FACULTY OF PSYCHOLOGY AND EDUCATIONAL SCIENCES

THE EFFECTS OF SPONTANEOUS EMOTION REGULATION ON STRESSRESPONSE

Spontaneous emotion regulation during stress-induction tasks: associations with *subjective*, *parasympathetic*, and *sympathetic* stress recovery measures

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FOREWORD

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ABSTRACT

Objective:

The vagus nerve, a key part of the parasympathetic nervous system, is crucial for emotion regulation (ER) and stress management. Vanderhasselt and Ottaviani (2022) highlight the potential for enhanced resilience through vagal stimulation. This study examines how spontaneous ER-strategies can serve as practical interventions for stress recovery, focusing on their impact on stress response and recovery following a stress-induction task.

Methods:

This research investigates the effects of spontaneous ER on stress recovery using an adjusted version of the Trier Social Stress Test (TSST). We hypothesize that participants who spend more time on adaptive ER-strategies will show significant large improvements in stress recovery, as indicated by a large increase in HRV scores, and a large decrease in VAS-Tensed and PEP scores, between the stress and recovery phase (**Hypothesis 1a**). Conversely, those utilizing maladaptive ER-strategies are expected to show minimal improvements in stress recovery (**Hypothesis 1b**). Furthermore, we explore which specific adaptive ER-strategies are effective in the TSST context, motivated by previous research questioning the universal effectiveness of these strategies (**Hypothesis 1c**).

Results:

Our finding did not support Hypotheses 1a and 1b, yielding non-significant results. This may be attributed to small sample sizes and limited statistical power. Our findings partially supported Hypothesis 1c, revealing that positive reappraisal significantly improved subjective stress recovery only (VAS-Tensed scores), while other adaptive ER-strategies did not significantly affect HRV, PEP, or VAS-Tensed.

Conclusion:

While positive reappraisal demonstrated effectiveness in stress recovery (VAS-Tensed), other adaptive ER-strategies did not yield significant results. Future research should include larger samples and consider non-linear models to better capture complex relationships between ER-strategies and stress recovery. Additionally, exploring sex differences and improving measurement accuracy could further enhance the understanding and application of ER-strategies in stress management.

Keywords: *Emotion Regulation, Stress Recovery, Trier Social Stress Test, Vagus Nerve, Autonomic Nervous System*

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INTRODUCTION

The past few years researchers have shown us a great interest in topics about stress and emotion regulation. This is not surprising as everyone experiences stressful moments. Dealing with stress and emotions seem to have an important role in mental health. Many studies have linked the vagus nerve (which is thought to be the most important nerve of the parasympathetic nervous system) with the ability to regulate stress and emotions. Vanderhasselt & Ottaviani (2022) stated that there is a two-way interaction between the heart and the brain, which makes it possible to increase resilience by stimulating the vagus nerve. The vagus nerve is recognized as a key player in emotion regulation (ER), with empirical findings suggesting a positive correlation between heightened vagal activity and improved ER (Denson et al., 2011; Porges et al., 1994; Thayer & Lane, 2000; Vögele et al., 2010). Research has demonstrated the significance of regulating emotions during the recovery phase following stress (Jordan et al., 2017; Martin & Dahlen, 2005). This, for example, includes the mitigation of consequences arising from prolonged stress. (Chaby et al., 2015; Gomez-Bernal et al., 2019). Usually, a distinction is made between applying ER-strategies independently (i.e., spontaneous ER) and being instructed to apply specific ER-strategies (i.e., instructed ER). Both spontaneous and instructed ER are equally important to investigate in order to gain insight into which strategies are effective and which strategies are less effective in promoting stress recovery.

Therefore, this research focusses on **the effects of spontaneous emotion regulation on stress recovery.** Given the significant impact of stress on various groups, including workers, students, and parents, it is crucial to identify and test ER-strategies that can help reduce stress levels and promote recovery. Additionally, exploring how the time spent on specific strategies affects stress recovery would provide insights beyond individual preferences for certain strategies. This approach would consider individual differences in the allocation of time to different strategies, offering a more comprehensive understanding of their effectiveness in a stress context. This research could serve as a foundation for additional studies, where interventions aimed at teaching/stimulating adaptive ER-skills (i.e. instructed ER) could be implemented. These interventions could potentially contribute to facilitating stress recovery, as ER might have a stimulating effect on the vagus nerve. In therapy, ER appears to be a transdiagnostic factor that can be applied across various issues (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Harvey et al., 2004; Kring & Caponigro, 2010; Svaldi et al., 2012; Weiss et al., 2015). This implies that teaching specific ER-skills can be effective in promoting health (Barlow et al., 2016; Ehrenreich-May & Bilek, 2012; Roemer et al., 2008). Consequently,

this study provides opportunities to support a diverse range of individuals (ranging from healthcare providers to patients and parents).

Feasible techniques, namely adaptive ER-strategies, will be explored to stimulate the vagus nerve with the purpose of increasing resilience (i.e. promoting stress recovery). Moreover, exploring the link between spontaneous ER and physiological measures, such as heart rate variability (HRV) or the Pre-ejection Period (PEP), can offer insights into how ER influences our physiological state, specifically our autonomic nervous system, and consequently our (psychological) resilience. If a connection is established, this study could provide valuable, non-invasive biofeedback tools to assess the effects of ER on stress recovery. That is why this study will emphasize the importance of feasible interventions and tools, which may also be used outside a clinical context.

We will start discussing key concepts relevant to the research question, using scientific insights and theories (e.g., neurovisceral integration model; Thayer & Lane, 2000; Thayer et al., 2009), to provide a foundation for understanding the complex processes involved in ER and stress recovery. Next, the effects of ER and different measurements of stress recovery will be described, and underlying processes will be explored. Subsequently, we will outline the specific research question and hypotheses, and the significance of the research. The formulation of hypotheses will be based on the expected impact of spontaneous ER on stress recovery, considering the literature and theoretical frameworks discussed.

Stress and (spontaneous) emotion regulation

Stress and the nervous system

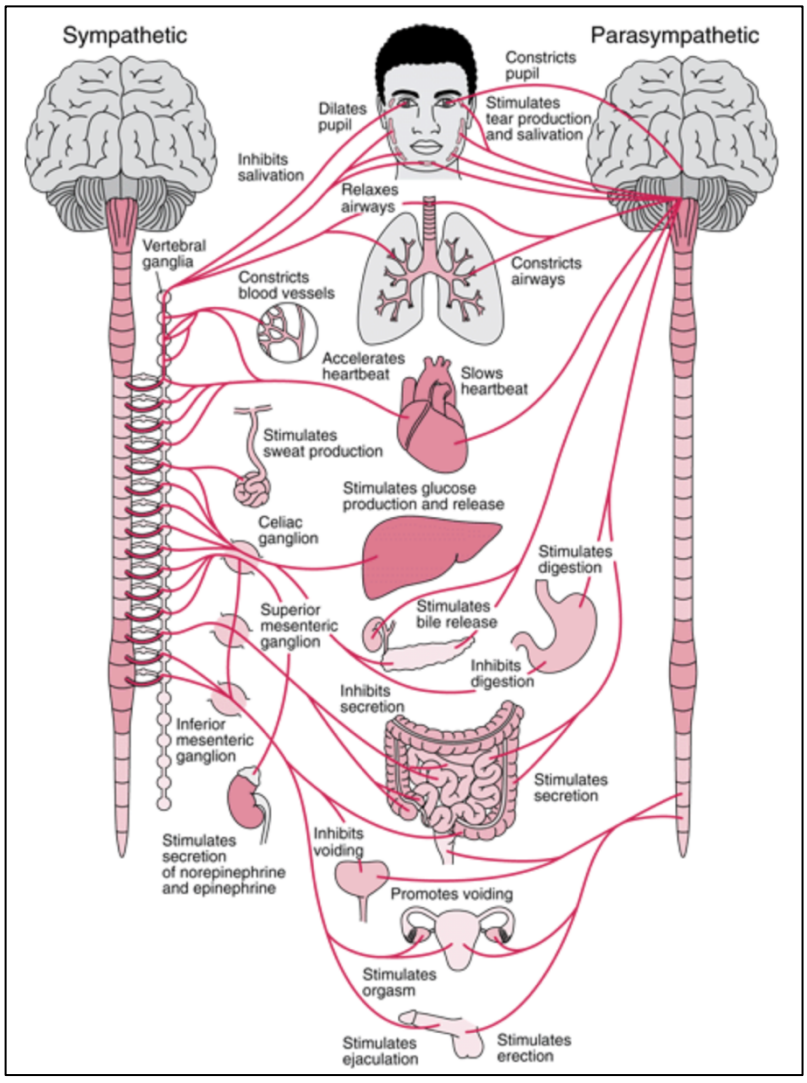
Organisms live in constantly changing environments, where dealing with stress becomes an 'almost normal' part of life. However, this does not mean that it should simply be accepted as 'part of life', as stress remains something best avoided as much as possible (Greenberg et al., 2002). Throughout our life we face numerous challenges. These range from serious events (e.g., a traumatic experience) to milder events (e.g., exams), and can each be seen as stressors. In addition, it is important to point out that not all changes and not all stressors lead to harmful effects. In cases where stressors become chronic or exceed our ability to deal with them, it may lead to an imbalance in our body (McEwen & Wingfield, 2003), and to the development of psychopathologies and/or health problems (Chaby et al., 2015; Gomez-Bernal et al., 2019). A stress response is the body's reaction to a particular stressor and has its roots in the evolutionary (attempted) survival of the organism (Greenberg et al., 2002). Vanderhasselt and Ottaviani (2022) describe it as an attempt to promote adaptation and energy mobilization (e.g., increased heart rate and blood pressure), through a physiological response by the body to a situation where personal or environmental demands appear to tax or exceed the person's adaptive capacity. To better understand this reaction and the effects of stress on our body, the complex functioning of our body will be briefly explained.

The autonomic nervous system (ANS) consists of the parasympathetic nervous system (PNS) and the sympathetic nervous system (SNS) and is responsible for the maintenance of the homeostasis in our body (McCorry, 2007). According to McCorry (2007), both systems (PNS and SNS) are tonically active (i.e., to be in a state of continuous unremitting action), however, under certain conditions one system (PNS or SNS) will dominate. **Figure 1** shows the effects of PNS- and SNS-activation (Low, 2023). The PNS is activated when a person is in a state of rest, which is why it is also referred to as the 'rest-and-digest system'. The SNS, on the other hand, is activated when a person exerts effort or experiences stress, which is why it is also referred to as the 'fight-or-flight system'. We may speak of body-mind interactions. So, when confronted with a stressor, SNS-activity will increase. **Figure 1** shows the effects of SNS-activity, and thus our body's response to a stressor (Low, 2023). This increase in SNS-activity is adaptive (i.e., to respond appropriately to optimize performance, survival, or well-being) (Epel et al., 2018), however prolonged SNS-activity may increase the likelihood of developing health problems (e.g., see association between chronic stress and ANS-dysregulation; Ottaviani et al, 2016; cardiovascular diseases; Graham et al., 2004; Grassi, 1998; Leimbach et al., 1986), or even lead

to exhaustion and death (Selye, 1946). Hence, the reason to focus on the relevance of coping with stress, and thus interventions aimed at stress reduction.

FIGURE 1

Areas associated with SNS- and PNS-activation



Note. Taken from "*Overview of the Autonomic Nervous System*", by P. Low, 2023 (https://www.msdmanuals.com/professional/neurologic-disorders/autonomic-nervous-system/overview-of-the-autonomic-nervous-system). Copyright © 2023 Merck & Co., Inc., Rahway, NJ, USA and its affiliates. All rights reserved.

The effects of stress on the body

It is important to understand the mechanisms that become active when experiencing stress, to get a more accurate idea of the body-mind connection. How our body reacts to stress depends on different characteristics of stress stimulus (e.g., the type, severity, and timing;

Yaribeygi et al., 2017), person and environment characteristics (e.g., diathesis–stress model for depression; Colodro-Conde et al., 2018; Gazelle & Ladd, 2003), and more relevant to this research, the ER-skills (e.g., neurovisceral integration model which will be discussed later; Thayer & Lane, 2000). A stressor may trigger the mind-body interactions, in which the vagus nerve is a central component (Dedoncker et al., 2021). Evidence has demonstrated a link between stress and the human nervous system (Blase et al., 2021; La Rovere et al., 2022; Lupien et al., 2009; Morris, 1956; Ziegler, 2012), as well as the effect of stress on psychological (Siddique & D'Arcy, 1984), and physical (Larzelere & Jones, 2008) levels of human functioning.

As discussed above, stressors can trigger the body-mind interaction (with the ANS at its core) (Dedoncker et al., 2021), with an increase in the SNS and a decrease in the PNS likely to be observed. The experience of stress triggers the activation of the primary stress response system 'hypothalamic-pituitary-adrenal axis', which serves as the neuroendocrine link between perceived stress and our body's response to stress (Breedlove and Watson, 2013). This in turn releases hormones (e.g., glucocorticoids, catecholamines, growth hormone and prolactin; Ranabir & Reetu, 2011), and the so-called 'stress-hormone' cortisol (King & Hegadoren, 2002; Ranabir & Reetu, 2011). These hormones will activate some systems and inhibit others (as seen in Figure 1; Low, 2023) to ensure survival and eventually return to homeostasis (i.e., a stable internal environment) in our body.

When a stressor disappears, a person needs to recover, this is what is called 'resilience' or 'stress recovery' (the terms are used interchangeably throughout the paper) (Epel et al., 2018). Herrman et al. (2011) describe resilience as 'referring to positive adaptation, or the ability to maintain or regain mental health, despite experiencing adversity'. Recovering from a stressor implies a decrease in SNS-activity and an increase in PNS-activity. Interventions targeting the vagus nerve, therefore the PNS via release of acetylcholine, can promote recovery from stress-related damage (Jerath et al., 2006; Laborde et al., 2017; Pal et al., 2004).

Due to the body-mind connection, the vagus nerve can be targeted through various interventions, ranging from bottom-up methods (e.g., slow-paced breathing; Borges et al., 2021; Jerath et al., 2006; Laborde et al., 2017; Ma et al., 2017; Sevoz-Couche & Laborde, 2022; Tatschl et al., 2020; Zaccaro et al., 2018; Zou et al., 2018) to top-down approaches (e.g., non-invasive brain stimulation such as transcranial magnetic stimulation and transcranial direct current stimulation; Smits et al., 2020). Additionally, literature has linked the vagus nerve with ER through its connection to the ANS (e.g., neurovisceral integration in ER and dysregulation; Thayer & Lane, 2000). Therefore, exploring and implementing this avenue in research is promising due to its favorable characteristics.

Stress and the role of (spontaneous) emotion regulation

Emotion regulation (ER) plays an important role during the experience of a stressor (Jamieson et al., 2013a; Jamieson et al., 2013b; Lazarus & Folkman, 1984; Thayer & Lane, 2000). (Cognitive) ER refers to (mental) processes and strategies (e.g., monitoring, evaluating, modifying) that individuals use to regulate their emotional reactions, in order to accomplish certain goals (Thompson, 1994). Based on a large body of research on ER and psychopathologies, two main categories are predominantly identified: adaptive and maladaptive ER-strategies (Aldao et al., 2010; Gross, 1998; Nolen-Hoeksema & Watkins, 2011). Adaptive strategies in ER refer to techniques that individuals use to effectively manage and cope with their emotions in a positive and constructive manner. These strategies typically lead to improved emotional well-being, better stress management, and enhanced overall functioning (Aldao et al., 2010; Billings & Moos, 1981; Goldin et al., 2008; Campbell-Sills et al., 2006; Hayes et al., 1999; Jamieson et al., 2013a; Lazarus & Folkman, 1984; Richards & Gross, 2000). Examples include cognitive reappraisal (Jamieson et al., 2013a; Lazarus & Folkman, 1984*)*, acceptance *(*Harris, 2006*)*, and problem-solving. In contrast, maladaptive ER-strategies refer to strategies that individuals employ to manage their emotions in ways that are ineffective to one's well-being. These strategies often fail to alleviate distress and may even exacerbate negative emotions or lead to additional problems (Aldao et al., 2010; Beck, 1975; Campbell-Sills et al., 2006; Gross, 1998; Hofmann et al., 2005; Richards et al., 2003; Salkovskis, 1998; Wegner et al., 1997). Examples include suppression of emotions/thoughts (Carver et al., 1989; Gross, 1998; Hayes et al., 2004; Wenzlaff & Wegner, 2000), avoidance, worrying, rumination (Nolen-Hoeksema, 1991; Watkins, 2008*),* self-blame (e.g., MA; Jannati et al., 2020), other-blame (Tennen & Affleck, 1990), and catastrophizing (Garnefski & Kraaij, 2006). However, Aldao and Nolen-Hoeksema (2012) note that maladaptive ER-strategies show a stronger association with psychopathologies compared to adaptive ER-strategies (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010). The effectiveness of adaptive ER-strategies may depend on contextual demands, meaning they might only yield positive outcomes when used appropriately within a specific context (Aldao & Nolen-Hoeksema, 2010; Aldao & Nolen-Hoeksema, 2011; Aldao et al., 2010). For example, problem-solving and seeking social support may be effective in a high-stress work environment, while cognitive reappraisal and empathy might be more appropriate in a personal relationship conflict. This raises a critical issue: if the efficacy of adaptive ER-strategies is so context-dependent, their application in certain settings becomes more complex. Emphasizing context-specific effectiveness highlights the need for flexibility in ER, which can be challenging for some individuals. This complexity may undermine the practicality of promoting adaptive

ER-strategies as universally beneficial, indicating the need for more nuanced approaches in understanding and teaching ER. Lastly, it is important to distinguish between instructed and spontaneous ER. Strategies can be applied independently (spontaneous ER) or be taught and guided (instructed ER).

The link between the vagus nerve and ER may be understood by using the model of 'neurovisceral integration in emotion regulation and dysregulation' (Thayer & Lane, 2000). This model includes the integration of autonomic, attentional, and affective systems which can help us understand the (dys)regulation of emotions (Thayer & Lane, 2000). Furthermore, it highlights the importance of the ANS in ER (Thayer & lane, 2000; Porges et al., 1994). This model is based on the central autonomic network (CAN) which includes brain areas associated with the ANS, that seem to be involved with emotional, cognitive, and cardiac processes regulation (Benarroch, 1993). The CAN is thought to play an important role in stress response as the associated brain areas affect sympathetic or parasympathetic output via the preganglionic autonomic neurons (Lamotte et al., 2021). Thayer and Lane (2000) claim that emotions are 'self-regulating responses' and represent the adjustments people make to a constantly changing environment. The neurovisceral integration model suggests that vagally-mediated Heart Rate Variability (vmHRV), which will be discussed next, can be used as an 'indicator of heart-brain interaction' (Thayer and Lane, 2009) and reflects the effectiveness of emotional, cognitive, and cardiac regulation (Mather & Thayer, 2018; Sevoz-Couche & Laborde, 2022).

As previously discussed, there is a well-established connection between pulmonary activity and the vagus nerve (Zagon, 2001), facilitating bi-directional communication between organs (e.g., stomach, pancreas, liver, bowels, heart, and lungs) and the brain, influencing ER (Porges et al., 1994; Thayer & Lane, 2000) and stress recovery (Pal et al., 2004; Sevoz-Couche & Laborde, 2022). This link between stress experiences and psychophysiological activity underscores the importance of interventions focused on ER. Consequently, this study aims to induce short-term stress (i.e., eliciting acute stress responses) and explore interventions like spontaneous ER to observe their impact on stress response.

The Vagus Nerve and Heart Rate Variability

What is the Vagus Nerve and Heart Rate Variability, and how are they connected?

What exactly is the significance of the vagus nerve in our body, and why is this nerve so important to investigate? The vagus nerve is thought to be the most important nerve of the PNS (i.e., nervous system that is dominant during rest), and plays an important role in the regulation of emotions (Porges et al., 1994; Thayer & Lane, 2000) and stress recovery (Jerath et al., 2006; Laborde et al., 2017; Pal et al., 2004). Moreover, it is the longest nerve of the ANS and consists of approximately 80% afferent (sensory) fibers, carrying information from the body to the brain (Dedoncker et al., 2021), and 20% efferent (motor) fibers, sending signals from brain to body (Howland, 2014). Through the vagal afferent/sensory fibers, information from the stomach, pancreas, liver, bowels, heart and lungs is send to the brain (Zagon, 2001), as can be seen in **figure 1**. Stimulating the vagus nerve seems to be an important intervention when it comes to stimulating resilience and ER.

To measure the vagal-mediated influence on the ANS, specifically the PNS, Heart Rate Variability (HRV) is a commonly used measurement. Many researchers have proposed to use HRV as an indicator that reflects the influence of the ANS on the heart (e.g., neurovisceral integration model; Thayer & Lane, 2000; Vanderhasselt & Ottaviani, 2022; Dedoncker et al., 2021; Laborde et al., 2017; Laborde et al., 2022). HRV represents the physiological phenomenon by which the heart rate changes from beat to beat, producing oscillations (i.e., moving back and forth in a regular rhythm) in time intervals between consecutive heartbeats, also known as R-R intervals (Thayer & Lane, 2009). As shown in **figure 2** (Laborde et al., 2017, p. 2), the variation in time in an R-R interval is measured, which means that via HRV cardiac vagal tone can be determined. Cardiac vagal tone reflects the contributions of the PNS to cardiac regulation and ER (Laborde et al., 2017). In a healthy human heart, the PNS can regulate the heart rate to an average of 75 beats per minute (bpm) and can even reduce it to as low as 20-30 bpm (Shaffer & Ginsberg, 2017). The neurovisceral integration model suggests that HRV, more specifically vagal-mediated HRV (vmHRV), reflects the output of the CAN (Sevoz-Couche & Laborde, 2022), hence also the PNS-activity, on the heart (Thayer & Lane, 2000). The variability in the time interval between consecutive heartbeats (i.e., HRV) can be measured and conclusions about the degree of vagal activity can be made (Dedoncker et al., 2021). Acharya et al. (2006) claim that HRV shows “the heart's ability to respond to various physiological and environmental stimuli", and thus stress response.

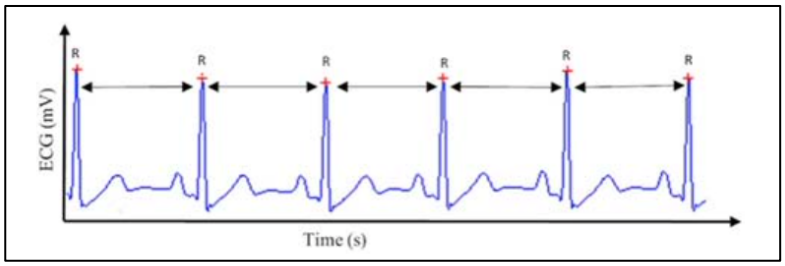
HRV is often used, presumably because of its desirable characteristics: non-invasive, low cost, pain-free and because of its broad applicability across different settings (Laborde et al.,

2017). Furthermore, the two-way communication between heart and brain (Vanderhasselt & Ottaviani, 2022), as indicated at the beginning of the introduction, becomes clearer when HRV is included.

In summary, normal heartbeat variability is primarily influenced by parasympathetic activation through the vagus nerve (via release of acetylcholine). During stressful events, increased sympathetic activation and vagal withdrawal elevate an individual's heart rate. The variability in R-R intervals, whether high or low, can reflect the body's capacity to adapt to stress (will be discussed next).

FIGURE 2

Heart Rate Variability (HRV)



Note. Taken from "Heart Rate Variability and Cardiac Vagal Tone in Psychophysiological Research – Recommendations for Experiment Planning, Data Analysis, and Data Reporting", by S. Laborde, E. Mosley & J.F. Thayer, 2017, *Frontiers in Psychology, 08*, p. 2 (https://doi.org/10.3389/fpsyg.2017.00213).

How to interpret HRV?

Heart rate variability (HRV) can be assessed using various methods, with the most common being time-domain and frequency-domain methods. **Time-domain measurements** involve evaluating the variations in the time intervals between consecutive heartbeats, known as R-R intervals or inter-beat intervals (IBIs) (Malik et al., 1996). During an electrocardiogram (ECG) recording (whether short- or long-term), consecutive QRS complexes are detected, and the normal-to-normal intervals (NN intervals) between these QRS complexes, as well as the heart rate, are determined. As shown in **Table 1a** (Malik et al., 1996, p. 358), numerous statistical variables can be calculated within time-domain analysis. These include the standard deviation of the NN intervals (SDNN), the standard deviation of the average NN intervals (SDANN), the square root of the mean squared differences of successive NN intervals (RMSSD), NN50, and pNN50. These variables can vary between long-term and short-term measurements, respectively

estimating overall HRV, long-term components of HRV, and short-term components of HRV. The widely used RMSSD measurement calculates the square root of the mean of the squares of successive differences between adjacent NN intervals. It reflects vagally mediated changes in HRV, primarily representing short-term HRV and parasympathetic activity. Higher RMSSD values indicate stronger vagal modulation (Shaffer & Ginsberg, 2017).

Frequency domain measurements analyze the distribution of power (variance) across different frequency bands in the heart rate signal. These can be performed using short-term (e.g., two to five minutes) or long-term (24-hour) recordings. This analysis provides insights into the autonomic regulation of the heart, distinguishing between the influences of the sympathetic and parasympathetic nervous systems (Malik et al., 1996). Short-term recordings are easier to perform and avoid 'stationarity problems' (Furlan et al., 1990). During these recordings, HRV is categorized into frequency bands (Malliani et al., 1991), as shown in **Table 1b (**Malik et al., 1996, p. 360). Across different studies, HRV values (in high frequency components; HF-HRV, usually between 0.15-0.40 Hz), are indicative of PNS control (Malliani et al., 1991; Malik et al. 1996; Pomeranz et al., 1985), whereas low frequency components (LF-HRV, usually between 0.04-0.15 Hz) reflects a mixture of PNS and SNS (Bilmann, 2013). Additionally, there is a very low frequency (VLF) band, typically below 0.04 Hz, that can be detected. However, its physiological significance is questionable, particularly for short-term ECG measurements (Malik et al., 1996).

If a person's heart rate is relatively more variable, it may indicate that the body can adapt to different types of changes, and possibly be considered more resilient. On the other hand, when a person's heart rate has a lower variability, it may indicate that the body is less likely to adapt to certain situations. Accordingly, low HRV has been related to atypical activity in brain regions responsible for emotional and cognitive functioning, and to reactions to psychosocial stressors (Thayer et al., 2009), poor self-regulation (Porges, 1992), and emotion dysregulation (Thayer & Lane, 2000). High HRV has been found to be associated with greater resilience to stress (Hirten et al., 2020) and the ability to self-regulate (Porges, 1992, p. 208). Interestingly, individuals with high resting-state HRV recovered faster in their responses to stress (Kaniusas et al., 2019; Weber et al., 2010). Thayer et al. (2012) conducted meta-analyses on studies examining the effect of neuroimaging interventions on HRV, finding that higher resting HRV correlated with activity in brain areas related to ER (e.g., prefrontal and limbic regions).

Table 1a

Time Domain Measures of HRV

Variable	Units	Description
		Statistical measures
SDNN	ms	Standard deviation of all NN intervals.
SDANN	ms	Standard deviation of the averages of NN intervals in all 5 min segments of the entire recording.
RMSSD	ms	The square root of the mean of the sum of the squares of differences between adjacent NN intervals.
SDNN index	ms	Mean of the standard deviations of all NN intervals for all 5 min segments of the entire recording.
SDSD	ms	Standard deviation of differences between adjacent NN intervals.
NN50 count		Number of pairs of adjacent NN intervals differing by more than 50 ms in the entire recording. Three variants are possible counting all such NN intervals pairs or only pairs in which the first or the second interval is longer.
pNN50	%	NN50 count divided by the total number of all NN intervals.

Note. Taken from "Heart rate variability: Standards of measurement, physiological interpretation, and clinical use.", by Malik, M., Bigger, J. T., Camm, A. J., Kleiger, R. E., Malliani, A., Moss, A. J., & Schwartz, P. J., 1996, *European Heart Journal, 17(3),* 354–381, p. 358 (https://doi.org/10.1093/oxfordjournals.eurheartj.a014868). Copyright © 1996 American Heart Association Inc.; European Society of Cardiology. All rights reserved.

Table 1b

Frequency Domain Measures of HRV

Variable	Units	Description	Frequency range
		Analysis of short-term recordings (5 min)	
5 min total power	ms^2	The variance of NN intervals over the temporal segment	approximately $\leq$0·4 Hz
VLF	ms^2	Power in very low frequency range	$\leq$0·04 Hz
LF	ms^2	Power in low frequency range	0·04–0·15 Hz
LF norm	n.u.	LF power in normalised units LF/(Total Power–VLF) × 100	
HF	ms^2	Power in high frequency range	0·15–0·4 Hz
HF norm	n.u.	HF power in normalised units HF/(Total Power–VLF) × 100	
LF/HF		Ratio LF [ms^2]/HF [ms^2]	

Note. Taken from "Heart rate variability: Standards of measurement, physiological interpretation, and clinical use.", by Malik, M., Bigger, J. T., Camm, A. J., Kleiger, R. E., Malliani, A., Moss, A. J., & Schwartz, P. J., 1996, *European Heart Journal, 17(3),* 354–381, p. 360 (https://doi.org/10.1093/oxfordjournals.eurheartj.a014868). Copyright © 1996 American Heart Association Inc.; European Society of Cardiology. All rights reserved.

When using HRV, it is important to remain careful because it may reflect activity of other systems related to the functioning of the heart (e.g., mixed activity of PNS and SNS, or other systems) (Laborde et al., 2017), and different physiological influences (Gordan et al., 2015). Different authors have recommended using the RMSSD as a biomarker, as it more accurately reflects PNS activity (Laborde et al., 2017). Hence we used the RMSSD (time domain) as an index of HRV. Moreover, when interpreting HRV results, we must take into consideration that interpretations of the results can differ by age group (e.g., reduction of vmHRV with age; Laborde et al., 2022), and sex (e.g., higher HRV in woman compared to men; Koenig & Thayer, 2016; Tobaldini et al., 2020). Thus, to enable more accurate conclusions, it is highly recommended to adjust the interpretation of HRV results to the participants.

Other measures of autonomic nervous system activity

In the context of sympathetic activity, alternative physiological measures can be utilized. Research (e.g., Cacioppo et al., 1994; Hartley et al., 2012; Kelsey, 2012; Mackersie & Calderon-Moultrie, 2016; Mehler et al., 2012; Schächinger et al., 2001) has demonstrated that Pre-ejection Period (PEP) and skin conductance (SC; the skin's electrical conductance, which varies with moisture levels; Schmidt & Walach, 2000) are valid indicators of SNS-activity. PEP represents the duration between the initiation of electrical stimulation of the heart (depolarization) and the opening of the aortic valve, which allows blood to be ejected from the heart into the aorta. PEP serves as a measure of SNS-activity because sympathetic activation accelerates the heart's depolarization process, resulting in a shorter PEP duration (Cacioppo et al., 1994). When individuals encounter a stressor, they typically show an increase in PEP and SC, indicating activation of the SNS. This means that higher stress levels are associated with a greater increase in PEP measurements (Cacioppo et al., 1994) and heightened sweat production, leading to elevated SC levels (Mackersie & Calderon-Moultrie, 2016).

Current study

The literature has shown the detrimental effects of stress on physical and psychological levels of functioning, emphasizing the importance of ER in managing stress. The vagus nerve, a key component of the parasympathetic nervous system, appears to have an important function in stimulating stress recovery, and more so, resilience. Therefore, it is essential to investigate what effect stimulating the vagus nerve, via easily implementable ER-strategies, has on stress recovery. This study aims to examine the relationship between spontaneous ER and stress recovery, with the goal of gaining a better understanding of how ER contributes to resilience. Additionally, this research seeks to identify which ER-strategies are effective in stress-inducing contexts. By tailoring interventions to individual needs, the findings may lead to more effective stress-management programs within specific contexts.

The research on spontaneous ER may contribute to a comprehensive understanding of its benefits and challenges, thereby promoting further research and the application of instructed ER as a stress management tool in diverse contexts. Additionally, such research may provide a better understanding of how to stimulate a more adaptive stress response (thereby buffering the negative side effects of stress on health), by using adaptive strategies, considering that stress is often inevitable and beyond our control. This research may even contribute to the further development of accessible and personalized interventions to improve ER and stress management skills in high-risk groups where stress is prominent (e.g., workers, students, parents, patients, minority groups). If a strong link is found, physiological measures such as HRV or PEP could also be used as a biofeedback tools to teach and improve spontaneous ER-strategies.

Research question and hypotheses

The literature indicates that how individuals manage their emotional state, especially during stress-inducing situations, is crucial for their recovery process. Given the established impact of adaptive (Jamieson et al., 2013a; Jamieson et al., 2013b; Lazarus & Folkman, 1984; Thayer & Lane, 2000) and maladaptive (Aldao et al., 2010; Beck, 1975; Campbell-Sills et al., 2006; Gross, 1998; Hofmann et al., 2005; Richards et al., 2003; Salkovskis, 1998; Wegner et al., 1997) ER on stress recovery—measurable through methods like HRV, PEP, and SC—it would be valuable to further investigate how these relationships manifest in a stress context. Exploring how each participant's stress response (i.e., subjective responses, parasympathetic, and sympathetic activity) changes over time or in response to different conditions (i.e., those using more adaptive or more less adaptive strategies) would be highly interesting. Given the extensive evidence from numerous studies on the impact of ER-strategies on stress response (such as Aldao

et al., 2010; Billings & Moos, 1981; and Goldin et al., 2008) it is relevant to investigate whether the time spent on these strategies significantly affects stress recovery metrics within groups using more adaptive or maladaptive strategies. Specifically, this research aims to explore if the amount of time spent on adaptive ER-strategies can explain differences in stress recovery scores among participants who predominantly use adaptive ER-strategies. Similarly, this research examines whether the time spent on less adaptive ER-strategies (i.e., maladaptive ER-strategies) can account for variations in stress recovery scores among those who primarily use maladaptive ER-strategies.

Therefore, this study will investigate the following research question and hypotheses*:* (1) "**What are the effects of spontaneous emotion regulation on stress response during stress-induction tasks?**". We hypothesize that participants who spontaneously employ more time on adaptive ER-strategies (such as relaxation, reappraisal, acceptance) will demonstrate significantly great improvements in stress recovery. Specifically, we expect that HRV scores will show a large increase, and VAS-Tensed and PEP scores will show a large decrease between the stress and recovery phase (**Hypothesis 1a**). Whereas participants who spontaneously utilize more time on less adaptive ER-strategies (such as self-blame, rumination, catastrophizing, blame-others) will exhibit no/low improvements in stress recovery. Specifically, we expect that HRV scores will show a small increase, and VAS-Tensed and PEP scores will show a small decrease between the stress and recovery phases (**Hypothesis 1b**).

Additionally, we do not have a priori hypothesis regarding the specific adaptive ER-strategies' effect on stress recovery within a Trier Social Stress Test (TSST) context. Therefore, we aim to explore which adaptive ER-strategies are effective in a TSST context within the group of participants that spent more time on adaptive ER-strategies (**Hypothesis 1c**). This exploration is motivated by previous research questioning the universal effectiveness of adaptive ER-strategies (Aldao & Nolen-Hoeksema, 2010; Aldao & Nolen-Hoeksema, 2011; Aldao et al., 2010), prompting us to examine which strategies demonstrate effectiveness in this stress-inducing context.

METHOD

Ethics

The study followed the guidelines provided by the Medical Ethical Committee [1]of the Ghent University hospital and was conducted in accordance with accepted standards for scientific and ethical behavior. The researchers adhered to good research practices and followed the principles of research ethics as described in 'Ethics in Social Science and Humanities' (EU, 2018)[2]. Furthermore, all participants were provided written informed consent (**attachment 1**) prior to participation. During the experiment, we used deception by not disclosing the true purpose of our research beforehand. However, a thorough debriefing was conducted after the experiment and participants were informed of the study procedure and purpose, potential risks and benefits, confidentiality, the storage of the data, their rights, and the contact information (**attachment 2**). Prior to this study, an online Data Management Plan (DMP) and a General Data Protection Regulation (GDPR)[3] form were created to outline the management and sharing of data while ensuring the protection of participants' personal information. These documents were regularly updated. Furthermore, participants received a compensation of 10 euros for participating.

This study employed an experimental within-subject design, collecting and analyzing repeated measures for each participant across different phases (i.e., baseline, preparation, stress, feedback, recovery). The main analyses (regarding research question 1) were conducted using the scores differences between the stress and recovery phase as the outcome measures (i.e., stress recovery measures). This approach transformed the data from a repeated measures design to a single measurement design per subject.

Participants and research design

We recruited higher education students. There were several inclusion criteria: 1) *higher education students of age 18 and older*; 2) *sufficient comprehension of the Dutch language* (i.e., the ability to speak and understand); 3) *no known cardiovascular conditions*, 4) *no current psychiatric diagnosis,* 5) *no smoking*, and 6) *not pregnant*. Individuals were excluded if they met the following criteria: 1) *students not following the terms of participation* (see below), 2) *students pursuing education related to medicine* (e.g., medicine, pharmacy, …), and 3) *students having an elevated score on a subtest of the Mini International Neuropsychiatric Interview, version*

[1] https://www.uzgent.be/student-en-onderzoeker/commissie-voor-medische-ethiek
[2] https://ec.europa.eu/research/participants/data/ref/h2020/other/hi/h2020_ethics-soc-science-humanities_en.pdf
[3] See https://dmponline.be

5.0.0 (M.I.N.I.; Sheehan et al., 1998). This study was conducted entirely in Dutch, therefore, proficiency in the Dutch language was required. Due to ethical considerations, participants with elevated scores on the M.I.N.I. (Sheehan et al., 1998) were excluded. Additionally, students who were (ab)using drugs and/or alcohol were excluded because these substances can influence the autonomic nervous system (ANS) and therefore interfere with the results (Boschloo et al., 2011; Szasz et al., 2011). Factors such as being pregnant, smoking, and/or having cardiovascular difficulties were also considered potentially disruptive to psychophysiological measurements, and were thus excluded (Laborde et al., 2017). Moreover, it would have been considered unethical to include them due to the stress-inducing nature of the experiment. Furthermore, students with relevant medical knowledge were excluded from the study because the stress-induction task involved a presentation on blood-related information. Including such participants might have reduced the effectiveness of the task, as they might not have experienced a significant enough increase in stress compared to other students.

Desired sample size was determined based on established parameters such as effect size and power, informed by relevant literature. Certain studies (e.g., Jentsch & Wolf, 2020; Wang et al., 2016) have found medium to large effect sizes (e.g., d= -0.79; η^2_p = .11). Other studies (e.g., Grol & De Raedt, 2021; Knepp et al., 2015) have reported smaller to moderate effect sizes. Our goal was to obtain .80 power (1-ß) to detect a medium effect size d = .50 (Cohen's f^2 = 0.15) at the standard .05 alpha error probability. Given the structure of our within-subject repeated measures design, which typically offers higher statistical power, detecting a medium effect size seemed appropriate. We conducted a power analysis for Linear Regression in R using the 'stats' package, regarding hypotheses 1a and 1b (i.e., a model using one predictor: ER_Time-adaptive/maladaptive). The calculated minimum sample size needed to achieve a power of .80 for detecting an effect size of 0.15 in this linear regression model, with an alpha level of 0.05, is **53** participants (*n*). To calculate the minimum sample size needed to test hypothesis 1c, with five predictors (i.e., acceptance, concentrating on positive, concentrating on planning, positive reappraisal, relativation), **86** participants (*n*) are required to achieve a power of .80 at an alpha level of 0.05.

Procedure

Before the experiment

Before the experiment, questionnaires and other relevant documents had been exchanged online. The participants were given an informed consent, to which they had to agree, screening questionnaires about the inclusion and exclusion criteria, and other questionnaires (CERQ, EMO-Check, DASS, PANAS). Participants who met the screening criteria were able to make an appointment and were given a brief explanation of the terms of participation. The following terms were listed for participants to consider: (**a**) *the participant is not allowed to use psychoactive drugs (cannabis, cocaine, MDMA) in the last 72 hours before the experiment*; (**b**) *the participant is not allowed to use alcohol, in the last in the last 24 hours before the experiment*; (**c**) *the participant is not allowed to eat or drink coffee/other caffeinated drinks (e.g., energy drinks/tea), in the last two hours before the experiment*; (**d**) *the participant cannot perform intense activities, the day before and the day of the experiment*; (**e**) *the participant should maintain a normal sleep pattern the day before the experiment*.

Seven days before the experiment, participants received an invitation to fill out the questionnaires. If the questionnaires were not completed, participants received daily reminders. Four days before the start of the experiment, participants received a reminder email providing all practical information again and clearly reiterating the conditions for participation.

During the experiment

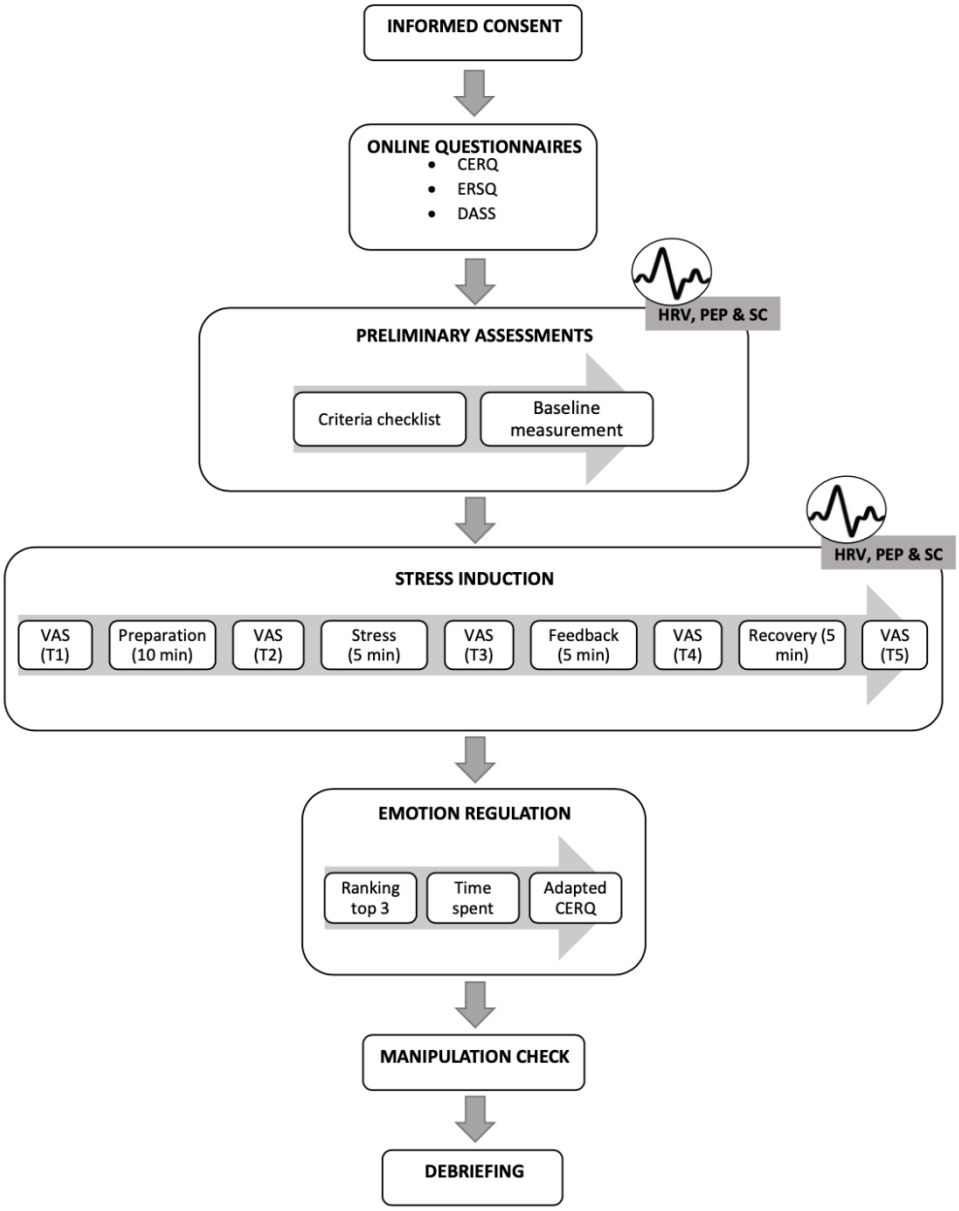
During the **first phase (**i.e., **preparation phase**), participants completed another informed consent (on paper). During this phase, the inclusion and exclusion criteria, and the terms of participation were questioned again, after which the Vrije Universiteit Ambulatory Monitoring System (VU-AMS) was installed. Participants had to sit on the chair with knees bent at 90 degrees and both feet flat on the floor. As the final step of this phase, the M.I.N.I. (Sheehan et al., 1998) was administered for additional screening of potential mental health vulnerabilities. If participants did not show elevated risks, they proceeded to the next phase.

The **second phase** of the experiment (i.e., **experiment phase**) started with a baseline measurement (seven minutes), participants were instructed to remain seated in the same position for seven minutes to facilitate the collection of heart rate and skin conductance measurements for comparative purposes. Participants then completed the VAS T1 (anger, sadness, happiness, tension; **attachment 4**) through which baseline stress level results were obtained (i.e., subjected measurements of stress response). During the preparation time (10 min.), participants were given instructions (**attachment 3** for the Dutch scripts). After the preparation participants filled in the

VAS T2. Following this, the stress induction took place. Psychosocial stress was induced using an adapted version of the Trier Social Stress Test (TSST; Kirschbaum et al., 1993), a widely used experimental method (Allen et al., 2014; Dickerson & Kemeny, 2004; Eagle et al., 2021). Participants underwent a five-minute preparation period followed by a five-minute presentation via a 'Teams meeting' on information about blood (**attachment 5**) to a panel of judges. After the stress induction phase, participants were asked to complete the VAS T3 and respond to an additional question regarding their satisfaction with their performance. In a next phase, negative feedback was provided to the participants about their presentation. Negative feedback provided by the jury regarding their presentation served to heighten the induced stress even further. It's important to note that participants were not told that the jury feedback was from pre-recorded video segments featuring actors. Slightly different video segments were used depending on the participant's status (e.g., first-year student vs. not-first-year student), with jury members using only one different phrase tailored to each group. This feedback was given by the jury members and lasted five minutes (see **attachment 3**), followed by the completion of VAS T4 and a question regarding their satisfaction with their performance. After feedback was given, a recovery phase was introduced to complete the physiological measurements (five minutes). Participants were instructed to maintain the same position. Afterwards they were asked to fill in the last VAS T5 and additional questionnaires that assess the way participants spontaneously managed their emotions (five minutes (i.e., top three strategies they have used and how much time they spent on each strategy; CERQ; **attachment 6**). Next, we conducted a manipulation check to assess whether the participants believed the video feedback, by asking them directly about their belief in it.

The **third** and final **phase** of the experiment (i.e., **debriefing phas**e); contained a debriefing about the research and the tasks performed, as well as a question time for the participants, after which details for the payment process were collected.

Figure A
Visualisation of the procedure of this research



Materials and measurements

Online questionnaires: emotional state and emotion regulation

To measure spontaneous ER, this research used a Dutch-translated version of the Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski et al., 2001). Seven coping strategies were selected and assessed during the experiment (along with the addition of another strategy, 'relaxation') (**attachment 6**). This questionnaire evaluated the ER-strategies used at three consecutive points after receiving feedback (part 1) and measured the time spent on each strategy (part 2). The CERQ (Garnefski & Kraaij, 2006) measures nine cognitive coping strategies (e.g., '*self-blame*', '*blaming others'*, '*acceptance*', '*positive refocusing*', '*refocus on planning'*, *'rumination or focus on thought*', '*positive reappraisal'*, '*putting into perspective*' and '*catastrophizing*') consisting of each two items referring to two indictors of emotional problems (i.e., depressive and anxiety symptoms). *Self-Blame* refers to blaming yourself for what happened (e.g., '*I feel that I am the one who is responsible for what has happened*'), *blaming others* refers to blaming the environment or another person for what happened (e.g., '*I feel that others are responsible for what has happened*'), *acceptance* refers to letting go and accepting what has happened (e.g., '*I think that I have to accept that this has happened*'), *positive refocusing* refers to focusing your attention on positive experiences (e.g., '*I think of pleasant things that have nothing to do with it*'), *refocusing on planning* refers to thinking about the next steps and dealing with the negative event (e.g., ' *I think about how to change the situation*'), *rumination or focus on thought* refers to thinking about the negative event and its associated feelings and thoughts (e.g., '*I often think about how I feel about what I have experienced*'), *positive reappraisal* refers to redefining a situation in a more adaptive/positive way (e.g., '*I think I can learn something from the situation*'), *putting into perspective* refers to comparing your own situation with others, putting the importance of an event into perspective (e.g., '*I tell myself that there are worse things in life*'), and *catastrophizing* refers to drastically magnifying the negative effects of your situation (e.g., '*I keep thinking about how terrible it is what I have experienced*'). The 36 items were rated on a five-point Likert scale, ranging from *'(almost) never'* (scored as one) to *'almost all the time'* (scored as five). Significant results were found of the validity (Ireland et al., 2017) and reliability of the CERQ and CERQ-short (Betegón et al., 2022).

The EMO-Check was used to measure the past week's emotional state and coping style. This self-reporting questionnaire is based on the original, German questionnaire called 'Selbsteinschätzung emotionaler Kompetenzen – 27' (SEK-27) from Berking & Znoj (2008) and includes 50 items measuring emotions and moods, and a 27-item questionnaire measuring the handling of these emotions. The items in this questionnaire indicate emotions like stress, anxiety,

anger, sadness, depression, shame, coping, and positive and negative affect, rated on a five-point Likert scale (from '*not at all'* scored as zero, to '*very*' scores as four). The other 27 items measure the following nine adaptive ER-skills (measured by three items each): attention to own emotions (e.g., '*During the last week I paid attention to my feelings*'), emotional clarity (e.g., '*During the last week I could clearly tell what I was feeling*'), bodily sensations (e.g., '*During the last week I had good body awareness regarding my feelings*'), emotional understanding (e.g., '*During the last week I knew why I felt the way I was feeling*'), resilience (e.g., '*During the last week I could do what I had intended to do even while experiencing negative feelings'*), acceptance (e.g., '*During the last week I was sure to be able to tolerate negative emotions*'), emotion regulation (e.g., '*During the last week I could accept negative feelings*'), readiness to confront distressing situations (e.g., '*During the last week I knew I could influence my feelings*') and self-support (e.g., '*During the last week I remained myself in tough situations*'). The SEK-27 (Berking & Znoj, 2018) is based on the Adaptive Coping of Emotion model (ACE; Berking & Whitley, 2014) and the items are rated on a five-point Likert scale, ranging from '*not at all*' (scored as zero) to '*almost always*' (scored as four). Significant results were found of the validity and reliability of this questionnaire (Berking & Znoj, 2008; Grant et al. 2018).

The Depression Anxiety Stress Scale 42 (DASS; Lovibond & Lovibond, 1995) is a 42-item, self-reported scale of depression (e.g., '*I felt like my life had no meaning*'), anxiety (e.g., '*I noticed that my mouth felt dry*'), and stress (e.g., '*I noticed that I was rather touchy*'. The three scales of the DASS correspond with the tripartite model of Clark and Watson (Clark & Watson, 1991; Watson & Kendall, 1989) in which three groups of symptoms were identified (e.g., symptoms of negative affect, of absence of positive affect, and, of physiological hyperarousal). For each item, participants were required to indicate how they felt over the past week, on a four-point Likert scale, ranging from '*not at all/never*' (scored as zero) to '*most definitely/mostly*' (scored as three). The DASS seems to be reliable and valid for measuring depression, anxiety, and stress (Brown et al., 1997; Clara et al., 2001; Henry & Crawford, 2005). This study used the Dutch version of the DASS, which showed good psychometric characteristics (i.e., good internal consistency between subscales, adequate test-retest reliability, and good discriminant validity; De Beurs et al., 2001).

The Positive and Negative Affect Schedule (PANAS) was employed to assess mood (Watson et al., 1988). This scale comprises 20 items that assesses both positive (e.g., energetic) and negative (e.g., anxious) affect. Participants rated their feelings using a five-point Likert scale, from *'very slightly or not at all*' (scored as one) to *'most definitely/mostly*' (scored as five), reflecting their general or specific timeframe emotions (e.g., present moment, past

day/week/year). The researchers also reported high internal consistency and provided evidence supporting convergent and discriminant validity for the scales (Watson et al., 1988). For the current research question, data of this questionnaire was not used and will therefore not be further discussed.

Psychological assessments

To screen participants, this study used the Dutch version of the Mini International Neuropsychiatric Interview, version 5.0.0 (M.I.N.I.; Sheehan et al., 1998). This tool evaluates a wide range of psychiatric disorders based on the criteria established by the Diagnostic and Statistical Manual of Mental Disorders (DSM-III-R; DSM-IV) and the International Classification of Diseases (ICD-10; World Health Organization, 1993). Typically, two screening questions are asked. If both are answered negatively, no further questions are posed for that disorder, suggesting the patient may not have it. If one or both are answered positively, more detailed symptom questions follow. Participants are screened for various disorders (such as Major Depressive Disorder, Dysthymic Disorder). The authors have also successfully demonstrated the reliability and validity of the M.I.N.I. as a screening tool for psychiatric disorders (Sheehan et al., 1998).

The Visual Analoge Scale (VAS) was used to visualize perceived emotions like tension, sadness, joy, and anger (**attachment 4** for Dutch version). It consists of a horizontal line (100mm) where the extreme left represents '*not at all*' and the extreme right represents '*very much'*. Participants were required to indicate to what extent they experienced a certain emotion (e.g., tension, sadness, joy, or anger) by placing a cross on the dimensional axis.

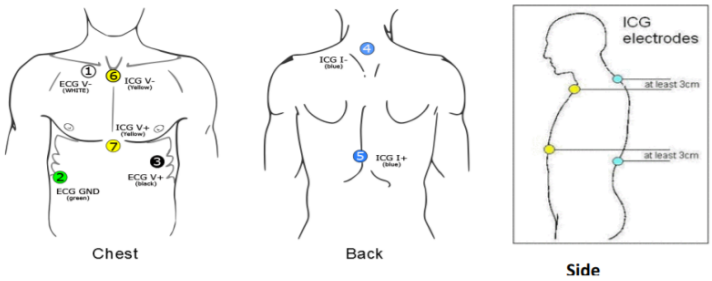
Physiological assessments

To collect physiological data, we utilized the 'Vrije Universiteit Ambulatory Monitoring System' (VU-AMS) to measure electrocardiogram (ECG) and Impedance Cardiography (ICG) for various cardiac parameters (e.g., heart rate, cardiac output), and electrodermal activity (EDA) for skin conductance (i.e., SC; the skin's electrical conductance, which varies with moisture levels; Schmidt & Walach, 2000). We used three electrodes on the chest for ECG-measures, four electrodes (two on the front, two on the back) for ICG-measures, and two electrodes on the non-dominant hand of the participant EDA measures. All electrodes were placed appropriately and collected data throughout the experiment (as shown in **Figure 4** & **Figure 5**; Vrije Universiteit Amsterdam, 2022, p.16). To collect, inspect and analyze the measurements, the Data Analysis and Management Software (VU-DAMS) program was used (version 5.4.13.). The measures

included subjective stress recovery (VAS measures), sympathetic activity (PEP), and parasympathetic activity (RSSMD) to assess stress recovery.

Figure 4

Placement ECG, ICG and EDA electrodes



ECG:

1 Slightly below the right collar bone 4 cm to the right of the sternum

2 (GND) On the right side, between the lower two ribs

3 At the apex of the heart on the left lateral margin of the chest approximately at the level of the processus xiphodius

ICG:

Electric current generating electrodes

4 At the back, on the spine, at least 3 cm (1") above electrode 6

5 At the back, on the spine, at least 3 cm (1") below electrode 7

Impedance measuring electrodes

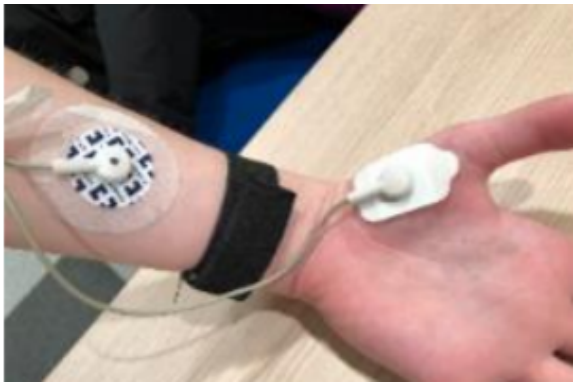
6 At the suprasternal notch above the top of the sternum

7 At the processus xiphodius at the bottom of the sternum

Note. Adapted from VU-DAMS manual, by Vrije Universiteit Amsterdam, 2022, p. 16.

Figure 5

Placement electrodes for skin conductance



Note. Adapted from VU-DAMS manual, by Vrije Universiteit Amsterdam, 2022, p. 16.

Statistical analyses

As mentioned in the procedure, psychophysiological data was analyzed first (collecting, inspecting and analyzing the measurements) by using the VU-DAMS, version 5.4.13. During each phase (i.e., baseline, preparation, stress, feedback, recovery), averages for HRV and PEP levels were calculated, and perceived stress levels (VAS-Tensed) were collected. For the baseline measurement only the last five minutes were used. The 10-minute preparation phase was divided into two five-minute segments, with average scores calculated for each segment and then for the entire 10 minutes. These measures represented the physiological response during each phase. For our final analyses, SC-measures were excluded as indicators for stress recovery because of their slow decline, which requires longer observation periods to yield more interpretable data. Furthermore, for several participants we had problems collecting the SC-measures, which served as an additional reason for not including this measure in the analysis.

All analyses were conducted in R 4.4.1 (2023.06.0+421). We computed the RMSSD, PEP, and VAS-Tensed (VAS-T) as stress recovery measures by subtracting the stress score (i.e. stress-RMSSD and stress-PEP; VAS T1-Tensed) from the recovery score (i.e., recovery-RMSSD, recovery-PEP, and VAS T3-Tensed). For the following analyses we utilized these score differences between stress and recovery phase, reflecting participants' recovery from an acute stressor, as dependent variables (i.e., Stressrecovery_RMSSD, Stressrecovery_PEP, Stressrecovery_VAS_T).

Firstly, we cleaned the data by removing missing and incorrect data (i.e., noisy ECG readings) and conducted descriptive analyses (e.g., calculating mean scores, standard deviations, relative and/or absolute frequencies of the demographic variables) in order to describe the complete sample (i.e., sample before separating into two groups). Then we conducted multiple multivariate analyses of variances (MANOVAs) to identify pertinent control variables, on our complete sample. Additionally, we assessed the impact of the stress-induction procedure by performing paired-sample t-tests for all stress recovery measures (VAS-T, RMSSD, PEP), for 'satisfaction' before and after inducing stress, on this sample. We also conducted the Welch Two Sample t-test to examine whether there was a significant difference in the effectiveness of the feedback video between participants who believed the video and those who did not.

Furthermore, to test our hypotheses (1a, 1b, 1c) relevant to our research question (1), we separated the sample into two different groups after data-collection (i.e., adaptive_group_ER_Time and maladaptive_group_ER_Time), allowing us to examine the effects of time spent on ER-strategies on stress recovery metrics within each group independently. We manipulated the allocation of time spent on specific groups of strategies

(adaptive vs. maladaptive), ranging from 0 to 100 percent, which participants had to distribute among these strategies. This resulted in two distinct groups: one characterized by spending more time (≥ 50%) on adaptive ER-strategies (i.e., adaptive group), and the other by spending more time (≥ 50%) on maladaptive ER-strategies (i.e., maladaptive group). Our decision to base the grouping on time allocation rather than solely on the use of specific strategies was made to ensure more accurate interpretations. For instance, if a participant used four adaptive ER-strategies and one maladaptive ER-strategy but allocated 80% of their time to the maladaptive strategy, they would be classified into the maladaptive group rather than the adaptive group, based on our grouping criteria.

Before testing our hypotheses, we conducted additional analyses to explore the normality assumptions both statistically, using the Shapiro-Wilk test, and graphically, using QQ plots. Then we conducted separate linear models for each outcome measure within each group, by using the 'stats' R package, for hypotheses 1a and 1b. To test **hypothesis 1c**, we fitted a multiple linear regression model for each outcome measure (i.e., stress recovery measures) within the adaptive group. Since we use the score differences between the stress and recovery phase as the outcome measures (i.e., Stressrecovery_RMSSD, Stressrecovery_PEP, Stressrecovery_VAS_T), the data transforms from a repeated measurement design to a single measurement design per subject. This simplified the analysis to a standard linear model, where each subject contributes a single data point. This approach eliminated the need for mixed-effects modeling, as there are no longer multiple measurements per subject to account for.

For these analyses, F statistics were reported along with the p-values and R-squared adjusted ($R^2_{Adjusted}$).

Results

Preliminary analysis

Sample characteristics

A total of 101 people completed the screening list. During the process several participants were excluded due to elevated DASS scores, deviant scores on the M.I.N.I, noisy ECG readings , and other factors (e.g. not filling in the subsequent questionnaires). Our *final* sample size consisted of a total of **51** participants, comprising 14 males (27.5%) and 37 females (72.5%), with an average age of 21 years (*SD*= 1.87). All participants were Belgian college or university students, except for one student with dual nationality. Recruitment was conducted through flyers and social media platforms such as Facebook and Instagram. Of the 51 participants, 23 were first-year students (45.1%) and 28 were not first-year students (54.9%). The sample included 31 bachelor's degree students (60.8%), 15 master's degree students (29.4%), and five students in a bridging course to a master's program (9.8%). Additionally, 22 students (43.1%) had an additional higher education degree, while 29 students (56.9%) did not.

Descriptive statistics

Table 2 presents the mean scores and standard deviations for the DASS, CERQ, and EMO-Check. The DASS results indicate normal mean scores across all subscales.

Tabel 2

Mean Scores and Standard Deviations for the questionnaires

Outcome variables	Mean (*M*)		Standard Deviation (*SD*)	
DASS				
Subscale stress	9.86^{pre}		6.42^{pre}	
Subscale anxiety	4.82^{pre}		4.22^{pre}	
Subscale depression	6.61^{pre}		5.49^{pre}	
CERQ				
Self-blaming	10.76^{pre}	10.25^{exp}	3.07^{pre}	3.75^{exp}
Blaming others	6.57^{pre}	5.73^{exp}	1.82^{pre}	2.21^{exp}

Acceptance	13.24^{pre}	14.16^{exp}	3.65^{pre}	3.71^{exp}
Rumination	13.45^{pre}	10.76^{exp}	4.23^{pre}	3.88^{exp}
Concentrating_other_ positive	11.00^{pre}	8.06^{exp}	3.47^{pre}	4.62^{exp}
Concentrating_planning	13.75^{pre}	11.24^{exp}	3.94^{pre}	3.71^{exp}
Positive reappraisal	12.69^{pre}	10.94^{exp}	4.18^{pre}	3.78^{exp}
Relativation	12.69^{pre}	10.98^{exp}	3.63^{pre}	4.09^{exp}
Catastrophizing	6.86^{pre}	5.59^{exp}	2.56^{pre}	1.98^{exp}
EMO-Check				
Awareness	6.92^{pre}		2.73^{pre}	
Bodily sensations	6.88^{pre}		2.39^{pre}	
Emotional clarity	6.98^{pre}		2.54^{pre}	
Emotional understanding	7.78^{pre}		2.57^{pre}	
Modification	6.75^{pre}		2.30^{pre}	
Acceptance	7.55^{pre}		2.32^{pre}	
Tolerance	6.55^{pre}		2.28^{pre}	
Readiness to confront	6.73^{pre}		2.27^{pre}	
Self-support	7.57^{pre}		2.15^{pre}	

Note. Mean scores and standard deviations of ratings on a 4-point Likert scale (ranging from 0 to 3 for DASS-21), and a 5-point Likert scale (ranging from 1 to 5 for CERQ-short; from 0 to 4 for EMO-Check) for the questionnaires. See previous *'Online questionnaires: emotional state and emotion regulation*' section for details.

^{pre}: scores pre-experiment

^{exp}: scores during the experiment

Table 3 displays the mean scores and standard deviations for stress recovery measurements (i.e., RMSSD, PEP, VAS-T) across different phases, as well as the mean score and standard deviation for participants' recovery after stress induction. The results indicate a slight increase in mean RMSSD (M = -19.13, SD = 19.47) and PEP (M = -16.02, SD = 15.74) measures, along with a moderate decrease in mean VAS-T (M = -33.35, SD = 28.53) measures across phases.

Table 3

Mean Scores and Standard Deviations for the stress recovery measurements across phases

Outcome variables	Mean (*M*)	Standard Deviation (*SD*)	Range
Baseline			
RMSSD	48.69	25.99	4.21 – 105.16
PEP	112.76	15.10	67.91 – 141.00
VAS-Tensed	31.33	24.49	0.00 – 87.00
Stress			
RMSSD	35.82	18.52	7.58 – 82.72
PEP	94.52	19.03	57.26 – 134.22
VAS-Tensed	64.69	23.30	16.00 – 100.00
Recovery			
RMSSD	54.94	27.52	4.70 – 127.86
PEP	110.54	15.50	69.00 – 136.00
VAS-Tensed	30.69	22.62	0.00 – 78.00
Stress recovery			
RMSSD	-19.13	19.47	-75.04 – 14.54
PEP	-16.02	15.74	-63.26 – 2.00
VAS-Tensed	-33.35	28.53	-81.00 – 35.00

Note. Mean scores and standard deviations for baseline, stress and recovery phase, as well as the difference score between stress and recovery for RMSSD, PEP, and VAS-Tensed. Stress recovery measures for RMSSD, PEP, and VAS-Tensed refer to the difference in mean scores between the stress and recovery phase.

Table 4 displays the results of how much time is spend on ER-strategies on average presented in percentages (standard deviations are shown as well). Results show that participants spent more time using adaptive ER-strategies (*M*= 78.25%, *SD*= 23.57) compared to maladaptive ones (*M*= 21.75, *SD*= 23.57). Additionally, a significant portion of our participants utilized both adaptive and less adaptive ER-strategies (71% used both), where other participants used only adaptive ER-strategies (29%)

Table 4

Mean Percentage Scores and Standard Deviations for time spent on ER-strategies

Outcome variables	Mean (*M*)	Standard Deviation (*SD*)
Adaptive ER-strategies	78.25	23.57
Relaxation	9.86	6.42
Acceptance	4.82	4.22
Relativation	6.61	5.49
Maladaptive ER-strategies	21.75	23.57
Self-blaming	10.76	3.07
Rumination	6.57	1.82
Blaming others	13.24	3.65
Catastrophizing	13.45	4.23

Note. Mean scores and standard deviations of time spent (%) on ER-strategies. The results are based on the questionnaire taken at the end of the experiment.

Based on our specific hypotheses (1a and 1b) examining how variations in time spent on adaptive and maladaptive ER-strategies within distinct groups relate to stress recovery outcomes, participants were categorized into either the adaptive or maladaptive group based on their strategy engagement (i.e., adaptive_group_ER_Time or maladaptive_group_ER_Time). We assessed whether increased time spent on adaptive or maladaptive ER-strategies correlated with significant stress recovery differences (as outlined in **Hypotheses 1a** and **1b**). As a result, *45* participants were included in the adaptive group, and *seven* participants were included in the maladaptive group. It's important to note that *one* participant allocated an equal percentage of time to adaptive and maladaptive ER-strategies (50/50) and was therefore included in both groups. Furthermore, within the adaptive group (i.e., adaptive_group_ER_Time), we examined which adaptive ER-strategy contributed most positively to stress recovery outcomes (as outlined in **Hypothesis 1c**).

Controlling for other variables

Multiple Multivariate Analyses of Variances (MANOVAs) were conducted (on the complete sample; 51 participants) to examine the effects of age, education level, gender,

academic year, and nationality on stress recovery metrics (Stressrecovery_RMSSD, Stressrecovery_PEP, Stressrecovery_VAS_T). The MANOVAs revealed non-significant effects for age (V= 0.26, F(21, 129)= 0.57, p= .93), education level (V= 0.019, F(6, 94)= 0.15, p = .99), academic year (V= 0.026, F(3, 47) = 0.42, p = .74), and nationality (V= 0.046, F(3, 47)= 0.81, p = .53), and a significant effect for gender (V= 0.18, F(3, 47)= 3.55, p < .05). Although the analysis revealed a significant effect of gender on the combined dependent variables for stress recovery (p< .05), the variable 'gender' was not included in further tests or analyses to maintain focus on the primary research question and hypotheses. Thus, none of these variables were included in subsequent analyses.

Manipulation check

Participants were asked whether they believed the feedback given after their presentation. 33 participants (64.71%) indicated they did not believe the feedback, while 18 participants (35.29%) indicated they believed it. A paired sample t-test was conducted (on the complete sample; 51 participants) to examine whether there was a significant difference in satisfaction scores with the presentation before and after feedback was provided. The paired t-test showed a statistically significant difference between satisfaction scores before and after feedback was given (t = 3.99, df = 50, p< .001). The mean difference in satisfaction scores was 7 points (95% CI [3.47, 10.53]). This indicates that, on average, participants reported lower satisfaction with the presentation after receiving feedback compared to before. Additionally, we conducted three Welch Two Sample t-tests to determine if there was a significant difference in the effectiveness of the feedback video between participants who believed the feedback video (believers) and those who did not (non-believers). The stress recovery measures HRV (i.e., RMSSD), PEP, and VAS-T were used as outcome measures, and were compared between the group believers vs non-believers. The comparison included 18 participants in the believer group and 33 participants in the non-believer group. The results showed no significant differences between the two groups on any of the stress recovery measures: RMSSD (t(36.89)= 1.03, d= -5.79, p= .31), PEP (t(46.91)= 1.14, d = -4.66, p = .26), and VAS-T (t(39.47)= -1.08, d= -8.64141, p= .29).

To assess whether the stress-induction procedure significantly increased stress across phases, several paired sample t-test were conducted on all measure of stress recovery (VAS-T, RMSSD, PEP) on the complete sample (51 participants). We conducted a test for VAS-T scores (i.e., subjective stress measure) between baseline (VAS-T1) and stress (VAS-T3), and between stress (VAS-T3) and feedback (VAS-T4). The t-test yielded a significant result (t= -8.35, df= 50, p< .001), indicating a substantial difference in subjective stress levels between baseline and

stress phase. The mean difference in VAS-T scores between these phases was -33.35 (95% CI [-41.38, -25.33]). This suggests that participants experienced significantly higher stress during the stress phase compared to baseline. Additionally, the t-test comparing subjective stress levels between the stress and the feedback phase yielded a significant result (t= 6.45, df= 50, p< .001). The mean difference in VAS-T scores between these phases was 15.12 (95% CI [10.41, 19.82]). The same analyses were repeated for RMSSD and PEP, for baseline and stress phase, and stress and feedback phase. We found a significant effect between baseline and stress phase for RMSSD (t= 4.57, df = 50, p< .001), with a mean difference of 12.88 (95% [7.218, 18.539]), and a significant effect between stress and feedback phase (t= -12.54, df = 50, p < .001), with a mean difference of -66.50 (95% [-77.151, -55.853]). As for PEP, we found a significant effect between baseline and stress phase (t= 39.31, df = 50, p < .001), with a mean difference of 128.77 (95% [122.193,135.353]), and a significant effect between stress and feedback phase (t= -40.54, df = 50, p< .001), with a mean difference of -120.59 (95% [-126.57, -114.62]).

Main analysis for the adaptive group (Hypotheses 1a and 1c)

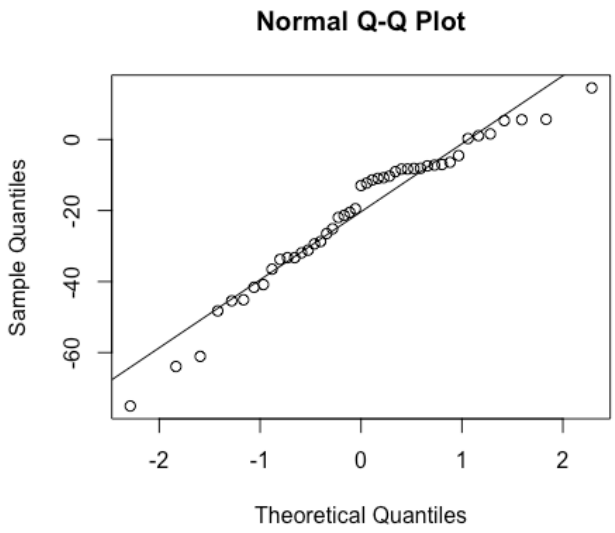
Parasympathetic stress recovery: RMSSD

First, we conducted analyses within the adaptive group (n= 45), meaning the group of participants that spent more time (≥ 50 % of their time) on adaptive ER strategies (i.e., relaxation, acceptance, positive reappraisal, relativation). To examine the effect of time spent on adaptive ER-strategies (ER_TIME_adaptive) on stress recovery (RMSSD, PEP, VAS-T), we performed linear regressions to examine the effect of ER_Time_adaptive on each stress recovery measure individually.

We began with the dependent variable RMSSD, which represents objective parasympathetic activity, thus vagally mediated HRV (vmHRV). To test the normality assumptions of the data (for RMSSD) for the adaptive group, we conducted the Shapiro-Wilk test and examined the graphical normality distribution using plots. The data for Stressrecovery_RMSSD appeared to be approximately normally distributed based on the Shapiro-Wilk test (p< .05). Visual inspection of additional plot further confirmed the normality assumption for RMSSD (see **Figure 6**).

Figure 6

Normality assumption of RMSSD via plots



Note. Visual plot testing the normality assumption of RMSSD for stress recovery in de adaptive group.

The aim was to test whether the amount of time spend on adaptive ER-strategies (ER_Time_adaptive) predicts the score difference between stress and recovery phase (i.e.,

Stressrecovery_RMSSD), which reflects HRV changes (**Hypothesis 1a**). We fitted a linear model with Stressrecovery_RMSSD as the outcome measure and ER_Time_adaptive as the predictor. The results suggested that the amount of time spent on adaptive ER-strategies did not significantly predict the score difference between stress and recovery phases ($F(1,43) = 0.075$, $p = .79$, $R^2_{adjusted} = -0.022$), in this study sample.

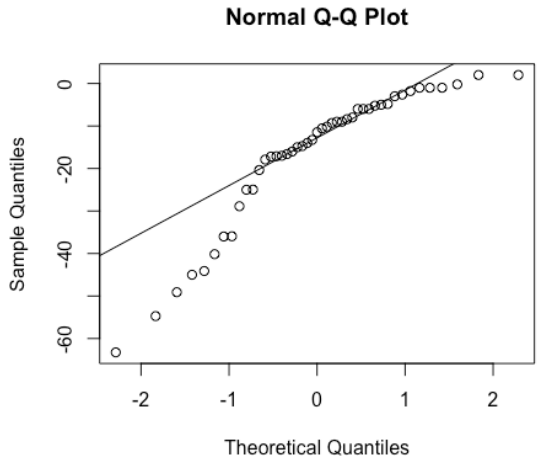
To explore the effects of adaptive ER-strategies on stress recovery measure, RMSSD, within the adaptive group (**Hypothesis 1c**), we used mean scores of CERQ (for adaptive ER-strategies only; acceptance, relativation, positive reappraisal, planning, and concentrating on other positive) collected at the end of the experiment. We fitted a multiple linear regression model with Stressrecovery_RMSSD as dependent variable, and the mean scores of the five adaptive ER-strategies, measured by CERQ, as independent variables. The linear regression analysis (exploratory) indicated that none of the CERQ mean scores significantly predicted the Stressrecovery_RMSSD outcome ($F(5,39) = 0.53$, $p = .76$, $R^2_{adjusted} = -0.057$). This means that the average use of adaptive ER-strategies (measured by CERQ) did not predict the differences in stress recovery as indicated by RMSSD, in this study sample.

Sympathetic stress recovery: PEP

We repeated the same steps for PEP, testing the normality assumption with the Shapiro-Wilk test and via plot, and investigating whether the amount of time spent on adaptive ER-strategies predicted the score difference between stress and recovery phase (Stressrecovery_PEP), which reflected sympathetic activity changes (**Hypothesis 1a**). Based on the Shapiro-Wilk test for normality, the p-value was very low ($p < .001$). This suggested that the distribution of Stressrecovery_PEP was significantly different from a normal distribution. Additional visual inspections through QQ plots supported the conclusion that the distribution of Stressrecovery_PEP was deviated from normal (see **Figure 7**).

Figure 7

Normality assumption of PEP via plots



Note. Visual plot testing the normality assumption of PEP for stress recovery in the adaptive group.

We still fitted a linear model with Stressrecovery_PEP as the outcome and ER_Time_adaptive as the predictor (**Hypothesis 1a**). The results suggested that the amount of time spent on adaptive ER-strategies did not significantly predict the score difference between stress and recovery phase for PEP ($F(1,43) = 0.013$, $p = .91$, $R^2_{adjusted} = -0.023$). Therefore, the amount of time spent on ER-adaptive strategies did not significantly predict changes in Stressrecovery_PEP during stress and recovery periods in this study sample (adaptive group).

To explore the relationship between adaptive ER-strategies (measured by CERQ mean scores) and stress recovery as indicated by Stressrecovery_PEP (**Hypothesis 1c**), a multiple linear regression model analysis was performed with stressrecovery_PEP as dependent variable and the mean scores of CERQ, for adaptive ER-strategies only, as predictors (i.e., acceptance, relativation, positive reappraisal, planning, and concentrating on other positive). The exploratory, linear regression analysis indicated that none of the CERQ mean scores significantly predict the Stressrecovery_PEP outcome ($F(5,39)= 1.29$, $p = .29$, $R^2_{adjusted}= - 0.032$). This means that the average use of adaptive ER-strategies (measured by CERQ) did not predict the differences in stress recovery as indicated by PEP, in this study sample.

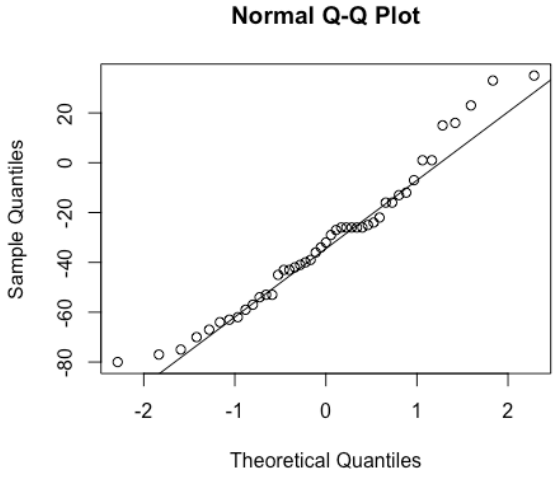
Subjective stress recovery: VAS-Tensed (VAS-T)

Finally, we conducted the same analyses for VAS-T (Stressrecovery_VAS_T) as for RMSSD and PEP. The data for Stressrecovery_VAS_T within the adaptive group appeared to

be normally distributed based on the Shapiro-Wilk test (p= .18). The additional plot confirmed this conclusion (see **Figure 8).**

Figure 8

Normality assumption of VAS-T via plots



Note. Visual plot testing the normality assumption of VAS-T for stress recovery in the adaptive group.

We fitted a linear model with Stressrecovery_VAS_T as the outcome and ER_Time_adaptive as the predictor (**Hypothesis 1a**). The results suggested that the amount of time spent on adaptive ER-strategies did not significantly predict the score difference between stress and recovery phase for VAS-T (F(1,43) = 1.73, p = .20, $R^2_{adjusted} = 0.016$). Therefore, the amount of time spent on adaptive ER-strategies did not significantly predict differences in subjective stress recovery, within the adaptive group.

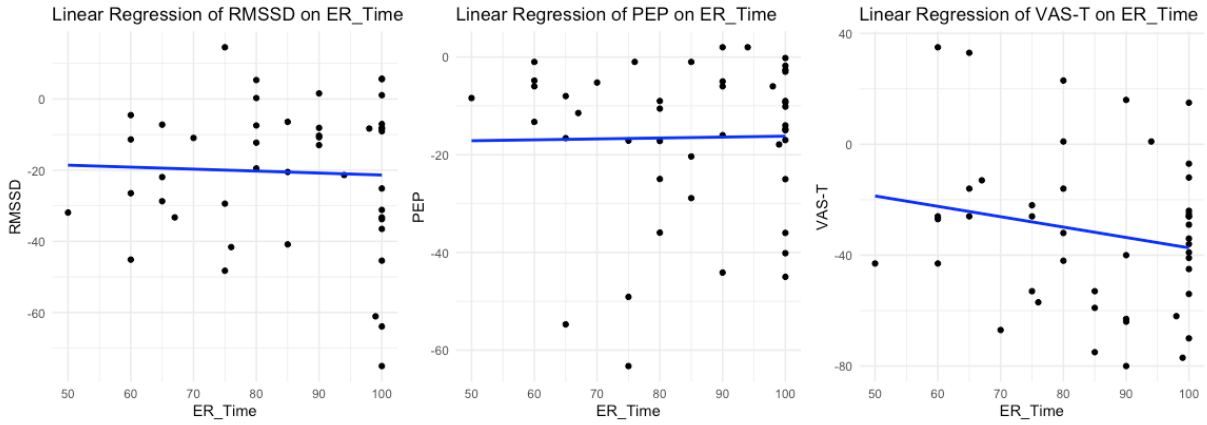
To explore the relationship between adaptive ER-strategies (measured by CERQ mean scores) and stress recovery, as indicated by Stressrecovery_VAS_T, within the adaptive group, a linear regression analysis was performed with subjective stress recovery (Stressrecovery_VAS_T) as dependent variable and the mean scores of CERQ, for adaptive ER-strategies only, as predictors (i.e., acceptance, relativation, positive reappraisal, planning, and concentrating on other positive) (**Hypothesis 1c**). The exploratory analysis revealed that positive reappraisal was a significant predictor of subjective stress recovery (p< .05) in this sample, indicating that higher scores in positive reappraisal strategies were associated with improved stress recovery, measured by VAS-T. In contrast, other adaptive ER-strategies did not show

significant predictive value for Stressrecovery_VAS_T in this sample ($F(5, 39) = 1.51$, $p = .21$, $R^2_{Adjusted} = 0.055$).

Figure 9 shows the visual representation of the findings discussed above (**Hypothesis 1a**).

Figure 9

Plots linear regression time spend on adaptive ER-strategies and RMSSD, PEP, and VAS-T.



Note. Plots for linear regression with ER_Time as the predictor and stress recovery scores (RMSSD, PEP, VAS-T) as the outcomes, within the adaptive group (**Hypothesis 1a**).

Main analysis for the maladaptive group (Hypothesis 1b)

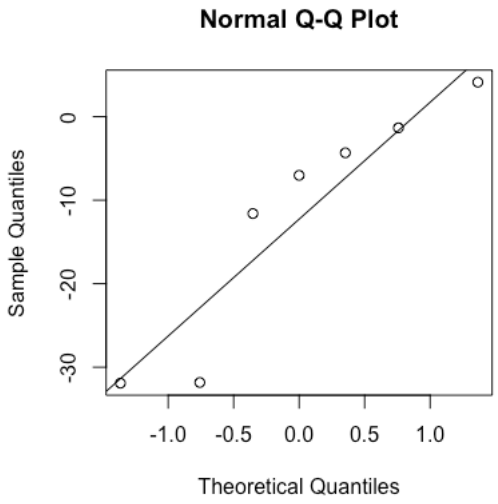
Parasympathetic stress recovery: RMSSD

We conducted the same analyses within the maladaptive group ($n = 7$), meaning the group of participants that spent more time (≥ 50 % of their time) on maladaptive ER-strategies (e.g., self-blaming, rumination, catastrophizing, blaming others). We realized that this group is too small to yield enough power, but we nevertheless executed these analyses for the completeness of this master thesis. To examine the effect of the amount of time spent on maladaptive ER-strategies (i.e., ER_Time_maladaptive) on stress recovery measures (Stressrecovery_RMSSD, Stressrecovery_PEP, and Stressrecovery_VAS_T) within the maladaptive group (**Hypothesis 1b**), we fitted linear regressions on each stress recovery measure individually.

The data for Stressrecovery_RMSSD within this group appeared to be normally distributed based on the Shapiro-Wilk test ($p = .15$). Additional visual inspections through QQ plots supported this conclusion (see **Figure 10**).

Figure 10

Normality assumption of RMSSD via plots



Note. Visual plot testing the normality assumption of RMSSD for stress recovery in the maladaptive group.

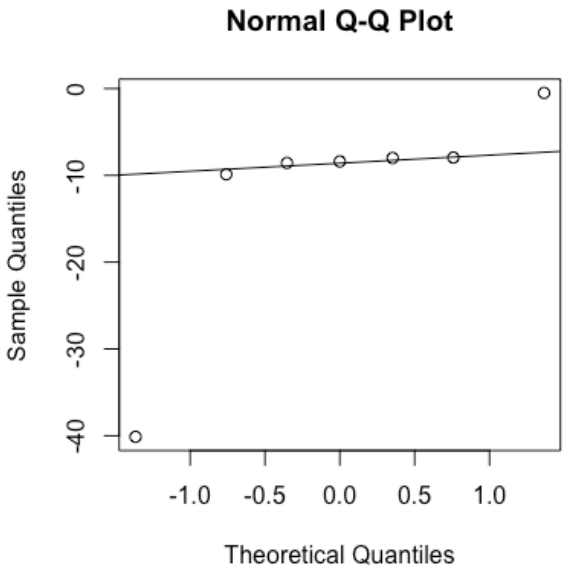
We fitted a linear model with Stressrecovery_RMSSD as outcome measure and time spent on maladaptive ER-strategies (i.e., ER_Time_maldaptive) as predictor (**Hypothesis 1b**). The results suggested that the amount of time spent on maladaptive ER-strategies did not significantly predict the score difference between stress and recovery phase ($F(1,5) = 1.32$, $p = .30$, $R^2_{adjusted} = 0.050$), in this study sample.

Sympathetic stress recovery: PEP

We repeated the same steps for PEP, testing the normality assumption with the Shapiro-Wilk test and via plots, and investigating whether the amount of time spent on maladaptive ER-strategies predicted the score difference (Stressrecovery_PEP) between stress and recovery phase (**Hypothesis 1b**). The data for Stressrecovery_PEP within this group appeared to not be normally distributed based on the Shapiro-Wilk test ($p < .001$), but this analysis was not necessarily reliable given the small number of subjects. However, additional visual inspections through QQ plots supported to some extent the conclusion that the distribution of Stressrecovery_PEP was not too much deviated from a normal distribution (see **Figure 11**).

Figure 11

Normality assumption of PEP via plots



Note. Visual plot testing the normality assumption of PEP for stress recovery in the maladaptive group.

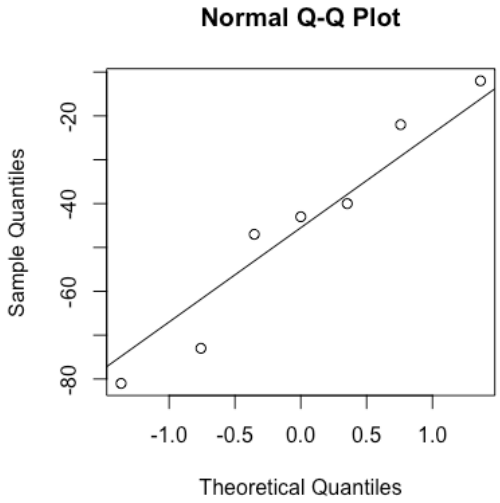
We fitted a linear model with Stressrecovery_PEP as outcome measure and time spent on maladaptive ER-strategies (i.e., ER_Time_maldaptive) as predictor within the maladaptive group (**Hypothesis 1b**). The results suggested that the amount of time spent on maladaptive ER-strategies did not significantly predict stress recovery as measured by PEP ($F(1,5) = 0.00$, $p = .99$, $R^2_{adjusted} = -0.20$), in this study sample.

Subjective stress recovery: VAS-Tensed (VAS-T)

Finally, we conducted the same analyses for VAS-T (Stressrecovery_VAS_T) as for RMSSD and PEP within the maladaptive group. The data for Stressrecovery_VAS_T appeared to be normally distributed based on the Shapiro-Wilk test ($p = .70$). The additional plot confirmed this conclusion (see **Figure 12**).

Figure 12

Normality assumption of VAS-T via plots



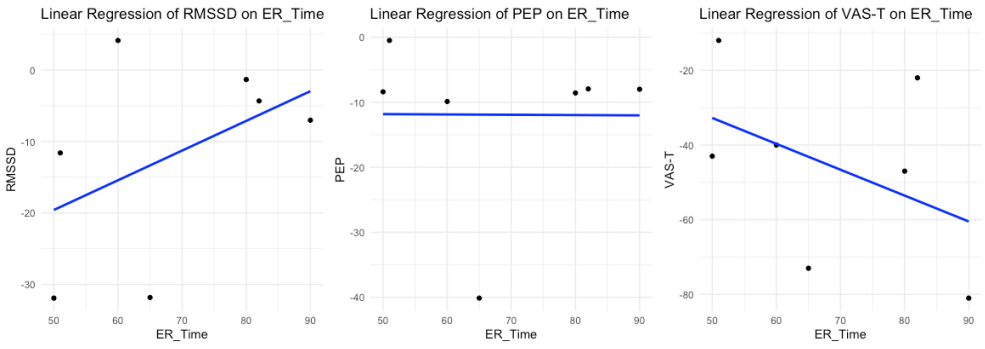
Note. Visual plot testing the normality assumption of VAS-T for stress recovery in the maladaptive group.

We fitted a linear model with Stressrecovery_VAS-T as outcome measure and time spent on maladaptive ER-strategies (i.e., ER_Time_maldaptive) as predictor within the maladaptive group (**Hypothesis 1b**). The results suggested that the amount of time spent on maladaptive ER-strategies did not significantly predict subjective stress recovery as measured by VAS-T ($F(1,5) = 0.00$, $p = 1.21$, $R^2_{adjusted} = 0.033$), in this study sample.

Figure 13 shows the visual representation of the findings discussed above.

Figure 13

Plots linear regression time spend on maladaptive ER strategies and RMSSD, PEP, and VAS-T.



Note. Plots for linear regression with ER_Time as the predictor and stress recovery scores (RMSSD, PEP, VAS-T) as the outcomes, within the maladaptive group.

Discussion

Discussion

Stress is a prominent aspect of our lives and significantly impacts our well-being. As explained by the 'neurovisceral integration in emotion regulation and dysregulation' model (Thayer & Lane, 2000), ER seems like an important player effecting individuals stress response, where certain strategies could promote and other diminish stress recovery (Aldao et al., 2010; Barlow et al., 2016; Ehrenreich-May & Bilek, 2012; Gross, 2015; Roemer et al., 2008). Adaptive ER-strategies, such as acceptance, positive reappraisal, and planning, have been consistently associated with better mental health outcomes and improved stress recovery (Troy & Mauss, 2011; Webb et al., 2012). Our study builds on this literature by examining how these strategies specifically impact stress recovery, measured through both subjective reports (VAS-T) and physiological markers like RMSSD and PEP for stress recovery.

How can this study contribute to further research?

Our research fills a crucial gap by exploring the effectiveness of specific adaptive ER-strategies, such as acceptance and positive reappraisal, in facilitating recovery from stress. By examining the differential effects of these strategies, we aim to offer insights that can inform the development of targeted interventions and support systems designed to enhance individuals' adaptive capacities in the face of stress. Furthermore, the challenges and limitations encountered in this research, such as the non-significant results for other ER-strategies and physiological measures, can guide future research in refining methodologies. This might include more precise measurement tools, better control for confounding variables, or alternative ways to categorize and assess ER-strategies. Additionally, our findings may contribute to the existing body of ER-research by emphasizing not only whether individuals employ certain ER-strategies but also how these strategies are used in conjunction with others over time. This approach may provide a nuanced understanding of ER, considering individual differences in strategy use and the duration of their application. Our approach aligns with the growing recognition that stress is an integral part of life, necessitating effective coping mechanisms to maintain psychological resilience and overall well-being. As ER-interventions seems to be a useful transdiagnostic approach to promote an individual's overall well-being (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010; Harvey et al., 2004; Kring & Caponigro, 2010; Svaldi et al., 2012; Weiss et al., 2015).

Discussion of the results: preliminary results

Descriptive statistics indicated that nearly all participants used adaptive ER-strategies (around 88%), and most participants spent more time on adaptive ER-strategies (78.25% of the participants) compared to maladaptive ER-strategies (21.75%) (see **Table 4**). This trend may be explained by the characteristics of the participants in our study, indicating overall good mental health. This is evidenced by the normal mean scores across all DASS subscales (see **Table 2**). Additionally, a significant portion of our participants utilized both adaptive and less adaptive ER-strategies (71% used both), where other participants used only adaptive ER-strategies (29%), highlighting the importance of categorizing groups based on the amount of time spent on specific categories ER-strategies—adaptive versus maladaptive – rather than merely whether they used a particular strategy or not.

To examine the effects of several independent variables on our outcomes (i.e., stress recovery measures), we performed several MANOVAs on the complete sample (i.e., sample before separating into the two groups; 51 participants). The analyses only revealed a significant effect of gender ($p < .05$). Given the small sample size ($n = 51$) and the disproportionate distribution of genders, with 14 males and 37 females, this imbalance may have influenced the observed significant effect. Although gender showed significance, we decided not to include it in further analyses to maintain the focus on our primary research question. However, future research should consider the impact of gender distribution more carefully, as gender differences in HRV and physiological emotional responses have been documented in literature, therefor making it possible to influence the interpretation of the results (Chentsova-Dutton & Tsai, 2007; Koenig & Thayer, 2016; Tobaldini et al., 2020). By conducting gender specific analyses, gender effects could be accounted for, making interpretations more accurate or generalizable.

We further evaluated the effectiveness of the stress-induction procedure by performing paired-sample t-tests for all stress recovery measures (RMSSD, PEP, VAS-T), as well as for participants' ratings for satisfaction of their presentation, before and after the stress induction. Based on previous literature that has proven TSST to be a valid and widely used method for inducing stress (Allen et al., 2014; Eagle et al. 2021), we expected VAS-T and PEP (Cacioppo et al., 1994) scores to be elevated, and RMSSD (Malliani et al., 1991; Malik et al. 1996; Pomeranz et al., 1985) scores to be low, after stress induction. Our tests yielded significant effects of the stress-inducing procedure across those phases for all three measures of stress recovery, confirming that participants showed more stress after the stress-induction task within this study. However, it is important to consider that other factors might influence elevated physiological measures, as we could not fully control for variables described in the exclusion

criteria (such as the use of alcohol or other substances). The only way to account for the influence of such factors was by questioning participants, which relied on subjective measurement and on the honesty of participants. Additionally, our results showed that participants were less satisfied with their performance after receiving negative feedback, indicating that the feedback video was effective. Notably, a significant number of participants reported not believing the feedback video to be real (64.71%). We investigated whether the credibility of the feedback video influenced the effect of stress induction on stress recovery by conducting Welch Two Sample t-tests for each stress recovery measure. We expected to find no significant difference between the group who believed the video and those who did not, which would indicate that the feedback video had similar effects on stress recovery for both groups, regardless of their belief in its content. The results showed no significant differences between the two groups (believers vs non-believers) on any of the stress recovery measures within this sample. This suggests that the stress induction procedure was effective for both believers and non-believers, indicating that belief in the feedback video did not influence the effectiveness of stress induction on stress response. A plausible explanation for the stress experienced by participants, despite their claims of not believing the feedback video, could be that questioning the video's credibility acted as an ER-strategy, allowing individuals to attribute their experienced tension to external factors rather than internalize it. This explanation was further supported by our tests, which confirmed that the feedback significantly influenced participants' satisfaction with their presentation ($p < .001$). Participants reported lower satisfaction with their presentation after receiving the feedback compared to before. Another explanation for the elevated stress experienced by the non-believers, could be that the stress induction procedure itself, such as the TSST or the experimental context in which the feedback was given, was inherently stressful. Even if participants consciously dismissed the feedback as unbelievable, the social evaluation or the pressure of the situation might have still triggered a stress response on a physiological level. This suggests that their bodies reacted to the situation, not just the content of the feedback or the stress-induction task.

Discussion of the results: Hypotheses 1a, 1b

To address our research question, we hypothesized that participants who spontaneously spent more time using adaptive ER-strategies, such as relaxation, reappraisal, and acceptance, would demonstrate significantly great improvements in stress recovery (**Hypothesis 1a**). Based on previous research, this improvement would be measured by a large decrease in VAS-Tensed (subjective) and PEP scores (sympathetic response; Cacioppo et al., 1994), and a large increase

in HRV scores (parasympathetic response; Malliani et al., 1991; Malik et al., 1996; Pomeranz et al., 1985), between the stress and recovery phase. This hypothesis was supported by previous research indicating the benefits of adaptive ER-strategies for mental health and resilience (Troy & Mauss, 2011; Webb et al., 2012).

Conversely, we hypothesized that participants who spent more time on less adaptive ER-strategies, such as self-blame, rumination, catastrophizing, and blaming others, would exhibit low improvements in stress recovery, as indicated by a small decrease in VAS-Tensed and PEP scores (Cacioppo et al., 1994), and a small increase in HRV scores (Malliani et al., 1991; Malik et al., 1996; Pomeranz et al., 1985) (**Hypothesis 1b**). The literature suggested that maladaptive ER-strategies were associated with poorer stress recovery and worse psychological outcomes (Aldao et al., 2010; Gross, 2015).

To test hypotheses 1a and 1b, we conducted a linear regression analysis to examine the effects of the amount of time spent on adaptive ER-strategies on stress recovery within the group of participants who used more adaptive ER-strategies. Similarly, we analyzed the effects of time spent on maladaptive ER-strategies on stress recovery for those who used more maladaptive ER-strategies. However, contrary to our hypotheses (1a and 1b), our results did not provide significant evidence for changes in the three recovery scores between the stress and recovery phase for either the adaptive group using adaptive ER-strategies or the maladaptive group using maladaptive ER-strategies. Additionally, when testing for normality of Stressrecovery_PEP, both the Shapiro-Wilk test ($p < .001$) and visual inspection through QQ-plots indicated that the data was not normally distributed (see **Figure 7**). A plausible explanation for this could be the presence of outliers; if these were excluded, the data might have become normally distributed. To address this possibility, an outlier detection analysis should have been performed, and if necessary, alternative methods should have been used to handle this kind of data.

The non-significant results (Hypotheses 1a and 1b) could potentially be explained by the small number of participants (n=45 for hypothesis 1a and 1c, and n=7 for hypothesis 1b). A post-hoc power analysis revealed a power of .74 for **Hypothesis 1a**, and a power of .13 for **Hypothesis 1b**, at an alpha level of 0.05. This indicates that the probability of detecting a true effect, should one exist, is 74% and 13%, respectively. While the power for Hypothesis 1a is above the .50 mark, it falls short of the commonly accepted threshold of .80, suggesting that our study may have been underpowered. Consequently, the likelihood of committing a Type II error (failing to reject a false null hypothesis) was relatively high at 26%, and 87%, for Hypotheses 1a and 1b respectively. The non-significant findings in this study may therefore be a result of the study

being underpowered rather than the absence of a true effect. Additionally, the variability in participant characteristics, such as baseline stress levels (RMSSD) ranging from 4.21 to 105.16, might also have diluted the effects. This variability could mask potential differences and contribute to the non-significant results observed. Another plausible explanation for the non-significant results could be that the method used to categorize participants based on whether they spent a minimum of 50% of their time on certain ER-strategies (adaptive or maladaptive) may not have been effective. For example, if a participant spends 80% of their time attempting to reappraise and accept but was unsuccessful, and 20% of their time successfully blaming and ruminating, it is likely that no significant effects will be observed.

Considering these limitations, future research should aim to increase the sample size to enhance the statistical power and reduce the risk of Type II errors. Furthermore, controlling for participant characteristics like baseline stress levels or using more nuanced methods to assess the effectiveness of ER strategies—such as evaluating the success of strategy use through self-report measures or applying alternative criteria for categorization—may help clarify the effects of adaptive and maladaptive ER-strategies on stress recovery.

Discussion of the result: Hypothesis 1c

The exploratory analysis of the relationship between specific adaptive ER-strategies and stress recovery within the context of the Trier Social Stress Test, aimed to identify which adaptive ER-strategies were effective among participants who spent more time on these strategies (**Hypothesis 1c**). This exploration was motivated by previous research that questioned the universal effectiveness of adaptive ER-strategies (Aldao & Nolen-Hoeksema, 2010; Aldao & Nolen-Hoeksema, 2011). Our linear regression results partially supported **Hypothesis 1c**. Consistent with findings from other researchers (e.g., Jamieson et al., 2013a; Lazarus & Folkman, 1984), we found that cognitive reappraisal was significantly associated with subjective stress recovery (VAS-T scores), but not with stress recovery measures, RMSSD (HRV) and PEP. These results suggested that participants who used positive reappraisal as an adaptive ER-strategy experience a significant recovery from the stress induced during the stress phase, in this study. The significant finding with VAS-T scores but not with HRV or PEP may suggest that cognitive reappraisal primarily influenced how participants perceived and evaluated their stress, rather than directly altering their physiological stress responses. Another plausible explanation may be that subjective measures like VAS-T could have been better at capturing the immediate benefits of reappraisal within the short time frame of the study, leading to a faster subjective improvement (feeling better) that was not immediately reflected in physiological markers like

HRV and PEP, which could have taken longer to respond or may have required a stronger intervention to show change.

Despite previous evidence supporting the effectiveness of other adaptive ER-strategies such as acceptance (Harris, 2006), no other adaptive ER-strategies significantly predicted improvements in HRV, PEP, or VAS-T in this study. The non-significant effects of other ER-strategies (i.e., acceptance, reappraisal, concentrating on planning, and positive thinking) could potentially be explained by the method used to measure these strategies. Participants were asked about their use of adaptive ER-strategies at the end of the experiment, relying on their subjective interpretation and retrospective reporting. Additionally, our analysis considered participants' responses on the CERQ, which were based on a five-point Likert scale ranging from *'not at all'* (scored as zero) to *'almost always'* (scored as four). However, we did not control for the variations in the frequency of use of different adaptive ER-strategies. This lack of control might explain why comparisons between different adaptive ER-strategies in our study yielded non-significant results, and why this comparison may be considered unjustified. Some participants might have used certain strategies more frequently than others, but if this variability wasn't accounted for, it could have diluted the perceived effectiveness of those strategies. Additionally, if one strategy was used more frequently than another, it might have seemed more or less effective simply because of its frequency of use, rather than its inherent effectiveness. The sentence is mostly correct but can be refined for clarity. Another explanation could be the small sample size used to test this hypothesis (n=45), with a post-hoc power analysis revealing a power of .49 for Hypothesis 1c at an alpha level of 0.05, suggesting that the study may have been underpowered. Consequently, as with Hypotheses 1a and 1b, the likelihood of committing a Type II error was relatively high at 51%.

Considering these limitations, future studies should consider that participants may use certain ER-strategies more frequently than others. To make comparisons between ER-strategies more valid, future research should control for the variations in how often each specific adaptive ER-strategy was used. This approach will provide a more accurate understanding of the effectiveness of different adaptive ER-strategies in stress recovery. Additionally, future research should aim to increase the sample size to enhance the statistical power and reduce the risk of Type II errors.

Theoretical and practical implications

Despite finding only one significant relationship between positive reappraisal as an adaptive ER-strategy and subjective stress recovery (VAS-T) within the adaptive group, and no significant relationships between time spent on specific ER-strategies and stress recovery in either group, our current findings may still have important theoretical and practical implications.

Theoretical Implications

Despite existing research linking ER to psychopathologies (Aldao & Nolen-Hoeksema, 2010; Aldao et al., 2010) and stress recovery measures like HRV to difficulties (Porges, 1992; Thayer & Lane, 2000; Thayer et al., 2009), our study suggests that differences in time spent on ER-strategies may not sufficiently account for variations in stress recovery measures in this context. This study may also indicate that the relationship between ER and HRV observed in previous research (e.g., Thayer et al., 2012) might not be universally applicable. This calls for a reassessment of how ER is conceptualized and measured in relation to physiological indicators of stress recovery. Additionally, this research might highlight the role of cognitive reappraisal within a stress context, suggesting that this strategy may be particularly effective in modulating how individuals perceive and experience stress. Future research might explore whether subjective recovery alone is sufficient for overall well-being or if both subjective and physiological recoveries need to be aligned for optimal health outcomes. This could lead to a deeper understanding of how different ER-strategies affect various facets of stress recovery, including both subjective experiences and physiological responses.

Practical Implications

The study highlights the importance of increasing sample sizes in future research to improve the power of statistical tests and reduce the likelihood of Type II errors. A larger sample size could provide more robust evidence regarding the relationship between ER and stress recovery. Additionally, this study suggests that employing non-linear models and including interaction terms in future analyses may better capture the complex relationships between time spent on ER-strategies and stress recovery measures. This approach might reveal more nuanced patterns and enhance the understanding of how varying allocations of time spent on ER-strategies impact stress recovery. Furthermore, considering alternative or additional methods for measuring ER and stress recovery might enhance the accuracy and reliability of findings. For example, incorporating objective measures alongside subjective self-reports could provide a more comprehensive assessment of ER-strategies and their effects.

These implications offer valuable directions for future research and practical applications, potentially leading to a more nuanced understanding of the role of ER in stress recovery and the development of more effective interventions.

Limitations

Despite the strengths of our study, several potential limitations need to be acknowledged. First, the small number of participants (n=45 for Hypothesis 1a and 1c, and n=7 for Hypothesis 1b) limits the generalizability of our findings and amplifies the risk of Type II errors. Second, while we included both subjective and objective measures of stress recovery, the reliance on self-reported data for ER-strategy use could introduce biases. Participants' retrospective accounts of the strategies they used may not accurately reflect their actual behavior during the stress and recovery phase. Then, there was considerable variability in baseline stress levels (RMSSD) among participants (ranging from 4.21 to 105.16). This variability could dilute the effects of the ER-strategies on stress recovery, making it harder to detect significant relationships (for **Hypotheses 1a**, **1b**, and **1c**). Further, we did not control for the variations in the frequency of use of different adaptive ER-strategies, or for other relevant variables such as gender (where a significant effect has been found). This limits our ability to make justified comparisons between different adaptive ER-strategies (**Hypothesis 1c**), reduces the accuracy of conclusions about the research question, and may cause generalizability issues (e.g., across genders).

Moreover, the use of a specific stress-induction method (video feedback) ensures consistency but may not generalize to other types of stressors or real-world situations. The controlled environment of the study may not fully capture the complexity of stress and ER in everyday life. Additionally, our study was cross-sectional, capturing only the immediate effects of ER-strategies on stress recovery. Longitudinal studies are needed to investigate the sustained effects of ER on stress resilience over time. Lastly, our analysis was primarily linear, which might not fully capture the complex interactions between variables. Considering non-linear models and interaction terms in future research could provide a deeper understanding of these relationships.

Benefits and considerations for future research

Our study aimed to contribute to the field of ER- research in several keyways. First, we advocated for a more nuanced understanding and approach to ER by considering individual differences in strategy use, including the duration each strategy was employed in conjunction with others. As a portion of our participants utilized both adaptive and less adaptive ER-strategies (71% used both), where other participants used only adaptive ER-strategies (29%), this study highlighted the relevance of categorizing groups based on the amount of time spent on specific categories ER-strategies—adaptive versus maladaptive – rather than merely whether they used a particular strategy or not. This perspective acknowledges the complexity of ER and the variability in how individuals manage stress. Second, our research highlighted the value of using multiple outcome measures for stress recovery. By incorporating both subjective and objective assessments, we provided a more complete picture of the stress recovery process. This integration of objective measures alongside subjective assessments responds to calls for more robust and multi-faceted approaches in ER-research. By including physiological data, we addressed the limitations of self-report methods and offered a more comprehensive understanding of stress recovery. Additionally, this study utilized an easy-to-use and implementable method to induce stress, namely video feedback. Consequently, all participants underwent the same stress-induction procedure, ensuring consistency across the study and enhancing the reliability of our findings.

In summary, our study contributes to ER-research by emphasizing individual differences in strategy use, employing a diverse set of outcome measures, and utilizing a standardized stress-induction method. These methodological strengths help to provide a more detailed and reliable understanding of the stress recovery process.

Future studies should aim to enhance methodological rigor by including randomized groups, large sample sizes, and heterogeneous participant samples. These measures are necessary to establish the generalizability of ER-strategies as effective vagal stimulation techniques. Additionally, conducting longitudinal studies can help investigate the sustained effects of ER on stress resilience over time, providing deeper insights into how ER-strategies influence long-term stress recovery. Further, future research could benefit from incorporating more objective stress recovery cut-offs to make results more interpretable. This would help in establishing clearer benchmarks for stress recovery and improving the reliability of findings. Moreover, it could be valuable to consider sex differences in the interpretation of HRV, as research has shown that women display higher HRV and physiological emotional responses compared to men

(Chentsova-Dutton & Tsai, 2007; Koenig & Thayer, 2016; Tobaldini et al., 2020). Although our study showed no significant effects of most of the demographic variables, and one significant effect of gender via MANOVA, taking these differences into account will allow for more accurate interpretation of HRV data, tailoring of stress interventions more effectively, and ensuring that findings are valid and generalizable across both sexes. This approach can then lead to a better understanding and management of sex-specific responses to stress and recovery.

Increasing the sample size in future studies is crucial for enhancing statistical power and reducing the likelihood of Type II errors. Additionally, considering non-linear models and including interaction terms can help capture more complex relationships between variables. Therefore, this approach could potentially provide a better understanding of how different allocations of time spent on ER-strategies impact stress recovery.

Conclusions

This thesis examined the relation between spontaneous ER and stress recovery, measured by subjective recovery (VAS-Tensed), parasympathetic (RMSSD) and sympathetic (PEP) activity. Considering the main research question, we examined the effects of time spent on adaptive and maladaptive ER-strategies within defined groups (adaptive or maladaptive), as well as specific adaptive ER-strategies within a TSST-context.

In this study, despite participants expressing skepticism about the feedback video, the stress-induction procedure—namely, the TSST—effectively induced stress. Participants' subjective stress levels (measured via VAS-T) and objective stress markers (measured via RMSSD and PEP) indicated that the TSST was successful in eliciting stress.

However, our results did not reveal significant effects of the time spent on specific adaptive or maladaptive ER-strategies, within the defined ER-strategy groups (adaptive and maladaptive), on all three measures of stress recovery (i.e., HRV, PEP, VAS-T) (**Hypotheses 1a** and **1b**). This lack of significant findings was likely due to the small sample sizes (n=45 for the adaptive group and n=7 for the maladaptive group), which resulted in lower statistical power and an increased risk of failing to detect smaller effects. Nevertheless, we did find some evidence supporting positive reappraisal as an effective adaptive ER-strategy for stress recovery (**Hypothesis 1c**). However, the study provided limited significant evidence regarding the effectiveness of other adaptive ER-strategies in promoting stress recovery within the TSST context (**Hypothesis 1c**). This raises the question of whether alternative analyses might reveal more robust effects. For instance, considering individual variations in the use of specific ER-strategies or categorizing groups based on criteria other than the time spent on ER-strategies, could lead to more reliable conclusions about their effectiveness. Further research with larger sample sizes is needed to identify more robust effects of time spent on specific ER-strategies on stress recovery and to better understand the relationship between spontaneous ER and stress recovery.

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ATTACHMENTS

ATTACHMENT 1: informed consent

Informatiebrief voor de deelnemers aan een experiment

Titel van de studie: Help ik heb stress! Wat gebeurt er in ons lichaam wanneer we stress ervaren?

Officiële titel: Een blik op de psychofysiologische processen tijdens het ervaren van stress; wat kunnen Hartslagvariabiliteit, Pre-ejection period en Huidgeleiding ons vertellen?

Beste,

U wordt uitgenodigd om deel te nemen aan een klinische studie. Neem, voor u beslist om deel te nemen aan deze studie, voldoende tijd om deze informatiebrief aandachtig te lezen en dit te bespreken met de onderzoeker of zijn/haar vertegenwoordiger, of met andere personen van uw keuze. Neem ook de tijd om vragen te stellen indien er onduidelijkheden zijn of indien u bijkomende informatie wenst. Dit proces wordt 'informed consent' of 'geïnformeerde toestemming' voor deelname aan een experiment genoemd. Eens u beslist heeft om deel te nemen aan de studie zal men u vragen om het toestemmingsformulier achteraan deze bundel te ondertekenen.

1 WAT IS HET DOEL VAN DE STUDIE?

Wij nodigen u uit om deel te nemen aan een klinische studie met als basisdoelstelling een zicht te krijgen op de psychofysiologische processen die plaatsvinden tijdens het uitvoeren van een prestatie die als stresserend kan ervaren worden. In deze studie zal u een spreektaak krijgen die door een vakkundige jury zal beoordeeld worden. Tijdens deze taak zullen we een aantal psychofysiologische processen zoals hartslag en huidgeleiding meten. De opdrachtgever van deze studie is UGent, met als hoofdonderzoeker Prof. dr. Kristof Hoorelbeke van de Vakgroep Experimenteel-Klinische en Gezondheidspsychologie (Universiteit Gent). Masterstudenten klinische psychologie aan de Universiteit Gent zullen deel uitmaken van het onderzoeksteam.

Niet iedereen zal kunnen deelnemen aan de studie. U wordt gevraagd om vooraf online een korte screeningsvragenlijst in te vullen om te bepalen of u geschikt bent voor onze onderzoeksdoeleinden. Afhankelijk van deze vragenlijst ontvangt u een melding of u kunt deelnemen aan het onderzoek of niet.

2 WAT HOUDT DEELNAME AAN DE STUDIE IN VOOR U?

Studenten hoger onderwijs met een leeftijd ouder dan 18 jaar zullen worden gerekruteerd. Enkel wanneer u voldoet aan alle inclusie en exclusie criteria (zie punt 5), zal u worden toegelaten om deel te nemen aan de studie. Vervolgens zal met u een afspraak moment vastgelegd worden waarop de studie kan doorgaan. 7 dagen voor de start van deze studie zal u gevraagd worden om online vier vragenlijsten in te vullen.

Bij de start van het onderzoek zullen we u eerst enkele algemene vragen stellen om te peilen naar de eerder meegegeven voorwaarden (bijvoorbeeld niet gerookt hebben). Vervolgens zullen we enkele elektrodes op uw lichaam bevestigen zodat we het apparaat dat uw hartslag, ademhaling en huidgeleiding zal meten hieraan kunnen bevestigen. Daarna zullen we u enkele vragen stellen omtrent uw mentale gezondheid. Hierna zal u instructies krijgen voor de taak die u dient uit te voeren. U zal 10 minuten de tijd krijgen om een tekst te lezen. Vervolgens dient u gedurende vijf minuten aan een vakkundige jury een samenvatting van deze tekst te bieden. De jury zal hierop volgend feedback geven op de geleverde prestatie, waarna een rustperiode van 5 minuten volgt. Hierna wordt u gevraagd om nogmaals een vragenlijst in te vullen. Op het einde van het onderzoek volgt een debriefing waarbij u eventueel nog extra uitleg kan krijgen over de studie. De studie zal ongeveer 1 uur en 15 minuten in beslag nemen.

Het onderzoek zal doorgaan in de Faculteit Psychologie en Pedagogische Wetenschappen van de Universiteit Gent

3 HOEVEEL DEELNEMERS ZULLEN AAN DEZE STUDIE DEELNEMEN?

Er zullen in totaal 200 personen aan deze studie deelnemen.

4 WAT IS DE DUUR VAN DEZE STUDIE?

De verwachte totale duur van de studie voor u is 1u en 15 minuten.

5 WAT WORDT VERWACHT VAN DE DEELNEMER?

Voor het welslagen van de studie is het uitermate belangrijk dat u volledig meewerkt met de onderzoeker en dat u zijn/haar instructies nauwlettend opvolgt.

Voorafgaand aan de studie zal u worden gevraagd om een korte screeningsvragenlijst in te vullen om te bepalen of u geschikt bent voor onze onderzoeksdoeleinden. Afhankelijk van deze vragenlijst ontvangt u een melding of u kunt deelnemen aan het onderzoek of niet.

Bij deelname aan het onderzoek vragen we u

- Geen intense fysieke activiteiten te ondernemen op de dag voor deelname aan de studie alsook de dag van deelname.
- In de twee uur voorafgaand aan uw deelname niet te eten of koffie of andere dranken met cafeïne te nuttigen (bijvoorbeeld energiedrankjes of thee).
- In de 24 uur voorafgaand aan uw deelname geen alcohol te nuttigen.
- In de 72 uur voorafgaand aan uw deelname geen psychoactieve drugs te gebruiken (bvb. Cannabis, cocaïne, MDMA).
- Een normaal slaappatroon te volgen de dag voor deelname (Op uw normale tijdstip gaan slapen en opstaan).

Voor uw deelname aan de studie dient u er ook zeker van te zijn dat er geen tegenindicaties bestaan:

- Jonger dan 18 jaar
- Roker
- Huidige psychiatrische diagnose
- Zwangerschap

6 WELKE PROCEDURES VINDEN TIJDENS DE STUDIE PLAATS?

6.1 Procedures:

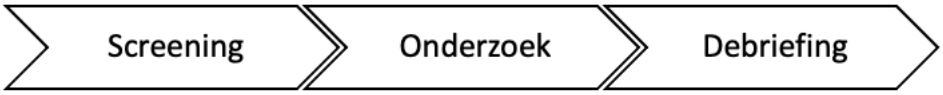
Eerst en vooral zult u voor het experiment een online informatiebrief kunnen lezen waarna u ook uw toestemming voor deelname moet geven. Tijdens een screeningsvragenlijst zullen we enkele inclusie- en exclusiecriteria bevragen (zie punt 5). Indien deze screening gunstig is, wordt een datum vastgelegd waarop het experiment zal plaatsvinden. 1 week voor de start van het experiment zult u gevraagd worden vier beknopte vragenlijsten in te vullen. Deze vragenlijsten bevragen depressieve-, angst- en stresskenmerken alsook hoe u normaal omgaat met uw emoties.

Bij aankomst op de dag van het experiment zult u een papieren versie van de informatiebrief krijgen, waarbij we u nogmaals zullen vragen deze te ondertekenen. Vervolgens zullen we u de inclusie- en exclusiecriteria nogmaals bevragen. Alvorens de studie te starten, zullen we de voorwaarden (zie punt 5) aftoetsen. Vervolgens zullen elektroden op het lichaam geplaatst worden die met de meetapparatuur verbonden worden. Dit is een volledig pijnloze methode en kan vergeleken worden met de afname van een elektrocardiogram bij een dokter. Dit toestel zal een aantal psychofysiologische parameters registreren doorheen de studie. De elektroden blijven dus gedurende het volledige experiment op uw lichaam bevestigd. Wanneer de elektroden zijn aangesloten, zullen we u via een gestructureerd interview enkele vragen stellen omtrent uw mentale gezondheid.

Daarna zal de eigenlijke studie van start gaan waarbij u tijd krijgt om zich voor te bereiden op het geven van een uiteenzetting over een wetenschappelijke tekst die u zal krijgen. Na het uitvoeren van deze taak zal u ook feedback krijgen op uw prestatie. Ten slotte zullen we u vragen om nog een vragenlijst in te vullen. Nadien zullen we u nog wat extra informatie geven over het onderzoek en krijgt u zelf ook de kans om eventuele vragen te stellen.

6.2 Studieverloop:

Indien u besluit om deel te nemen aan de studie en aan alle voorwaarden voor deelname voldoet, zal u onderstaande testen en onderzoeken doorlopen:



Screening (volledig online):
- Lezen en akkoord verklaren informatiebrief
- Bevraging inclusie en exclusie criteria (zie punt 5)

- Invullen vragenlijsten (zie punt 6.1)

Onderzoek:
- Lezen en akkoord verklaren informatiebrief
- Bevraging inclusie en exclusie criteria (zie punt 5)
- Bevraging voorwaarden deelname (zie punt 5)
- Bevraging psychiatrische problemen
- Installatie elektroden voor psychofysiologische metingen (zie punt 6.1)
- Taak + Feedback (zie punt 6.1)

Debriefing:
- Extra uitleg door onderzoeker
- Mogelijkheid tot stellen van vragen

7 WAT ZIJN UW RECHTEN BIJ DEELNAME AAN DEZE STUDIE?

De deelname aan deze studie is volledig vrijwillig, er kan op geen enkele manier sprake zijn van dwang. U kunt weigeren om deel te nemen aan de studie en u kunt zich op elk ogenblik terugtrekken uit de studie zonder dat u hiervoor een reden moet opgeven en zonder dat dit op enige wijze een invloed zal hebben op uw studieresultaten of de verdere relatie met de onderzoeker.

Uw deelname aan deze studie zal beëindigd worden als de onderzoeker meent dat dit in uw belang is. U kan ook voortijdig uit de studie teruggetrokken worden door de onderzoeker als u de in deze informatiebrief beschreven procedures niet goed opvolgt of u de beschreven items niet respecteert.
Indien u uit de studie gehaald wordt, zullen de reeds verzamelde gepseudonimiseerde gegevens in de databank blijven voor analyse, maar er zal geen nieuwe data toegevoegd worden.

Deze studie werd vooraf goedgekeurd door een onafhankelijke Commissie voor Medische Ethiek verbonden aan het Universitair Ziekenhuis van Gent en de Universiteit Gent. De studie wordt uitgevoerd volgens de richtlijnen voor de goede klinische praktijk (ICH/GCP) en de verklaring van Helsinki opgesteld ter bescherming van mensen deelnemend aan klinische studies. In geen geval dient u de goedkeuring door de Commissie voor Medische Ethiek te beschouwen als een aanzet tot deelname aan deze studie.

Indien u verdere vragen heeft bij deze informatie, kan u uw vragen altijd stellen aan de onderzoeksmedewerker of mailen naar Kristof.Hoorelbeke@UGent.be, vooraleer u beslist om toe te stemmen.

7.1 Vertrouwelijkheid

In overeenstemming met de Algemene Verordening Gegevensbescherming (of GDPR) (EU) 2016/679 van 27 april 2016 (die vanaf 25 mei 2018 in voege is) en de Belgische wet van 30 juli 2018, betreffende de bescherming van natuurlijke personen in verband met de verwerking van persoonsgegevens en betreffende het vrije verkeer

van die gegevens, zal uw persoonlijke levenssfeer worden gerespecteerd en kan u toegang krijgen tot de over u verzamelde gegevens. Elk onjuist gegeven kan op uw verzoek verbeterd worden.
Uw toestemming om deel te nemen aan de studie betekent dat we gegevens van u verwerken voor het doel van de klinische studie. Deze verwerking van gegevens is wettelijk voorzien op basis van artikel 6, paragraaf 1 (a) en artikel 9, paragraaf 2 (j) van de Algemene Verordening Gegevensbescherming.

Alle informatie die tijdens deze studie verzameld wordt zal gepseudonimiseerd worden (hierbij kan men uw gegevens nog terug koppelen naar uw persoonlijk dossier). In het geval van pseudonimisering zal de sleutel tot deze codes enkel toegankelijk zijn voor de onderzoeker of de door hem/haar aangestelde vervanger. Enkel de gepseudonimiseerde gegevens zullen gebruikt worden voor analyse van de gegevens en in alle documentatie, rapporten of publicaties (in medische tijdschriften of congressen) over de studie. Vertrouwelijkheid van uw gegevens wordt dus steeds gegarandeerd. Zowel verzamelde persoonsgegevens als gegevens aangaande uw gezondheid zullen verwerkt en bewaard worden gedurende minstens 10 jaar. De verwerkingsverantwoordelijke van de gegevens is de instelling van de hoofdonderzoeker van de studie, Prof. dr. Kristof Hoorelbeke (UGent). Zijn onderzoeksteam zal toegang krijgen tot uw persoonsgegevens. In het kader van de gegevensbescherming zullen de gegevens verwerkt worden door personen behorend tot het onderzoeksteam en aangeduid door en onder de verantwoordelijkheid van de hoofdonderzoeker, inclusief interne medewerkers met een niet-gezondheidszorgberoep.

Met uw uitdrukkelijke toestemming, kunnen de gepseudonimiseerde onderzoeksdata op multidisciplinaire open access onderzoeksplatformen gepubliceerd worden, voor toekomstig wetenschappelijk onderzoek. Dergelijke studies dienen steeds ingediend en goedgekeurd te worden door het ethisch comité.
Stel dat uw gegevens overgemaakt worden aan een land buiten de Europese Economische Ruimte (EER) of aan een internationale organisatie, dan zal UGent er zich van vergewissen of het land van bestemming een passend beschermingsniveau biedt. Wanneer het land waarnaar UGent gegevens wenst over te maken geen passende waarborgen biedt, zal UGent via modelovereenkomsten, ter beschikking gesteld door de Europese Commissie, of andere aanvaarde maatregelen zelf passende waarborgen afdwingen. Uw uitdrukkelijke toestemming voor deze gegevensoverdracht wordt gevraagd in het toestemmingsformulier onderaan.

De Data Protection Officer kan u desgewenst meer informatie verschaffen over de bescherming van uw persoonsgegevens. Contactgegevens: Hanne Elsen, privacy@ugent.be.

Vertegenwoordigers van de opdrachtgever, auditoren, de Commissie voor Medische Ethiek en de bevoegde overheden, allen gebonden door het beroepsgeheim, hebben rechtstreeks toegang tot uw medische dossiers om de procedures van de studie en/of de gegevens te controleren, zonder de vertrouwelijkheid te schenden. Dit kan enkel binnen de grenzen die door de betreffende wetten zijn toegestaan. Door het toestemmingsformulier, na voorafgaande uitleg, te ondertekenen, stemt u in met deze toegang.

De Belgische toezichthoudende instantie die verantwoordelijk is voor het handhaven van de wetgeving inzake gegevensbescherming is bereikbaar via onderstaande contactgegevens:

Gegevensbeschermingsautoriteit (GBA)
Drukpersstraat 35 – 1000 Brussel
Tel. +32 2 274 48 00
e-mail: contact@apd-gba.be
Website: www.gegevensbeschermingsautoriteit.be

7.2 Verzekering

De opdrachtgever voorziet in een vergoeding en/of medische behandeling in het geval van schade en/of letsel ten gevolge van deelname aan deze klinische studie. Voor dit doeleinde is een verzekering afgesloten met foutloze aansprakelijkheid conform de wet inzake experimenten op de menselijke persoon van 7 mei 2004 (Allianz Global Corporate & Specialty – polisnummer opdrachtgever UZ Gent BEL001889 – polisnummer opdrachtgever UGent BEL000862). Indien de onderzoeker van mening is dat er verband met de studie mogelijk is (er is geen verband met de studie bij schade ten gevolge van het natuurlijke verloop van de ziekte of ten gevolge van gekende bijwerkingen van de standaardbehandeling), zal hij/zij de aangifteprocedure bij de verzekering starten. Op dat ogenblik kunnen uw gegevens doorgegeven worden aan de verzekeraar. In het geval van onenigheid met de onderzoeker of met de door de verzekeringsmaatschappij aangestelde expert, en steeds wanneer u dit nodig acht, kunnen u, of in geval van overlijden uw rechthebbenden, de verzekeraar rechtstreeks in België dagvaarden (Allianz Global Corporate & Specialty; Uitbreidingstraat 86, 2600 Berchem; Tel: +32 33 04 16 00).

8 WAT ZIJN DE RISICO'S EN VERWACHTE VOORDELEN BIJ DEELNAME AAN DEZE STUDIE?

Deelname aan deze studie brengt voor u waarschijnlijk geen onmiddellijk therapeutisch voordeel. Uw deelname aan de studie kan wel nuttige informatie opleveren voor verder onderzoek naar wat er in ons lichaam gebeurt wanneer we stress ervaren. Door hier meer zicht op te krijgen, kunnen we in de toekomst mogelijks handvatten aanreiken rond het omgaan met stress.

De waarschijnlijkheid dat u door deelname aan deze studie enige schade ondervindt, is laag. U hebt het recht om op elk ogenblik vragen te stellen over de mogelijke en/of gekende risico's van deze studie. Als er in het verloop van de studie gegevens aan het licht komen die een invloed zouden kunnen hebben op uw bereidheid om te blijven deelnemen aan deze studie, zult u daarvan op de hoogte worden gebracht. Mocht u door uw deelname aan de studie toch enig nadeel ondervinden, zal u een gepaste behandeling krijgen.

9 ZIJN ER KOSTEN VERBONDEN AAN DE DEELNAME AAN DEZE STUDIE?

Deelname aan deze studie brengt geen extra kosten mee voor u.

10 IS EEN VERGOEDING VOORZIEN BIJ DEELNAME AAN DEZE STUDIE?

Er zal bij de beëindiging van het volledige studieprotocol een vergoeding van 10 euro voorzien worden (studieprotocol duurt ongeveer 1 uur en 15 minuten).

11 TOT WIE KUNT U ZICH RICHTEN IN HET GEVAL VAN PROBLEMEN OF INDIEN U VRAGEN HEEFT?

Als u aanvullende informatie wenst over de studie of over uw rechten en plichten, kunt u in de loop van de studie op elk ogenblik contact opnemen met de onderzoeker of een medewerker van zijn of haar team:

Naam: Prof. dr. Kristof Hoorelbeke

Adres: Kristof.Hoorelbeke@UGent.be

Telefoonnummer: +32 (0)9 264 64 74

Naam: Prof. dr. Rudi De Raedt

Adres: Rudi.DeRaedt@UGent.be

Telefoonnummer: +32 (0)9 264 64 47

Naam : Jente Depoorter

Adres : Jente.Depoorter@UGent.be

Telefoonnummer : +32 (0)9 264 94 14

TOESTEMMINGSFORMULIER VOOR DE DEELNEMERS AAN EEN EXPERIMENT

Referentienummer van de deelnemer voor deze studie	

Ik heb het document "Informatiebrief voor de deelnemers aan een experiment" pagina 1 tot en met 7 gelezen en begrepen en ik heb er een kopij van gekregen. Ik heb uitleg gekregen over de aard, het doel, de duur, de te voorziene effecten van de studie en over wat men van mij verwacht. Ik heb uitleg gekregen over de mogelijke risico's en voordelen van de studie. Men heeft me de gelegenheid en voldoende tijd gegeven om vragen te stellen over de studie en ik heb op al mijn vragen een bevredigend antwoord gekregen, ook op medische vragen.
Ik begrijp dat deelname aan de studie vrijwillig is en dat ik mij op elk ogenblik uit de studie mag terugtrekken zonder een reden voor deze beslissing op te geven en zonder dat dit op enigerlei wijze een invloed zal hebben op mijn verdere relatie met de onderzoeker.
Ik begrijp dat niet deelnemen of mijn deelname aan het onderzoek stopzetten op geen enkele manier invloed heeft op eventuele evaluatie en/of studiebegeleiding.
Ik begrijp dat auditors, vertegenwoordigers van de opdrachtgever, de Commissie voor Medische Ethiek of bevoegde overheden, mijn gegevens mogelijks willen inspecteren om de verzamelde informatie te controleren. Bovendien ben ik op de hoogte dat bepaalde gegevens doorgegeven worden aan de opdrachtgever van de studie. Te allen tijde zal mijn privacy gerespecteerd worden. Door dit document te ondertekenen, geef ik toestemming voor deze controle.
Ik ben me ervan bewust dat deze studie werd goedgekeurd door een onafhankelijke Commissie voor Medische Ethiek verbonden aan het UZ Gent en de Universiteit Gent en dat deze studie zal uitgevoerd worden volgens de richtlijnen voor de goede klinische praktijk (ICH/GCP) en de verklaring van Helsinki, opgesteld ter bescherming van mensen deelnemend aan experimenten. Deze goedkeuring was in geen geval de aanzet om te beslissen om deel te nemen aan deze studie.
Men heeft mij ingelicht dat zowel persoonlijke gegevens als gegevens aangaande mijn gezondheid worden verwerkt en bewaard gedurende minstens 10 jaar. Ik ben op de hoogte dat ik recht heb op toegang en op verbetering van deze gegevens. Aangezien deze gegevens verwerkt worden in het kader van medisch-wetenschappelijke doeleinden, begrijp ik dat de toegang tot mijn gegevens kan uitgesteld worden tot na beëindiging van het onderzoek. Indien ik toegang wil tot mijn gegevens, zal ik mij richten tot de onderzoeker die verantwoordelijk is voor de verwerking ervan.
Ik weet dat de UGent de verantwoordelijke eenheid is m.b.t. persoonsgegevens verzameld tijdens het onderzoek. Ik weet dat de data protection officer me meer informatie kan verschaffen over de bescherming van mijn persoonlijke informatie. Contact: Hanne Elsen (privacy@ugent.be)
Ik ben ervan op de hoogte dat ik op aanvraag een samenvatting van de onderzoekbevindingen kan krijgen.
Men heeft mij ingelicht dat studenten meewerken aan dit onderzoek in het kader van hun Masterproef. De studenten staan onder supervisie van Jente Depoorter, Prof. dr. Rudi De Raedt en Prof. dr. Kristof Hoorelbeke.

Aankruisen door de deelnemer indien akkoord

Ik stem in om deel te nemen aan de studie:

1) Ik stem ermee in om volledig samen te werken met de onderzoeker. Ik zal hem/haar op de hoogte brengen als ik onverwachte of ongebruikelijke symptomen ervaar.	
2) Ik stem ermee in om de vragenlijsten in te vullen.	
3) Ik stem ermee in dat er elektrodes op mijn lichaam zullen worden bevestigd om mijn hartslag, ademhaling en huidgeleiding te meten.	
4) Ik stem ermee in om de taak uit te voeren.	
5) Ik geeft toestemming aan de onderzoeker om mijn resultaten op vertrouwelijke wijze te bewaren, te verwerken en te rapporteren, met de mogelijkheid dat de gepseudonomiseerde dataset online beschikbaar gesteld wordt voor hergebruik voor toekomstige wetenschappelijke doeleinden. Deze toekomstige studies dienen steeds ingediend en goedgekeurd te worden door een ethisch comité.	
6) Ik ben akkoord dat gepseudonimiseerde data voor onderzoeksdoeleinden gedeeld kan worden met een land buiten de Europese Economische Ruimte (EER) of een internationale organisatie. Dit houdt mogelijks publicatie in van de	

gepseudonimiseerde onderzoeksdata op daarvoor voorzien multidisciplinaire open access onderzoeksplatformen ter bevordering van Open Science Practice.	

Naam en voornaam van de deelnemer	Handtekening	Datum
Naam en voornaam van de onderzoeker*	Handtekening	Datum

2 kopieën dienen te worden vervolledigd. Het origineel wordt door de onderzoeker bewaard in het ziekenhuis gedurende 10 jaar, de kopie wordt aan de deelnemer gegeven.

*Aankruisen door de onderzoeker indien akkoord

Ik verklaar de benodigde informatie inzake deze studie (de aard, het doel, en de te voorziene effecten) mondeling te hebben verstrekt evenals een exemplaar van het informatiedocument aan de deelnemer te hebben verstrekt.	
Ik bevestig dat geen enkele druk op de deelnemer is uitgeoefend om hem/haar te doen toestemmen tot deelname aan de studie en ik ben bereid om op alle eventuele bijkomende vragen te antwoorden.	

ATTACHMENT 2: debriefing

Beste participant,

Bedankt voor uw deelname aan dit onderzoek. We willen U graag inlichtingen geven over het doel en de ware aard van deze studie.

De basisdoelstelling van deze studie is om tot een beter begrip te komen rond hoe we onze emoties spontaan reguleren wanneer we geconfronteerd worden met negatieve gevoelens zoals stress, angst of verdriet. Er zijn verschillende manieren om onze emoties te reguleren, maar sommige strategieën lijken hiertoe meer adaptief. Voorafgaand onderzoek heeft aangetoond dat het gebruik van maladaptieve emotieregulatiestrategieën geassocieerd is met meer psychologische klachten. Tijdens dit onderzoek wilden we nagaan hoe mensen spontaan omgaan met de emoties die ze ervaren. Door het meten van uw hartslag en huidgeleiding kunnen we zien hoe uw lichaam reageert op een stressor. Het ervaren van stress en hoe u daar mee omgaat heeft namelijk ook heel wat lichamelijke gevolgen (u gaat bijvoorbeeld meer zweten, wat voor een stijging in de huidgeleiding zorgt, maar ook uw hartslag gaat omhoog). Door deze zaken te meten kunnen we nagaan wat het effect is van het gebruik van emotieregulatiestrategieën op onze stressrespons. We hebben u op voorhand bewust niet verteld dat deze studie een onderzoek is naar spontane emotieregulatie. Mochten we dit wel doen, dan lopen we het risico dat uw emotieregulatie niet meer spontaan plaatsvindt.

Omdat het voor deze studie heel belangrijk was dat u stress of negatieve gevoelens zou ervaren, hebben we over enkele zaken misleidende informatie verschaft. Het is helemaal niet zo dat er aangetoond werd dat uw prestatie op deze taak samenhangt met uw academische prestaties. Hoe goed u de presentatie hebt gedaan, heeft geen enkel effect op uw academisch presteren. Tevens is de feedback die u van de jury ontving vooraf opgenomen en dus helemaal niet representatief voor uw prestatie op de spreektaak. Iedere participant krijgt dezelfde vooraf opgenomen feedback, onafhankelijk van hun prestatie, wat het dus niet relevant maakt voor u.

Nogmaals bedankt voor uw deelname aan deze studie! Indien u nu, of later, nog vragen hebt, wees vrij om deze te stellen aan de onderzoeker.

ATTACHMENT 3: scripts

script for preparation:

"Deze taak analyseert hoe goed u een wetenschappelijke tekst kunt begrijpen, verwerken en presenteren onder tijdsdruk. Deze vaardigheid is een belangrijke vereiste voor een succesvol studieverloop. Uit eerder onderzoek is gebleken dat studenten die hier slecht op scoren vaker problemen ervaren in hun studieloopbaan. Je zal zo meteen 10 minuten voorbereidingstijd krijgen om deze tekst te lezen. Vervolgens zul je hierover 5 minuten moeten presenteren, probeer een zo'n duidelijk mogelijke en samenhangende presentatie te geven, waarbij je 5 minuten vol praat. Het is niet toegestaan om tijdens de presentatie gebruik te maken van notities. Tijdens de voorbereiding mag dit wel, maar tijdens de presentatie nemen we deze weg. Deze presentatie zal door een jury gevolgd worden via een teams-meeting en zij zullen jou achteraf ook feedback geven. Het is niet toegestaan om vragen te stellen aan de juryleden of deze te onderbreken. De jury zal u kunnen zien en horen tijdens uw presentatie, maar u zal uzelf niet zien zodat er geen afleiding mogelijk is. Heeft u hier nog vragen over? Dit is het enige moment waarop u vragen kunt stellen."

script for feedback:

Jurylid 1 (+/- 2 min) – Peter Depoorter – Professor Waeselynck

Begint te spreken….

Proefleider: "Beste prof. Waeselynck, u staat nog gemute"

Kan u ons mij horen?
(Proefleider bevestigd)
Oké perfect. Bedankt voor uw presentatie. In het begin van uw presentatie leek u zelfzeker over te komen en had ik ook het gevoel dat u goed voorbereid was. Maar al snel had ik door dat mijn eerste indruk over uw capaciteiten niet correct was. Misschien heb ik het verkeerd ingeschat, maar u leek nerveus, ook wat onzeker en ik had het gevoel dat u zelf niet goed wist waarover u sprak. De algemene zaken over de samenstelling van het bloed heeft u correct verwoord, maar dat is dan natuurlijk ook wel basiskennis. Bij de specificaties van de erytrocyten ging u echter compleet de mist in. Ik had ook mijn twijfels over uw uitleg omtrent leukocyten, maar mijn collega zal hier mogelijks straks verder op ingaan, gezien zij hieromtrent veel kennis heeft. Nochtans hebben we de tekst zo opgesteld dat deze voor iedereen met een diploma middelbaar onderwijs perfect te begrijpen is. Ten slotte heb ik ook genoteerd dat u niet alle belangrijke informatie uit de tekst heeft vermeld. Ik zou u dus als advies meegeven om op een andere manier wetenschappelijke teksten te verwerken anders zal u tijdens uw opleiding zeker in de problemen komen.

Proefleider: Bedankt prof. X. Dan geef ik nu graag het woord aan prof. Depaepe. (10 seconden)

Jurylid 2 (+/- 1min22) – Davina Van der Heggen (Professor Depaepe)

Ik kan mij wel vinden in de beoordeling van mijn collega. Het is niet de slechtste presentatie die ik al heb gezien, maar gemiddeld gezien dient men hier toch beter op te scoren. U wist inderdaad wel de basissamenstelling van het bloed, maar raakte verloren bij de specificaties die hiermee samen gaan. Nochtans zijn sommige van deze specificaties heel verstaanbaar uitgelegd in de tekst. Ik heb in mijn professionele carrière vooral onderzoek naar leukemie gedaan en hierdoor heb ik dus inderdaad een zeer gedegen kennis omtrent leukocyten, zoals mijn collega daarnet vermelde. Ik merkte tijdens u presentatie dat u over dit stuk niet zeker was. Ik vind dat u hieromtrent niet voldoende kennis had en daardoor toch een aantal grote fouten hebt gemaakt in uw uitleg. Ik zou u graag ook nog wat feedback geven omtrent het presenteren. Ik denk dat u hier nog heel wat verbetermogelijkheden heeft. Ik vond dat u in het begin een duidelijk verhaal volgde, maar merkte naar het einde toe dat het chaotischer werd. Hierdoor werd ik minder geïnteresseerd in wat u vertelde en merkte ik dat ik mijn aandacht verloor. In de toekomst dient u er dus zeker op te letten dat u een presentatie goed opbouwt zodat u de aandacht van alle luisteraars ten alle tijden kan behouden.

Jente: Bedankt prof. Depaepe. Dan geef ik nu graag het woord aan dr. Van der Heggen. (10 seconden)

Jurylid 3 (+/- 1min) – David Van der Heggen (dr. Van der Heggen)

ATTACHMENT 4: VAS

VAS – Versie 1 dd. 04/10/2021

VAS - SCHALEN

Zet op onderstaande lijn duidelijk zichtbaar een haaks streepje op de plaats die overeenkomt met uw mening.

Hoe gespannen voelt u zich?

Hoe verdrietig voelt u zich?

Hoe gelukkig voelt u zich?

Hoe boos voelt u zich?

ATTACHMENT 5: preparation blood

Het bloed

Bloed is een ondoorzichtige rode vloeistof die bestaat uit een geelachtig plasma en de daarin gesuspendeerde rode bloedcellen (erytrocyten), witte bloedcellen (leukocyten) en bloedplaatjes (trombocyten).

Rode bloedcellen - Erytrocyten

Het grootste deel van het bloedvolume (ongeveer 44%) bestaat uit rode bloedcellen of erytrocyten. Bij mannen zijn er ongeveer 5,1 miljoen erytrocyten per microliter, bij vrouwen zijn dit er 4,6 miljoen. Naast water vormt hemoglobine het grootste deel van de erytrocyten. Het eiwit hemoglobine is goed voor 34% van het natte gewicht van de erytrocyten en 90% van het droog gewicht.

Menselijke erytrocyten zijn platte, ronde, kernloze schijven met inkepingen in het midden, waarvan de grootste dikte (aan de rand) slechts 2 µm is en waarvan de diameter bij gezonde mensen rond een gemiddelde waarde van 7,5 µm (normocyten) ligt, in de vorm van een normaalverdeling.

Erytrocyten worden gevormd in de hemopoëtische weefsels, d.w.z. in het embryo in de dooierzak, in de foetus in de lever en milt en bij de volwassene in het rode merg van de platte botten. Hier bevinden zich de pluripotente stamcellen, de uniforme voorouders van alle soorten bloedcellen. De volgende fase van differentiatie zijn de bepaalde voorlopercellen, die alleen in staat zijn om ofwel erytrocyten, leukocyten of trombocyten te produceren. Er wordt een onderscheid gemaakt tussen verschillende stadia van differentiatie en rijping tot wanneer de jonge, genucleëerde erytrocyten het beenmerg verlaten als reticulocyten (ofwel onvolgroeide erytrocyten). Erytrocyten circuleren 100-120 dagen in het bloed. Vervolgens worden ze afgebroken door cellen van het reticulo-endotheliaal systeem (het immuunsysteem) in het beenmerg. Onder pathologische omstandigheden gebeurt dit ook in de lever en milt.

Witte bloedcellen - Leukocyten

Leukocyten of witte (kleurloze) bloedcellen zijn genucleëerde, hemoglobinevrije cellen. Bij gezonde mensen bevinden er zich 4000-10000 leukocyten per microliter bloed. In tegenstelling tot het relatief constante aantal erytrocyten bij gezonde mensen, verandert het aantal leukocyten in het bloed binnen veel ruimere grenzen, afhankelijk van het tijdstip van de dag en de functionele toestand van het organisme. Als er meer dan 10.000 leukocyten per microliter bloed zijn, spreekt men van een leukocytose, bij minder dan 4000 van een leukopenie. Leukocytosen komen vooral voor bij ontstekingsziekten en - in de meest ernstige vorm - bij leukemie.

De leukocyten zijn geen uniforme groep cellen. Naargelang morfologische en functionele aspecten en de plaats van vorming wordt een onderscheid gemaakt tussen de volgende drie grote groepen: granulocyten, monocyten en lymfocyten. Granulocyten en monocyten ontstaan in het beenmerg onder invloed van bepaalde glycoproteïneweefselhormonen van mesenchymale oorsprong (CFF, "koloniestimulerende factoren"). De specifieke groeifactor voor de lymfocyten is interleukine-2.

Bloedplaatjes - Trombocyten

Met de in de kliniek gebruikelijke methode voor het bepalen van het aantal trombocyten in het bloed vindt men bij gezonde volwassenen 15.000-350.000 bloedplaatjes per microliter. De platte, onregelmatig ronde, kernloze bloedplaatjes hebben een lengtediameter van 1-4 micrometer en een dikte van 0,5-0,75 micrometer.

Ze ontstaan in het beenmerg als een vernauwing van het cytoplasma van megakaryocyten. Uit een gigantische beenmergcel kunnen 1000 bloedplaatjes worden gevormd. Net als de vorming van erytrocyten, wordt de vorming van bloedplaatjes gereguleerd door een glycoproteïnehormoon van renale oorsprong, trombopoëtine. De bloedplaatjes blijven 5-11 dagen in het bloed. Bloedplaatjes worden afgebroken in de lever, longen en milt.

ATTACHMENT 6: CERQ

SPONTANE EMOTIEREGULATIE

***Deel 1a**: Nadat u alle feedback had ontvangen, wat is het eerste dat u deed?*

Ontspannen: Ik probeerde me te ontspannen na wat me overkomen is.	☐
Jezelf de schuld geven: Ik bedacht me dat ik zelf verantwoordelijk ben voor wat me overkomen is.	☐
Accepteren: Ik accepteerde wat me overkomen is.	☐
Rumineren: Ik bleef stilstaan bij de gevoelens en gedachten over wat me overkomen is.	☐
Positief herinterpreteren: Ik dacht na over de positieve kanten van wat me overkomen is.	☐
Relativeren: Ik dacht dat er ergere dingen waren in de wereld dan wat me overkomen is.	☐
Catastroferen: Ik dacht na over hoe vreselijk het is wat me overkomen is.	☐
Anderen de schuld geven: Ik bedacht me dat anderen verantwoordelijk zijn voor wat me overkomen is.	☐

SPONTANE EMOTIEREGULATIE

***Deel 1b**: Wat deed u vervolgens? (Indien u niets anders deed dan daarnet gelieve dan te klikken op 'Ik deed niets anders dan daarnet'.)*

Ontspannen: Ik probeerde me te ontspannen na wat me overkomen is.	☐
Jezelf de schuld geven: Ik bedacht me dat ik zelf verantwoordelijk ben voor wat me overkomen is.	☐
Accepteren: Ik accepteerde wat me overkomen is.	☐
Rumineren: Ik bleef stilstaan bij de gevoelens en gedachten over wat me overkomen is.	☐
Positief herinterpreteren: Ik dacht na over de positieve kanten van wat me overkomen is.	☐
Relativeren: Ik dacht dat er ergere dingen waren in de wereld dan wat me overkomen is.	☐
Catastroferen: Ik dacht na over hoe vreselijk het is wat me overkomen is.	☐
Anderen de schuld geven: Ik bedacht me dat anderen verantwoordelijk zijn voor wat me overkomen is.	☐

SPONTANE EMOTIEREGULATIE

***Deel 1c**: Wat deed u hierna? (Indien u hetzelfde deed als bij stap 1, dan mag u opnieuw dit antwoord aanduiden. Indien u hetzelfde deed als bij stap 2, dan mag u het antwoord 'Ik deed hetzelfde als de vorige stap (stap 2)' aanduiden.*

Ontspannen: Ik probeerde me te ontspannen na wat me overkomen is.	☐
Jezelf de schuld geven: Ik bedacht me dat ik zelf verantwoordelijk ben voor wat me overkomen is.	☐
Accepteren: Ik accepteerde wat me overkomen is.	☐
Rumineren: Ik bleef stilstaan bij de gevoelens en gedachten over wat me overkomen is.	☐
Positief herinterpreteren: Ik dacht na over de positieve kanten van wat me overkomen is.	☐
Relativeren: Ik dacht dat er ergere dingen waren in de wereld dan wat me overkomen is.	☐
Catastroferen: Ik dacht na over hoe vreselijk het is wat me overkomen is.	☐
Anderen de schuld geven: Ik bedacht me dat anderen verantwoordelijk zijn voor wat me overkomen is.	☐

SPONTANE EMOTIEREGULATIE

***Deel 2**: Nadat u alle feedback had ontvangen, hoeveel percentage van de tijd (5 minuten) heb je aan de volgende zaken besteed? Het totaal dient 100 te zijn.*

Ontspannen: Ik probeerde me te ontspannen bij wat me overkomen is.	☐
Jezelf de schuld geven: Ik bedacht me dat ik zelf verantwoordelijk ben voor wat me overkomen is.	☐
Accepteren: Ik accepteerde wat me overkomen is.	☐
Rumineren: Ik bleef stilstaan bij de gevoelens en gedachten over wat me overkomen is.	☐
Positief herinterpreteren: Ik dacht na over de positieve kanten van wat me overkomen is.	☐
Relativeren: Ik dacht dat er ergere dingen waren in de wereld dan wat me overkomen is.	☐
Catastroferen: Ik dacht na over hoe vreselijk het is wat me overkomen is.	☐
Anderen de schuld geven: Ik bedacht me dat anderen verantwoordelijk waren voor wat me is overkomen.	☐