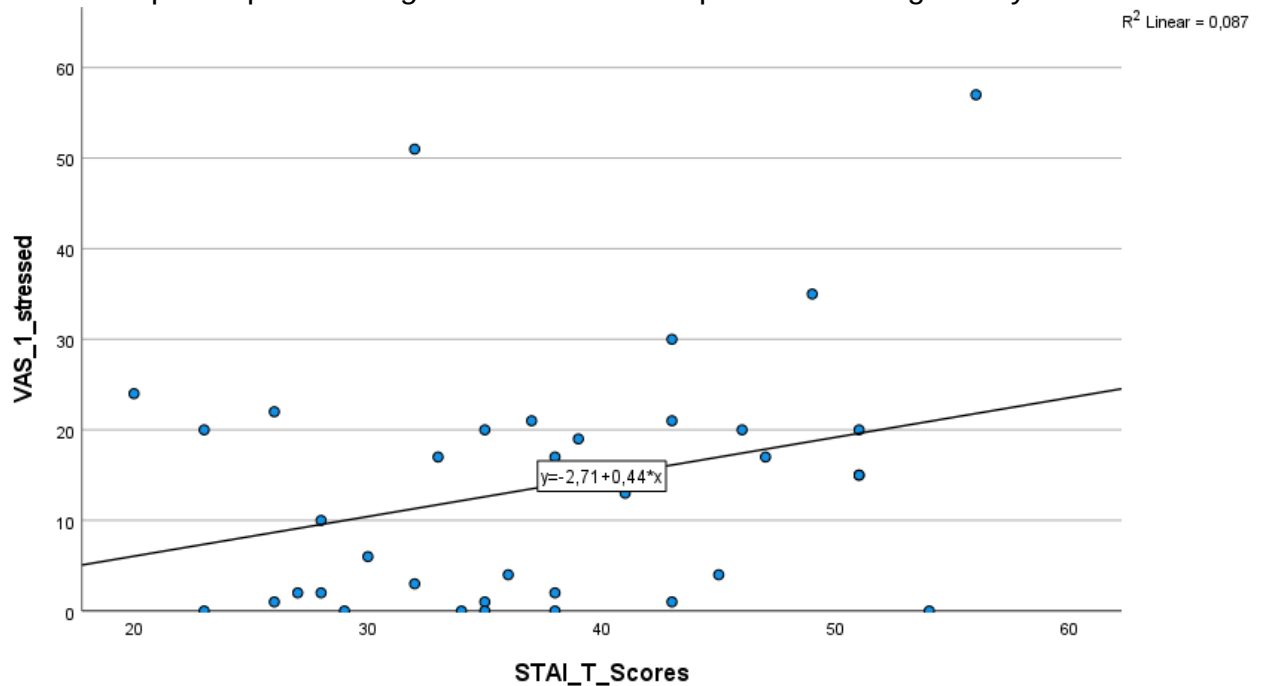


Figure 6. Scatterplot of STAI-T scores and perceived stress at baseline.

To investigate if the indicated trend of trait anxiety scores on the subjective stress at baseline persists throughout the experiment the according analyses were conducted using the data from the subjective perceived stress VAS immediately before and after the TSST. The simple linear regression using STAI-T scores and perceived stress before the TSST (VAS2) indicated that the model explained 6% ($R^2 = 0.06$) of the variance, but the model was not significant, $F(1,34) = 2.06$, $p = .160$. Therefore, STAI-T scores did not predict subjective stress before the TSST ($\beta_1 = .62$, $p = .160$).

As for the effect of STAI-T scores on perceived subjective stress after the TSST (VAS3) the model explained 3% of the variance in VAS3, and the model was not significant $F(1,34) = 0.87$, $p = .357$. STAI-T scored did not predict subjective stress after the TSST ($\beta_1 = .43$, $p = .357$).

Discussion

The study aimed to shed light on the influences of trait anxiety on the physiological stress response in the form of a change in heart rate variability after an acute stressor and, to my knowledge, is among the first to consider retrospective stress appraisal as a mediator in the given context. To investigate the effects of non-clinically relevant anxiety in healthy individuals, a very specific group of female participants was recruited, thereby eliminating possible influences of hormonal as well as gender differences.

Below, the results and their implications for the hypotheses presented above will be discussed, followed by possible shortcomings and limitations. Finally, some suggestions for further future research will be given.

The first analysis concerning the expected main effect of trait anxiety using the STAI-T score on stress-induced change in HRV could not show a greater reduction in HRV for individuals higher in trait anxiety after an acute stressor. Furthermore, I was unable to replicate the findings of Fuller (1992), who reported a significant inverse relation between trait anxiety scores and HRV, which has been estimated by the LF/HF ratio, in female baccalaureate students, or Watkins et al. (1998), who, likewise, found a significant inverse relation between trait anxiety and respiratory sinus arrhythmia (RSA) as well as baroreflex control of the heart (BRC). Both RSA and BRC reflect the parasympathetic influence on the HF component of the LF/HF ratio and therefore represent HRV.

There might have been various reasons for the failure to replicate possible effects. First, the participant's trait anxiety scores ranged from 20 to 56 points out of a possible 80. During their attempt to define the construct repression-sensitization via trait anxiety and defensiveness Krohne and Rogner (1986) categorized STAI-T scores of ≥ 56 as high while everything up to 55 was considered low. Following this cut-off score, out of the sample given, only one participant would qualify for the classification of high trait anxiety, with a score of exactly 56. Because of this interpretation, a limitation of the information value given by the data can be assumed. These rather medium to low scores within the STAI-T scale might have been due to the very strict exclusion criteria of the study, which automatically excluded everyone who reported having problems with anxiety as well as anyone who has ever been to therapy due to ethical issues with putting participants with already existing anxiety

problems in a situation which might worsen their condition. In combination with the small sample size due to the COVID-19 restrictions, only a rather small number of participants were tested. Furthermore, it needs to be considered, that the participants had to voluntarily contact us, which might have already filtered out individuals with high trait anxiety scores since they might not have had the courage to overcome their anxiety and apply for this study. Since the possible applicants were informed through the handed-out flyers that an acute stress situation would be part of the study, this might have deterred some possible participants.

The explanations given above also apply to the result of the second hypothesis concerning the influence of trait anxiety scores on appraisal. No effect of STAI-T scores on retrospective appraisal has been found, therefore contradicting the effect of trait anxiety on cognitive appraisal found by Abdullatif (2006). This difference in findings might be explained on the one hand by the low number of participants and, on the other hand, by the lack of participants with high STAI-T scores, which could lead to a lower informative value of the gathered data. Furthermore, while retrospective appraisal should give a more precise insight into the stress experienced because of the TSST procedure in contrast to anticipatory stress, reappraisal of the situation might have already taken place. This could skew the estimate of the experienced stress, as the participants' appraisal of the situation might have been intensified, mitigated, or even reversed.

Since no direct effects between STAI-T scores and the change in HRV or stress appraisal have been found, the requirements for the proposed mediation analysis using PROCESS by Andrew Hayes (2013) were not fulfilled. Therefore, the third hypothesis must be considered as not supported.

The exploratory analysis of the effect of trait anxiety on subjective stress arose from considerations of the complex nature of heart rate variability and its numerous implications. As stated in the literature part of my thesis, a rise in heart rate as well as a reduction in its variability are not exclusive to negative stress and anxiety but could also be interpreted as anticipation or physical demands such as having to use the toilet or running up the stairs to the examination room because the participant was late. Therefore, I concluded that the most direct way to identify if the participants were stressed was to use the visual analogue scale that asked them to indicate how subjectively stressed they felt, ranging from 0-100. The analysis of the effect of the

STAI-T score on subjective stress at baseline, using the data of the whole sample, including the previously excluded participants, fell just short of significance with $p=.080$. Although no conclusions can be drawn from this result, it might have been significant if the originally planned sample size of 80-100 participants had been reached, therefore indicating a trend towards a possible influence of trait anxiety on perceived stress at the baseline measurement. Further analysis, however, showed no correlation between the trait anxiety scores and the subjective stress measurements before or after the TSST. This result makes sense, as it would coincide with the very definition of trait anxiety. As already mentioned during the literature part of this thesis it is described as the personality disposition that describes a person's tendency to perceive situations as threatening (Gaudry et al., 1975) and react with increased state anxiety (Morales, 2012). Therefore, individuals high in trait anxiety more easily tend to appraise an objectively neutral situation as threatening, hence the higher subjective stress during the baseline measurement. Since the TSST is supposed to evoke anxiety within every individual, the difference in subjective stress diminishes, and no further correlation can be seen.

This might be accompanied by ceiling effects. If a highly anxious person reports a baseline score of 90 out of 100 points on subjective stress, they cannot rise above the maximum of 100 points, whereas a non-anxious person may report a much stronger increase. Therefore, the change in subjective stress is much greater than in individuals who reported high subjective stress at baseline.

Although baseline differences as well as ceiling effects might have affected the measure of the participants' perceived subjective stress, it seems improbable that this was the case with the change in HRV. First, the analysis showed no baseline differences that could explain why a correlation was not found when testing for the direct effect of trait anxiety on the change in HRV. Second, the HRV measure is not done using a subjective VAS ranging from 0-100 but by using a chest strap, which provides objective data. It can be assumed that even small changes within individuals that have a low HRV at baseline can be reliably detected, therefore rendering ceiling effects rather unlikely.

Based on the findings and limitations presented, it can be assumed that trait anxiety does not affect the physiological stress response after an acute stressor, as would have been shown by the HRV, but it might be able to influence the subjective

stress experienced. A non-matching change in the subjective and objective stress parameters has been reported before by Koriat et al. (1972), who found reduced subjective stress in participants who were trying to detach themselves from a stressful situation but no differences in the physiological stress response. Hence, it may be possible for trait anxiety to increase the intensity of the subjective stress, but not the objectively measured physiological stress response, thereby increasing the experienced state anxiety, as has been found by Morales (2012).

There are several implications for future studies derived from the information presented. First, while distinctions between clinically relevant and non-clinically relevant anxiety need to be made, it should be considered that a certain score on the trait anxiety scale of the STAI might be needed to display the proposed effects in the given literature. Since this study's recruitment methods solely relied on the applicants reaching out to us, we might have been unable to reach certain groups ranking higher on the requested score. A targeted recruitment based on the STAI-T score to generate two groups (high and low scores) and compare differences could give insight into possible variations between individuals scoring low, middle, or high on the trait anxiety scale. In addition, due to the difficulties of the COVID-19 pandemic explained above, my sample size only consisted of roughly half the desired target range. As a result, the chances for a type II error are vastly increased due to the seemingly high variability in HRV between participants and the possibly small effect of STAI-T scores on HRV. To increase the statistical power of the analysis, therefore reducing the chance for a type II error, a larger sample size would be needed.

Second, I am fully aware of the many more parameters for the measurement of HRV that are available. A full-on analysis of further time-domain measures as well as the known frequency measures, such as an in-depth analysis of possible uses of the LF/HF parameters, could shed light on the complex nature of the HRV response under acute stress.

Finally, because of the TSST's objective stressful nature, it may be impossible to demonstrate the effect of high trait anxiety on everyday stress. Since trait anxiety, per definition, increases the likelihood of appraising situations as threatening and therefore leading to more frequent experiences of stress, a situation known to induce stress within almost every individual fails to depict the very core of the concept. Therefore, it might be needed to evaluate another experimental situation that may be

stress-inducing but does not intentionally provoke it. The trend towards a possible effect of the STAI-T score on perceived subjective stress at the baseline TSST, which diminished at later timepoints, could point towards this assumption.

In conclusion, there were no effects of trait anxiety on the change in HRV after an acute stressor or on retrospective stress appraisal. However, an almost significant positive trend ($p=0.08$) indicating an effect of trait anxiety on perceived subjective stress at the baseline measure could be shown. This trend diminished at other time points of measure over the course of the objectively stressful TSST. Since the requirements for the proposed mediation were not fulfilled, it can be assumed that retrospective appraisal does not act as a mediator on the effect of trait anxiety on the change in HRV after an acute stressor. Considering all the limitations discussed above, it is possible to conclude that trait anxiety does not predict HRV at baseline or the change in HRV after an acute stressor in individuals with low to medium trait anxiety scores. Furthermore, trait anxiety did not influence the retrospective appraisal of the TSST as an acute stressor.

Literature

- Ader, R. (1980). Psychosomatic and Psychoimmunologic Research1: *Psychosomatic Medicine*, 42(3), 307–321. <https://doi.org/10.1097/00006842-198005000-00001>
- Agorastos, A., Heinig, A., Stiedl, O., Hager, T., Sommer, A., Müller, J. C., Schruers, K. R., Wiedemann, K., & Demiralay, C. (2019). Vagal effects of endocrine HPAC axis challenges on resting autonomic activity assessed by heart rate variability measures in healthy humans. *Psychoneuroendocrinology*, 102, 196–203. <https://doi.org/10.1016/j.psyneuen.2018.12.017>
- Allen, A. P., Kennedy, P. J., Dockray, S., Cryan, J. F., Dinan, T. G., & Clarke, G. (2017). The Trier Social Stress Test: Principles and practice. *Neurobiology of Stress*, 6, 113–126. <https://doi.org/10.1016/j.ynstr.2016.11.001>
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders* (Fifth Edition). American Psychiatric Association. <https://doi.org/10.1176/appi.books.9780890425596>
- Arnold, M. B. (1960). *Emotion and Personality* (2 vols.). New York: Colombia University Press.
- Bandura, A. (1982). Self-efficacy mechanism in human agency. *American Psychologist*, 37(2), 122–147. <https://doi.org/10.1037/0003-066X.37.2.122>
- Barber, T. X., & Coules, J. (1959). Electrical skin conductance and galvanic skin response during “hypnosis”. *International Journal of Clinical and Experimental Hypnosis*, 7(2), 79–92. <https://doi.org/10.1080/00207145908415811>
- Bowlby, J. (1961). Processes of mourning. *The International Journal of Psycho-Analysis*, 42, 317–340.
- Brod, J. (1963). Haemodynamic basis of acute pressor reactions and hypertension. *Heart*, 25(2), 227–245. <https://doi.org/10.1136/hrt.25.2.227>
- Childs, E., Vicini, L. M., & De Wit, H. (2006). Responses to the Trier Social Stress Test (TSST) in single versus grouped participants. *Psychophysiology*, 43(4), 366–371. <https://doi.org/10.1111/j.1469-8986.2006.00414.x>
- Clamor, A., Hartmann, M. M., Köther, U., Otte, C., Moritz, S., & Lincoln, T. M. (2014). Altered autonomic arousal in psychosis: An analysis of vulnerability and specificity. *Schizophrenia Research*, 154(1–3), 73–78. <https://doi.org/10.1016/j.schres.2014.02.006>
- Crocq, M.-A. (2015). A history of anxiety: From Hippocrates to DSM. *Dialogues in Clinical Neuroscience*, 17(3), 319–325. <https://doi.org/10.31887/DCNS.2015.17.3/macrocq>

- DeMorrow, S. (2018). Role of the Hypothalamic–Pituitary–Adrenal Axis in Health and Disease. *International Journal of Molecular Sciences*, 19(4), 986.
<https://doi.org/10.3390/ijms19040986>
- Dishman, R. K., Nakamura, Y., Garcia, M. E., Thompson, R. W., Dunn, A. L., & Blair, S. N. (2000). Heart rate variability, trait anxiety, and perceived stress among physically fit men and women. *International Journal of Psychophysiology*, 37(2), 121–133.
[https://doi.org/10.1016/S0167-8760\(00\)00085-4](https://doi.org/10.1016/S0167-8760(00)00085-4)
- Ehlert, U., Gaab, J., & Heinrichs, M. (2001). Psychoneuroendocrinological contributions to the etiology of depression, posttraumatic stress disorder, and stress-related bodily disorders: The role of the hypothalamus-pituitary-adrenal axis. *Biological Psychology*, 57, 141–152.
- Elliott, G. R., Eisdorfer, C., & Institute of Medicine (U.S.) (Hrsg.). (1982). *Stress and human health: Analysis and implications of research: a study*. Springer Pub. Co.
- Folkins, C. H. (1970). Temporal factors and the cognitive mediators of stress reaction. *Journal of Personality and Social Psychology*, 14(2), 173–184.
<https://doi.org/10.1037/h0028688>
- Folkins, C. H., Lawson, K. D., & Opton, E. M. (1968). Desensitization and the experimental reduction of threat. *Journal of Abnormal Psychology*, 73(2), 100–113.
<https://doi.org/10.1037/h0025490>
- Friedman, B. H., & Thayer, J. F. (1998). Autonomic balance revisited: Panic anxiety and heart rate variability. *Journal of Psychosomatic Research*, 44(1), 133–151.
[https://doi.org/10.1016/S0022-3999\(97\)00202-X](https://doi.org/10.1016/S0022-3999(97)00202-X)
- Fuller, B. F. (1992). The effects of stress-anxiety and coping styles on heart rate variability. *International Journal of Psychophysiology*, 12(1), 81–86.
[https://doi.org/10.1016/0167-8760\(92\)90045-D](https://doi.org/10.1016/0167-8760(92)90045-D)
- Gaab, J., Rohleder, N., Nater, U. M., & Ehlert, U. (2005). Psychological determinants of the cortisol stress response: The role of anticipatory cognitive appraisal. *Psychoneuroendocrinology*, 30(6), 599–610.
<https://doi.org/10.1016/j.psyneuen.2005.02.001>
- Gaudry, E., Vagg, P., & Spielberger, C. D. (1975). Validation of the State-Trait Distinction in Anxiety Research. *Multivariate Behavioral Research*, 10(3), 331–341.
https://doi.org/10.1207/s15327906mbr1003_6

- Gomes, A. R., Faria, S., & Vilela, C. (2017). Anxiety and burnout in young athletes: The mediating role of cognitive appraisal. *Scandinavian Journal of Medicine & Science in Sports*, 27(12), 2116–2126. <https://doi.org/10.1111/sms.12841>
- Grinker, R. R., & Spiegel, J. P. (1945). *Men under stress*. New York: McGraw-Hill.
- Hackett, T. P., & Weisman, A. D. (1964). Reactions to the imminence of death. In G. H. Grosser, H. Wechsler, & M. Greenblatt (Eds.), *The threat of impending disaster*. Cambridge, MA: The MIT Press.
- Hall, J. E., & Hall, M., (2020). *Guyton and hall textbook of medical physiology* (14. Aufl.). Elsevier.
- Harvard Health. (2020, 6. Juli). *Understanding the stress response*.
<https://www.health.harvard.edu/staying-healthy/understanding-the-stress-response>
- Hayes, A. F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. The Guilford Press.
- Hinkle, L. E. (1974). The Concept of “Stress” in the Biological and Social Sciences. *The International Journal of Psychiatry in Medicine*, 5(4), 335–357.
<https://doi.org/10.2190/91DK-NKAD-1XP0-Y4RG>
- Hoyer, J., & Knappe, S. (Hrsg.). (2020). *Klinische Psychologie & Psychotherapie*. Springer Berlin Heidelberg. <https://doi.org/10.1007/978-3-662-61814-1>
- Kasl, S. V., & Cobb, S. (1970). Blood pressure changes in men undergoing job loss: A preliminary report. *Psychosomatic Medicine*, 32, 19-38.
- Kerr, J. I., Naegelin, M., Weibel, R. P., Ferrario, A., La Marca, R., von Wangenheim, F., Hoelscher, C., & Schinazi, V. R. (2020). The effects of acute work stress and appraisal on psychobiological stress responses in a group office environment. *Psychoneuroendocrinology*, 121, 104837.
<https://doi.org/10.1016/j.psyneuen.2020.104837>
- Kim, H.-G., Cheon, E.-J., Bai, D.-S., Lee, Y. H., & Koo, B.-H. (2018). Stress and Heart Rate Variability: A Meta-Analysis and Review of the Literature. *Psychiatry Investigation*, 15(3), 235–245. <https://doi.org/10.30773/pi.2017.08.17>
- Kirschbaum, C., & Hellhammer, D. H. (1999). Hypothalamus-Hypophysen-Nebennierenrindenachse. In C. Kirschbaum & D. H. Hellhammer (Hrsg.), *Enzyklopädie der Psychologie: Psychoendokrinologie und Psychoimmunologie* (S. 79–140). Göttingen: Hogrefe.
- Kirschbaum, C., Kudielka, B. M., Gaab, J., Schommer, N. C., & Hellhammer, D. H. (1999). Impact of Gender, Menstrual Cycle Phase, and Oral Contraceptives on the

Activity of the Hypothalamus-Pituitary-Adrenal Axis: *Psychosomatic Medicine*, 61(2), 154–162. <https://doi.org/10.1097/00006842-199903000-00006>

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

Koriat, A., Melkman, R., Averill, J. R., & Lazarus, R. S. (1972). The self-control of emotional reactions to a stressful film¹. *Journal of Personality*, 40(4), 601–619. <https://doi.org/10.1111/j.1467-6494.1972.tb00083.x>

Krohne, H.W., Rogner, J. (1985). Mehrvariablen-Diagnostik in der Bewältigungsforschung. In H.W. Krohne (Hrsg.), *Angstbewältigung in Leistungssituationen* (S. 45-62). Weinheim: edition psychologie.

Kudielka B. M., Hellhammer D. H., Kirschbaum C. (2007). "Ten years of research with the Trier Social Stress Test (TSST)—Revisited," in Harmon-Jones, E., & Winkielman, P. (Eds.). (2008). *Social neuroscience: Integrating biological and psychological explanations of social behavior*. (pp. 56–83) New York, NY: Guilford Publications.

Kusumoto, F., & Bernath, P. (2012). *ECG interpretation for everyone: An on-the-spot guide*. Wiley-Blackwell.

Laux, L., Glanzmann, P., Schaffner, P., & Spielberger, C. D. (1981). Das state-trait-angst inventar [German state-trait anxiety inventory]. Göttingen, Germany: Hogrefe.

Lazarus, R. S. (1966). *Psychological stress and the coping process*. New York: McGraw-Hill.

Lazarus, R. S. (1984). On the primacy of cognition. *American Psychologist*, 39(2), 124–129. <https://doi.org/10.1037/0003-066X.39.2.124>

Lazarus, R. S., & Alfert, E. (1964). Short-circuiting of threat by experimentally altering cognitive appraisal. *The Journal of Abnormal and Social Psychology*, 69(2), 195–205. <https://doi.org/10.1037/h0044635>

Lazarus, R. S., & Cohen, J. B. (1977). Environmental Stress. In I. Altman & J. F. Wohlwill (Hrsg.), *Human Behavior and Environment* (S. 89–127). Springer US. https://doi.org/10.1007/978-1-4684-0808-9_3

Lazarus, R. S., & Folkman, S. (1984). *Stress, appraisal, and coping*. Springer.

Lazarus, R. S., Speisman, J. C., Mordkoff, A. M., & Davison, L. A. (1962). A laboratory study of psychological stress produced by a motion picture film. *Psychological Monographs: General and Applied*, 76(34), 1–35. <https://doi.org/10.1037/h0093861>

- Lovallo, W. R. (2016). *Stress & health: Biological and psychological interactions* (Third edition). SAGE.
- Lumsden, D. P. (1981). Is the concept of "stress" of any use, anymore? In D. Randall (Ed.), *Contributions to primary prevention in mental health: Working papers*. Toronto: Toronto National Office of the Canadian Mental Health Association
- Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, 10(6), 434–445. <https://doi.org/10.1038/nrn2639>
- Malik, M., & Camm, A. J. (Hrsg.). (1995). *Heart rate variability*. Futura Pub. Co.
- Maslach, C., Schaufeli, W. B., & Leiter, M. P. (2001). Job Burnout. *Annual Review of Psychology*, 52(1), 397–422. <https://doi.org/10.1146/annurev.psych.52.1.397>
- McEwen, B. S. (1998). Protective and Damaging Effects of Stress Mediators. *New England Journal of Medicine*, 338(3), 171–179. <https://doi.org/10.1056/NEJM199801153380307>
- McEwen, B. S., Stellar, E., (1993). Stress and the Individual: Mechanisms Leading to Disease. *Archives of Internal Medicine*, 153(18), 2093. <https://doi.org/10.1001/archinte.1993.00410180039004>
- Morales, A. S. (Hrsg.). (2012). *Trait anxiety*. Nova Science Publishers, Inc.
- Munck, A., Guyre, P. M., & Holbrook, N. J. (1984). Physiological Functions of Glucocorticoids in Stress and Their Relation to Pharmacological Actions*. *Endocrine Reviews*, 5(1), 25–44. <https://doi.org/10.1210/edrv-5-1-25>
- Natelson, B. H., Krasnegor, N., & Holaday, J. W. (1976). Relations between behavioral arousal and plasma cortisol levels in monkeys performing repeated free-operant avoidance sessions. *Journal of Comparative and Physiological Psychology*, 90(10), 958–969. <https://doi.org/10.1037/h0077280>
- Nomikos, M. S., Opton, E., & Averill, J. R. (1968). Surprise versus suspense in the production of stress reaction. *Journal of Personality and Social Psychology*, 8(2, Pt.1), 204–208. <https://doi.org/10.1037/h0025274>
- Pittig, A., Arch, J. J., Lam, C. W. R., & Craske, M. G. (2013). Heart rate and heart rate variability in panic, social anxiety, obsessive–compulsive, and generalized anxiety disorders at baseline and in response to relaxation and hyperventilation. *International Journal of Psychophysiology*, 87(1), 19–27. <https://doi.org/10.1016/j.ijpsycho.2012.10.012>

- Rapee, R. M., & Heimberg, R. G. (1997). A cognitive-behavioral model of anxiety in social phobia. *Behaviour Research and Therapy*, 35(8), 741–756.
[https://doi.org/10.1016/S0005-7967\(97\)00022-3](https://doi.org/10.1016/S0005-7967(97)00022-3)
- Reiche, E. M. V., Nunes, S. O. V., & Morimoto, H. K. (2004). Stress, depression, the immune system, and cancer. *The Lancet Oncology*, 5(10), 617–625.
[https://doi.org/10.1016/S1470-2045\(04\)01597-9](https://doi.org/10.1016/S1470-2045(04)01597-9)
- Rimmele, U., Zellweger, B. C., Marti, B., Seiler, R., Mohiyeddini, C., Ehlert, U., & Heinrichs, M. (2007). Trained men show lower cortisol, heart rate and psychological responses to psychosocial stress compared with untrained men. *Psychoneuroendocrinology*, 32(6), 627–635.
<https://doi.org/10.1016/j.psyneuen.2007.04.005>
- Ruiz-Padial, E., & Thayer, J. F. (2014). Resting heart rate variability and the startle reflex to briefly presented affective pictures. *International Journal of Psychophysiology*, 94(3), 329–335. <https://doi.org/10.1016/j.ijpsycho.2014.10.005>
- Sarason, I. G., & Sarason, B. R. (1990). Test Anxiety. In H. Leitenberg (Hrsg.), *Handbook of Social and Evaluation Anxiety* (S. 475–495). Springer US.
https://doi.org/10.1007/978-1-4899-2504-6_16
- Selye, H. (1946). The general adaptation syndrome and the diseases of adaptation. *The Journal of Clinical Endocrinology & Metabolism*, 6(2), 117–230.
<https://doi.org/10.1210/jcem-6-2-117>
- Shaffer, F., & Ginsberg, J. P. (2017). An Overview of Heart Rate Variability Metrics and Norms. *Frontiers in Public Health*, 5, 258. <https://doi.org/10.3389/fpubh.2017.00258>
- Shaffer, F., McCraty, R., & Zerr, C. L. (2014). A healthy heart is not a metronome: An integrative review of the heart's anatomy and heart rate variability. *Frontiers in Psychology*, 5. <https://doi.org/10.3389/fpsyg.2014.01040>
- Sloan, R. P., Shapiro, P. A., Bagiella, E., Boni, S. M., Paik, M., Bigger, J. T., Steinman, R. C., & Gorman, J. M. (1994). Effect of mental stress throughout the day on cardiac autonomic control. *Biological Psychology*, 37(2), 89–99. [https://doi.org/10.1016/0301-0511\(94\)90024-8](https://doi.org/10.1016/0301-0511(94)90024-8)
- Smith, W. D. (1994). *Hippocrates. Volume VII*. Harvard University.
- Speisman, J. C., Lazarus, R. S., Davison, L., & Mordkoff, A. M. (1964). Experimental analysis of a film used as a threatening stimulus. *Journal of Consulting Psychology*, 28(1), 23–33. <https://doi.org/10.1037/h0047028>
- Spielberger, C. D. (Ed.). (1966). *Anxiety and behavior*. New York: Academic Press.

- Spielberger, C. D., Oorsuch, R. L., & Lushene, R. E. (1970). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
- Step toe, A., & Kivimäki, M. (2012). Stress and cardiovascular disease. *Nature Reviews Cardiology*, 9(6), 360–370. <https://doi.org/10.1038/nrcardio.2012.45>
- Task Force of the European Society of Cardiology the North American Society of Pacing and Electrophysiology (1996). Heart Rate Variability: Standards of Measurement, Physiological Interpretation, and Clinical Use. *Circulation*, 93(5), 1043–1065. <https://doi.org/10.1161/01.CIR.93.5.1043>
- Taylor, J. A. (1953). A personality scale of manifest anxiety. *The Journal of Abnormal and Social Psychology*, 48(2), 285–290. <https://doi.org/10.1037/h0056264>
- Thayer, J. F., Friedman, B. H., & Borkovec, T. D. (1996). Autonomic characteristics of generalized anxiety disorder and worry. *Biological Psychiatry*, 39(4), 255–266. [https://doi.org/10.1016/0006-3223\(95\)00136-0](https://doi.org/10.1016/0006-3223(95)00136-0)
- Thayer, J. F., & Sternberg, E. (2006). Beyond Heart Rate Variability: Vagal Regulation of Allostatic Systems. *Annals of the New York Academy of Sciences*, 1088(1), 361–372. <https://doi.org/10.1196/annals.1366.014>
- Vale W, Spiess J, Rivier C, Rivier J (September 1981). Characterization of a 41-residue ovine hypothalamic peptide that stimulates secretion of corticotropin and beta-endorphin. *Science (journal)* **213** (4514): 1394–7.
- Watkins, L. L., Grossman, P., Krishnan, R., & Sherwood, A. (1998). Anxiety and Vagal Control of Heart Rate: *Psychosomatic Medicine*, 60(4), 498–502. <https://doi.org/10.1097/00006842-199807000-00018>
- World Health Organization. (2022). *World mental health report: Transforming mental health for all*. World Health Organization. <https://apps.who.int/iris/handle/10665/356119>
- Xi, Y. (2020). Anxiety: A concept analysis. *Frontiers of Nursing*, 7(1), 9–12. <https://doi.org/10.2478/fon-2020-0008>
- Yang, L., Zhao, Y., Wang, Y., Liu, L., Zhang, X., Li, B., & Cui, R. (2015). The Effects of Psychological Stress on Depression. *Current Neuropharmacology*, 13(4), 494–504. <https://doi.org/10.2174/1570159X1304150831150507>

Appendix

Abstract

Trait anxiety is a personality disposition that describes a person's tendency to perceive a situation as threatening. According to Lazarus' transactional model of stress and coping, the emergence of stress is determined by a person's appraisal of the situation and their available coping resources. Individuals with a higher likelihood of appraising situations as threatening might be at a higher risk of developing diseases related to chronic stress due to the increased odds of appraising a situation as threatening. This paper investigates the relationship between trait anxiety and the change in variability of the heart rate as a physiological indicator of stress after an acute stressor, as well as its possible mediation by retrospective appraisal. To control for hormonal influences on the physiological stress response, only female participants (N=33) were recruited. The study failed to replicate earlier findings indicating an effect of trait anxiety on stress-induced changes in heart rate variability. No differences in baseline HRV could be found that would explain the failed replication. Further analysis of subjective stress as an alternative stress parameter indicated a trend towards the influence of trait anxiety on perceived subjective stress at baseline but not right before or after the TSST. This indicates that individuals high in trait anxiety seem to experience more subjective stress in objectively non-stress-inducing situations, which coincides with the definition of trait anxiety, but not in situations that are objectively stressful. As a result, highly trait anxious people appear to experience stress more frequently, but not to a greater extent.

Trait Angst beschreibt die Tendenz einer Person Situationen als bedrohlich wahrzunehmen. Nach dem Transaktionalen Stressmodell von Lazarus entsteht Stress, wenn eine Person eine auftretende Situation als bedrohlich, und ihre verfügbaren Bewältigungsressourcen als unzureichend einschätzt. Es ist daher anzunehmen, dass Personen, die von Natur aus häufiger Angst verspüren und Situationen als bedrohlich einschätzen häufiger Stress verspüren und dadurch anfälliger für die gesundheitlichen Folgen von chronischem Stress sind. Diese Studie untersucht den Zusammenhang von Trait Angst und der Veränderung der Variabilität der Herzrate, als physiologischer Indikator einer Stressreaktion, nach einem akuten Stressor. Zudem wird eine mögliche Mediation des Effekts durch retrospektive Bewertung untersucht. Aufgrund hormoneller

Einflüsse auf die physiologische Stressreaktion wurden 33 weibliche Teilnehmerinnen rekrutiert. Als akuter Stressor, wurde der Trier Social Stress Tests (TSST) angewandt. Bisherige Ergebnisse die mögliche Effekte von Trait Angst auf die Variabilität der Herzrate indizierten konnten nicht repliziert werden. Es konnten keine Unterschiede bei der Baseline Messung festgestellt werden, die erklären, warum keine Effekte gefunden wurden. Eine explorative Analyse von subjektivem Stress als alternativer Stressparameter zeigt einen Trend bezüglich eines Effekts von Trait Angst auf subjektiven Stress bei der Baseline Messung. Dieser Trend konnte nicht für spätere Messzeitpunkte gezeigt werden. Das deutet darauf hin, dass Personen die hohe Trait Angst zeigen mehr subjektiven Stress in objektiv nicht stressigen Situationen zeigen, jedoch nicht in allgemein stressauslösenden Situationen. Dies stimmt mit der grundlegenden Definition von Trait Angst überein. Personen mit hohen Werten im Bereich Trait Angst scheinen daher häufiger Stress zu empfinden, jedoch nicht in stärkerem Ausmaß.

Trait Anxiety Scale of the STAI (Laux et al., 1981)

Anleitung: Im folgenden Fragebogen finden Sie eine Reihe von Feststellungen, mit denen man sich selbst beschreiben kann. Bitte lesen Sie jede Feststellung durch und wählen Sie aus den vier Antworten diejenige aus, die angibt, wie Sie sich **im Allgemeinen** fühlen. Kreuzen Sie bitte bei jeder Feststellung die von Ihnen gewählte Antwort an.

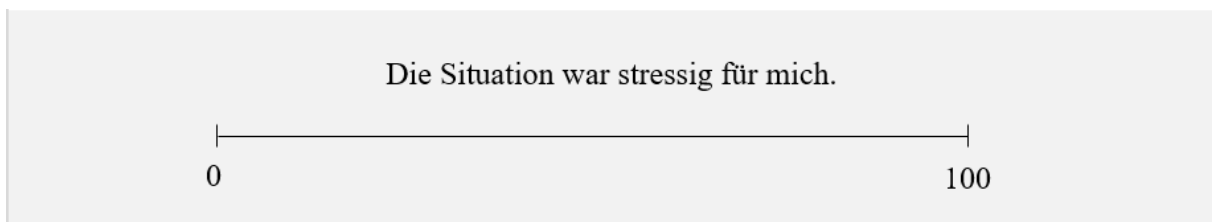
Es gibt keine richtigen oder falschen Antworten. Überlegen Sie bitte nicht lange und denken Sie daran, diejenige Antwort auszuwählen, die am besten beschreibt, wie Sie sich im **Allgemeinen** fühlen.

	FAST NIE	MANCHMAL	OFT	FAST IMMER
001. Ich bin vergnügt	1	2	3	4
002. Ich werde schnell müde	1	2	3	4
003. Mir ist zum Weinen zumute	1	2	3	4
004. Ich glaube, mir geht es schlechter als anderen Leuten	1	2	3	4
005. Ich verpasse günstige Gelegenheiten, weil ich mich nicht schnell genug entscheiden kann	1	2	3	4
006. Ich fühle mich ausgeruht	1	2	3	4
007. Ich bin ruhig und gelassen	1	2	3	4
008. Ich glaube, dass mir meine Schwierigkeiten über den Kopf wachsen	1	2	3	4
009. Ich mache mir zu viel Gedanken über unwichtige Dinge	1	2	3	4
010. Ich bin glücklich	1	2	3	4
011. Ich neige dazu, alles schwer zu nehmen	1	2	3	4
012. Mir fehlt es an Selbstvertrauen	1	2	3	4
013. Ich fühle mich geborgen	1	2	3	4
014. Ich mache mir Sorgen über ein mögliches Missgeschick	1	2	3	4
015. Ich fühle mich niedergeschlagen	1	2	3	4
016. Ich bin zufrieden	1	2	3	4
017. Unwichtige Gedanken gehen mir durch den Kopf und bedrücken mich	1	2	3	4
018. Enttäuschungen nehme ich so schwer, dass ich sie nicht vergessen kann	1	2	3	4
019. Ich bin ausgeglichen	1	2	3	4
020. Ich werde nervös und unruhig, wenn ich an meine derzeitigen Angelegenheiten denke	1	2	3	4

TSST-Visual Analogue Scale Retrospective Stress Appraisal

(Kirschbaum & Hellhammer, 1993)

Bitte zeichnen Sie bei den folgenden Fragen an der Stelle auf der Linie einen senkrechten Strich ein, die Ihrer persönlichen Einschätzung am meisten entspricht. Dabei geht es darum, wie Sie die vorangegangene Situation jeweils erlebt haben. Die Wertung 0 bedeutet, dass die Aussage überhaupt nicht für Sie zutrifft und die Wertung 100, dass die Aussage voll und ganz zutrifft. Sie können natürlich auch zwischen 0 und 100 einen Strich einzeichnen, je nachdem, wie Sie die Situation empfunden haben.



Die Situation war stressig für mich.

0 100

A horizontal line with vertical tick marks at each end, representing a scale from 0 to 100. The text 'Die Situation war stressig für mich.' is centered above the line.

Visual Analogue Scale 1, 2 and 3 (at baseline, before and after TSST)

(Kirschbaum & Hellhammer, 1993)

Bitte zeichnen Sie bei der folgenden Frage an der Stelle auf der Linie einen senkrechten Strich ein, die Ihrer persönlichen Einschätzung am meisten entspricht.

